

# **COURT PROCEDURES FOR IDENTIFYING PROBLEM DRINKERS**

## **Volume 2: Supplementary Readings**

Barbara Mudge  
Margaret W. Kerlan  
David V. Post  
Rudolf G. Mortimer  
Lyle D. Filkins

*Highway Safety Research Institute  
The University of Michigan  
Ann Arbor, Michigan 48105*

June, 1971

National Highway Traffic Safety Administration  
U.S. Department of Transportation  
Washington, D.C. 20591



## NOTICES

Sponsorship. Prepared for U.S. Department of Transportation, National Highway Traffic Safety Administration, under contract Number FH-11-7615. The work reported herein was conducted under this contract by The University of Michigan's Highway Safety Research Institute. Contracts and grants to The University of Michigan for the support of sponsored research by the Highway Safety Research Institute are administered through the Office of the Vice-President for Research. The National Highway Traffic Safety Administration Contract Technical Manager has been Robert B. Voas, Ph.D., of the Office of Alcohol Countermeasures.

The opinions, findings, and conclusions expressed in this publication are those of the authors and not necessarily those of the National Highway Traffic Safety Administration.

COURT PROCEDURES FOR  
IDENTIFYING PROBLEM DRINKERS  
VOLUME II: SUPPLEMENTARY READINGS

Barbara Mudge  
Margaret W. Kerlan  
David V. Post  
Rudolf G. Mortimer  
Lyle D. Filkins



## CONTENTS

Introduction. . . . .	1
Interviewer Qualifications. . . . .	2
Questions and Answers on Program Philosophy . . . . .	3
Rationale for Selection of Questionnaire Items. . . . .	9
The Consequences of Alcohol Use and Abuse . . . . .	15
The Body's Response to Alcohol. . . . .	15
Introduction . . . . .	15
The Metabolism of Ethyl Alcohol. . . . .	15
Intoxication . . . . .	20
Complications of Alcohol Abuse. . . . .	23
Injury and Trauma During Intoxication. . . . .	23
Adverse Effects of Alcohol Withdrawal. . . . .	26
Medical Complications of Alcohol Abuse . . . . .	30
Summary. . . . .	45
Appendix A: The Development of a Court-Related Rehabilitation Program. . . . .	47
Appendix B: A Description of the Services Provided by Volunteers in Probation, Inc. . . . .	51
Appendix C: Resources Available to Stimulate the Development of Probation Services . . . . .	55
References. . . . .	58



## INTRODUCTION

These supplementary readings should prove useful for people working with the DUI offender, such as judges, court counselors, and other personnel dealing with rehabilitation. The following is a description of each reading.

### I. INTERVIEWER QUALIFICATIONS

Part I provides a description of interviewer qualifications necessary to perform the prescribed tasks involved in the Identification Procedure.

### II. QUESTIONS AND ANSWERS ON PROGRAM PHILOSOPHY

It is anticipated that personnel working with the DUI offender may have further questions concerning the philosophy behind the development of the Identification Procedures.

### III. RATIONALE FOR SELECTION OF QUESTIONNAIRE ITEMS

Since most of the Questionnaire items are not obvious drinking questions, personnel may want to know what relationship the questions do have to problem drinking identification.

### IV. CONSEQUENCES OF ALCOHOL USE AND ABUSE

A discussion is presented of what happens when a person drinks to excess. Physical changes are described which will be temporary or permanent, depending upon the degree and length of alcohol abuse.

## INTERVIEWER QUALIFICATIONS

There will be a great deal of latitude allowed on the type of person selected as a pre-sentence investigator or court counselor. He may have little or no special training or be highly trained, e.g., a psychiatric social worker. The choice will depend on several factors, including the number of drinking offenders who come through a single court, money allotted for special programs and personnel, and people available for the position.

However, there are basic minimum requirements that can be discussed within the context of the jobs to be performed. (1) The person will be required to administer a Questionnaire and Interview-- both of which demand reading skill and verbal comprehension. (2) The interviewer must be able to follow the directions for administering the procedure and scoring the results. (3) He must have simple, basic mathematical skills in order to score the procedure. (4) He must be able to communicate his findings in writing in order to maintain a file on each offender. (5) He may have to speak a second language (e.g. Spanish) in courts where there is a high ratio of non-English speaking people. (6) Most importantly, the counselor should be a sensitive, empathetic person who is truly concerned about the people he deals with and is readily able to communicate this concern to them. He should be capable of establishing rapport quickly and easily.

One possibility for a court that has little or no funding available for extra personnel is a professional volunteers program. This approach was initially developed in Royal Oak, Michigan and is now in use in some 500 courts throughout the country. A description of the program and people to contact for further information is provided in Appendices A-C.

In summary, the counselor need not necessarily have a great deal of education or special training. The job does require innate intelligence, as well as concern and respect for others.



## QUESTIONS AND ANSWERS ON PROGRAM PHILOSOPHY

In this section we hope to answer questions concerning the rationale for using the procedure previously described, together with traditional legal sanctions; and concomitant consideration about problem drinking and the person who commits a drinking-related offense.

### Q.1. Why is this procedure used only with persons arrested for drinking-related offenses?

Given the time and cost constraints within the present court system, it would be impossible for many courts to carry on a pre-sentence investigation for every offender who comes through the system. Since it is already known that a large percentage of persons convicted of drinking-related offenses are problem drinkers, then anyone arrested for a drinking-related offense essentially has already identified himself as a potential problem drinker.

Also, a significant number of people commit crimes of lesser to major seriousness while under the influence of liquor. It may be one of the largest known single factors related to crime, and needs to be probed further.

### Q.2. Why do you think offenders who commit a drinking-related offense are problem drinkers?

One researcher who compared histories of DUIL offenders with the general driving population, found that, "Persons convicted of drunk driving tend to have more problems as a result of their drinking: alcohol seems to upset their spouses, affect their budget, interfere with their jobs and accompany crimes more often than for persons never convicted of drunk driving."<sup>1</sup> For example, 37% of the first offense DUILs had a previous alcohol-related arrest as compared to 3% of the driver sample. Forty-one percent of the first time DUIL offenders and 68% of the third time DUIL offenders had previous criminal records of less serious crimes as compared to 7% of the driver sample.

One psychiatrist concluded that 78% of the DUIs he interviewed were alcoholics, probable alcoholics, or prealcoholics.<sup>2</sup> Another researcher contended that 74% of the DUIs he interviewed demonstrated multiple symptoms of problem-oriented drinking, as shown by their past histories of medical or social drinking problems.<sup>3</sup> Other studies have consistently revealed that DUI offenders and those who commit other types of drinking-related offenses, often have problems that are outgrowths of their drinking habits. Thus, we can conclude that they tend, as a group, to be problem drinkers.

Q.3. Why are traditional legal sanctions alone not adequate in dealing with the drinking offender?

Traditional sanctions, such as a fine, jail, or license revocation are adequate deterrents for some drinking offenders. But for the problem drinker the traditional approach is often not adequate. He will probably continue to drink to excess and, according to his previous history, get into trouble again. The following sample driver record (Table 1) is a vivid illustration of this pattern. If we want to prevent a repetition of this pattern, or something worse, such as a fatal crash, then we must deal with the causes of the behavior related to the crime, and excessive drinking is often the cause.

Q.4. What is meant by the word "treatment?"

Treatment, in this context, would denote any effort outside of, or in addition to, the traditional approach of fine or jail. Treatment may range from driver retraining or alcohol education to clinical treatment for alcoholism.

Q.5. Why do you think the treatment approach will work?

For convenience let's look at the results of alcoholism treatment. Courts that have instigated court-related treatment programs claim successes which range from 38-90% of their groups.<sup>4</sup> Industrial programs which deal with the alcoholic claim a 60-80% success rate.<sup>5</sup>

TABLE 1. Sample Driver Record of a DUIL Offender\*

Date	Event	Response
DEC 1959	Reckless Driving.	
SEP 1960		Re-exam, instructions given.
DEC 1960		Re-exam, restricted license issued may drive to and from work, and on road for testing cars.
NOV 1961		License appeal board hearing, restrictions lifted.
APR 1963	DUIL	Mandatory suspension of 3 months.
OCT 1963	Prohibited turn.	
JAN 1964	Speed 50/35.	
JAN 1965	Crash: 2 veh., 1 injury; DUIL conviction.	Mandatory suspension from NOV 1965 - AUG 1966.
SEP 1965		Re-exam, license revoked.
OCT 1966		License denied on basis of 2 previous DUILs.
NOV 1966		License appeal board hearing, restricted license issued, no pleasure driving, NOV 1966 - NOV 1967.
MAR 1968	Crash: 4 veh., 1 injury; Improper driving, illness not known if drink- ing.	
JUL 1968		Referred for review.
SEP 1968	Crash: 2 veh., 2 injuries; DUIL 0.34% W/V.	
OCT 1968		Re-exam, corrected physical condition.
DEC 1968		Mandatory suspension, DEC 1968 - Jun 1970.
FEB 1969		Financial responsibility denied because of 2 or more convictions of DUIL.
MAR 1969		Favorable doctor's statement held by driver improvement.

---

\*From: Proceedings of a Conference on Community Response to Alcoholism and Highway Crashes, Filkins & Geller (Ed.), Michigan, University of Michigan, Highway Safety Research Institute, 1970.

Aside from the group of chronic alcoholics who go through the courts, there is also a younger group which has problems with drinking but may or may not be physically dependent upon alcohol. Fifty-five percent of the DUIs going through the Fifteenth District Court in Ann Arbor, Michigan are under 35 years of age. This suggests that the opportunity for early intervention exists for a significant portion of the court population. Early intervention is believed to be a key factor in successful treatment outcome.

Q.6. Is treatment less likely to work with the drinking offender who is physically dependent upon alcohol?

In the past we have largely dealt ineptly with the alcoholic drinking offender. He has often been called the "revolving door alcoholic" because he would be arrested, jailed, and released, only to be re-arrested shortly thereafter, to repeat the process. In this approach there was no attempt to recognize the basic problem: the inability of the chronic alcoholic to control his drinking.

Jail would not change this drinking behavior any more than it would that of the heroin addict. The chronic alcoholic may first of all need medical attention so he can "dry out." After that, rehabilitative efforts can be made to deal with the various facets of his life that need "repair."

Q.7. How do you get someone to enter treatment?

A term which is now frequently used to describe the process of talking someone into treatment is called "constructive coercion." Industry coerces the alcoholic into treatment by threatening job loss if he does not choose treatment.<sup>6</sup> The constructive element is the assumption that, in many cases, the alcoholic cannot help himself and is very defensive, denying vehemently that he has a drinking problem.

Some courts, depending on local ordinance or statute, may be

able to use this approach by giving the offender a choice: fine, jail, treatment, or a combination of these. Obviously, there will be pressure on the offender to choose treatment, if in the courts' opinion, this form of intervention is believed to be most effective for the offender.

Q.8. Does the pressure involved in making the decision to enter treatment make this person a poor treatment risk?

There is no indication that the pressure per se, or the individual's initial reticence will make him a poorer risk. One researcher who studied 1038 people and their motivations for entering a hospital for alcoholism treatment concluded that few would have sought treatment if it had not been for pressure from wife, employer, or someone else.<sup>7</sup> Ultimately many who entered treatment under those conditions were successfully treated.

Q.9. How do you prevent pulling the social drinker into the treatment system?

You do not. One of the biggest concerns is how to deal with the social drinker who, on occasion, drinks too much and drives, and at least on that occasion becomes a problem drinking driver. Obviously, the social drinker who winds up being arrested for drinking and driving has some incorrect assumptions about his own ability to drive safely after drinking. Besides the traditional sanctions, alcohol education would be appropriate for this person, and depending on his driving record, perhaps even driver re-training.

Q.10. Why does the procedure have to be a standard one?

A standard procedure minimizes errors due to differences in training of the persons administering the technique, or differences in the conditions of administration. Use of a standard technique also makes it possible to measure the effectiveness of the technique and provides a documented base technique from which improved diagnostic methods can evolve. It can be more readily compared to the same technique used in other programs for screening persons for intake, and it can be compared with other techniques.

More effective research into improvement of existing programs or potential effectiveness of proposed new programs is thus made possible. Standardization also permits conclusions to be drawn about how the populations under test differ from one locale to another or how they change over time. All of the above factors will provide greater insight into the problem and will aid in developing innovative countermeasures in dealing with it.

Q.11. Because of its uniformity, will this approach hinder the interaction between the interviewer and the client?

No. Open-ended questions have been provided specifically so the interviewer will not be hindered but will feel free to elaborate upon questions or delve further into the answers given to him.

Q.12. Will this procedure be more costly to the courts?

At this point we do not know. At a minimum, it would require one person to administer the Questionnaire and Interview (depending on the size of the court load). Other expense would include printing the Questionnaire and Interview, searching for criminal and driver records, and related costs. However, the cost of developing and applying a solution to the problem must be considered in the context of the present cost of nonsolution. According to a U.S. Senator, "In economic terms, alcohol and related problems cost the private and public economy more than \$15 billion annually."<sup>8</sup> This cost of "nonsolution" involves lives lost in fatal accidents; 8 billion dollars to industry; costs to the legal system; and most importantly, the loss to society of a viable human being.

Q.13. Do both Questionnaire and Interview always have to be used together?

The Questionnaire and Interview have been designed to use the minimum time necessary to assure an adequate assessment. Since they are sensitive to somewhat different areas of the person's life, and attempt to get at similar kinds of information through

different routes, both kinds of evidence are valuable. Either one may off-set any inaccuracy which occurs for any reason in the other one. For example, a person who is very defensive about his drinking and gives dishonest or evasive answers to the Interview questions may still answer the more subtle items of the Questionnaire in such a manner that his drinking problem is apparent. In addition, there may be handicaps or other special circumstances which interfere with the efficient use of the Questionnaire or the Interview. For example, a person who reads poorly may have difficulty in answering the Questionnaire items, whereas a person with difficulty in hearing or speaking may have difficulty with the Interview. The two halves of the procedure, used together, will tend to compensate for errors caused by such difficulties. In any event, there can be no doubt that the more information gathered about the person, the better. It is a recognized scientific principle that any characteristic or property of a person or object can be better assessed by two or more independent kinds of measurement than by relying exclusively on the single kind.

#### RATIONALE FOR SELECTION OF QUESTIONNAIRE ITEMS

Empirical and eclectic methods were used in choosing the appropriate items to include in the Questionnaire-Form A. The empirical method selects appropriate questions on a topic (e.g., problem drinking) by means of statistics rather than logic. A large number of questions are presented to criterion groups, such as problem drinkers and social drinkers, and the answers are analyzed. Those questions that the groups answer differently to a significant degree can then be used as predictors of group membership; at this point such a procedure is termed "initially validated" as it has been tested once for its ability to differentiate groups. If these initially validated questions are then given to new populations of problem drinkers and social drinkers, their ability to significantly differentiate the criterion groups

can be reassessed. The questions which the groups answer differently to a significant degree on the second administration, as well as on the first administration, are then said to have been "cross-validated." These questions will then have demonstrated their ability to discriminate on two different samples, whereas initially validated items have only proven their discriminability on one sample. Therefore, more confidence can be placed on cross-validated predictor items than on initially validated predictor items even though they both have shown an ability to differentiate members of one criterion group from those of another criterion group.

The items used in developing the Questionnaire were derived from various sources. Many questions were previously used by Mortimer and Lower.<sup>9</sup> They will not be discussed here but can be found by referring to the following numbers which correspond to the items in the Questionnaire-Form A: 12, 13, 15, 16, 18, 19, 20, 21, 23, 24, 26, 28, 29, 31, 33, 34, 39, 40, 41, 42, 46, 47, 48, 52, 53, 54, 55, 56, 57, 58.

The rationale for the use of the other items found in the literature is discussed below.

Q.1. What is your present marital status?

Marital status was found to differentiate a random sample group taken from the California Department of Motor Vehicles from a group with three or more drunk driving offenses.<sup>1</sup> Those indicating that they were separated or divorced comprised 5.5% of the random sample compared to 34.4% of the sample with three or more drunk driving convictions.

Q.2. With whom do you live?

Living arrangement was also found to differentiate a random sample from those with three or more drunk driving convictions. Pollack<sup>1</sup> reports persons living with their spouses to comprise



70.3% of the random sample as compared to 41.1% of the sample having three or more drunk driving convictions. Persons living alone comprised 8.7% of the random sample. Of those having three or more drunk driving convictions, 27.9% lived alone.

Q.3. How many times have you and your wife (husband) seriously considered divorce in the last two years?

"That the drinking accidents may be selective of individuals with current marital adjustment problem(s)" is a statement supported by Barmack and Payne (1961).<sup>10</sup> Pollack's results<sup>1</sup> would tend to confirm that there is at least a strong association between marital adjustment problems and drunk driving convictions.

Q.4. Does (did) your wife (husband) often threaten you with divorce?

Threat of divorce will indicate marital maladjustment which is not necessarily reflected in marital status, living arrangement, or number of marriages.

Q.7. Do you smoke?

Nonsmokers were found by Pollack<sup>1</sup> to comprise 54.9% of the random sample versus 11.4% of those having three or more drunk driving convictions.

Q.8. About how many packs of cigarettes do you smoke per week?

Pollack<sup>1</sup> has demonstrated the strong relationship between amount of smoking and number of drunk driving convictions.

Q.9. Were you ever arrested?

The relationship between having been arrested and drunk driving is shown to be substantial.<sup>1</sup> Guze et al.,<sup>11</sup> also noted the significance of the number of arrests in discriminating alcoholics and reported that criminals arrested only once were significantly more likely to be nonalcoholics.

Q.10. Are your relatives upset with the way you live?

"Relative upset" stems from similar ideas in initially validated questions 35, 80, 230, 235, and 240 by Mortimer and Lower.<sup>9</sup> Question 3 on the MAST<sup>12</sup> is also similar.

Q.11. Is your income sufficient for your basic needs?

Insufficient income for basic needs was found by Pollack<sup>1</sup> among 33.1% of those with three or more drunk driving convictions versus 15.7% of the random sample.

Q.14. Have you recently undergone a great stress (such as something concerning your job, your health, your finances, your family, or a loved one)?

The concept of stress (job, health, finances, family, or death) leading to excessive drinking and driving is supported by Pollack<sup>1</sup> and Selzer.<sup>13</sup>

Q.22. Do you feel that you have abnormal problems?

Brown et al.<sup>14</sup> reported on the relationship between fatal accidents, alcohol, and paranoid features. Persons having paranoid features feel that they have abnormal problems because of their persecution by others.

Q.25. Does drinking help you make friends?

This item about using drinking to help make friends is a paraphrase of item 21 of the Drinking History Questionnaire reported by Horn and Wanberg.<sup>15</sup>

Q.27. Do you think that creditors are much too quick to bother you for payments?

This item is included as an assessment of financial problems and stress related to alcohol abuse.<sup>1</sup>

Q.30. Do you usually perspire at night?

"Usually perspiring at night" was found to be the case for 15.9% of those having three or more drunk driving convictions, but for only 1.7% of a random sample.<sup>1</sup>

Q.32. About how many years has it been since your last out-of-town vacation?

"Time elapsed since last out-of-town vacation" was included as an assessment of financial affairs and long-term stress, both of which are related to alcohol abuse.<sup>1</sup>

Q.35. Have you ever had your driver's license suspended or revoked?

Pollack<sup>1</sup> reported that the difference between those in the three or more drunk driving convictions sample and the random sample for minor traffic violations was 59.0% versus 16.9% respectively; for major traffic violations this difference was 66.8% versus 0.8%. He also reported that approximately 55% of the drunk driver group had one or more alcohol-related vehicle code conviction, whereas only approximately 0.5% of the drivers license groups met this conviction criterion. This high incidence of minor and major traffic violations and alcohol-related convictions is associated with license suspension and revocation.

Q.36. About how many times have you asked for help for your problems (personal, family, marriage, money, or emotional)?

This was asked because the amount of help sought should be indicative of the amount of stress one has undergone.

Q.37. Is there a history of alcoholism in your family?

Guze et al.<sup>11</sup> reported that 50% of the alcoholic criminals he studied had a "family history" of alcoholism and that this incidence is significantly greater than that of the nonalcoholic criminal population.

Q.38. Do you have a relative who is an excessive drinker?

This item is used because Cisin and Cahalan<sup>16</sup> showed that more abstainers and heavy drinkers than average drinkers reported having a close relative with a drinking problem.

Q.43. Drinking seems to ease personal problems.

Pollack<sup>1</sup> reported that 29.2% of those with three or more drunk

driving convictions admitted drinking to cope with personal problems while only 4.7% of the random sample did so. Of those with three or more drunk driving convictions 48.8% reported drinking to ease tension when worried or upset while only 11.0% of the random sample did likewise.

Q.44. How many drinks can you handle and still drive well?

The number of drinks that one can have and still drive well was found to be a significant discriminator by Pollack.<sup>1</sup> He reported the following sample differences for those with three or more drunk driving convictions versus the random sample.

Over 4: 63.9% (3+DD) versus 36.2% (DMV)

Over 5: 53.2% (3+DD) versus 25.5% (DMV)

Over 6: 32.7% (3+DD) versus 8.0% (DMV)

8 or over: 31.1% (3+DD) versus 7.2% (DMV)

Q.45. In the last year, how many times have you drunk more than you could handle, but still been a good driver when you got behind the wheel?

Drinking "more than one can handle," but "still being a good driver" was found to be a significant discriminator.<sup>1</sup> Those persons answering six or more times a year comprised 52.5% of the three or more drunk driving convictions sample compared to only 11.4% of the random sample. Similarly, those answering "never" comprised only 20.5% of the three or more drunk driving convictions sample compared to 63.5% of the random sample.

Q.49. Do you feel sinful or immoral?

"Feeling sinful or immoral" was implicated as a predictor by an initially validated item found significant by Mortimer and Lower.<sup>9</sup>

Q.50. A drink or two gives me energy to get started.

"Drinking gives me energy" was implicated as a predictor by an initially validated item found significant by Mortimer and Lower.<sup>9</sup>

Q.51. Does drinking help you work better?

"Drinking helps me work better" is a paraphrase of the significantly discriminating item 24 from the Drinking History Questionnaire used by Horn and Wanberg.<sup>15</sup>

Q.57. Would you say that 4 or 5 drinks affect your driving?

See item 44.

THE CONSEQUENCES OF ALCOHOL USE AND ABUSE

THE BODY'S RESPONSE TO ALCOHOL

INTRODUCTION. Most voluntary human consumption of alcohol is either a manifestation of sociability, or an attempt to produce a therapeutic effect consisting of either a feeling of pleasure or relief from distress. The following discussion will deal with the effects of alcohol on the physiological system of any drinker, and trace the progression of disease states which develop as a result of persistent and continuous alcohol abuse.

THE METABOLISM OF ETHYL ALCOHOL. Ethyl alcohol, or ethanol, is the active ingredient in beer, wine, whiskey, gin, brandy, and the less common alcoholic beverages. These beverages differ from one another in their concentration of alcohol; but when they are consumed in quantity, any one is capable of producing intoxication. The habitual abuse of any one, or a combination, of these alcoholic beverages will almost surely lead to problems of one sort or another.

Hard liquor or distilled spirits contain congeners--small, organic molecules other than ethanol--which contribute to the beverage's unique flavor and smell. These congeners have been blamed for hangover symptoms and a variety of the medical complications of alcoholism.<sup>17</sup> However, there is little evidence that these molecules have any physiological effect at the concentration found in the alcoholic beverages. At the present time it must be assumed that ethyl alcohol is the primary, if not the sole, toxic ingredient in distilled spirits and the agent responsible for the direct side effects of alcohol consumption.

The body's oxidation of ethanol produces seven calories per gram of alcohol, almost double the amount as is produced by the oxidation of sugar or protein.<sup>18</sup> However, it is important to emphasize that alcohol cannot be stored in the body as can sugar, nor can it be used in the replacement of destroyed tissue as can protein. Alcohol has no food value and serves no nutritional purposes; it simply adds unwanted adipose tissue to the body. If an individual's dietary history shows little food intake and yet the person is able to maintain his weight, the calories are coming from somewhere, and alcohol is a good first guess.<sup>19</sup>

Alcohol is absorbed into the blood stream from the stomach and the whole length of the intestine. The rate of absorption is greatest in the first part of the small intestine (the duodenum); alcohol absorption is considerably slower in the stomach. Drinking when the stomach is full extensively reduces the liquor's intoxicating effects. Analyses of blood alcohol levels have shown that if alcohol is consumed after or during a meal, the peak blood alcohol concentration (BAC) is reduced (with respect to the BAC that would have been reached on an empty stomach) and the time from onset of drinking to peak is lengthened. This is probably explained by the fact that when the stomach contains solid material the valve leading from the stomach to the intestine remains closed and only opens intermittently to release a small portion of the stomach contents into the intestine. Consequently, when alcohol is introduced into a full stomach it is held there for longer periods and the absorption rate is thus reduced. A number of foods and chemical substances will alter the rate at which alcohol is absorbed from both the stomach and intestine. Fatty foods and "coating agents" (for example, antacids like Maalox) will retard absorption, while irritants such as aspirin will significantly increase the absorption rate.

Approximately 10% of the ingested alcohol is eliminated through the lungs, kidneys, and sweat glands without being metab-

olized.<sup>18</sup> This free alcohol, which is excreted from the pores and released in each exhaled breath, accounts for the intoxicated person's distinct odor. The alcohol on his breath is ethanol and it does not matter whether the ethanol entered the system initially as beer, wine, or the various distilled spirits. As mentioned earlier, the congeners may have distinctive smells themselves, but with or without their simultaneous presence, ethanol can be detected on the breath. Thus, it is a naive individual who thinks vodka is undetectable on the breath. Our nerve endings for smell are very sensitive to alcohol, and are dulled by even small quantities of alcohol. For this reason the drinker quickly becomes unaware of his own odor.

The remaining ethanol (amounting to approximately 90% of the amount ingested) is metabolized primarily in the liver. In the first step in this metabolism, the enzyme alcohol dehydrogenase, catalyzes the oxidation of alcohol to acetaldehyde. The acetaldehyde thus formed is converted to acetyl coenzyme A in most tissues of the body. The acetyl coenzyme A is fed into the universal Krebs metabolic cycle and converted to carbon dioxide and water.<sup>20</sup> Tremendous scientific effort has recently been applied toward understanding the metabolism of alcohol and identifying possible enzymatic differences between the social drinker and the alcoholic. Unfortunately, a biochemical explanation for alcoholism has not yet been found.

Factors Altering The Metabolism Of Ethyl Alcohol. Perhaps erroneously, it was felt for a long time that chronic heavy drinking increased the rate of ethanol metabolism. Alterations in ethanol metabolism have been demonstrated in both alcoholic and nonalcoholic subjects following their consumption of large amounts of alcohol over several days' duration. Unfortunately, the alteration was observed to occur in both directions; some subjects showed an increased rate of ethanol metabolism, while others showed a

decreased rate.<sup>21,22</sup> More moderate alcohol consumption produced no rate changes for either controls or alcoholics.<sup>23</sup> A microsomal enzyme system found in liver tissue has definitely been shown to increase with prolonged drinking, but this system accounts for such a small part in ethanol metabolism that it does not change the overall metabolic rate.<sup>24</sup>

An atypical alcohol dehydrogenase enzyme, which has a slightly different molecular structure from the commonly found enzyme, has been isolated. However, despite this difference in structure, the atypical enzyme is still capable of catalyzing the metabolic step and persons possessing this enzyme are able to handle alcohol normally.<sup>25</sup>

In short, there presently is no convincing evidence that alcoholics and nonalcoholics metabolize alcohol at different rates or by different enzymatic pathways. Of course, an enzymatic defect which correlates highly with the clinical symptoms of alcoholism may yet be found, but such a defect is not known.

There is little question that alcoholics can reach very high blood alcohol concentrations seldom seen in normal drinkers, and that at a given alcohol concentration the alcoholic appears to be less intoxicated than the normal drinker. The first phenomenon, achieving high blood alcohol levels, appears to result from the simple fact that the alcoholic can and does drink more. Mendelson found that control drinkers, encouraged to drink as much as possible, developed gastrointestinal pain, nausea, and vomiting as the blood alcohol concentration approached 0.10% W/V (100 mg%).\* At that point the nonalcoholic generally stops drinking because of discomfort or because he vomits the liquor from his stomach before absorption can take place. The alcoholic, on the other hand, does not experience this gastrointestinal distress and, therefore, is able to absorb significantly greater amounts of ethanol.<sup>26</sup> The reason for the alcoholic's gastrointestinal tolerance is not clear.

---

\*Blood alcohol concentrations in percent weight by volume will hereafter be referred to by the decimal portion only; i.e., 0.10% will indicate 0.10% W/V.



The behavioral tolerance also is not well understood, but it may be a function of practice. With frequent intoxication, the alcoholic can learn how to partially compensate for his inebriation. For example, he learns to shut one eye to prevent double vision, and to walk with his feet further apart to provide a broader base and better balance. Or possibly, there may exist a cellular adaptation in which ethanol's effect on the individual body cells is lessened. Explanations along this line are appealing, but although considerable research is being done in this area<sup>27</sup> no evidence substantiating such explanations presently exists.

Both the intoxicated individual and those about him have, at some time, sought methods to speed the sobering process. Many a drunken person has been walked around the block, induced to perform simple exercises, and had coffee poured into him with the hope of hastening his sobriety. These efforts have no effect on the rate of ethanol metabolism but may serve the dual purpose of preventing further alcohol consumption and occupying the time while the normal sobering process takes place. A word of caution might be inserted at this point: coffee and other stimulants do not essentially alter the depressant effects of alcohol and may in some instances be detrimental. Nothing is gained by producing a wide-awake and hyperactive drunken individual from one who was previously sleepy and lethargic. Also, if the action of the stimulant extends into the withdrawal period, it may add to the withdrawal reaction causing the person to be still more uncomfortable. In most instances it is probably more prudent to let the body handle the alcohol in its own fashion without chemical or physical intervention.

Scientists have found a number of drugs which slow or otherwise deter the metabolism of ethanol. The most widely used of these is disulfiram, frequently known by the Ayerst Laboratories trade name Antabuse. The action of disulfiram can be understood if the metabolism of ethanol is again reviewed. Ethanol is converted to acetaldehyde in the liver and the acetaldehyde is metabolized to acetyl coenzyme A in most body tissue. The conversion of

acetaldehyde to acetyl coenzyme A is very rapid and even with heavy drinking the concentration of acetaldehyde is low, which is fortunate because acetaldehyde is a very toxic substance, even in moderate concentrations, and it produces very unpleasant side effects. Disulfiram interferes with the conversion of acetaldehyde to acetyl coenzyme A, thus allowing the concentration of acetaldehyde to increase to a point at which the side reactions occur.

A person taking disulfiram who ingests alcohol may experience flushing, a quickening and pounding of the heart, difficulty in breathing, low blood pressure, faintness, nausea, and vomiting. The severity of the reaction depends upon the amount of disulfiram in the system and the quantity of alcohol ingested. Even a mild form of the reaction just described is usually sufficiently frightening and unpleasant to discourage the patient from drinking while he continues to receive disulfiram. When this drug is used therapeutically it is usually administered daily; however, the action of a single dose extends over several days. Therefore, therapy should be discontinued for a week or more before the use of alcohol can be considered safe.

#### INTOXICATION.

Physiological Changes During Intoxication: Alcohol's Tranquilizing Effects. Ethyl alcohol is a very effective tranquilizer, and when it is taken in moderate to high doses it will lessen or totally abolish anxiety, apprehension, and caution. When anxiety is inappropriate to the situation, tranquilization is therapeutic; but when tranquilizers are taken in such doses that dangerous situations are no longer perceived as threatening, then the drug's effects may lead to injury. At intoxicating dosages, alcohol removes a portion of the necessary adjustive anxiety. Consequently, the individual's judgment is faulty; his built-in caution signal has been temporarily short-circuited.

Alcohol's Anesthetic Effects. Just as alcohol has many of the properties of a tranquilizer, it also has most of the properties of a general anesthetic--albeit a very poor and dangerous one. At an intoxicating dosage, alcohol significantly decreases the accuracy of most of the senses.

Visual problems, which are the sensory problems most frequently complained about, involve a blurring of near vision and an inability to focus; at slightly higher doses, diplopia (double vision) develops because alcohol interferes with the very complex central nervous system reflexes necessary to cause the eyes to converge and maintain a single image. Pupillary response is slowed by alcohol, and there is increased difficulty in adapting to rapid light changes. The "moment of blindness" experienced when one confronts a bright light or darkened room is, thus, considerably extended for the drunken individual. Depth and distance perceptions become faulty. Consequently, an intoxicated person who is trying to cross a street often may stumble off the curb, surprised by its existence; and when he reaches the other side he may stand a foot or more from the curb and make high stepping movements with his lead foot, unable to get up on the curb he sees.

Most of the apparent hearing problems of the intoxicated person result from inattention rather than physiological defect. However, Borg demonstrated that for a person with a blood alcohol concentration of 0.13% to demonstrate the same middle ear reflex he demonstrated while sober, the intensity of a tone had to be increased 10 decibels.<sup>28</sup> Because it is a natural response for a person who is hard of hearing to raise his own voice, this decrease in hearing acuity may partially account for the loud talking that follows several drinks. The middle ear reflex, which Borg measured, is designed to protect the ear from damage by loud noises. Since the reflex is significantly altered during intoxication, the hearing mechanism is left vulnerable to permanent damage from loud noises.

Very little is written about alcohol's effect on the sense of taste, but most heavy drinkers will report that food becomes rather bland and tasteless after they have consumed a considerable amount of alcohol. Many a hostess will attest to the fact that her specially prepared dinner went unappreciated after a long cocktail hour. And although small amounts of alcohol are sometimes prescribed to stimulate the appetite of older or debilitated persons, it is doubtful whether the alcohol has any effect on the appetite center in the brain; the increased appetite may appear as a consequence of the relaxation and general sense of well-being provided by a small amount of liquor.

The analgesic, or pain-reducing, properties of alcohol are considerably overrated. In most cases aspirin would do a better job. There are two separate aspects of pain perception: the pain itself, and a psychological response to the sensation of pain. Both contribute to the discomfort of pain. While commercial medications for pain act on the pain itself, narcotics act on both aspects, and alcohol primarily acts on the psychological discomfort. Since intoxicating doses of alcohol affect this psychological aspect and, thus, lessen the perception of pain, the drunken individual frequently does not recognize injury or, if he recognizes it, he is not sufficiently uncomfortable to seek medical attention. Consequently, significant injury may go unattended for hours or even days. Contributing to this is the common reluctance to go to a doctor or emergency room while intoxicated. A study of emergency room patients at Massachusetts General Hospital showed a direct relationship between delay in arriving for medical treatment and positive Breathalyzer<sup>®</sup> Readings.<sup>29</sup>

Personality And Behavioral Changes During Intoxication. Mild to moderate personality changes usually accompany the ingestion of intoxicating dosages of alcohol. The type and degree of change varies with different individuals and within the same individual from one time to another. Some individuals become euphoric and

have grandiose ideas about their importance and capabilities, while others become depressed and despondent so that life, for a period, loses meaning. Still others become hostile and openly aggressive. A common motivation for drinking is discomfort with one's present feelings, whatever they are, and the view that alcohol will afford relief. One or two drinks frequently will have an uplifting effect, but heavier drinking often antagonizes the situation by intensifying the original oppressive state.

It is estimated that between 17%<sup>30</sup> and 31%<sup>31</sup> of the suicide attempts are by alcoholics; these figures are particularly impressive if only about 4% of the general adult population are true alcoholics, as Rushing suggests.<sup>31</sup>

Quarrels and fights are not infrequent accompaniments to drinking. The removal of self-control by alcohol may result in a fatal ending to a minor disagreement. In a study of emergency service patients at Massachusetts General, Wechsler found that 45% of the persons treated for injuries incurred in fights or assaults had previously been drinking.<sup>29</sup> In a study conducted in Australia, Birrell found that approximately one-fourth of 100 homicide victims had blood alcohol levels of at least 0.15%.<sup>32</sup> The close relationship between crime and alcohol has long been known by those in the legal field. While serving as Dean of the Faculty of Law of the University of Adelaide, Morris made the following statement: "Such studies as there are, and some 12 years relatively close observation of this problem, have led me to the belief that probably 50% of those convicted of the more serious crimes were, at the time of the commission of their crimes, under the influence of alcohol sufficient to have had an appreciable effect on their inhibitions."<sup>33</sup>

#### COMPLICATIONS OF ALCOHOL ABUSE

INJURY AND TRAUMA DURING INTOXICATION. Violence and trauma are probably the only complications of alcohol abuse that are not

dependent upon the existence of a long-standing problem. The young drinker who experiments with liquor and becomes intoxicated places his life in the same jeopardy as does the veteran alcoholic. However, since the probability of occurrence is related to exposure, the frequently intoxicated problem drinker is more likely, in the long run, to suffer trauma than is the individual who confines his heavy drinking to one or two episodes a year.

Frostbite And Other Reactions To Cold. The intoxicated individual's apparent indifference to cold creates many problems. Exposure leads to injury from frostbite and may be related to the alcoholic's increased susceptibility to pneumonia; in particular, to those types of pneumonia which are difficult to treat. Hypothermia (the lowering of body temperature) is always a threat to the inebriate. Alcohol dilates the blood vessels in the skin, resulting in an increased flow of blood through the skin and loss of body heat to the surroundings.<sup>34</sup> (The increased peripheral blood flow accounts for the well known red face, red nose, and warm hands of the drunk.) Normally, as the body cools, the circulation to the hands and feet is reduced to a minimum in an effort to conserve body heat. It leaves the extremities more vulnerable to the deleterious effects of cold, but the vital centers of the body are protected.

Pressure Paralysis. The injury most frequently observed following a drinking bout is the partial paralysis of a limb resulting from pressure on a peripheral nerve. The condition is usually reversible, recovery requiring about six weeks, but occasionally the nerve damage is so great that there is no improvement.<sup>35</sup> "Saturday-night paralysis" is a well defined peripheral nerve paralysis caused by pressure on the radial nerve from sleeping with the arm hanging over the back of a chair.<sup>36</sup> Normally when pressure begins to damage a peripheral nerve the limb supplied by that nerve begins to ache and the posture is

changed to relieve the pressure. However, if an individual is unconscious or stuporous from alcohol, he will not respond to the pain and considerable nerve damage may result.

Head Injury: Subdural Hematoma. Head injury deserves special attention because it is such a common injury following intoxication and because it is so often missed by both lay and medical personnel. The most frequent and severe head injury is the subdural hematoma which results when one of the blood vessels lying within the covering of the brain is torn. This injury most often follows a blow to the head. Blood is lost from the injured vessel and is trapped between the skull and the brain, thus placing pressure on the brain beneath. If this pressure is not removed by surgically evacuating the blood, permanent brain injury and death may result. The subdural hematoma is often missed because the individual does not remember injuring his head and because the symptoms of the hematoma are very similar to those of acute intoxication. Both sets of symptoms result from the malfunctioning of nerves within the brain. The subdural hematoma may be diagnosed or highly suspected despite intoxication; thus, if a person is known to have sustained a significant blow to the head, he should be evaluated by a physician.

Asphyxiation And Aspiration. Asphyxiation and aspiration (drawing fluid into the lungs) are also common perils to the drunken individual. Vomiting is common following significant alcohol ingestion and aspiration of vomitus may occur. Such aspiration causes a severe and often fatal lung reaction. Choking on food is more frequent in the intoxicated person because the mechanisms regulating swallowing are affected as are those for walking and speaking. Drunken individuals have been known to suffocate on their own bedclothing or in an inch of water at the bottom of a ditch. In fact, it has been shown that many drowning victims were drunk at the time of falling into the water.

General Injuries. The lack of coordinated body movements results in a great many of the injuries to intoxicated individuals. The execution of smooth and precise movements requires optimal functioning throughout all the levels of the nervous system. Alcohol interferes with many levels of nerve functioning and, therefore, seriously impairs coordinated movement. Walking with a staggering gait or dropping things held in the hands are examples of alcohol's interference with even the most basic and well practiced body movements. The unconscious reflex movements immediately called into play when the sober individual loses balance are retarded or absent during intoxication; thus, the inebriate is subject to many falls--perhaps down a flight of stairs, through a window, or against a sharp object.

Fire presents another hazard. The unnoticed dropped cigarette accounts for a large number of clothing, bed, and building fires; and forgotten food cooking on the stove may cause a kitchen fire. When in a stupor, an individual may not awaken with smoke, or if he is aroused, he may be too confused to find an exit.

Studies conducted in most of the major cities in the United States and in numerous other countries have uniformly found alcohol to be implicated in at least 50% of fatal traffic accidents.<sup>4</sup> Driving is a very complex task requiring the individual to be optimally alert and coordinated. Since even moderate amounts of alcohol markedly impair an individual's alertness and coordination, it is not surprising to find this high association.

ADVERSE EFFECTS OF ALCOHOL WITHDRAWAL. Withdrawal symptoms such as tremulousness, nausea and vomiting, hallucinations, seizures, and delirium tremens following abstinence are manifestations of physical dependence on alcohol. Development of dependence is related to the amount of alcohol taken per unit time and the length of the drinking episode. Initially it was felt that a high alcohol intake over a 3- to 20-year period was required for



dependence or addiction to develop; however, recent investigations have found tremulousness occurring after several days of steady ethanol intake,<sup>26</sup> and hallucinatory states have appeared after two weeks of imbibing.<sup>37</sup> Severe delirium tremens appear to require a background of months of inebriation.<sup>18</sup>

The pathogenesis of the withdrawal state is not well understood. Physical examination and autopsy evidence reveal no clear-cut explanation for the serious mental and behavioral abnormalities found. Withdrawal states are presumed both to be related to biochemical and/or physiological changes in the body induced by an addicting drug, and to be slowly reversible in the prolonged absence of the drug. While the drug is present, the, biochemical or physiological changes seem to exert little or no adverse effect on the body. It is as if the induced change and the drug neutralized one another. However, once these changes are induced, withdrawal of the drug can have devastating effects upon normal body functioning. The nature of the changes induced by alcohol is presently unknown. All the withdrawal symptoms can be reversed or at least modified by giving alcohol or another drug capable of substituting for alcohol. In clinical practice, withdrawal states are treated by using a substitute drug in sufficient quantity to control the symptoms, and then gradually decreasing the dosage over a period of time.

Tremulousness. Tremulousness is the mildest of the withdrawal symptoms. Frequently it only involves the hands and consists of shaking and difficulty in grasping and holding objects. In its more extreme form, tremulousness can extend to the whole body and the person will shake so violently that he is unable to speak clearly or stand without help. Tremulousness can develop as long as three days after cessation of drinking; however, it occurs more often after a relatively short period of abstinence, such as a period of sleep. Associated with the observable tremor

is a subjective feeling of being "shaky inside," which is akin to marked anxiety states. This feeling is persistent and frequently lasts from 10 to 14 days; the gross tremor usually disappears after 3 to 4 days of abstinence. While in the tremulous state, the person is frequently nauseated and disinterested in food. On occasion he will vomit, and in extreme cases this can be severe enough to cause the esophagus (lower throat) to tear resulting in hemorrhaging, known as the Mallory-Weiss syndrome which is a true medical emergency. Withdrawal nausea and vomiting can be quelled with alcohol while the vomiting which occurs while drinking is only antagonized by alcohol. Because tremulousness is nicely controlled by several drinks it probably serves as the stimulus for most morning drinking. It may also be treated with mild sedation. However, tremulousness may herald impending delirium tremens and treatment may partially mask the warning symptoms.

The tremulous patient may complain of insomnia and yet crave rest and sleep. He generally is preoccupied with his misery, inattentive, and disinclined to answer questions. If he chooses to answer, he may give rude or perfunctory responses. He may be mildly disoriented as to time but show no significant confusion about his surroundings and his illness.<sup>18</sup> He is often filled with remorse and guilt for his previous behavior, fearful of the future, and physically uncomfortable in his present situation.

Disorientation. Various degrees and kinds of disorientation may accompany alcohol withdrawal; they usually appear within 24 to 48 hours after the cessation of drinking.<sup>37</sup> Approximately one-fourth of all tremulous patients will show symptoms of disordered sense perception. Such patients may complain of nightmares which they find difficult to separate from real experiences. Sounds and shadows may be misinterpreted, or familiar objects may be distorted and assume unreal forms. These are not hallucinations in the strict sense of the term.<sup>18</sup> However, hallucinations may also occur.

Hallucinations. The most common hallucinations are visual and animate comprising various forms of human, animal, or insect life. Such forms may appear shrunken or enlarged, natural or distorted, and hideous. Auditory hallucinations are less commonly encountered: the patient hears extremely real and vivid voices which he frequently attributes to his family, friends, or neighbors; he rarely attributes them to God, radio, or radar. The voices most frequently discuss the patient in the third person and the nature of their conversation is maligning, reproachful, or threatening. The patient's emotional response is usually appropriate to the hallucinatory content: he may call the police for protection, barricade himself against invaders, or even attempt suicide to avoid what the voices threaten. The duration of hallucinations varies greatly--they may be momentary or they may occur intermittently for several days. A small percentage of these patients develop chronic auditory hallucinations. The relationship between this chronic state and schizophrenia is presently disputed. However, there is some evidence that repeated attacks of acute auditory hallucinations render the patient more vulnerable to the chronic form of the illness.

Seizures. Epileptic grand mal seizures may also be a part of the withdrawal symptomatology. Usually they occupy a discrete period 12 to 36 hours after the drinking episode. A single seizure may occur, but one is more likely to encounter bursts of two to six seizures, after which the patient remains free of seizures until his next drinking episode. This condition is quite distinct from the more common idiopathic epilepsy. The idiopathic epileptic experiences convulsive episodes earlier in the period of abstinence--usually five to six hours after drinking. When a careful history is taken, he will reveal seizures not associated with drinking episodes.

Seizures may also be a clue to brain injury secondary to head

trauma sustained while the person was intoxicated, and they may indicate that neurosurgical intervention is necessary as a life-saving measure. For this reason, anyone who has convulsed during withdrawal deserves a thorough neurological examination and observation over an extended period. Approximately one-third of the patients who have seizures during withdrawal will develop full-blown delirium tremens.<sup>18</sup>

Delirium Tremens. Delirium tremens is a grave complication of an alcoholic episode; approximately 15% of these cases end fatally. The state is characterized by profound confusion, delusions, vivid hallucinations, tremor, agitation, sleeplessness, dilated pupils, fever, fast heart rate, and profuse sweating. The delirium frequently begins and ends abruptly after several days' duration. Following the delirium the individual goes into a deep sleep and awakens lucid, quiet, hungry, exhausted, and with virtually no memory of the delirium. Only in rare cases is the period of delirium recurrent.

A patient may either show one of the withdrawal symptoms discussed earlier or he may show all of them. If he develops all of the symptoms, he does so in a predictable sequence; first he shows tremulousness and hallucinosis (and/or seizures), followed by delirium tremens.

**MEDICAL COMPLICATIONS OF ALCOHOL ABUSE.** The following section discusses many of the disease states manifested by persons whose alcohol intake is excessive. It includes both the disease states directly related to the toxic effects of alcohol, and those common conditions which are secondary to disease states, such as malnutrition. This information is intended to serve as a reference source for the nonmedical worker who may encounter clients with the diseases or conditions described, and for the nonmedical reader who may have various questions about the following: the general physiologic and pathologic changes occurring with alcohol abuse, the specific points at which these changes are likely to occur,

and whether the changes are amenable to treatment. This will neither enable the reader to diagnose these diseases nor to fully understand their treatment. Instead, it should help him get a more complete picture of alcoholism, and better understand the alcoholic's particular problems. It is also an attempt to correct some misconceptions about the medical aspects of alcoholism in the belief that before we can offer effective therapy we must understand the problem.

#### Gastrointestinal Complications.

Gastritis. Probably the most common medical problem related to over indulgence in alcohol is gastritis. This term simply refers to the inflammation of the stomach lining by an irritating substance, in this case by alcohol. Beverages which are at least 40% ethanol are quite irritating to the stomach and their long-term use leads to glandular destruction, erosion, and the formation of cysts.<sup>3 8</sup> The surface inflammation and erosion may involve blood vessels and the patient may vomit specks or larger quantities of blood. Some degree of gastritis always follows a drinking bout, but if it is a mild case the discomfort subsides in a matter of days. However, more severe forms of gastritis may leave scars and cause permanent dysfunction. The symptoms of gastritis are abdominal swelling, stomach pain, belching, and a general food disinterest. Without a history of excessive drinking, alcoholic gastritis is indistinguishable from the common viral gastritis experienced by everyone at one time or another. For this reason alcoholic gastritis is frequently missed as a symptom of excessive drinking. If abusive drinking is identified when it first begins to cause gastritis and drinking behavior is successfully altered, the high morbidity and mortality associated with alcoholism can be avoided.

Peptic Ulcer. A common lesion, the peptic ulcer, is discussed here because its occurrence is exceptionally high in

the alcoholic population. An ulcer is a localized erosion of the inner surface of the stomach or the first part of the intestine. If the erosion involves blood vessels, extensive bleeding may result, and if the erosion is deep enough it may perforate the outer wall producing a hole from the stomach or intestine into the abdominal cavity. The last two complications are infrequent, but when they occur they are serious and require vigorous medical therapy. The symptoms of a peptic ulcer are similar to those of gastritis, with high abdominal pain being the most characteristic. No matter what the etiology of the ulcer, all drinking of alcoholic beverages should be discontinued while the ulcer is symptomatic. Very moderate drinking is permitted only at times when the ulcer is quiescent. Heavy drinking by a person with a history of an ulcer is indicative of poor drinking controls and alcoholism should be suspected.

Pancreatitis. A third gastrointestinal complication of alcohol abuse is pancreatitis. The pancreas is a digestive organ lying just below the stomach. It has two major functions: to produce a great many digestive enzymes which flow into the intestine, and to produce insulin which regulates the blood sugar level. Exactly how or why alcohol causes an inflammatory reaction in the pancreas is not known, but during pancreatitis the digestive enzymes appear to be activated within the organ itself, rather than in the intestine. Thus, auto-digestion of the pancreas results in and causes severe inflammation. Normally the insulin function of the pancreas is not significantly altered, but if the organ is largely destroyed by the inflammatory process, insulin deficiency may ensue. The pain associated with acute pancreatitis is extremely severe and very debilitating. Two forms of this disease exist: one is the acute type, and the other the chronic, relapsing type in which the episodes are less severe but tend to be recurrent or persistent. Pancreatitis is an uncommon pathology, but when it occurs it is highly related

to alcoholism. Cox found that 25% of the pancreatitis patients in his study had a history of alcoholism.<sup>39</sup>

Hypoglycemia. Another pathologic condition related to the pancreas and the liver is alcohol-induced hypoglycemia (low blood sugar). Sugar is the only food brain cells can utilize. Therefore, low blood sugar will cause brain cell starvation. If the hypoglycemia is severe or prolonged, it can lead to coma and death. Following the ingestion of a large amount of alcohol some persons experience hypoglycemic episodes which frequently result in loss of consciousness. Predisposing factors to this reaction are diabetes mellitus and cirrhosis; however, persons with neither of these conditions may have hypoglycemic reactions.<sup>40,41,42</sup> The use of alcohol by diabetic patients is ill-advised because of the high carbohydrate content of the alcoholic beverages and because alcohol may alter the body's response to the diabetic medication. Unfortunately, the symptoms of hypoglycemia closely resemble those of acute intoxication and the hypoglycemia may not be recognized because the person is presumed to be drunk. It is a safe rule that all diabetic patients in a stupor, regardless of the apparent cause of the stupor, be evaluated and supervised by a physician.

Fat Infiltration. Several liver changes precede the development of cirrhosis. Through a mechanism not well understood at this time, alcohol causes a redistribution of body fat and the fat is deposited within the liver cells. This fatty infiltration of the liver occurs in anyone who drinks regardless of his drinking pattern. It is usually reversible if the period of abstinence from alcohol is long enough to allow the body to mobilize the liver's fat stores and return them to their normal places in the body. With heavy or sustained alcohol use there is no chance to reverse the process and the deposition of fat in the liver increases; consequently, the liver expands. It

is this increasing size which leads to the positive correlation between alcoholism and "big livers." Fatty infiltration of the liver occurs in many conditions other than alcoholism, for example, obesity, diabetes, and nutritional diseases.<sup>18</sup> However, the fatty livers in the other conditions do not generally progress to cirrhosis, while in alcoholism this progression is common. Viel postulates that fat deposited in the alcoholic's liver may be different from fat present in the obese individual, and that "alcoholic fat" may injure the liver cells leading to the development of cirrhosis.<sup>43</sup>

Cirrhosis And Related Complications. Cirrhosis is probably the disease complication most frequently thought of in relation to heavy alcohol intake and alcoholism. However, its incidence in the total alcoholic population is small; Jellinek estimates it to be about 3%.<sup>44</sup> Generally cirrhosis develops only after many years of alcohol misuse. Viel, studying the occurrence of cirrhosis in persons autopsied following their violent death in Santiago de Chile, found a positive correlation between cirrhosis and increasing age, number of years of heavy drinking, and total quantity of alcohol consumed.<sup>45</sup>

Cirrhosis is a process which starts with localized inflammation followed by death of liver cells. Adjacent living tissue makes new cells to replace the dead ones, but in the process considerable amounts of scar tissue are produced and the normal architectural pattern of the liver is lost.<sup>46</sup> Normally, within the liver the cells line up in cords extending out from a collecting vein like spokes in a wheel. Blood trickles from the hub through the channels formed by the lines of cells to the collecting vein. But with the formation of scar tissue the cords and channels are disrupted, blood flow is impaired, and individual liver cells are no longer in immediate contact with blood; thus, they cannot function as normal liver tissue.



When the cirrhotic process begins, the scarred areas are minute and diffusely scattered throughout the liver. As the disease progresses, the number of involved areas increases and the scarred areas enlarge. So long as the normal liver tissue is sufficient to meet the demands of the body, the cirrhotic process causes few problems. However, as more of the normal tissue is converted to scar tissue, the liver, as a whole, is increasingly unable to function and the complications of cirrhosis develop. These complications frequently lead to the death of the individual. Cirrhosis itself is irreversible because, as mentioned previously, it is a process of scarring and scar tissue cannot revert to normal tissue. But if alcohol is completely eliminated from the diet, the cirrhotic process in the liver will be stopped or substantially slowed. If at the time the cirrhotic degeneration is stopped there is enough healthy tissue to carry out the essential liver functions, a normal life can be anticipated.

A definitive diagnosis of either fatty liver or cirrhosis can only be made by liver biopsy which requires a physician skilled in that procedure and involves the risk of complications. Consequently, the diagnosis of cirrhosis is usually made by presumptive evidence: a hard, nodular liver that can be felt below the ribs (the normal size liver lies entirely beneath the rib cage and cannot be palpated), and tests for abnormal liver function. Of course alcohol abuse is not the only predisposing factor for the development of cirrhosis, but in the United States an estimated 75% to 90% of the persons with cirrhosis have a history of alcoholism.<sup>18,46</sup> For a long time it was felt that cirrhosis was a result of the alcoholic's nutritional deficiency rather than the ethanol itself. However, present theories stress that alcohol is a necessary factor in the development of cirrhosis.<sup>47,48</sup> Alcohol alone has not been shown to be cirrhotogenic, but an increased amount of liver enzyme (SGOT) in the blood following intoxication has been found by Mendelson using normal drinkers,<sup>21</sup>

and by Bang using alcoholics.<sup>49</sup> Thus enzyme's presence in the blood is indicative of damage to liver cells; therefore, alcohol must at least have acute toxic effects on the liver. The ultimate development of cirrhosis is probably dependent upon three variables: (1) the individual liver's constitutional susceptibility to damage, (2) the amount of ethanol consumed daily and the duration of excessive consumption, and (3) the degree and duration of malnutrition, particularly of protein deficiency.<sup>46</sup>

The liver is essential to life; its functions include the following: (1) metabolism of protein, fats, and sugars, (2) production of important substances secreted into the blood, (3) purification or cleansing of the blood to remove useless and toxic substances, bacteria, and other particulate matter, and (4) storage of certain food stuffs and minerals. Serious disease states arise when the liver is unable to perform one or more of these functions, as is the case in far advanced cirrhotic degeneration. Four of the complications of cirrhosis, esophageal varices, ascites, coagulation problems, and feminization of the male, are discussed below.

Esophageal varices develop when the scarred liver is unable to handle the normal amount of blood presented to it. It then acts as a dam in the circulatory system. Blood which is backed up behind the liver at an increased pressure finds alternate pathways to maintain circulation. One such shunt is through the esophageal (lower throat) veins. These veins are not constructed to handle the increased amount of blood or to withstand blood under increased pressure. Consequently, the weak walls of the veins balloon out, become very fragile, and are subject to spontaneous rupture. Bleeding from these veins (which are called esophageal varices) is among the most serious medical emergencies and the mortality rate is high. Medical and surgical treatment of esophageal varices is less than adequate. A person with this

condition has a substantially reduced life expectancy.

Another consequence of blood backing up behind the liver, in combination with the liver's inability to make enough of a specific blood protein (albumin), is the leakage of fluid through the walls of blood vessels into the abdominal cavity. The accumulation of fluid is called ascites and the amount of fluid in the cavity can be as much as two gallons. A person with this condition has a distended abdomen and may resemble a woman with a term pregnancy. A person with substantial ascites is usually severely ill and in considerable pain. With intensive medical treatment, rigid control of fluid intake and diet and abstinence from alcohol, the fluid may be reduced but the person still has a poor prognosis.

In advanced cirrhosis the liver is unable to make a number of substances necessary for normal body function. Among these are various factors involved in the formation of blood clots.<sup>50</sup> Consequently, bleeding episodes in cirrhotics are not infrequent and can be serious.

In addition to the liver's inability to synthesize various substances, it also is unable to degrade and detoxify certain body chemicals. Ammonia, normally formed in the breakdown of proteins, is not detoxified in the cirrhotic liver and is allowed to circulate through the brain.<sup>51</sup> When the ammonia and other toxins are sufficiently concentrated in the brain, impaired mental states and coma develop.

Estrogen (a female hormone) normally produced in very small quantities in men also, is degraded in the liver; thus, it generally has little physiological effect in the male.<sup>52</sup> However, in the cirrhotic the degradation of estrogen is retarded; it accumulates and can reach a concentration sufficient to cause early breast development in the male.

These complications of cirrhosis--esophageal varices, ascites, coagulation problems, and feminization of the male, are late and frequently irreversible manifestations of alcohol abuse. They are, for practical purposes, diagnostic of alcoholism but in many cases the diagnosis occurs too late for effective therapy or rehabilitation.

The numerous gastrointestinal complications of long-standing alcoholism frequently antagonize one another, thus leading through a vicious circle to a more severe clinical picture. Frequent bleeding episodes from gastritis, peptic ulcers, or varices are prolonged by the coagulation defects. The breakdown of large quantities of blood in the intestine following a bleeding episode produces an abnormally large amount of ammonia and other toxins which cannot be handled by the liver. When these toxic substances reach the brain, they produce changes in consciousness, behavior, and neurological status; collectively these changes are known as hepatic coma. On occasion, coma has been reversed but it is a grave condition with a poor prognosis. Many a patient has been carried through an acute bleeding episode by transfusions and rigorous medical treatment only to succumb a few days later to hepatic coma.

While the list of gastrointestinal complications of alcohol abuse is long it represents a far from complete picture of the total medical problems resulting from the misuse of alcohol. The musculo-skeletal and cardiovascular systems may also suffer.

Musculo-Skeletal Complications. For normal functioning muscles depend on their own intact cells and on proper nerve supply. A number of muscular problems resulting from nerve defects will be covered in greater detail in the section on neurologic complications; consequently, they will only briefly be mentioned here.

Neuromuscular Problems. One such neurologic problem is polyneuritis, a disease of the peripheral nerves involving particularly those of the arms and legs. Denied their normal innervation these muscles lose substance and begin to waste away, resulting in profound muscle weakness and pain. The normal, smoothly coordinated muscular movement, made possible by highly integrated nerve circuits in the cerebellus, is lost when alcohol attacks the brain cells in this area, as in the case of alcoholic cerebellar degeneration or advanced Wernicke's disease. Consequently, movements become jerky and inaccurate, balance becomes poor, and tremor may appear. These neurologic complications are mentioned here because they may initially appear to be primary muscular disease.

Primary Alcoholic Myopathy. Primary alcoholic myopathy is a muscle disease which affects persons with a history of prolonged, severe, over-indulgence in alcohol. Both acute and chronic syndromes of skeletal muscle involvement in alcoholics have been delineated.<sup>4 3</sup> In the acute disease the patient complains of muscle aching and tenderness, often with cramps. Electrical studies disclose abnormalities in the muscle functions; analyses of blood constituents show an elevation in muscle cell enzymes; biopsy specimens show degeneration and death of muscle cells; living cells appear swollen and there is destruction of the subcellular structures.<sup>5 3</sup>

The predominant feature of the chronic syndrome is muscle weakness which primarily affects the muscles of the pelvis and shoulders. The biochemical and cellular abnormalities are similar to, although less intense than, those found in the acute phase, and it seems probable that chronic alcoholic myopathy results from repeated heavy drinking associated with episodes of acute muscle damage. While the acute symptoms are reversible if drinking is stopped, at least part of the damage already caused may well be permanent; continued drinking may result in severe muscle wasting and weakness.<sup>5 4</sup>

Cardiovascular Complications. Heart failure and sudden death are frequent complications of long-standing alcoholism. Of 119 patients with primary heart or myocardial disease Tobin found that 33% were alcoholic and 75% of these had been excessive drinkers for more than 10 years.<sup>55</sup> Postmortem examination of such patients revealed a huge, dilated heart, and microscopic examination of the heart muscle showed small areas of muscle degeneration and scar formation. This is a picture of chronic atrophy suggesting the effects of a continuing, or often repeated, insult.<sup>54</sup>

The correlation between this condition and the incidence of beer as the preferred alcoholic beverage is striking.<sup>56,57</sup> Why this association exists is a mystery, but it may be related to the generally low vitamin content of beer.<sup>58</sup> This theory is consistent with the prevalent feeling that alcoholic cardiomyopathy is a manifestation of the vitamin deficiency disease beriberi. However, if heart failure is secondary to the vitamin deficiency one would expect the heart condition to respond better to the administration of thiamine than it does.

Alexander, who does not subscribe to this theory, has examined affected heart muscle under the electron microscope and noted both that the contractile elements of the muscle cells are fragmented and arranged in a more or less haphazard fashion, and that there is considerable storage of sugar within the cells.<sup>59</sup> He suggests that in alcoholic heart disease the heart's pumping ability is seriously compromised not only mechanically, by the loss of contractile elements, but also chemically, by a failure to convert chemical energy to contractile energy.<sup>60</sup>

Excessive short- or long-term consumption of alcohol is equally toxic to the nervous system. The following section discusses these complications.

Neurologic and Neuropsychiatric Complications. The neurologic complications can be divided into two groups: those that are potentially reversible, and those that are permanent. No attempt will be made to cover all the conditions described in the literature.

Reversible Neurologic Complications. There is no currently known neurological defect to explain the "blackout" phenomenon which is a form of amnesia for certain events that occurred while the person was drinking. The period of amnesia may be short, lasting only a few minutes, or it may last as long as a week during binge drinking; it may be intermittent, leaving the person with numerous memory gaps throughout the drinking episode. The amnesic period is not accompanied by a loss of consciousness or even stupor; in fact, during a blackout the person may not even appear to be intoxicated. It is sometimes difficult for persons who have been with such a drinker to believe that later he has no memory of what he said or did. Chafetz postulates that a blackout may be part of a psychological defense system which allows the person to repress knowledge of unacceptable behavior during intoxication.<sup>4 5</sup> However, the forgotten behavior is frequently trivial and not unacceptable.

Many persons who work with alcoholics consider blackouts to be an early sign of alcoholism. However, Goodwin found that blackouts were reported by only two-thirds of the 100 alcoholics interviewed. In contrast, he found that 30% of the young social drinkers had experienced at least one blackout episode.<sup>6 1</sup> The exact correlation between a blackout and alcoholism is perhaps unimportant. Multiple or repeated blackouts are strongly indicative of alcoholic drinking and their occurrence usually precedes the onset of physiological changes associated with alcoholism. Blackouts are not associated with permanent memory damage. Furthermore, psychiatric and neurologic complications are not more common in alcoholics experiencing blackouts than they are in other alcoholics.

Polyneuritis, which was discussed earlier, is a complication which results from a diet lacking thiamine rather than any direct toxic effects of alcohol. It is decreasing in incidence because of the vitamin supplementation of many basic foods.<sup>44</sup> Frequent symptoms of polyneuritis are numbness, tingling sensations, and severe pain in the feet, the lower legs, and the hands. In addition to these sensory disturbances, weakness of the legs may be present. Usually the condition is sufficiently uncomfortable and debilitating to motivate the individual to seek early medical attention. Fortunately, in its early stages polyneuritis can be almost completely reversed by giving the vitamin thiamine, which allows the fatty cuff surrounding the nerve to reform, and normal nerve conduction and functioning to thus be restored.

Wernicke's encephalopathy is also primarily attributable to thiamine deficiency. Persons suffering from this acute disturbance of brain function have double vision, loss of balance, staggering gait, and, usually, extreme mental confusion with a loss of temporal and spatial orientation.<sup>18</sup> The brain swells within the skull and small vessels may bleed.<sup>54</sup> If this condition goes untreated, various areas of the brain will die; the condition will then become permanent. However, if it is treated with large doses of vitamin B<sub>1</sub> (thiamine), the condition will improve and leave little or no residual effect. Wernicke's encephalopathy usually manifests itself acutely and constitutes an urgent medical problem requiring rapid administration of thiamine.

Permanent Neurologic Complications. Permanent neurologic and neuropsychiatric changes associated with chronic alcoholism require that the patient either be nursed at home or, as is usually the case, become an inmate of a state V.A. mental hospital.



One permanent complication is Korsakoff's psychosis, which may be an extension of untreated Wernicke's encephalopathy. Dementia is present and can be improved with thiamine therapy, but the patient is seldom successfully rehabilitated. The most characteristic symptom of this psychosis is severely disturbed memory, with a tendency on the part of the patient to cover this defect by fabricating remarkably detailed accounts to fill the gaps in memory. Superficially the patient may not seem psychotic, but upon closer analysis, much of what he says is seen to be purely confabulatory. At times hallucinations are present. Typically the patient is jovial, although he may become depressed or irritable. Because of his disorientation and loss of memory, a patient with Korsakoff's psychosis is seldom able to function outside an institution.

Brain atrophy, in which nerve cells die and brain tissue is lost, accounts for a large group of permanent neurological defects associated with alcoholism. The extent of the atrophy can be measured by infusing air into the ventricles and then x-raying the head. The skull and the air in the ventricles are apparent on the x-ray; the brain tissue fills the remaining area. When there is marked atrophy of the brain, the ventricles enlarge and the distance between the skull and ventricles decreases.

Atrophy is an end result of irreversible nerve damage; it has numerous causes such as head trauma, brain infections, hereditary disease, neurotoxins, or the aging process. However, because brain atrophy is more common in chronic alcoholics than in the general population of the same age, and because the atrophy in alcoholics is characteristically found in certain areas of the brain, researchers believe that if alcohol is consumed in large doses over extended periods it can cause irreversible brain cell damage and thus result in brain atrophy.<sup>6 7</sup>

The symptomatology associated with brain atrophy depends

upon the area of the brain that has been destroyed, and upon the amount of brain tissue lost. The frontal lobes, lying just behind the forehead, are commonly involved, leading to decreased mental functioning, poor memory, and, frequently, to child-like behavior. The cerebellum, located at the base of the brain, is another common area of involvement. Because the cerebellum is intimately involved in the integration and coordination of movement, disturbances of its function result in poor balance, awkward walking, and general movement difficulties. Other areas of the brain may also become atrophied leading to different symptomatology; however, they are too numerous to discuss here.

Neuropsychiatric Complications. The neuropsychiatric complications of alcoholism are numerous and constitute an area of considerable controversy. Many patients when sober are found to have underlying psychotic or severely neurotic conditions. Some authorities maintain that to become an alcoholic a person must have some predisposing psychiatric problem. The successful and happy readjustment of many alcoholics would seem to speak against this rigid position.

However, persons afflicted with psychiatric and neurotic disorders have greater difficulty meeting social demands, experience more anxiety, and are troubled more frequently with depression. These persons may find that alcohol initially fulfills many of their needs; it decreases their anxiety and self-consciousness, lifts their depression, and strengthens their self-importance.<sup>62</sup> Alcohol can thus become a tool with which they can make themselves more like other people. Thus, alcohol may offer far greater rewards for some neurotics than it does for the normal person, and the neurotic may begin to use it as a cure-all medicine. However, as tolerance and dependence develop, the quantity of alcohol needed to achieve these ends increases to the point that no amount of alcohol can produce the looked-for

results. The individual may now drink just to become oblivious of his environment. As his misuse of alcohol increases, social condemnation of his behavior also increases which not only confirms any original low self-esteem but forces him to drink in social isolation. He no longer tries to cope with the outside world and his grasp on reality (and perhaps his wish to grasp reality) lessens. In such persons alcohol abuse contributes to the severity of the psychiatric problems, but probably, in itself, does not cause them. Treatment of such a person must be directed both at the alcoholism and at the underlying emotional problems, if successful and happy readjustment is to be achieved. However, therapy must attack the alcohol problem first and treat the adjustment problem secondarily. Only when he is sober is this patient amenable to therapy for his basic psychiatric problem.

#### SUMMARY

There are individual differences in the physical symptoms following ingestion of alcohol, but the symptoms follow a characteristic course. Initially, there may be feelings of euphoria, comfort, and elation. However, as the dose increases, less desirable effects occur, such as increased emotionalism. A person may become quarrelsome, affectionate, or both, and then become depressed. At moderate to high doses, alcohol will cause an individual to be less cautious, and will decrease the accuracy of most of the senses, including an individual's perception of pain, thus posing a real threat to the safety of the intoxicated person.

The body may eventually begin to react adversely to frequent and prolonged alcohol abuse. Withdrawal symptoms may occur with the cessation of a drinking episode, creating an extremely unpleasant experience and one which can be alleviated with further alcohol intake. Withdrawal, and other bodily dysfunctions which occur with continual abuse, can be reversed with a decrease or

cessation of alcohol intake. However, if there is continued abuse in the face of these adverse conditions, then irreversible disease states may develop.

A discussion of the disease states and conditions associated with alcohol abuse and alcoholism could be extended, but either the incidence of the diseases not covered is very small or their effects are rather benign. The foregoing discussions should be more than sufficient to show that the indiscriminate use of alcohol over extended periods can seriously injure most of the important organs and organ systems in the body. Ethyl alcohol is truly a potent and toxic agent.

## Appendix A

### THE DEVELOPMENT OF A COURT-RELATED REHABILITATION PROGRAM

#### 1. VOLUNTEERS IN PROBATION, INC.

(FORMERLY)

(PROJECT MISDEMEANANT FOUNDATION, INC.)  
February 1, 1969

In 1960, the Judge of the Royal Oak, Michigan Municipal Court invited eight citizens to serve as volunteer probation officers. Completely without funds to establish effective rehabilitative services, the court had no alternative but to seek citizen participation. Punishment, alone, simply was not effective.

The eight citizens responded. In a few years, over 500 citizens volunteered their time, talent and money. By 1965, the court was able to furnish \$300,000 a year in rehabilitative services on a \$17,000 a year city budget.

The volunteers perform extremely effective services on a one-to-one basis as Volunteer Inspirational Personalities. They also stimulated a total response from the community. As a result, many professionals volunteered to establish such services as psychiatric evaluations and counseling, marriage counseling, professional counseling, etc. Businessmen donated funds. For the first time, the city provided funds for the rehabilitation of offenders. As a result of the volunteer, complete rehabilitative services were established.

They were, and are, effective. A grant from the National Institutes of Mental Health proved that offenders' attitudes improved and recidivism (those committing a second crime while on

probation) was greatly reduced. By a battery of psychological tests and retests, the hostility of defendants was found to be reduced in 73% of the cases in Royal Oak as compared with only 18% in a comparable court. Recidivism in Royal Oak was only half that of the other court. The difference? The \$17,000 spent by Royal Oak included hours of volunteer involvement. The other court received the services of one professional for its \$17,000. With an insurmountable case load of some 500 probationers, he could do very little. (Both cities had similar populations, each around 100,000.)

In 1965, the National Board of Christian Social Concerns of the Methodist Church provided funds to spread the idea throughout the country. By the end of 1968, about 125 cities had started similar programs in Adult Misdemeanant or Juvenile Courts due to Project Misdemeanant. These cities range in size from Denver, Houston, Dallas and Seattle to Bergenfield, New Jersey, Fort Smith, Arkansas, Olympia, Washington and Hastings, Michigan (Population 7,000). The larger cities have gone from the pre-existing professional programs to the volunteer. The smaller cities have usually started with the volunteer and have, through the volunteer, obtained professional assistance. The best answer is the volunteer and the professional working together.

In February, 1969, the funds from the church were exhausted.

Rather than let the work die, several private donors made Project Misdemeanant Foundation, Inc. possible. This non-profit Foundation is ready to assist you to utilize volunteers and citizen participation in your court. It furnishes, without cost, speakers, consultants, work-shops, demonstrations, films, literature, tapes of speeches, etc. Now, as we begin the new decade of the 1970's an estimated 500 or more courts are using volunteers.

We believe that attitudes are not changed by platitudes; that human conduct is changed by human contact. The key to

rehabilitation is to insert inspiring personalities in the lives of young offenders. The difference between success and failure is the presence or absence of a giant, who seemed to be ten feet tall, in our childhood and adolescence. We are putting giants in the lives of the youthful offender. The answer is in personalities. We cannot buy the services of personalities in sufficient quality and quantity but our citizens, motivated by Jewish-Christian concepts, will give their personalities to us, if we but ask.

The first probation officer in history was a volunteer, a Boston cobbler. This was in 1849. The idea was lost and the over-burdened professional, and much more often no one at all, provided services. In 1960, the idea was rediscovered.

Would you like to become part of the nationwide movement? Let us know. We will help you in any way we can. We would only suggest that you start small, build spirit and do not carbon-copy the mechanics of any other program.

We hope to hear from you.

Keith J. Leenhouts  
Judge, Municipal Court & District Court, 1959-1969  
Director, Volunteers in Probation, Inc.  
200 Washington Square Plaza  
Royal Oak, Michigan 48067

## 2. VOLUNTEER PROGRAM-BOULDER, COLORADO

In 1963, a volunteer program began in the Boulder (Colorado) Juvenile Court. Completely without knowledge of each other, this fine program developed in a manner philosophically similar to Royal Oak. Boulder has also helped to stimulate other courts to use volunteers. For information write to Dr. Ivan Scheier, Juvenile Court, Hall of Justice, Boulder, Colorado 80302. Research in Boulder and Denver has further proven the effectiveness of volunteers.



## Appendix B

### A DESCRIPTION OF SOME OF THE SERVICES PROVIDED BY VOLUNTEERS IN PROBATION, INC.

#### PROBATION SERVICES

##### PRESENTENCE:

Presentence reports are essential. The investigator interviews offenders gathering factual information including family background and police reports. This is completed before sentencing and appropriate recommendations are presented to the judge to assist him in sentencing. Often presentence evaluations include evaluations by the staff and volunteer psychiatrists. Psychological evaluations are also available. Many volunteer psychiatrists and psychologists are involved. The use of retirees keep the cost minimal.

##### CHIEF COUNSELORS:

When probation is desirable, offenders are assigned to one of 12 Chief Counselors. These are professionally trained part-time staff members who counsel and supervise probationers. Each is responsible for about 15 individuals. They meet regularly with probationers and are responsible for coordinating other prescribed services. They are the heart of the professional counseling program. They supervise the volunteers.

##### PSYCHIATRIC-PSYCHOLOGICAL SERVICES:

For the offender in need of such assistance, these free services are available: (1) Psychiatric evaluations and psychological testings, (2) Individual treatment by a part-time psychiatrist, (3) Individual therapy on a private basis, (4) Group therapy conducted by staff and volunteer psychiatrists. Thirty volunteer professionals are involved.

#### VOLUNTEER SPONSORS:

Often a probationer is assigned to one of about 150 volunteer men and women sponsors representing just about every sector of our community. Those not trained as counselors are selected with care, receive orientation and are carefully supervised.

The sponsor is the inspirational personality with whom the young probationer can identify...build a relationship based upon trust and confidence. The sponsor listens and seeks to understand, love, correct and inspire. This can make the difference in changing lawless behavior.

The sponsor is dedicated, concerned and desires to serve his court and community. He is not paid for his service. It has been said that he is not an answer but the answer to rising crime and delinquency. His main function is to be a friend of the probationer. He does everything that you would do for a friend.

#### OTHER VOLUNTEER ASSISTANCE:

Professional men including lawyers, doctors and optometrists lend their skills to the program whenever called upon. Volunteer secretaries help out.

Businessmen, large and small, assist through financial contributions and job placement.

#### MARRIAGE AND FAMILY COUNSELING:

This service is also available at no cost. A Chief Counselor is specifically trained in this area and works solely with the cases needing such counseling individually and in groups.

#### CHURCH REFERRAL SERVICE:

If a probationer indicates a desire to have a church "home" the clergy or layman who represents that church is asked to make

contact with the probationer and support him in establishing or re-establishing his church affiliation. Some 90 churches are involved.

#### ALCOHOLICS ANONYMOUS:

The serious problem of alcoholism is known to every court. The failure of routine court "treatment" is also well known. With a real desire to cope with this problem, the court A.A. group was launched. It is directed by a retired gentleman with many years of A.A. membership. Referrals are made directly by the court. Our success ratio compares favorably with that of A.A. programs in general.

#### NONSUPPORT ENFORCEMENT AND RESTITUTION:

A retired senior citizen and the Probation Administrator carry out this important task. The service is also performed without cost. In all, some 13 retired citizens serve the court.

#### EMPLOYMENT COUNSELING:

Helping young people discover their work potentials is most important. Employment counseling and aptitude testing are available. A retired citizen, formerly with the Employment Securities Commission, directs this free service. This is a great aid in rehabilitation.

#### THE WORK DETAIL:

The basic idea is to punish without creating a permanent court record. Worthy defendants without a criminal record are assigned to the Work Detail. The case is then adjourned without sentencing for up to two years and then dismissed if all "terms of adjournment" are satisfied.

The offender works for the City four Saturdays a month for one to six months. This program is self-supporting, each offender paying \$48 per month to cover all costs.

This work includes cleaning city parks, snow removal, collecting litter and painting park tables.

In addition, the offender reports to the Probation Department and is usually assigned to a volunteer sponsor. The Work Detail is ordinarily granted only where there is parental concern and the police approve. The combined deterrent of punishment, unofficial probation including all of the probationary services and the incentive of avoiding a criminal record by earning a dismissal has been most effective.

RETIRED CITIZENS:

Seven retirees working full-time for what is allowed under Social Security, administer the entire program.

ADDITIONAL INFORMATION:

Your attention is directed to a 50 page report entitled, "Concerned Citizens and a City Criminal Court." It is available upon request as is other literature.

## Appendix C

### RESOURCES AVAILABLE TO STIMULATE THE DEVELOPMENT OF PROBATION SERVICES

FROM

VOLUNTEERS IN PROBATION, INC.

(Formerly Project Misdemeanant Foundation, Inc.)

200 Washington Square Plaza

Royal Oak, Michigan 48067

---

1. LEAFLETS: "Royal Oak Aids Its Problem Youth," a reprint from the October, 1965 Reader's Digest describing the need and possibilities for probation services in courts handling the misdemeanor offender.
2. REPORT: "Concerned Citizens and a City Criminal Court." (55 pages) A report of the Royal Oak Municipal Court Probation Department. This includes a description of the development and use of professionals and volunteers and key resource materials used in the program.
3. FILMS: "Big Help for Small Offenders," a 16mm. sound, black and white film. 35 or 45 minutes, last ten minutes being optional depending on audience. Updates (1969) "City With A Heart." Describes the research and the spread of the concept throughout the United States through Project Misdemeanant. A small rental fee is suggested although special arrangements are also possible. Low cost purchase is also available.  
  
"TV Report -- Royal Oak -- City With A Heart." 16mm. sound, black and white film, 22 minutes. Describes the operation of the Royal Oak Probation Program and includes interviews with volunteers and a boy on probation. Loan copy available.

4. TAPE: A 30 minute tape of excellent quality is available of a speech given by Judge Leenhouts at a city which was interested in starting to use volunteers and citizen participation in a rehabilitative probation program.
5. BROCHURE: A short brochure about Royal Oak Program entitled, "Citizen Participation in a Probation Department."
6. GUIDE: "Steps in Securing Community Support for Misdemeanant Probation Services." Briefly describes ways to go about developing more effective services for rehabilitation.
7. CONSULTANTS:
- 1) Professionals are available for consultation with community groups that are ready to develop probation services for the misdemeanor offender. We will come to your city.
  - 2) Professionals in the field of probation are available through the National Council on Crime and Delinquency to guide volunteers.
  - 3) Church leaders are available to make contact with community and church leaders in order to develop support for probation services for the misdemeanor offender.
8. VISIT  
ROYAL OAK: We extend you a cordial invitation to come to Royal Oak and learn more about the use of volunteers. We invite you to come anytime. Just let us know what airplane to meet and how long you can stay with us.

9. SOURCE OF ASSISTANCE: An excellent source of assistance is the Juvenile Court at Boulder, Colorado. You can write to Dr. Ivan H. Scheier or Judge Horace B. Holmes. Their address is: Hall of Justice, Boulder, Colorado, 80302. Ask to be put on their mailing list for the Volunteer Courts Newsletter.
10. THINGS YOU CAN DO: Attend the annual meetings of the: American Correctional Association, 1000 Shoreham Building, 15th and H Streets, N.W., Washington, D.C. 20005.  
National Council on Crime and Delinquency, 44 East 23rd Street, New York, New York 10010.  
North American Judges Association, Judge Phillip Lambert, Executive Director, Municipal Court Building, 700 Couch Drive, Oklahoma City, Oklahoma 73112.  
Write to them for further details.
11. COSTS: All services are available free or on a cost reimbursement basis.

IN 1960, VIRTUALLY NO COURTS WERE USING VOLUNTEERS. IN 1967, ABOUT 25 COURTS WERE USING THEM. NOW, AS WE BEGIN THE NEW DECADE OF THE 1970's, APPROXIMATELY 500 OR MORE COURTS ARE USING VOLUNTEERS. IF YOU WOULD LIKE TO BECOME PART OF THIS GROWING AND SIGNIFICANT MOVEMENT, LET US KNOW. WE WILL ASSIST YOU IN ANY WAY YOU FEEL IS APPROPRIATE.





## REFERENCES

1. Pollack, S. Drinking Driver and Traffic Safety Project. Vol. I. Public Systems Research Institute, Univ. of S. Calif., Los Angeles, 1969.
2. Selzer, M. Payne, C.E. Gifford, J.D. and Kelly, W.L. Alcoholism Mental Illness and the "Drunk Driver". Am. J. Psychiat. 120:326-331, 1963.
3. Smith, F. Blood Alcohol Level and Other Selected Factors As Indicators of the Problem Drinking Driver. (Ph.d. Thesis), Michigan Dept. of Secondary Education and Curriculum, Mich. State Univ. 1970.
4. Filkins, L.D. Clark, C.D. Rosenblatt, C.A. Carlson, W.L. Kerlan, M.W. and Manson, H. Alcohol Abuse and Traffic Safety: A Study of DWI Offenders, Alcoholics and Court-Related Treatment Approaches. Highway Safety Research Institute, The University of Michigan, Ann Arbor, 1970.
5. Purdy, L.A. Success Story: The Community Program Concept. In: Proceedings of a Conference on Community Response to Alcoholism and Highway Crashes. Filkins & Geller (ed) Highway Safety Research Institute, The University of Michigan, Ann Arbor, 1970.
6. Dana, A.H. Problem Drinking in Industry: A Study of Industry's Implications of Alcoholism in Florida. Res. Rep. Social Sci. 6:1-113, 1963.
7. Lemere, F. O'Halloren, P. and Maxwell, M.A. Motivation in the Treatment of Alcoholism. Quart. J. Stud. Alc. 19:428-431, 1958.
8. Hughes, Howard. Comment. National Council on Alcoholism, Inc. Number 2, 1971.
9. Mortimer, R.G. and Lower, J.S. Development of a Driver Alcoholism Test. Highway Safety Research Institute, The University of Michigan, Ann Arbor, 1970 (unpublished document).
10. Barmack, J.E. and Payne, E.D. Injury Producing Private Motor Vehicle Accidents Among Airmen. Bulletin 285, Highway Research Board, 1961.

11. Guze, S.B. Tuason, V.B. Gatfield, P. Stewart, M.A. and Picken, B. Psychiatric Illness and Crime with Particular Reference to Alcoholism: A Study of 223 Criminals. J. Nerv. and Ment. Dis. 134:512-521, 1962.
12. Selzer, M.L. The Quest for a New Diagnostic Instrument. Amer. Psychiat. Assoc., Annual Meeting, San Francisco, Calif., May 11-15, 1970.
13. Selzer, M.S. Payne, C.E. Westervelt, F.H. and Quinn, J. Automobile Accidents as an Expression of Psychopathology in an Alcoholic Population. Quart. J. Stud. Alc. 28:505-516, 1967.
14. Brown, S.L. Bohnert, P.J. Finch, J.R. Pokorny, A.D. and Smith, J.P. Alcohol Safety Study: Drivers Who Die. Baylor University College of Medicine, Houston, 1968.
15. Horn, J.L. and Wanberg, K.W. Symptom Patterns Related to Excessive Use of Alcohol. Quart. J. Stud. Alc. 30:35-58, 1969.
16. Cisin, I.H. and Cahalan, D. A Comparison of Abstainers and Heavy Drinkers in a National Survey. Amer. Psychiat. Assoc. Annual Meeting, Washington, D.C., 1966.
17. Murphree, H.B. Neuropharmacological Effects of Substances Other Than Ethanol in Alcoholic Beverages. Fed. Proc. 26:1468-73, 1967.
18. Harrison, T.R. Principles of Internal Medicine. 5th ed. McGraw-Hill Book Company, New York, 1966.
19. Bates, R.C. The Diagnosis of Alcoholism. Appl. Therapeutics 1965, 1966.
20. Lieber, C.S. Metabolic Derangement Induced by Alcohol. Ann. Rev. Med. 18:35-54, 1967.
21. Mendelson, J.H. Comparative Psychophysiological Studies of Alcoholic and Non-Alcoholic Subjects Undergoing Experimentally Induced Ethanol Intoxication. Psychosom. Med. 28:1-12, 1966.
22. Mendelson, J.H. Effects of Experimentally Induced Intoxication on Metabolism of Ethanol-1-C<sup>14</sup> in Alcoholic Subjects. Metabolism. 14:1255-66, 1965.

23. Mendelson, J.H. Ethanol-1-C<sup>14</sup> Metabolism in Alcoholics and Non-Alcoholics. Science. 159:319-20, 1968.
24. Lieber, C.S., et al. Ethanol Oxidation by Hepatic Microsomes Adaptive Increase after Ethanol Feeding. Science. 162:917-18, Nov. 22, 1968.
25. Edwards, J.A., et al. Ethanol Metabolism in Subjects Possessing Typical and Atypical Liver Alcohol Dehydrogenase. Clin. Pharmacol. Therapeutics. 8:824-829, 1967.
26. Mendelson, J.H. Serum Cortisol Levels in Alcoholic and Non-Alcoholic Subjects During Experimentally Induced Ethanol Intoxication. Psychosom. Med. 28:616-26, 1966.
27. Mendelson, J.H. Biologic Concomitants of Alcoholism. (Parts I & II). New Engl. J. Med. 283:24-32, 1970.
28. Borg, E. and Moller, A.R. Effect of Ethyl Alcohol and Pentobarbital Sodium on the Acoustic Middle Ear Reflex in Man. Acta Oto-Laryng. 64:415-426, 1967.
29. Wechsler, H., et al. Alcohol Level and Home Accidents. Pub. Health Repts. 84:1043-1050, 1969.
30. Drew, L.R. Alcoholism as a Self-Limiting Disease. Quart. J. Stud. Alc. 29:956-967, 1968.
31. Rushing, W.A. Suicide and the Interaction of Alcoholism (Liver Cirrhosis) with the Social Situation. Quart. J. Stud. Alc. 30:93-103, 1969.
32. Birrell, J.H. Blood Alcohol Levels in Drunk Drivers, Drunk and Disorderly Subjects and Moderate Social Drinkers. Med. J. Aust. 2:949, 1965.
33. Morris, N. Alcoholism in Relation to Crime and Punishment. In: Alcoholism, Foundation for Research and Treatment of Alcoholism. Sydney, 1960, p. 152.
34. Gillespie, J.A. Vasodilator Properties of Alcohol. Brit. Med. J. 2:274-277, 1967.
35. Ott, B. Paralysis Due to Nerve Pressure "Without" Trauma. Landarzt. 42:1632-1635, 1966.
36. Hart, R.H. Saturday Night Syndrome. Can. Med. Assoc. J. 100:799, 1969.

37. Marjot, D. The Length of the drinking Bout Preceding Alcohol Withdrawal States. Brit. J. Addict. 64:307-13, 1970.
38. Aspects of Alcoholism, Vol. 2. J.B. Lippincott Company, Philadelphia, 1966, p. 51.
39. Cox, W.D. and Gillesby, W.C. Longitudinal Pancreatico-jejunosotomy in Alcohol Pancreatitis. Arch. Surg. 94:469-475, 1967.
40. Verdy, M. and Daliou-Dialla, G. Hypoglycemia and Alcohol. Can. Med. Ass. J. 98:827-830, 1968.
41. Arky, R.A., et al. Irreversible Hypoglycemia; A Complication of Alcohol and Insulin. J.A.M.A. 206:575-578, 1968.
42. Carneiro-DeMoura, M., et al. Clinical Alcohol Hypoglycemia. Ann. Intern. Med. 66:893-905, 1967.
43. Perkoff, G. Dioso, M. Bleisch, V. Klinkerfuss, G. A Spectrum of Myopathy Associated with Alcoholism. I. Clinical and Laboratory Features. Ann. Intern. Med. 67:481-492, 1967.
44. Jellinek, E.M. Estimating the Prevalence of Alcoholism: Modified Values in the Jellinek Formula and an Alternative Approach. Quart. J. Stud. Alc. Vol. 20, 1959.
45. Viel, B., et al. Alcoholism, Accidents, Athrosclerosis, and Hepatic Damage. In: Alcohol and Alcoholism. R. E. Popham, Ed. University of Toronto Press, Toronto, 1970. p. 319.
46. Anderson, W. Pathology. 5th ed. C.V. Mosby Company, St. Louis, 1966. p. 899.
47. Caroli, J. and Pequignot, G. Enquete sur les circonstances dietetiques de la cirrhose alcoolique en France. Proceedings of the World Congress of Gastroenterology and Fifty-Ninth Annual Meeting of the American Gastroenterological Assoc. 1:661, 1958.
48. Sepulveda, B., et al. Malnutrition and Liver Disease. Gastroenterology. 33:249-, 1957.

49. Bang, N.U., et al. Serum Glutamic Oxalacetic Transaminase in Acute-Activity and Chronic Alcoholism. J.A.M.A. 168: 1956, 1958.
50. Ratnoff, O. Hemostatic Mechanisms in Liver Disease. M. Clin. North Am. 47:721, 1963.
51. Stahl, J. Blood Ammonia in Liver Disease. Ann. Intern. Med. 58:1, 1963.
52. Isreal, S.L., et al. Inactivation of Estrogen by the Liver. Am. J. Med. Sci. 194:835, 1937.
53. Klinkerfuss, G. Bleisch, V. Dioso, M. and Perkoff, G. A Spectrum of Myopathy Associated with Alcoholism II. Light and Electron Microscopic Observations. Ann. Intern. Med. 67:493-510, 1967.
54. Carson, D.J. Pathologic Findings Following Alcohol. Anesthesia and Analgesia. 48:671-675, 1969.
55. Tobin, J.R., et al. Primary Myocardial Disease and Alcoholism; the Clinical Manifestations and Course of the Disease in a Selected Population of Patients Observed for Three or More Years. Circulation. 35:754-764, 1967.
56. Sullivan, J.F., et al. Myocardiopathy of Beer Drinkers: Subsequent Course. Ann. Intern. Med. 70:277-282, 1969.
57. Kerr, A., Jr. Myocardiopathy, Alcohol, and Pericardial Effusion. Arch. Inter. Med. 119:617-619, 1967.
58. McDermott, P.H., et al. Myocardosis and Cardiac Failure in Men. J.A.M.A. 198:253-256, 1966.
59. Alexander, C.S. Idiopathic Heart Disease: Analysis of 100 Cases with Special Reference to Chronic Alcoholism. Amer. J. Med. 41:213-228, 1966.
60. Alexander, C.S. Idiopathic Heart Disease, Electron Microscopic Examination of Myocardial Biopsy Specimens in Alcoholic Heart Disease. Amer. J. Med. 41:229-234, 1966.
61. Goodwin, D., et al. Alcoholic "Blackouts": A Review and Clinical Study of 100 Alcoholics. Am. J. Psych. 126: 191-198, Aug. 1969.
62. English, O. and Finch, S. Introduction to Psychiatry. W.W. Norton & Company, Inc., New York, 1964.





