Alcohol Abuse and Alcoholism in Primary Health Care Settings

Kathryn Magruder-Habib, PhD, A. Mark Durand, MD, and Keith A. Frey, MD
Durham, North Carolina

Alcohol problems are common in primary care patients, yet they are often not detected and treated. Methods for improving the detection and diagnosis of alcohol problems in the primary care setting are reviewed in terms of pertinent history, physical examination, and laboratory findings. Screening instruments such as the CAGE questionnaire and the Michigan Alcoholism Screening Test are recommended for routine use by primary care physicians. Such instruments have been shown to have higher sensitivity than laboratory tests alone. Although less is known about intervention and management, earlier intervention with innovative (less costly) management techniques may be both efficacious and acceptable to the patient.

Key words. Alcoholism; primary health care; screening. *J Fam Pract 1991; 32:406-413.*

Alcohol abuse and alcoholism are an enormous burden to society, accounting for $13.5 billion a year in direct treatment costs and $116.7 billion per year in total (direct and indirect) costs in the United States.[1] Although an estimated 27% of men and 43% of women are abstainers (ie, they drink less than one alcoholic drink per month), 7%, or 10 million Americans 18 years old and older, are alcoholics.[2] According to a number of studies published in the 1980s, among all adults who seek outpatient care, an estimated 4% to 33% are alcoholics, with the lowest estimates obtained from patient self-administered screening tests, and the highest from interviewer-administered screening tests[3–13] (Table 1). Other studies have shown that of those who seek inpatient care, an estimated 15% to 61% are alcoholics[14–27] (Table 2). Clearly, alcoholics are overrepresented in patient populations, and their overrepresentation escalates as cost and intensity of treatment services go up.

These data indicate that the primary care health setting provides multiple opportunities for physicians and other health care professionals to diagnose and treat alcoholism. Physicians often encounter alcoholic patients, usually when they present with other problems[28]; such encounters provide opportunities for early detection and intervention, when there is a greater likelihood of successful treatment.

Screening and Diagnosis

Alcoholism, unlike other disorders, is a disease many primary care physicians do not want to detect. In addition, most alcoholic patients do not want their disease detected. Unfortunately, this creates a system of collusion. Nevertheless, the desire to avoid the diagnosis does not relieve the physician of his or her responsibilities. Physicians should approach alcoholism in much the same way they approach other disorders, that is, by asking pertinent historical questions, by being sensitive to physical presentation, and by ordering and interpreting laboratory and other diagnostic tests.

History

The following historical findings are considered risk factors for alcoholism: the patient has a family history of alcoholism; has a family history of moral constraints about alcohol use; has a spouse or family member of spouse with a history of alcoholism; was reared in a broken home; was the last child born into a large family; is of Irish, Scandinavian, or Native American descent; has female relatives of more than one generation with a high incidence of recurrent depression; is a heavy smoker; is single or divorced; is unemployed; has a history of hyperactivity during childhood; is a bartender, house

ISSN 0094-3509

Table 1. Reported Prevalence of Alcoholism in Outpatient Settings

Study	Prevalence (%)	Setting	Criteria
Hurt et al[3]	5	Mayo Medical Clinic	SAAST
Magruder-Habib et al[4]	31	Veterans Administration	MAST ≥6
Magruder-Habib et al[5]	25	Veterans Administration	Clinical judgment
Powers and Spickard[6]	4	General medical clinic	Self-administered MAST ≥7
Leckman et al[7]	19	Family practice center	MAST
Nicol and Ford[8]	33*	General practice	MAST ≥5
	4†		
Wiseman et al[9]	14	General practice	CAGE/QF/BAL
King[10]	9	General practice	CAGE ≥2
Coulehan et al[11]	14	Primary care patients	DIS
Cyr and Wartman[12]	20	Urban medical clinic	MAST ≥5
Woodhall[13]	12	Family practice center	SMAST ≥3

*Male patients.
†Female patients.
SAAST—Self-Administered Alcoholism Screening Test; MAST—Michigan Alcoholism Screening Test; SMAST—Short Michigan Alcoholism Screening Test; QF—quantity-frequency. BAL—blood alcohol level; DIS—Diagnostic Interview Schedule.

painter, or traveling salesman; or has an addiction to another substance.[29] Controversy exists over whether specific childhood personality traits (eg, impulsivity, poor self-esteem, low ego strength) are independent risk factors for alcoholism.[30–32] The risks associated with each factor above and the ways in which factors interact in the induction of alcoholism are not precisely known. Nevertheless, an appreciation of these factors can attune the physician to individuals in his or her practice who may be vulnerable to alcoholism. Furthermore, attention should be paid to social history details such as marital discord, job difficulties, disorderly conduct, violent behaviors, and arrests for driving while impaired, all of which can be early manifestations of alcoholism.[33]

Physical Presentation

Problem drinkers tend to present with certain complaints: palpitations, anxiety, sleep disturbances, depression, dyspepsia, nausea, diarrhea, impotence, and recurring minor trauma. These nonspecific complaints tend to occur earlier than manifestations such as gastrointestinal bleeding, pancreatitis, or liver disease, which most physicians more closely associate with alcoholism.[34] In addition, physicians need to be alert to tremulousness, tachycardia, elevated blood pressure, and the odor of ethanol on a patient's breath. While the positive predictive value of any one of the signs and presenting complaints enumerated above is probably low, when considered in the context of other information that the physician may have, these indicators may provide the important additional information needed to confirm a diagnosis of alcoholism.

Laboratory Results

Laboratory studies can often provide excellent additional information for the physician who suspects alcoholism. It should be noted, however, that laboratory findings provide cross-sectional information. Thus, periodic drinkers or those who have been abstinent for several weeks may

Table 2. Reported Prevalence of Alcoholism in Inpatient Settings

Study	Prevalence (%)	Setting	Criteria
Pearson[15]	29	Hospital	4 of 5 criteria from Jellinek*
Nolan[16]	15	Hospital	Staff evaluation
Green[17]	19	Hospital	History and evaluation
Barchha et al[18]	28	Hospital	3 of 4 categories and interview
McCusker et al[19]	60	Hospital, Harlem	Interview and history
Moore[20]	18	Hospital	MAST and records
Chakerian and Schenkel[21]	24	Veterans Administration	Diagnosis and staff consultation
Favazza and Pires[22]	29	General military hospital	MAST ≥5
Mayfield et al[23]	39	Veterans Administration	Multidisciplinary evaluation
Gomberg[24]	55	Veterans Administration	3 of 4 categories and interview
Quinn and Johnston[25]	26	Hospital	MAST and physician examination
Aderhold[26]	61†	Veterans Administration	CAGE ≥2
Mayfield and Johnston[27]	43	Veterans Administration	Psychiatric admissions evaluation
Moore et al[14]	13–43	Hospital (prevalence varied by service)	MAST ≥5 or CAGE ≥2

*Jellinek EM. The Disease Concept of Alcoholism. Highland Park, NJ: Hillhouse Press, 1960.
†Includes 36% current, 25% past patients.
MAST—Michigan Alcoholism Screening Test.

have normal test results. With the exception of blood alcohol level, the laboratory markers of alcoholism all indicate toxic effects of alcohol on the body. These effects occur late in the disease and thus are insensitive indicators of early disease. Younger alcoholic patients often have no clinical abnormalities, but have alcohol-related psychosocial problems. Older alcoholic patients, on the other hand, generally have both.

The blood alcohol level is a good measure of alcohol intake during the hours preceding the test, but is an indicator of problem drinking only when significantly high blood alcohol levels (>32.6 mmol/L or >150 mg/dL) exist in the absence of perceivable intoxication (indicating alcohol tolerance).[35]

Other well-studied, relatively early laboratory markers of heavy alcohol intake include the levels of gamma glutamyltransferase (GGT) and aspartate aminotransferase (AST, previously SGOT), and the mean corpuscular volume (MCV).[35–37] Of these, GGT is probably the most sensitive, and will be elevated after 2 to 4 weeks of steady ethanol intake in the range of 40 to 60 grams per day. From 33% to 75% of heavy drinkers have elevated GGT.[38,39] In one study, 12 of 15 adolescents who drank six or more alcoholic drinks per day had elevated GGT.[40] Elevated GGT values decline toward normal about 4 weeks after cessation of alcohol intake.[38,41,42]

It usually takes weeks or months of steady alcohol intake for AST and MCV to become elevated. They are not as sensitive as GGT, though they may be elevated in some patients in whom GGT is normal.[38,39] One study, which compared the questionnaire responses to the Michigan Alcoholism Screening Test (MAST) and to the test called CAGE of 385 adult psychiatric inpatients with laboratory markers (GGT, GOT, MCV, urate, glutamyl dehydrogenase) to identify problem drinkers and alcoholics, found the sensitivity of the best laboratory marker (GGT) to be only 33%.[37] In contrast, questionnaires correctly identified more than 90%. Thus, laboratory markers alone are of little use in screening for early alcoholism, except, perhaps, when used to further characterize individuals at high risk. In such preselected patients, the prevalence of alcoholism will be higher; thus, the predictive value of the laboratory markers will be higher.

It may soon be possible to detect genetically high-risk or prealcoholic individuals on the basis of monoamine oxidase function[43–52] using newly developed radioenzyme assay techniques.[44]

Other Diagnostic Tools

The primary care physician should be familiar with at least two other tools: the National Council on Alcohol-ism (NCA) criteria[53] and the *Diagnostic and Statistical Manual of Mental Disorders-III-Revised* (DSM-III-R) criteria[54] for the diagnosis of alcoholism. Although each provides important information, when used as prescribed, they tend to be cumbersome for daily practice.

The NCA criteria provide an exhaustive list of symptoms organized into major and minor criteria in two categories: track 1 covers physiological and clinical dimensions, and track 2 covers behavioral, psychological, and attitudinal dimensions. Each criterion is weighted: *classical*, *definite*, and *obligatory* equals 1; *probable*, *frequent*, and *indicative* equals 2; and *potential*, *possible*, and *incidental* equals 3. The criteria are further organized into early, middle, and late symptoms. To confirm a diagnosis of alcoholism, one or more major criteria from one track must be fulfilled, as well as some criteria from the other track. Alternatively, fulfillment of several minor criteria from both tracks could confirm a diagnosis of alcoholism. The major strength of the NCA criteria is comprehensiveness. Unfortunately, this tool's complex system for judging whether diagnostic criteria are met makes using it unrealistic in daily practice.

DSM-III-R criteria, while not as cumbersome as the NCA criteria, nevertheless have shortcomings within primary care. Chief among them is a psychiatric orientation that makes the criteria difficult for primary care physicians to interpret. Within the psychoactive substance use disorders of DSM-III-R, there are two alcohol-related diagnoses: alcohol dependence (303.90) and alcohol abuse (305.00). Patients qualifying as alcohol dependent must meet criteria in three of nine symptom areas, some of which must have persisted for a month or more. Those qualifying for alcohol abuse must meet not the dependence criteria but criteria in one of two symptom areas, some of which must have persisted for a month or more. Most nonpsychiatric physicians are not apt to use the DSM-III-R approach as they evaluate a patient for an alcohol disorder. Furthermore, many primary care patients do not meet diagnostic criteria, but nevertheless show early signs, symptoms, and impairment from excessive alcohol consumption and could benefit from early intervention.

After the diagnosis of alcoholism is made, considerations include whether a patient is a primary or secondary alcoholic,[55] and whether a patient is a type 1 (environmentally determined) or type 2 (genetically determined) alcoholic.[51] Since these distinctions may have implications for the type of treatment, they are important determinations to make. The development of biochemical laboratory tests to identify individuals at risk for type 2 alcoholism before the disease develops would be an important step. Such tests would present opportunities for true disease prevention in the primary care setting.

Table 3. Short Michigan Alcoholism Screening Test (SMAST)[61]

1. Do you feel you are a normal drinker? (By normal we mean do you drink less than or as much as most other people.) (No)*
2. Does your wife, husband, a parent, or other near relative ever worry or complain about your drinking? (Yes)
3. Do you ever feel guilty about your drinking? (Yes)
4. Do friends or relatives think you are a normal drinker? (No)
5. Are you able to stop drinking when you want to? (No)
6. Have you ever attended a meeting of Alcoholics Anonymous? (Yes)
7. Has drinking ever created problems between you and your wife, husband, a parent, or other near relative? (Yes)
8. Have you ever gotten into trouble at work because of drinking? (Yes)
9. Have you ever neglected your obligations, your family, or your work for two or more days in a row because you were drinking? (Yes)
10. Have you ever gone to anyone for help about your drinking? (Yes)
11. Have you ever been in a hospital because of drinking? (Yes)
12. Have you ever been arrested for drunken driving, driving while intoxicated, or driving under the influence of alcoholic beverages? (Yes)
13. Have you ever been arrested, even for a few hours, because of other drunken behavior? (Yes)

Alcoholism-indicating responses in parentheses.

Consumption

While the pattern of alcohol use may vary widely among affected individuals, the most common feature of alcoholism is heavy ethanol consumption, either on a regular or sporadic basis. Why then is a more direct approach to diagnosis not indicated? Why not just ask patients detailed questions about how much they drink? There are two reasons.

First, alcohol consumption in relatively homogeneous populations follows a smooth, unimodal distribution that approximates log normality.[56] Although problem drinkers certainly tend to be overrepresented under the high consumption tail of the curve, no clear-cut point distinguishes problem from nonproblem drinkers. Thus, alcoholism cannot be accurately defined by consumption.

Second, self-report of alcohol consumption is very unreliable, even for nonalcoholics. In a comparison of sales data with self-reported daily consumption (survey data) aggregated by geographic region, the ratio of consumption by sales to consumption by self-report ranges from 2.6 in the Northeast to 3.5 in the South.[57] In other words, individuals may consume as much as 3.5 times more ethanol than they report. Unfortunately, little information exists to determine whether heavy drinkers underreport more or less than light drinkers. Also, in our experience with interviewing, many patients do not consider "drinking" to include the consumption of beer or wine. It has been estimated that questions concerning quantity or frequency of consumption identify only about one half of the problem drinkers in a community.[36] Thus, patient self-report of consumption, although useful information, is not apt to be a reliable or clear indicator of disease.

Screening Instruments

Paper-and-pencil questionnaires that focus on specific aspects of problem drinking (eg, legal problems, fights) can be useful to the busy clinician. First, positive findings can alert the physician and lead to a more detailed workup to uncover other historical, laboratory, and physical signs or symptoms of alcoholism as described above. Second, a positive screening result can be used as another piece of information in the constellation of indicators available to the clinician.

The most widely used screening questionnaires are the Michigan Alcoholism Screening Test (MAST)[58] and the CAGE questionnaire.[23,59] The MAST is a 24-item, yes-or-no questionnaire with a weighted scoring system. Its chief drawback, particularly in screening older patients, is the wording of questions, which often leads the patient to indicate lifetime occurrence of problems rather than current occurrence. One solution has been to use a modification of the MAST that distinguishes between current, recent past, and distant past events.[60] A short MAST (SMAST) has also been developed.[61] It contains 13 MAST questions and is more easily scored than the MAST (Table 3).

The other widely used screening questionnaire is the 4-item CAGE questionnaire (Table 4), specifically designed for use in primary care.[23,59] The sensitivity and specificity of the CAGE are somewhat lower than those

Table 4. CAGE Questionnaire

1. Have you ever felt that you should Cut down on your drinking?
2. Have you every been Annoyed by criticism of your drinking?
3. Have you ever felt Guilty about your drinking?
4. Do you drink in the morning (Eye opener)?

of the MAST; however, when used as an indication of whether to do a more complex workup, the brevity of the CAGE may justify the loss in precision.

No instrument has been tested specifically on its ability to detect incipient alcoholism, although Skinner[33] has suggested that the range of possible MAST scores may approximate a continuum of severity. This suggestion implies that even low MAST scores (as low as 3 or 4) may indicate early alcohol problems. In addition, the inability to define alcoholism precisely and the absence of a reference standard for measuring actual consumption and alcohol-related problems is disquieting. Since alcoholism is a condition in which denial is a cardinal feature, there is no assurance that currently available questionnaires do not miss large numbers of problem drinkers. Despite the possibility of such occult insensitivity, however, these instruments are currently the best available means for identifying problem drinkers. They reveal significant numbers of problem drinkers who would otherwise remain unidentified in a general outpatient setting.[62,63]

One potential method for eliminating false-negative responses to items on the questionnaires is to address the questions to the patients' families. Anecdotal experiences suggest that family members will often reveal an alcohol problem that the patient denies; furthermore, there is some evidence that family members accurately report the patient's problems.[60]

Diagnosis

Recognizing alcoholism is not the same as diagnosing alcoholism. Many physicians fail to make the diagnosis after recognizing the problem because they feel uncomfortable confronting a patient with his or her alcoholism. They lack the skills and the training to deal with alcoholism, the knowledge of how and where to refer patients, and the needed confidence in the alcoholism treatment system. These blocks to diagnosis are formidable. Many programs sponsored by the National Institute on Alcohol Abuse and Alcoholism (NIAAA) have been initiated to overcome these obstacles. Such programs include the evaluation of medical school curricula and the initiation of projects to incorporate more material on alcoholism into medical education.

There are many reasons to make a formal diagnosis of alcoholism: (1) being told they have a disease legitimizes help-seeking behavior for many patients; (2) having a definite diagnosis legitimizes expenditures of health resources before severe alcoholism complications such as cirrhosis and pancreatitis are able to develop; (3) diagnosing alcoholism promotes early detection; and (4) diagnosing and treating alcoholism early may reduce health care use and expenditures in the long run. Also, regardless of whether the behavior of a patient changes as a result of being diagnosed as alcoholic, the physician may want to alter his or her system of prescribing drugs, monitoring compliance, and scheduling appointments for this patient, and will be more aware of manipulative patient behavior. Primary care physicians will treat these patients anyway, regardless of whether a diagnosis is made. It is best to deal with alcohol problems in a direct, open, and compassionate manner.

Intervention

Assuming that the incipient alcoholic can be accurately identified, two more hurdles must be surmounted before this information can be of benefit: the patient must be willing to go into treatment, and treatment must be effective. Both of these conditions present problems. In one study that addressed the willingness issue, less than 50% of problem drinkers identified by case finding in a general medical clinic agreed to participate in treatment.[62] Studies addressing the willingness of alcoholics to participate in various forms of treatment, however, including minimal treatments implemented in the primary care setting, have not been reported. It could be, for example, that while patients are unwilling to participate in intensive inpatient treatment, they may be willing to take disulfiram or to engage in brief counseling sessions with their primary care physician. A recent study at Johns Hopkins Hospital[14] indicates that self-reported patient intentions and compliance with treatment plans are highly associated with the strength of physician intervention. Those patients who had had no intervention reported no intentions and no compliance with treatment plans. Those whose physicians recommended they stop drinking, however, reported moderate levels of intention and compliance, and those whose physicians actually intervened through referral or consultation with an alcoholism counselor reported the highest levels of intention and compliance. Although willingness to participate in treatment was not measured, merely encouraging the patient to stop drinking appeared to have a moderate effect.

Should the patient be reluctant to become involved in treatment, the other approach to take is to work with the family. Clinical experience substantiates the effectiveness of formal interventions involving family members. Most treatment programs cite better effectiveness with family involvement.

The issue of the relative efficacy of early intervention as opposed to treatment of established alcoholism has never been directly studied. In fact, the efficacy of treat-

ment in any stage of alcoholism is not firmly established.[63] While a number of studies do show improvement in groups of alcoholics given various forms of treatment, they typically compare different treatment strategies with one another and do not contain a "benign neglect" control group. Studies by Vaillant,[29] however, suggest that even without treatment, the natural history of alcoholism is such that one can expect remission at the rate of 2% to 3% of patients per year. Furthermore, most of the studies comparing minimal intervention strategies with aggressive strategies show marginal or insignificant differences in rates of improvement. In the absence of control groups, it is impossible to ascertain whether these results indicate that alternative treatments are equally efficacious or ineffective.[64–67]

On the other hand, studies that have examined the determinants of successful treatment demonstrate that patients who enter treatment with more stable social profiles (eg, having jobs and family support) have a better long-term prognosis.[68] Since one of the hallmarks of advanced alcoholism is the loss of social support, these results imply that early case finding and treatment may offer some benefit.

Should direct efforts to intervene fail, the physician can always adjust the clinical decisions made about that patient. Useful indirect interventions include simplifying a patient's drug regimen and eliminating potential drugs of abuse, monitoring compliance more carefully, and being careful not to let the patient manipulate the situation.

Management

Because treatment has been dominated by the one-shot intensive inpatient episode, little has been written about the possibility of management (other than detoxification) in the primary care setting. Given the potential for early recognition, diagnosis, and intervention, a number of techniques less intensive than inpatient treatment seem feasible. One of the most effective options is to refer the patient to Alcoholics Anonymous or Adult Children of Alcoholics, and to refer family members to ALANON or ALATEEN. These programs are very successful; however, many patients will not attend self-help groups. Administration of disulfiram coupled with counseling is a logical alternative.[69] Counseling alone and even the giving of simple advice have shown some success.[65,66] In one Swedish study, heavy drinkers were identified on the basis of elevated GGT levels.[70] Physician instructions to reduce drinking and periodic monitoring of GGT levels resulted in significantly lower GGT levels in the experimental group. This approach is nonjudgmental; the physician approaches treatment in much the same way that he or she might approach the management of elevated blood pressure or blood glucose.

Conclusions

The high prevalence of alcohol problems in primary care populations provides many opportunities for physicians to have a positive impact on the health of their patients. Combining evidence from the patient's history, physical examination, and laboratory tests with patient responses on screening questionnaires such as the MAST or CAGE will allow the physician to detect considerable numbers of problem drinkers at a stage when they would have otherwise remained unrecognized and untreated.

Although there is no conclusive evidence that early treatment arrests the progression of incipient alcoholism, there is no evidence to the contrary. Given the relatively benign nature of the screening tools, more widespread incorporation of their use in primary care should be instituted. Trials of treatment and management techniques for early stage alcoholism should be conducted. With incorporation of current knowledge concerning screening and diagnosis into medical education and ultimately into medical practice, and with continued research in the areas of intervention and management, primary care physicians can better serve their patients who have alcohol problems.

References

1. Harwood HJ, Napolitano DM, Kristiansen PL, et al. Economic costs to society of alcohol and drug abuse and mental illness. Research Triangle Park, NC: Research Triangle Institute, 1980 (Publication 2734/00-01FR).
2. Niven RG. Alcoholism—a problem in perspective. JAMA 1984; 252:1912–4.
3. Hurt RD, Morse RM, Swenson WB. Diagnosis of alcoholism with a self-administered alcoholism screening test: results of 1002 patients receiving general examinations. Mayo Clin Proc 1980; 55: 365–70.
4. Magruder-Habib KM, Fraker GG, Peterson CL. Correspondence of clinicians' judgments with the Michigan Alcoholism Screening Test in determining alcoholism in Veterans Administration outpatients. J Stud Alcohol 1983; 44:872–84.
5. Magruder-Habib KM, Saltz CC, Barron P. Age-related patterns of alcoholism among veterans in ambulatory care. Hosp Community Psychiatry 1986; 37:1251–5.
6. Powers JS, Spickard A. Michigan Alcoholism Screening Test to diagnose early alcoholism in general practice. South Med J 1984; 77:852–6.

7. Leckman AL, Umland BE, Blay M. Prevalence of alcoholism in a family practice center. J Fam Pract 1984; 6:867–70.
8. Nicol EF, Ford MJ. Use of the Michigan Alcoholism Screening Test in general practice. J R Coll Gen Pract 1986; 36:409–10.
9. Wiseman SM, McCarty SN, Mitcheson MC. Assessment of drinking patterns in general practice. J R Coll Gen Pract 1986; 36:407–8.
10. King M. At risk drinking among general practice attendees: validation of the CAGE questionnaires. Psychol Med 1986; 16:213–7.
11. Coulehan JL, Zettler-Segal M, Block M, et al. Recognition of alcoholism and substance abuse in primary care patients. Arch Intern Med 1987; 147:349–52.
12. Cyr MG, Wartman SA. The effectiveness of routine screening questions in the detection of alcoholism. JAMA 1988; 259:51–4.
13. Woodall HE. Alcoholics remaining anonymous: resident diagnosis of alcoholism in a family practice center. J Fam Pract 1988; 26:293–6.
14. Moore, RD, Bone LR, Geller G, et al. Prevalence, detection, and treatment of alcoholism in hospitalized patients. JAMA 1989; 261:403–7.
15. Pearson WS. The "hidden" alcoholic in the general hospital: a study of "hidden" alcoholism in white male patients admitted for unrelated medical complaints. NC Med J 1962; 23:6–10.
16. Nolan JP. Alcohol as a factor in the illness of university service patients. Am J Med Sci 1965; 249:135–42.
17. Green JR. The incidence of alcoholism in patients admitted to medical wards of a public hospital. Med J Aust 1965; 1:465–6.
18. Barchha R, Stewart MA, Guze SB. The prevalence of alcoholism among general hospital ward patients. Am J Psychiatry 1968; 125:681–4.
19. McCusker J, Cherubin CE, Zimberg S. Prevalence of alcoholism in a general municipal hospital population. NY State J Med 1971; 71:751–4.
20. Moore RA. The prevalence of alcoholism in a community general hospital. Am J Psychiatry 1971; 128:638–9.
21. Chakerian A, Schenkel J. In support of a consolidated alcoholism treatment program. Newsletter Res Ment Health 1973; 5:37–45.
22. Favazza AR, Pires J. The Michigan Alcoholism Screening Test: application in a general military hospital. J Stud Alcohol 1974; 35:925–9.
23. Mayfield DG, McLeod G, Hall P. The CAGE questionnaire: validation of a new alcoholism screening instrument. Am J Psychiatry 1974; 131:1121–3.
24. Gomberg ES. Prevalence of alcoholism among ward patients in a Veterans Administration hospital. J Stud Alcohol 1975; 36:1458–67.
25. Quinn MA, Johnston RV. Alcohol problems in acute male medical admissions. Health Bull 1976; 34:253–6.
26. Aderhold RM. Determinants of alcoholism diagnosis and treatment. In: Proceedings of the annual meeting of the Robert Wood Johnson Foundation for Clinical Scholars. Princeton, NJ: Robert Wood Johnson, 1980.
27. Mayfield DG, Johnston RGM. Screening techniques and prevalence estimation in alcoholism. In: Fann WE, ed. Phenomenology and treatment of alcoholism. New York: Spectrum, 1981:33–4.
28. Kamerow DB, Pincus HA, MacDonald DI. Alcohol abuse, drug abuse, and mental disorders in medical practice: prevalence, cost, recognition, and treatment. JAMA 1986; 255:2054–7.
29. Vaillant GE. The natural history of alcoholism. Cambridge Mass: Harvard University Press, 1983:96–7.
30. Loper RG, Kammeier MJ, Hoffman H. MMPI characteristics of college freshman males who later become alcoholics. J Abnorm Psychol 1973; 82:159–62.
31. Jones MC. Personality correlates and antecedents of drinking patterns in adult males. J Consult Clin Psychol 1968; 32:2–12.
32. McCord J. Etiological factors in alcoholism: family and personal characteristics. J Stud Alcohol 1972; 33:1020–7.
33. Skinner HA. A multivariate evaluation of the MAST. J Stud Alcohol 1979; 40:831–44.
34. Hore BD. General practice study of commonest presenting complaints of alcoholics. J R Coll Gen Pract 1976; 26:140–2.
35. Holt S, Skinner HA, Israel Y. Early identification of alcohol abuse: 2: clinical and laboratory indicators. Can Med Assoc J 1981; 124:1279–99.
36. Wallace P, Haines A. Use of a questionnaire in general practice to increase the recognition of patients with excessive alcohol consumption. Br Med J 1985; 290:1949–53.
37. Bernandt MW, Mumford V, Taylor C, Smith B. Comparison of questionnaire and lab tests in the detection of excessive drinking and alcoholism. Lancet 1982; 1:325–8.
38. Rolaski SB, Rau D. Serum gamma glutamyl transpeptidase activity in alcoholism. Clin Chim Acta 1972; 39:41–7.
39. Rollason JG, Pincherle G, Robinson D. Serum gamma glutamyl transpeptidase in relation to alcohol consumption. Clin Chim Acta 1972; 39:75–80.
40. Westwood M, Cohen MI, McNamara MA. Serum gamma glutamyl transpeptidase activity: a chemical determinant of alcohol consumption during adolescence. Pediatrics 1978; 62:560–2.
41. Whitehead TP, Charlee CA, Whitfield AG. Biochemical and hematological markers of alcohol intake. Lancet 1978; 1:978–81.
42. Zein M, Discombe G. Serum gamma glutamyl transpeptidase as a diagnostic aid. Lancet 1970; 2:748–50.
43. Sullivan JL, Baenziger JC, Wagner DL, et al. Platelet MAO in subtypes of alcoholism. Biol Psychiatry 1990; 27:911–22.
44. Faraj BA, Lenton JD, Kutner M, et al. Prevalence of low monoamine oxidase function in alcoholism. Alcohol Clin Exp Res 1987; 11:464–7.
45. Brown JB. Platelet MAO and alcoholism. Am J Psychiatry 1977; 134:206–7.
46. Wiberg A, Gottfries CG, Oreland L. Low platelet monoamine oxidase activity in human alcoholics. Med Biol 1977; 55:181–6.
47. Sullivan JL, Cavenar JO, Maltbie AA, et al. Familial biochemical and clinical correlates of alcoholics with low platelet monoamine oxidase activity. Biol Psychiatry 1979; 14:385–94.
48. Major LF, Murphy DL. Platelet and plasma amine oxidase activity in alcoholic individuals. Br J Psychiatry 1978; 132:548–54.
49. Sullivan JL, Stanfield CN, Schanberg S, Cavenar JO. Platelet monoamine oxidase and serum dopamine-β-hydroxylase activity in chronic alcoholics. Arch Gen Psychiatry 1978; 35:1209–12.
50. Puchall LB, Coursey RD, Buchsbaum MS, Murphy DL. Parents of high risk subjects defined by levels of monamine oxidase activity. Schizophr Bull 1980; 6:338–46.
51. Cloninger CR. Neurogenetic adaptive mechanisms in alcoholism. Science 1987; 236:410–6.
52. Von Knorring AL, Bohman M, Von Knorring L, Oreland L. Platelet MAO activity in subgroups of alcoholism. Acta Psychiatr Scand 1985; 72:51–8.
53. National Council on Alcoholism, Criteria Committee. Criteria for the diagnosis of alcoholism. Am J Psychiatry 1972; 129:127–35.
54. Diagnostic and Statistical Manual of Mental Disorders, III-Revised. American Psychiatric Association, Washington, DC, 1987.
55. Schuckit MA. The clinical implications of primary diagnostic groups among alcoholics. Arch Gen Psychiatry 1985; 42:1043–9.
56. Schmidt W, deLint J. The single distribution theory of alcohol consumption. J Stud Alcohol 1978; 39:400–19.
57. Malin H, Coakley J, Kaelber C, et al. An epidemiologic perspective on alcohol use and abuse in the United States. In: National Institute on Alcohol Abuse and Alcoholism. Alcohol and health monograph 1: alcohol consumption and related problems. DHHS, Government Printing Office, 1982:99–153.
58. Selzer ML. Michigan Alcoholism Screening Test; the quest for a new diagnostic instrument. Am J Psychiatry 1971; 127:1653–8.
59. Ewing JA. Detecting alcoholism, the CAGE questionnaire. JAMA 1984; 252:1905–7.
60. Magruder-Habib KM, Harris KE, Fraker GG. Validation of the Veterans Alcoholism Screening Test. J Stud Alcohol 1982; 910–26.
61. Selzer ML, Vinokur A, vanRooijen L. A self-administered Short

Michigan Alcoholism Screening Test (SMAST). J Stud Alcohol 1975; 36:117–26.

62. Babor TF, Ritson EP, Hodgson RJ. Alcohol related problems in the primary health care setting: a review of early intervention strategies. Br J Addict 1986; 81:23–46.

63. Skinner HA, Holt S, Israel Y. Early identification of alcohol abuse. 1: critical issues and social indicators for a composite index. Can Med Assoc J 1981; 124:1141–52.

64. Miller WR, Taylor CA. Relative effectiveness of bibliotherapy: individual and group self-control training in the treatment of problem drinkers. Addict Behav 1980; 5:13–24.

65. Chick J, Ritson B, Connaughton J, Stewart A. Advice vs extended treatment for alcoholism: a controlled study. Br J Addict 1988; 83:159–70.

66. Edwards G, Orford J, Egert S, et al. Alcoholism: a controlled trial of treatment and advice. J Stud Alcohol 1977; 38:1004–31.

67. Orford J, Oppenheimer E, Edwards G. Abstinence or control: the outcome for excessive drinkers two years after consultation. Behav Res Ther 1976; 14:409–18.

68. Baekeland F. Methods for treatment of chronic alcoholism, a critical appraisal. In: Gibbins, RJ, ed. Research advances in alcohol and drug problems, vol 2. New York: John Wiley & Sons, 1975: 247–327.

69. Lundwall L, Baekeland F. Disulfiram treatment of alcoholism: a review. J Nerv Ment Dis 1971; 153:381–94.

70. Kristenson H. Studies on alcohol related disabilities in a medical intervention program in middle-aged males. Skurup, Sweden: Lindbergs Blankett AB, 1982.