

Early Diagnosis and Treatment of Alcoholism

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ANTONNETTE V. GRAHAM, RN, MSW (*Assistant Professor, Department of Family Medicine*): Today we are going to discuss what many people believe is the number-one health problem in the United States—alcoholism.¹ It is often felt that alcoholism affects the lives of more patients, either because of their own drinking or because of the drinking of a family member, than any other disease.

Current prevalency estimates of alcoholism and alcohol-related problems indicate that in many patients these problems go undetected by physicians until they become manifested by severe physical sequelae. Current thinking among experts is that early intervention leads to better prognosis. No longer is it felt that alcoholics must “bottom out” before help can be effective. These changing standards of medical care of patients with alcohol problems require residency programs to train family physicians for early detection of alcohol problems.

Residents’ education on alcoholism frequently concentrates on inpatients with medical manifestations of middle- or late-stage alcoholism.² The early diagnosis of alcoholism frequently must be made on psychosocial and behavioral indicators of problems with alcohol, and physicians trained to attend to the late medical problems can easily miss the early signs of alcoholism.

The patient being presented today is unlike the stereotypic alcoholic seen in the emergency room.

This patient is a very attractive, 34-year-old, single woman. She is currently employed as a school psychologist and is finishing her dissertation for her doctorate in psychology. She lives alone in a middle-class suburb of a large northeastern city. She is the eldest of four children. Her father is a retired college administrator, and her mother is currently employed as an executive secretary. There is no family history of alcoholism. Miss Carr’s parents and siblings drink socially. On Miss Carr’s initial visit to the Family Practice Center, her physical examination and laboratory values were normal.

MRS. GRAHAM: Thank you, Miss Carr, for attending our Grand Rounds today. I’d like to focus today’s discussion on how alcohol has affected your life—both while you were drinking and since you have stopped. Can you start by telling us about your drinking history?

MISS CARR: I had my first drink when I was 21 years old. In fact, it was to celebrate my birthday. I had never experimented with drinking during my teen years. I was the wholesome, cheerleader type. Also, I was quite religious and felt drinking was the wrong thing to do.

MRS. GRAHAM: What happened with your first drink when you were 21?

MISS CARR: Well, I didn’t stop with one. I had five screwdrivers and became drunk! I was in college and living at home, and I do remember having a hangover the next day. I spent that whole day in bed except when I was vomiting. After that, I began to drink regularly and to get drunk quite frequently. You’d think that with such a beginning I would never have touched alcohol again, but not so. I noticed that I seemed to drink faster than my

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This fixed combination drug is not indicated for initial therapy of hypertension. Hypertension requires therapy titrated to the individual patient. If the fixed combination represents the dose so determined, its use may be more convenient in patient management. The treatment of hypertension is not static, but must be re-evaluated as conditions in each patient warrant.

INDICATIONS AND USAGE: MINIZIDE (prazosin hydrochloride/polythiazide) is indicated in the treatment of hypertension. (See box warning.)

CONTRAINDICATIONS: RENESE (polythiazide) is contraindicated in patients with anuria, and in patients known to be sensitive to thiazides or to other sulfonamide derivatives.

WARNINGS: MINIPRESS (prazosin hydrochloride): MINIPRESS may cause syncope with sudden loss of consciousness. In most cases this is believed to be due to an excessive postural hypotensive effect, although occasionally the syncope episode has been preceded by a bout of severe tachycardia with heart rates of 120-160 beats per minute. Syncope episodes have usually occurred within 30 to 90 minutes of the initial dose of the drug. Occasionally they have been reported in association with rapid dosage increases or the introduction of another antihypertensive drug into the regimen of a patient taking high doses of MINIPRESS. The incidence of syncope episodes is approximately 1% in patients given an initial dose of 2 mg or greater. Clinical trials conducted during the investigational phase of this drug show that syncope episodes can be minimized by limiting the initial dose of the drug to 1 mg, by subsequently increasing the dosage slowly, and by introducing any additional antihypertensive drugs into the patient's regimen with caution (see DOSAGE AND ADMINISTRATION). Hypotension may develop in patients given MINIPRESS who are also receiving a beta-blocker such as propranolol.

If syncope occurs, the patient should be placed in the recumbent position and treated supportively as necessary. This adverse effect is self-limiting and in most cases does not recur after the initial period of therapy or during subsequent dose titration.

Patients with dizziness should be cautioned about these possible adverse effects and advised what measures to take should they develop. The patient should also be cautioned to avoid situations where injury could result should syncope occur during the initiation of MINIPRESS therapy.

RENESE: RENESE should be used with caution in severe renal disease. In patients with renal disease, thiazides may precipitate azotemia. Cumulative effects of the drug may develop in patients with impaired renal function.

Thiazides should be used with caution in patients with impaired hepatic function or progressive liver disease, since minor alterations of fluid and electrolyte balance may precipitate hepatic coma.

Sensitively reactions may occur in patients with a history of allergy or bronchial asthma.

The possibility of exacerbation or activation of systemic lupus erythematosus has been reported.

Thiazides may be additive or potentiative of the action of other antihypertensive drugs.

Potentiation occurs with ganglionic or peripheral adrenergic blocking drugs.

Periodic determinations of serum electrolytes to detect possible electrolyte imbalance should be performed at appropriate intervals.

All patients receiving thiazide therapy should be observed for clinical signs of fluid or electrolyte imbalance, namely, hypotatremia, hypokalemia, and hypocalcemia. Serum and urine electrolyte determinations are particularly important when the patient is vomiting excessively or receiving parenteral fluids. Medications such as digitalis may also influence serum electrolytes. Warning signs, irrespective of cause, are: dryness of mouth, thirst, weakness, lethargy, drowsiness, restlessness, muscle pains or cramps, muscular fatigue, hypotension, oliguria, tachycardia, and gastrointestinal disturbances such as nausea and vomiting.

Hypokalemia may develop with thiazides as with any potent diuretic, especially with brisk diuresis, when severe cirrhosis is present, or during concomitant use of corticosteroids or ACTH.

Interference with adequate oral electrolyte intake will also contribute to hypokalemia. Digitalis therapy may exaggerate the metabolic effects of hypokalemia, especially with reference to myocardial activity.

Ancient deficit is generally mild and usually does not require specific treatment except under extraordinary circumstances (as in hepatic or renal disease). Dilutional hyponatremia may occur in edematous patients in hot weather; appropriate therapy is water restriction rather than administration of salt, except in rare instances when the hyponatremia is life-threatening. In actual salt depletion, appropriate replacement is the therapy of choice.

Hypercalcemia may occur or frank gout may be precipitated in certain patients receiving thiazide therapy.

Insulin requirements in diabetic patients may be either increased, decreased, or unchanged. Latent diabetes mellitus may become manifest during thiazide administration.

The drug may increase responsiveness to tubocurarine.

The antihypertensive effects of the drug may be enhanced in the post-sympathectomy patient.

Thiazides may decrease arterial responsiveness to norepinephrine. This dimintion is not sufficient to preclude effectiveness of the pressor agent for therapeutic use.

If progressive renal impairment becomes evident, as indicated by a rising nonprotein nitrogen or blood urea nitrogen, a careful reappraisal of therapy is necessary with consideration given to withholding or discontinuing diuretic therapy.

Thiazides may increase the levels of serum cholesterol and triglycerides.

PRECAUTIONS: Carcinogenesis, Mutagenesis, Impairment of Fertility: No carcinogenic or mutagenic studies have been conducted with MINIZIDE (prazosin hydrochloride/polythiazide). However, no carcinogenic potential was demonstrated in 18-month studies in rats with either MINIPRESS (prazosin hydrochloride) or RENESE (polythiazide) at dose levels more than 100 times the usual maximum human doses. MINIPRESS was not mutagenic in *in vivo* genetic toxicology studies.

MINIZIDE produced no impairment of fertility in male or female rats at 50 and 25 mg/kg/day of MINIPRESS and RENESE respectively. In chronic studies (one year or more) of MINIPRESS in rats and dogs, testicular changes consisting of atrophy and necrosis occurred at 25 mg/kg/day (60 times the usual maximum recommended human dose). No testicular changes were seen in rats or dogs at 10 mg/kg/day (24 times the usual maximum recommended human dose). In view of the testicular changes observed in animals, 105 patients on long-term MINIPRESS therapy were monitored for 17-ketosteroid excretion and no changes indicating a drug effect were observed. In addition, 27 males on MINIPRESS alone for up to 51 months did not have changes in sperm morphology suggestive of drug effect.

Use in Pregnancy: Pregnancy Category C. MINIZIDE was not teratogenic in either rats or rabbits when administered in oral doses more than 100 times the usual maximum human dose. Studies in rats indicated that the combination of RENESE (40 times the usual maximum recommended human dose) and MINIPRESS (8 times the usual maximum recommended human dose) caused a greater number of stillbirths, a more prolonged gestation, and a decreased survival of pups to weaning than that caused by MINIPRESS alone. There are no adequate and well-controlled studies in pregnant women. Therefore, MINIZIDE (prazosin hydrochloride/polythiazide) should be used in pregnancy only if the potential benefit justifies the potential risk to the fetus.

Use in Nursing Mothers: It is not known whether MINIPRESS (prazosin hydrochloride) or RENESE (polythiazide) are excreted in human milk. Thiazides appear in breast milk. Thus, if use of the drug is deemed essential the patient should stop nursing.

Pediatric Use: Safety and effectiveness in children has not been established.

ADVERSE REACTIONS: MINIPRESS: The most common reactions associated with MINIPRESS therapy are: dizziness 10.3%, headache 7.8%, drowsiness 7.6%, lack of energy 6.9%, weakness 6.5%, palpitations 5.3%, and nausea 4.9%. In most instances side effects have disappeared with continued therapy or have been tolerated with no decrease in dose of drug.

The following reactions have been associated with MINIPRESS, some of them rarely. (In some instances exact causal relationships have not been established.)

Gastrointestinal: vomiting, diarrhea, constipation, abdominal discomfort and/or pain.

Cardiovascular: edema, dyspnea, syncope, tachycardia.

Central Nervous System: nervousness, vertigo, depression, paresthesia.

Dermatologic: rash, pruritus, alopecia, lichen planus.

Genitourinary: urinary frequency, incontinence, impotence, priapism.

ENT: blurred vision, reddened sclera, epistaxis, tinnitus, dry mouth, nasal congestion.

Other: diabetes.

Single reports of pigmentary mottling and serous retinopathy, and a few reports of cataract development or disappearance have been reported. In these instances, the exact causal relationship has not been established because the baseline observations were frequently inadequate.

In more specific slit-lamp and funduscopic studies, which included adequate baseline examinations, no drug-related abnormal ophthalmological findings have been reported.

RENESE: Gastrointestinal: anorexia, gastric irritation, nausea, vomiting, cramping, diarrhea, constipation, jaundice (intrahepatic cholestatic jaundice), pancreatitis.

Central Nervous System: dizziness, vertigo, paresthesia, headache, xanthopsia.

Hematologic: leukopenia, agranulocytosis, thrombocytopenia, aplastic anemia.

Dermatologic: purpura, photosensitivity, rash, urticaria, necrotizing angitis, (vasculitis) (cutaneous vasculitis).

Cardiovascular: Orthostatic hypotension may occur and be aggravated by alcohol, barbiturates, or narcotics.

Other: hypoglycemia, glycosuria, hyperuricemia, muscle spasm, weakness, restlessness.

OVERDOSAGE: RENESE: Accidental ingestion of at least 50 mg of MINIPRESS in a two-year-old child resulted in profound drowsiness and depressed reflexes. Blood pressure was noted. Recovery was uneventful.

Should overdosage lead to hypotension, support of the cardiovascular system is of first importance. Restoration of blood pressure and normalization of heart rate may be accomplished by keeping the patient in the supine position. If this measure is inadequate, shock should first be treated with volume expanders. If necessary, vasopressors should then be used. Renal function should be monitored and supported as needed. Laboratory data indicate that MINIPRESS is not dialysable because it is protein bound.

RENESE: Should overdosage with RENESE occur, electrolyte balance and adequate hydration should be maintained.

Gastric lavage is recommended, followed by supportive treatment. Where necessary, this may include intravenous dextrose and saline with potassium and other electrolyte therapy administered with caution as indicated by laboratory testing at appropriate intervals.

DOSAGE AND ADMINISTRATION: MINIZIDE (prazosin hydrochloride/polythiazide): Dosage: as determined by individual titration of MINIPRESS (prazosin hydrochloride) and RENESE (polythiazide). (See box warning.)

Usual MINIZIDE dosage is one capsule two or three times daily, the strength depending upon individual requirement following titration.

The following is a general guide to the administration of the individual components of MINIZIDE.

MINIPRESS: Initial Dose: 1 mg two or three times a day. (See Warnings.)

Maintenance Dose: Dosage may be slowly increased to a total daily dose of 20 mg given in divided doses. The therapeutic dosages most commonly used have ranged from 6 mg to 15 mg daily given in divided doses. Doses higher than 20 mg usually do not increase efficacy; however, a few patients may benefit from further increases up to a daily dose of 40 mg given in divided doses. After initial titration some patients can be maintained adequately on a twice-daily dosage regimen.

Use With Other Drugs: When adding a diuretic or other antihypertensive agent, the dose of MINIPRESS should be reduced to 1 mg or 2 mg three times a day and retitration then carried out.

RENESE: The usual dose of RENESE for antihypertensive therapy is 2 to 4 mg daily.

HOW SUPPLIED:

STRENGTH	COMPONENTS	COLOR	CAPSULE CODE	PKG. SIZE
MINIZIDE 1	1 mg prazosin + 0.5 mg polythiazide	Blue-Green	430	100's
MINIZIDE 2	2 mg prazosin + 0.5 mg polythiazide	Blue-Green/Pink	432	100's
MINIZIDE 5	5 mg prazosin + 0.5 mg polythiazide	Blue-Green/Blue	436	100's

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friends. I could never sip a drink. About the time I moved out of my parents' home and graduated from college, I got into some real heavy drinking. Most of my friends were college dropouts or college graduates who had just "dropped out" of the mainstream. I decided that I wanted to work with my hands and became a carpenter's apprentice. After a few months I realized this life wasn't for me, and I entered graduate school in another town.

MRS. GRAHAM: What about your drinking then?

MISS CARR: For a while I gave it up and was a model student. Then I discovered marijuana. I really liked the high I got with marijuana. It made me very anxious, however, so I began drinking to calm down. A group of us would get together over the weekend and get stoned on booze and marijuana. It got so bad that I'd be high from Thursday through Sunday.

MRS. GRAHAM: How were you managing in school?

MISS CARR: Surprisingly well. I was always a very good student and somehow managed to keep my grades up well enough to graduate with my master's degree.

MRS. GRAHAM: Then, after graduation, how did your first job go?

MISS CARR: I moved again. I was alone in a new town and felt overwhelmed with the responsibilities of the job. This gave me a good reason to drink.

MRS. GRAHAM: By this time, what difficulties were you getting into because of your drinking?

MISS CARR: Fortunately for me there was a real "drunk" in the office where I worked, and the people I worked with excused my little mishaps—like arriving late to work in the morning—because I had no problems compared with Dorothy. Socially, though, I was really having problems. I was spending many nights at a bar picking up new men. Although I'd hate myself in the morning and swear I'd never do it again, I always found myself back in the bar. I'm sure that's where I picked up herpes. I guess you'd say getting herpes was a side effect of my drinking. I just hated myself. But I couldn't seem to get out of the drinking rut.

MRS. GRAHAM: During that period, were you getting routine health care?

MISS CARR: Yes, but none of the doctors ever asked me about my drinking. Finally I decided to move back near my parents and to clean up my life.

MRS. GRAHAM: How did that go?

MISS CARR: Again, it went well for a while. I guess, in AA language, I was on a "dry drunk" for nine months. I was difficult to be around, very moody, and I couldn't figure out why. I wasn't drinking, but I was nowhere near getting treatment or even admitting I had an alcohol problem. Gradually my drinking got worse again.

MRS. GRAHAM: When did you begin having blackouts?

MISS CARR: Several years ago, during the period when I had my first job. Some of the things I did then, of course, I don't remember, but friends told me about my behavior. What I did would have been enough to make anyone but an alcoholic quit drinking. What surprised me was that people didn't know I was in a blackout period. Later they would tell me what I had done, and act very surprised that I couldn't remember. I guess my behavior in general seemed so normal.

MRS. GRAHAM: How did your family react to your drinking?

MISS CARR: At first my drinking was considered "Julie's problem" and no one discussed it. Finally, a week before I saw you, my mother did what I guess was an intervention. She sat me down and told me all about the times I had thrown up on the rug, embarrassed her in front of her friends, smashed up my car, and acted out of control after drinking. This made me think about getting my life in order. I still wasn't ready to admit that I had a drinking problem, however. I felt I was drinking because I had problems. When I called the Family Practice Center and requested an appointment with you, I wanted to work on my problems, not on my drinking. In fact, I was determined not to tell you about my drinking. As we began to focus, somewhat against my desire, on my drinking, I realized that alcohol treatment was the way I had to go. When you dialed the AA number and handed me the phone, I felt frightened but relieved that I had taken my first step toward treatment of my drinking problem.

MRS. GRAHAM: You've been a member of AA now for almost a year. What has it been like now that you are not drinking?

MISS CARR: It's great. I've never felt better.

Of course I have times when I'm down and times when I'm anxious, but it feels so much better to be in control of my life.

MRS. GRAHAM: Would you mind if we had a few questions from the audience?

MISS CARR: No, that would be fine.

RESIDENT PHYSICIAN: Miss Carr, since we are trying to find out how physicians can diagnose alcoholism earlier, what could the physician have asked you on your first visit here that would have helped discover you were having a drinking problem?

MISS CARR: I guess if I had been asked a little more about my drinking, especially if I was having blackouts, that would have made me real anxious, but I think I would have told the truth had I been pushed a little more. Instead it was easy for me to lie and just say I was having two to four glasses of wine or beer a week.

MEDICAL STUDENT: Miss Carr, you stated that you didn't drink when you were a teenager. Do you think if you had experimented earlier, you might not have developed such a problem with alcohol later?

MISS CARR: No, I think I just would have been a teenage alcoholic. I had a problem with alcohol right from the start. If I had started earlier, I just would have had the problem earlier.

MRS. GRAHAM: I'd like to thank you again, Miss Carr, for coming in today.

MISS CARR: I'm glad that I was able to share my story with you because I feel that physicians need to be informed about alcoholism, especially in people who don't look as if they're having problems.

(The patient then left the conference.)

RESIDENT PHYSICIAN: When I saw Miss Carr for her initial history and physical examination, I had a strange feeling about her. I couldn't quite put my finger on it, but the two of us didn't click. I felt as though she was hiding something.

DR. JAY S. THOMPSON (*Private practitioner in radiology*): Your statement is singularly perceptive. Ideally, the history-taking process is a factual and nonjudgmental accumulation of data. In actuality, however, we interact with our patient. Some patients produce very positive responses in the examiner. With others, the examiner may be uncomfortable. Particular lines of

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questioning may cause unease, both with the physician and with the patient. Whenever we begin to pursue such subjects as sexual dysfunction, alcoholism, homosexuality, child abuse, chemical dependency, venereal disease, and so on, it is important for us first to examine and come to terms with our own feelings and attitudes.

Listen for the things the patient doesn't tell you. Sometimes the quality of the response is more important than the response itself. Body language can be revealing. When the patient seems uncomfortable, or hesitant with a particular line of questioning, we must ask ourselves why.

Watch for mobilization of defense mechanisms. Denial is a constant theme in this illness. Rationalization, minimization, and projection may become apparent as the history-taking proceeds.

DR. DAVID SEDLACEK (*Assistant Clinical Professor, Department of Family Medicine*): This case is an excellent illustration of early-stage alcoholism, that is, alcoholism in which there is an absence of rapidly identifiable physical symptoms. Consequently, using our standard classification system can be misleading. According to the DSM-III, the diagnosis of alcoholism cannot be made without the presence of withdrawal symptoms.³ In Miss Carr's case there were no apparent withdrawal symptoms—such as tremors during the time of the examination or reported by the patient. In addition there were no obvious alcohol-related physical symptoms.

A more useful description of alcoholism is that it is a chronic, primary, and progressive disease with readily identifiable signs and symptoms.⁴ It can be defined as an inconsistent inability to drink alcohol safely; that is, the consequences of drinking are more important to diagnosis than the type of beverage used, the pattern of drinking, or the amount consumed. It is less important for the physician to ask about the amount of alcohol consumed than to find out about the consequences of that consumption.

There are several common barriers to the diagnosis of alcoholism⁵: (1) taking an inadequate alcohol-drug history, (2) accepting what the patient says as correct, (3) viewing alcoholism as a symptom of an underlying illness, and (4) viewing alcoholism as a unitary phenomenon. To explain this fourth point further, it is well known that not

all alcoholics show the same symptoms. Not only are there different types of alcoholism, there are also different stages in its progression. The earlier the alcoholism is detected, the easier it is to treat.

A physician must be aware of the existing treatment resources in a given locality in order to confidently diagnose early alcoholism.⁶ Alcoholics Anonymous is the best referral for those who express a desire to stop drinking and who will not experience problems with withdrawal. Outpatient services can be used as a primary treatment for those persons with a strong support system and an unwillingness or inability to enter an inpatient center. The treatment of choice for most alcoholics is an inpatient or residential treatment center, where intensive exposure to Alcoholics Anonymous, family treatment, and education to the disease concept of alcoholism can be accomplished in 14 to 28 days. This is especially important for the patient who is exhibiting a lot of denial. Physicians are advised to visit and investigate the alcoholism services provided in their locality in order to be able to make referrals appropriately. The local information and referral service of The Alcoholism Services Coordinating Agency can guide a physician with this task. Since alcoholism is a family disease, it is important for the physician to remember the family will need treatment regardless of whether the alcoholic chooses to be treated. Al-Anon is for persons close to an alcoholic and helps them work on their own problems. Families Anonymous is primarily for parents of alcoholic teenagers.

DR. THOMPSON: Remember, this is a *family* illness. The life of the alcoholic reaches out and touches significant others around him. Whenever alcoholism is clinically suspect, an alcohol history should be obtained from other family members as well. Not only is this necessary to confirm the examiner's suspicions, it may be the first step in intervention planning and referral to family counseling.

A good alcohol history should establish a drinking pattern. The following kinds of question should be asked: "When did drinking begin? Can the patient remember his first drink? Is drinking confined to weekends and social occasions, or is the patient a daily drinker? Is beer, wine, or hard liquor preferred? Remember that the quantity of alcohol consumed is not necessarily as important as the consequences of the drinking behavior."⁷

Is the patient using other medications or street drugs in addition to alcohol? Fifty percent of all women and 30 percent of men entering treatment programs for alcoholism are, in truth, polydrug dependent, and with adolescents, multiple drug use is the rule rather than the exception.

I have a number of screening questions I am particularly fond of using in obtaining an alcohol history:

1. Have you ever had a blackout?
2. Has a family member, friend, or business associate ever expressed concern over your drinking?
3. Have you ever tried to control your drinking?
4. Have you ever been worried that you might drink too much?
5. Have you ever been arrested for driving while intoxicated or hospitalized as the result of your drinking?

Finally, a word may be said on management of the recovering alcoholic. Abstinence from all mood-altering chemicals is basic to the recovery process. Alcohol in any form is absolutely contraindicated, and there is a strong relative contraindication to the use of tranquilizers, sleeping pills, mood elevators, and analgesics other than those such as aspirin. Even antihistamines may produce sedative side effects and are to be avoided. For the recovering alcoholic, sobriety is a precious commodity and a benchmark of living. It is important that we recognize this fact if we are to assist in the recovery process.

Working with the alcoholic can be a frustrating process or a rewarding experience. I know of no other illness in which patients get better than they were before they were sick. Those who survive their illness and recover know a depth of spirit one can barely measure. They have not suffered in vain.

DR. KENNETH G. REEB (*Associate Professor, Department of Family Medicine*): There are two general methods to improve physician effectiveness in the early detection of patients with alcohol problems:

1. Educate physicians about early signs and symptoms of alcoholism and improve their awareness of attitudinal problems that might interfere with their willingness to deal with alcoholic patients.⁸
2. Develop information systems that augment

physician memory and help organize some of the repetitive tasks involved in problem detection.

Thus far in this conference we have focused on physician learning. I want to propose a supplemental method to help our group accomplish its earlier detection goals. A structured form that incorporates a minimum alcohol utilization data base in each adult patient's chart will be combined with our existing adult health supervision protocol form. This alcohol utilization data base will remind each of us to collect this information on every patient. It will facilitate recording this information about any patient's or any patient's family's alcoholism status and will facilitate audit of charts for alcohol problems. Ultimately the form lends itself to computerization. Similar computerized reminder systems have been shown to improve physician compliance with practice policy.⁹

MRS. GRAHAM: We believe alcoholism is a treatable disease that causes less family disruption the earlier it is diagnosed. Family physicians are in a unique position to make an early diagnosis of alcoholism and to help the entire family in the treatment and recovery process.

This brings to a conclusion our Grand Rounds for today.

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