American Handbook of Psychiatry

ALCOHOLISM

A BIOBEHAVIORAL DISORDER

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Alcoholism

A Biobehavioral Disorder

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Alcoholism: A Biobehavioral Disorder[1]

Introduction

This chapter presents an overview of our current state of knowledge concerning the actions of alcohol, the disease of alcoholism, and patterns of use and abuse in contemporary American society. The basic pharmacology of alcohol effects is reviewed, and the medical, psychological, and social consequences of prolonged alcohol abuse and alcoholism are described. Comparisons are made between alcohol addiction and drug addiction wherever possible. The limitations in our current understanding of the factors which influence the development and maintenance of alcohol abuse and alcoholism are discussed, and the potential efficacy of existing treatment approaches is evaluated. We conclude that significant progress in combating alcohol abuse requires clarification of the basic mechanisms of the addictive process to permit development of effective therapies as well as productive strategies for prevention.

Alcohol: Beverage and Drug

All beverage alcohols, wine, beer and distilled spirits, contain the same primary ingredient, ethyl alcohol or ethanol. Ethanol is a relatively simple organic molecule which is produced in abundance by the fermentation

processes of microorganisms. Virtually all unicellular organisms have the capacity to produce ethanol given the availability of sugar, water, yeast, oxygen, warmth, and an appropriate acid-base balance. These basic ingredients have been present on earth since paleozoic times.

Beverage alcohols differ primarily in ethanol concentration which usually ranges from less than 4 percent for beer, to over 12 percent for wine, to 40-50 percent for distilled spirits. Under ordinary biological conditions, microorganisms do not produce alcohol concentrations in excess of 12-14 percent. However, man's discovery of distillation processes has permitted production of beverage alcohols with a higher concentration of ethanol. Alcohol boils at a lower temperature than water and therefore can be separated from its vehicle and concentrated. The term brandy derives from a German term for burnt distilled wine. Beverage alcohols also differ in terms of congener content or nonethanol impurities which include vitamins, organic and amino acids, minerals, salts, sugars, etc., as well as low concentrations of the higher alcohols (known as fusel oils) which are relatively toxic. The nonnutritional congener content of distilled spirits is higher than that of wine or beer and has been shown to increase as a function of aging.

It is well known that the rate of alcohol absorption into the blood varies markedly between beer, wine, and distilled spirits. Some congeners also affect the rate of alcohol absorption. Generally, the higher the alcohol concentration,

the more rapid its absorption, whereas the higher the congener concentration, the slower its absorption.

The nutrient value of alcohol is negligible following distillation. The caloric content of beverage alcohols varies between 100 and 200 calories per ounce. However, the extent to which calories in alcohol are equivalent to calories derived from food remains a subject of controversy. Alcoholics tend to eat poorly while they are drinking and it has often been suggested that this is due to the high caloric yield from alcohol. However, it has recently been observed that alcohol addicts receiving a daily total combined caloric intake from food (about 2000 calories) and alcohol averaging 4000 to 5000 calories did not gain weight over a two-month period. The small effective contribution of calories from alcohol to the total caloric pool may reflect the fact that utilization of calories from vitamin-deficient sources such as alcohol is impaired when food intake is adequate.

Alcohol Use in Historical Perspective

Alcohol was first discovered perhaps 200 million years ago and relics of the earliest civilizations show that alcohol was used in religious ceremonies, medical treatment, and in many aspects of daily life. It has been speculated that Paleolithic man learned to ferment honey and that the development of agriculture was paralleled by the improvement of techniques for

fermentation of fruits and grains, culminating in the process of distillation during the first century a.d. The legendary origins of alcohol are intermixed with the religious beliefs of many cultures, and it was commonly considered to be a gift of the Gods. In ancient Egypt and Greece, Osiris and Dionysus were worshipped as the givers of wine. It was believed that the Gods could use this gift to cause madness or enhance pleasure and awareness in the drinker. Later, alcohol itself was imbued with an autonomous power and a trace of animistic thinking about alcohol still persists.

Although festival drunkenness was condoned, the secular use of alcohol was accompanied by many warnings against excessive drinking throughout history. One of the oldest temperance tracts, entitled The Wisdom of Ani, was written in Egypt about 3000 years ago. Denunciation of excessive drinking can be found throughout ancient writings of Greece, Rome, India, Japan, and China as well as in the Old and New Testaments.

The early temperance movement of the 1830s recommended abstinence only from distilled spirits. Subsequently, there developed an increasing opposition to all alcoholic beverages, which were finally banned by the eighteenth amendment in 1920. National prohibition was repealed in 1933 by the twenty-first amendment in response to a complex series of attitude changes about self-regulation of drinking and the dangers of bootlegging. Legal sanctions[2] proved ineffective in controlling alcohol abuse

and in modifying the fact that many people like to drink. In contemporary American society, most adults consume some alcoholic beverages, and, apparently, most do so in a responsible and healthy way.

Medical Use of Alcohol

There is no current medical use of alcohol. Before the introduction of anesthesia, alcohol was used during surgical procedures in an effort to alleviate pain. However, since the dosage of alcohol required to produce loss of consciousness is close to a lethal dose for normal drinkers, alcohol is impractical as an anesthetic agent.

During the early part of the century, alcohol was occasionally used to counteract the alcohol-withdrawal syndrome. Although alcohol did reduce tremor briefly, its duration of action was too short to be practical. Moreover, prolonged use of alcohol merely reinstated the condition of chronic intoxication which leads to withdrawal symptoms following alcohol termination.

Until recently, alcohol was the drug of choice for the treatment of familial action tremor. This condition is now treated with propranolol (a beta-adrenergic blocking agent used in the clinical management of cardiac arrhythmias). The efficacy of both alcohol and propranolol in reducing symptoms of familial action tremor suggests that both may have a common

receptor site in the central nervous system (CNS).

Current Patterns of Alcohol Use and Abuse

According to a recent survey of American drinking practices, an estimated two thirds of all adults use alcohol occasionally. Total abstinence accounted for 32 percent of the 2746 representative persons surveyed. There were considerable regional variations in beverage preference and in the usual amount of alcohol consumed.

Of the alcohol users studied, 12 percent were categorized as "heavy" drinkers; i.e., persons who drank almost daily or once a week and often consumed five or more drinks per occasion. There are several problems associated with a compilation of a meaningful volume-frequency index of alcohol consumption. Although people may drink comparable total quantities of alcohol over a particular time interval, the extent to which they space drinking or concentrate drinking within a brief period may reflect distinctly different drinking patterns. In contrast to the "heavy" drinker, an alcohol addict may drink between 24 to 32 oz. per day in increments of 2 or 3 oz. per occasion. The length of a drinking spree may vary from a few days to two weeks or more.

A follow-up survey of American drinking practices revealed that there was considerable dynamic change in composition of the "heavy" drinking

group and of moderate and infrequent drinking groups. During the three-year interval between two successive surveys, 15 percent of the sample had moved out of or into the heavy drinking group. Even in the abstinent group, one third reported that they once used to drink. This high turnover rate is somewhat encouraging insofar as it indicates that heavy drinking does not invariably progress towards alcohol abuse or alcoholism.

Many interrelated sociological, demographic, economic, and psychological variables affect drinking patterns. The largest proportion of problem drinkers appear to be persons of lower socioeconomic status living in urban areas. The highest rates of alcohol-related problems were found in urban men, under 25, single and divorced, who often reported disrupted childhoods and a transition from rural to city living. The proportion of heavy drinkers among white and black males was comparable (22 vs. 19 percent), whereas black women showed a considerably higher rate of heavy drinking (11 percent) than white women (4 percent). However, estimates of alcohol abuse prevalence rates based on arrested or hospitalized alcoholics show a higher rate of severe drinking problems among ghetto-reared black males. American Indians and Eskimos have also been shown to be at high risk for the development of alcoholism.

The commonalities that have emerged from cross-sectional survey studies do not permit reliable prediction of the development of problem

drinking. Both heavy drinkers and abstainers have been described as more discontented and alienated from society than persons who use alcohol in moderation. Data from longitudinal studies suggest that the same childhood patterns predict drinking problems for ghetto-reared blacks and whites. Among both whites and blacks, adult drinking problems appear to be associated with early school problems, delinquency, drug use, and broken homes. These data have been interpreted to suggest that attention to *early* school problems might avert the subsequent progression of school failure and dropout, early drug exploration, and adolescent delinquency.

It is important to recognize that alcohol problems are not restricted to the disadvantaged, but can develop in anyone who drinks alcohol to excess. Alcoholism is considered to be the major drug-abuse problem in contemporary American society. It has been estimated that perhaps 5 million persons suffer from alcoholism. However, it should be emphasized that accurate case finding in alcoholism has been severely limited by the social stigma associated with this disorder. Consequently, accurate estimates of incidence and prevalence have been difficult to obtain. An additional 4 million persons are thought to abuse alcohol and therefore to be at high risk for the development of alcoholism. If these estimates are valid, perhaps 7 percent of the adult population have drinking problems.

The social costs to the afflicted individual and to his family are

incalculable. In its severest form, chronic alcoholism is associated with disruption of normal social and family ties; job loss and diminution of earning capacity; compromised physical and psychological health and decreased life expectancy. A profound and progressive isolation and alienation from self and society may sometimes terminate in violent death or suicide.

There is no simple formula to calculate the cost to society of the loss of a productive individual. The social costs of alcoholism have been estimated at levels of $750 million, $2 billion, and $15 billion dollars annually. Recent estimates suggest that $10 billion may be spent each year as a function of lost work time in every sector of the economy. Health and welfare services for alcoholics and their families cost an estimated $2 billion per year. An estimated 45 percent of all arrests in 1965 were for public intoxication, disorderly conduct, and vagrancy. Estimated medical expenses and property damage associated with alcohol problems bring the yearly cost of alcoholism to a total of $15 billion. These estimates, considered in connection with the human costs of alcohol-related traffic fatalities and disrupted lives, testify to the destructive toll of this poorly understood, complex bio-behavioral disorder.

Definitions of Alcoholism and Alcohol Abuse

The terms “alcohol abuse” and “alcoholism” are not synonymous, but

rather reflect stages in a continuum of severity from problem drinking to chronic alcohol addiction. Traditionally, there has been relatively poor agreement concerning definitions of alcoholism and alcohol abuse. Although the definition: "repetitive, excessive drinking that results in injury to an individual's health, adequate social function, or both," would be generally accepted, considerable variation continues to exist in the formulation of more precise definitions with concomitant criteria for differential diagnosis and implications for treatment (See references 7, 9, 66, 74, 119, 125, and 189). Definitions are important since they affect the management of the problem. The lack of an adequate definition has often impeded progress in our understanding of alcohol problems.

Sociocultural Definitions

Although at first glance it may not appear difficult to arrive at a consensus for definition of an alcohol-related problem, there are many conflicting social, cultural, and religious perceptions which contribute to continuing disagreement. Some attempts have been made to define alcohol abuse on the basis of a volume-frequency index of alcohol consumption. This approach is limited, since a particular drinking pattern will not be uniformly accepted in different regions or across various cultures. The Expert Committee of the World Health Organization defines abnormal drinking as that form of drinking which transgresses the normal social and dietary habits

of the community. Therefore, heavy drinking in a society which condones drinking would not constitute alcohol abuse, whereas consumption of any alcohol in a society which has rigorous standards of abstinence would be considered alcohol abuse. Depending on the standards employed, any definition of alcohol abuse results either in over-inclusion or under-inclusion of a large number of cases. Consequently, definitions which involve primarily social criteria are limited by the enormous variation in acceptable drinking habits within and between countries.

Pharmacological Definitions

One resolution of the difficulties associated with sociocultural definitions is to establish diagnostic criteria for alcoholism based on the pharmacological criteria of addiction: tolerance and physical dependence. Tolerance for and physical dependence upon alcohol is similar to phenomena which may develop following the abuse of other drugs which affect the central nervous system, such as opiates and barbiturates. Tolerance for alcohol refers to the fact that progressively larger quantities are required to produce changes in feelings or behavior which had previously been attained with smaller doses of alcohol. Physical dependence is demonstrated by the fact that the alcoholic experiences discomfort and physical illness when he stops drinking or decreases his alcohol intake. It is not necessary for an alcoholic to stop drinking completely before he experiences withdrawal

symptoms, since a relatively small decrease from high blood-alcohol levels may precipitate the onset of this disorder.

The pharmacological definition of alcoholism has the advantage of referring to a series of observable events, i.e., the alcohol withdrawal syndrome. This objective definition permits selection of comparable patients for research and treatment evaluation and thereby increases the generalizability of the results. However, the pharmacological definition is limited in that it applies only to the alcohol addict and does not include the early problem drinker. The skid-row alcoholic is the most visible victim of alcoholism and accounts for an estimated 3-5 percent of Americans with alcohol problems. The extent of physical dependence among the middle and upper socioeconomic classes is unknown.

The Disease Concept of Alcoholism

Demonstration that alcoholism is a form of addiction has led to a gradual acceptance of the idea that alcoholism is an illness and a medical problem. For many decades, alcoholism was considered primarily within the context of moral transgression and social deviancy, and public drunkenness was dealt with in a punitive, criminal-justice system. It has become increasingly evident that legal sanctions and moral pressures have not provided an adequate remedy for this problem. An unfortunate result of the

moralistic view of alcoholism was to limit interest in biomedical research on this condition. Our heritage from these years of scientific neglect is relative ignorance concerning even the basic phenomenology of alcoholism. Now that alcoholism, the disease, is considered an appropriate subject for laboratory and clinical investigation and federal resources are available for research, biomedical research interest has increased fourfold in six years (1966-1972).

The disease concept of alcoholism is sometimes criticized because it is thought to be overly restrictive and to imply a linear physical causality without sufficient attention to social and psychological factors. This criticism is based upon a misunderstanding of the disease model of alcoholism. In fact, the model assumes that expression of the disorder depends on an *interaction* between the individual, the agent of the disease (alcohol) and the environment in which the disease process develops (see Figure 15-1). It is well known that disease processes can rarely be explained on the basis of any specific factor within these three categories, but rather on their interrelationships. Even infectious disease is often more closely related to host-resistance factors and environmental variables than to the presence or even the virulence of any given infectious agent.

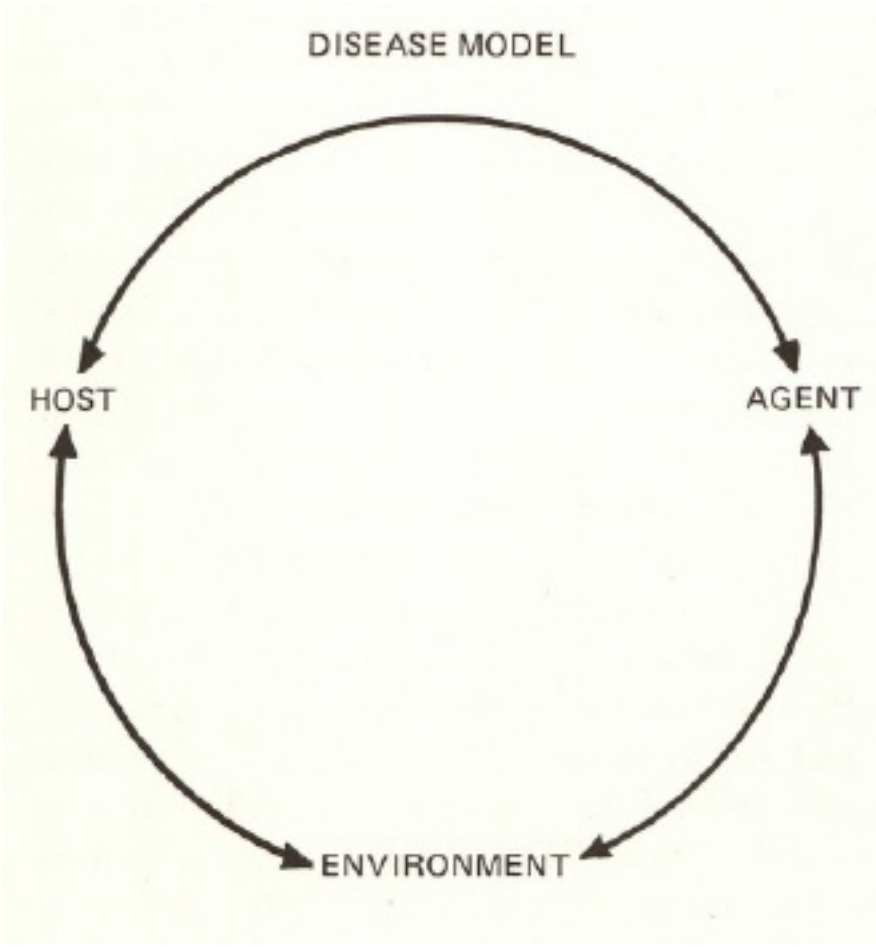

Figure 15-1.

Schematic diagram of a disease model of alcoholism depicting the interaction between host, agent (alcohol) and the environment.

One important advantage of a disease model of alcoholism is to

stimulate biomedical research and to redirect the attention of physicians to the medical aspects of this disorder. Traditionally, physicians have shown considerable reluctance to treat alcoholics for a variety of reasons and it has been difficult for alcoholics to gain admission to general hospitals. Insofar as alcoholism is now considered to be within the province of medicine, there should be an improvement in the quality of care for the medical complications of alcohol abuse. Moreover, as the physician begins to accept responsibility for the alcoholic patients, he may become more interested in and skillful at detecting early warning signals of the illness within the context of general medical practice.

A Practical Solution Comprehensive Diagnostic Criteria

A constructive and direct outgrowth of the general acceptance of the disease model of alcoholism is the development of comprehensive diagnostic guidelines by the National Council on Alcoholism. These criteria specify behavioral and attitudinal changes, as well as medical problems, to aid the physician in the early detection of alcohol problems and to permit differential diagnosis of late-stage alcoholism. There has been an attempt to compare the significance of certain subjective reports and objective signs in diagnosing alcohol problems. Common medical complications of alcoholism are specifically indicated and evaluation of possible concurrent psychiatric problems is urged.

Early detection is probably a critical aspect of prevention. Individuals at high risk for alcoholism characteristically deny the contribution of alcohol to any physical or psychological problems that led them to seek medical advice. By careful description of the types of behavioral and medical problems associated with excessive alcohol use, these diagnostic criteria provide a basis for increased awareness of the danger signs of alcoholism and the possibility of early intervention by the alert physician. The extent to which the availability of these diagnostic criteria may increase physicians' acceptance of the patient with alcohol problems remains to be determined.

Pharmacology of Alcohol and Alcohol Abuse

Mechanism of Action of Alcohol

The exact pharmacological mechanisms of the actions of alcohol are unknown. The targets of central and peripheral effects can be specified with some confidence, but the way in which alcohol produces physiological and behavioral changes is not understood. In addition to the effects of alcohol on the central nervous system, which are associated with the subjective and behavioral correlates of intoxication, alcohol ingestion may also result in a sensation of warmth and flushing of the skin (due to dilation of the peripheral blood vessels), muscular relaxation, and stimulation of gastric secretion and peristalsis.

Some of the physiological changes produced by alcohol may be determined by selective and perhaps even genetically controlled factors within an individual. Flushing and peripheral vasodilatation following alcohol consumption has been found to occur with greater frequency and intensity in Orientals than in occidentals. Studies of infant responses to alcohol indicate that this phenomenon is not due to cultural, cognitive, or experiential factors. These findings have recently been confirmed and extended to adults. This increased sensitivity of Orientals to alcohol may have some influence on their perception of drinking as relatively aversive or pleasurable.

Ethanol is absorbed primarily from the small intestine and its rate of absorption into the blood is influenced by several factors. Rapid drinking produces a higher concentration of alcohol in blood than slowly sipping a comparable amount of alcohol. The rate of absorption increases with increasing concentrations of ethanol (up to a maximum of about 40 percent). However, increasing concentrations of congeners are associated with a slower rate of ethanol absorption. The presence of food in the stomach substantially reduces the rapidity of alcohol absorption, as does a variety of other factors which may slow the emptying time of the stomach. Given the same alcohol intake under the same conditions, a 180-pound man has a lower blood-alcohol level than a 130-pound man. Body weight influences blood alcohol concentrations because alcohol is uniformly distributed throughout the body tissue fluids following absorption. Once alcohol attains equilibrium, its concentration in body tissues is directly proportional to their water content. Regional concentrations of alcohol are partly dependent on regional differences in water concentration.

Because of these interacting variables, it is impossible to accurately predict the blood-alcohol concentration following alcohol ingestion. However, it has been estimated that the usual legal limit of intoxication for operating a motor vehicle (100 mg. alcohol per 100 ml. of blood) is attained after consumption of 6 oz. of distilled spirits on an empty stomach. Three ounces of alcohol may yield a blood-alcohol level of 50 mg./100 ml. whereas 12 oz. yield

a level of 200 mg./100 ml. In general, the behavioral effects of alcohol are directly dose related.

Most alcohol is removed by metabolic processes in the liver and only an estimated 2-10 percent is excreted via the kidneys and lungs. The rate of alcohol metabolism is the same in normals and in abstinent alcohol addicts. However, it has been shown that rates of alcohol metabolism may increase as a function of the amount and duration of ethanol ingestion. At the present time, there are no pharmacological agents available which significantly enhance rates of ethanol metabolism with a concomitant reduction of acute intoxicating effects of alcohol.

Intoxication: Short-Term Effects of Alcohol

The behavioral level of intoxication is a function of the concentration of alcohol in the blood. It has been shown that low doses of alcohol act as a stimulant whereas higher doses result in central nervous system depression. Increases in intoxication as a function of increased alcohol dosage comprise a continuum from relaxation and mild euphoria to hyperactivity, garrulousness, and aggression to incoordination, confusion, disorientation, stupor, and possibly coma or death. A pleasant tranquility and mild sedation may accompany a blood alcohol level of about 50 mg./100 ml. Motor discoordination may occur at levels of about 100 mg./100 ml.

Above that level, intoxication is obvious and may produce unconsciousness in normal (non-tolerant) drinkers. Concentrations above 500 mg./100 ml. may be fatal. The exact mechanisms by which alcohol effects the brain to produce the behavioral expression of intoxication is unknown.

It was once thought that these sequelae of intoxication reflected a hierarchical disruption of brain centers progressing from structures which control complex cognitive and motor behaviors and critical faculties to structures which control essential life functions such as breathing. It was suggested that the hyperactivity and emotionality associated with intoxication could reflect stimulation of relevant neural structures *or* depression of other neural complexes which normally inhibit the expression of these behaviors. It now appears that alcohol acts on a regulatory system, the reticular activating system, which, in turn, concurrently effects the activity of both the cerebral cortex and subcortical structures.

There is no simple relationship between moderate levels of alcohol intoxication and an individual's capacity to perform a variety of cognitive[3] and motor tasks. In part because of the tranquilizing effects of alcohol, some individuals show improved performance at low doses. The behavioral tolerance for alcohol which accompanies alcohol addiction is defined by the lack of impairment of skilled-task performance even at very high blood-alcohol concentrations (150-200 mg./100 ml.).

Yet, it has been shown that moderate alcohol doses may impair visual sensory capacities, i.e., specifically brightness discrimination and readjustment after exposure to bright lights. Auditory and tactile sensitivities are not dramatically affected and sensitivity to taste and odor is somewhat impaired. It is attention, judgment, and the integration and evaluation of sensory information rather than the quality of sensory information per se that seems to be most affected by alcohol. For example, during intoxication, people tend to underestimate object speed and distance as well as the passage of time.

The contribution of alcohol intoxication to accidents is probably a complex combination of sensory-motor impairments, poor judgment, and emotional liability and aggressivity. The effects of alcohol on emotionality and aggressivity are poorly understood. Although alcohol is generally believed to increase emotional liability, considerable evidence suggests that the social context strongly influences both the type and direction of emotional expression during intoxication. Consumption of large amounts of alcohol may facilitate, and perhaps induce, violent and aggressive behavior including homicide, armed robbery with aggravated assault, and other crimes of violence. The possible relationship between alcohol-induced aggressivity and the preponderance of fatal accidents during intoxication remains to be determined.

The role of alcohol intoxication in fatal accidents has been clearly demonstrated. Over half the nonhighway accident fatalities have been shown to involve known alcoholics or alcohol abusers. In the sample studied, work-related fatal injuries occurred far less frequently than accidents in the home. A comparison of the number of positive blood-alcohol levels among accident victims admitted to a hospital emergency ward showed a decrease from highway accidents (30 percent), to home accidents (22 percent) to occupational accidents (16 percent).

The high concordance between automobile accidents and alcohol abuse is well known. The Injury Control Program of the Public Health Service has estimated that alcohol contributes to or is associated with 50 percent of fatal motor vehicle accidents. An estimated 28,000 highway fatalities were associated with alcohol intoxication during a recent 12 month period. Statistics on drunken-driving fatalities show that a preponderance of the victims had blood-alcohol levels above 150 mg./100 ml. These high blood-alcohol levels suggest that the single vehicle casualties studied were not social drinkers, but alcohol addicts with substantial behavioral tolerance for alcohol. The nontoloerant individual would show severe motor and cognitive dysfunction at these blood alcohol levels which could interfere with driving altogether.

The effect of alcohol intoxication on sexual function has been the subject

of many anecdotes in the nonscientific literature which usually concur with Shakespeare's observation that intoxication provokes desire but impairs sexual performance. Male alcohol addicts are usually described as experiencing diminished heterosexual desire and activity and sexual impotence. Female alcoholics tend to report initiation or increased drinking during the premenstrual period. There are biological data which indicate that chronic alcohol abuse may impair sexual function through a disruption of gonadal function. It is well known that chronic alcoholism in males may be associated with the development of gynecomastia and testicular atrophy, which in turn may be related to a derangement in androgen metabolism. Alcohol intoxication has recently been shown to suppress plasma testosterone levels to well below the normal range in alcohol addicts. Other centrally acting depressant drugs (i.e., heroin, barbiturates, and high dosages of methadone) also suppress plasma-testosterone levels in addicts. Following alcohol, methadone, and heroin withdrawal, plasma-testosterone levels return to predrug baseline or to normal levels. The possible endocrine mechanisms which influence the behavioral expression of sexuality (and aggression) are as yet unclear.

Following acute intoxication, a generalized somatic discomfort, commonly called "the hangover," may occur. The physiological determinants of the hangover are not understood. It has not been shown that mixing drinks or the congener content of drinks specifically leads to the development of a

hangover. There are no specific treatments for a hangover and little scientific support for the efficacy of the popular remedies.

Alcohol Addiction: Long-Term Effects of Alcohol Abuse

Drug addiction is defined by the pharmacological criteria of tolerance and physical dependence. Some chronic alcohol abusers eventually develop physical dependence upon alcohol. This end stage of alcohol abuse has been clearly shown to be an addictive disorder on the basis of both clinical and experimental observations. It was once thought that physical dependence, the alcohol withdrawal syndrome, reflected intercurrent illness or vitamin and nutritional deficiencies. It has now been shown that withdrawal signs and symptoms occur in healthy well-nourished alcoholics solely as a function of cessation of drinking. It has also been possible to induce physical dependence upon alcohol in a variety of experimental animals (See references 22, 27, 30, 32, 104, 137, 187, and 188).

The crucial determinants of the development of alcohol addiction are unknown, and the nature of the addictive process remains a matter of conjecture. Thus far, no single theory has proved adequate to explain the complex multilevel symptoms which we collectively term alcoholism. Most probably, alcoholism, like any other behavior disorder, derives from many diverse factors in the individual and his environment. The factors which effect

a particular individual's susceptibility to alcohol addiction, given a prolonged pattern of heavy drinking, remain to be determined.

Tolerance

The process described as "tolerance" occurs as a function of prolonged exposure to a drug. This term refers to the observation that progressively higher doses of a drug are required to produce comparable subjective and behavioral effects. The development of tolerance is far more rapid than that of physical dependence, both for alcohol and for narcotics. Tolerance and physical dependence were once believed to be sequential and inseparable aspects of the same underlying addictive process. However, our current understanding of the relationship between tolerance and physical dependence suggests that, although physical dependence is invariably accompanied by the development of tolerance, drug tolerance may occur independently of physical dependence.

The alcohol addict shows three types of tolerance which are common to other addictive disorders; i.e., behavioral tolerance, pharmacological tolerance and cross tolerance to other potentially addictive drugs. *Behavioral tolerance* for alcohol is illustrated by the fact that an alcohol addict can drink as much as a quart of bourbon per day without signs of gross intoxication. A number of investigators have found that task performance during sobriety

and inebriation may not differ significantly even when blood-alcohol levels are twice 100 mg/ml, the legal limit of intoxication in many states (See references 11 , 25, 103, 105, and 159-161).

Associated with behavioral tolerance is *pharmacological tolerance* for alcohol. Consistent consumption of as much as a quart of bourbon per day may result in unexpectedly low levels of alcohol in the blood. It has been shown that alcohol ingestion does increase the rate of alcohol metabolism in alcoholics and in controls as a function of both amount and duration of alcohol ingestion. However, the ethanol-induced enhancement of the rate of ethanol metabolism appears to be transient and does not persist long after cessation of drinking. A number of studies have shown that the rate of ethanol metabolism in abstinent alcoholics and nonalcoholics is not significantly different (See references 13, 53, 63, 110, 120, and 128). The dramatic behavioral tolerance for alcohol shown by the alcohol addict cannot be accounted for by a more rapid or effective capacity to metabolize alcohol. This is evidenced by the fact that alcoholics can perform well on difficult tasks even with very high blood-alcohol levels (above 200 mg./100 ml.). These data suggest that the adaptive processes subserving tolerance occur in the central nervous system rather than at a metabolic level.

Cross tolerance refers to the general phenomena of reduced responsivity to drugs other than the primary addicting agent. It has been shown that sober

alcoholics metabolize a number of drugs more rapidly than nonalcoholics, and there are reports that alcohol addicts undergoing surgery require far larger doses of anesthetics than nonaddict patients to induce a surgical level of anesthesia. Similarly, the alcohol addict may show cross tolerance to other potentially addictive agents such as barbiturates, hypnotics, and sedatives. No cross dependence has been shown between alcohol and opiate narcotics. Finally, the alcoholic shows tolerance for many toxic alcohols and is able to ingest these in quantities that would be fatal for nonalcoholics. It should be emphasized that the phenomena of cross tolerance occurs in the *sober* alcohol addict. Under conditions of intoxication, many drugs contribute to the effects of alcohol, and the alcoholic individual may metabolize these drugs more slowly than normal subjects.

Although alcohol tolerance is striking in the alcoholic, the degree of tolerance, on a comparable dosage basis, is much less for alcohol than for opiates or barbiturates. For example, barbiturate addicts may ingest twenty to thirty times the average hypnotic dose in a twenty-four-hour period. Heroin addicts show similar increases in self-administration over their initial dosage levels. It is well known that some heroin addicts go through withdrawal in order to reduce their tolerance and thereby lower their daily maintenance dosage requirements. The physical limit of tolerance for the alcoholic is more firmly fixed, and even though the alcoholic does develop behavioral tolerance, blood-alcohol levels rarely exceed 450 mg./100 ml. The

lethal level of alcohol dosage also remains close to that for normal drinkers, and blood-alcohol concentrations in excess of 500-600 mg./100 ml. result in severe respiratory depression.

Physical Dependence

The usual alcohol withdrawal syndrome occurs between twelve and seventy-two hours following cessation of drinking, although the course and severity are quite variable. For clinical description see pp. 383-385. Abstinence signs and symptoms may include tremor, profuse sweating, gastrointestinal disorders, nystagmus, hyperreflexia, sleep disturbances, hallucinations, and occasionally, seizures. Remission of major symptoms is usually complete within three to five days. The usual alcohol withdrawal syndrome is generally similar to mild barbiturate, opiate, and nonbarbiturate sedative withdrawal.

The biological mechanisms underlying alcohol withdrawal and abstinence syndromes generally are unknown. The development of physical dependence is thought to be multiply determined by a complex interaction of neural, endocrine, and metabolic variables. Most attempts to account for the characteristic psychomotor and autonomic hyperactivity associated with drug-withdrawal syndromes have postulated a heightened CNS excitability. The basis for CNS hyperexcitability during withdrawal has often been

attributed to a rebound effect following drug-induced depression of neural activity. There is considerable neurophysiological evidence consistent with the notion that the CNS becomes more excitable during drug withdrawal.

The role of alcohol in facilitating seizure disorders in persons with an underlying epileptic disorder has long been a subject of considerable controversy. It is now thought that alcohol intoxication rather than alcohol withdrawal may increase the probability of seizures in persons with idiopathic or traumatic epilepsy.

Recent data suggest that, following removal of the depressant effect of alcohol, there is an increased sensitivity of the respiratory center to carbon dioxide and hyperventilation. It was once thought that alcoholics became acidotic during alcohol withdrawal. However, it has recently been shown that alcoholics develop a significant respiratory alkalosis which correlates both with susceptibility to stroboscopically induced seizures and the occurrence of delirium tremens. These data are of particular interest since several other hyperventilation syndromes, also associated with a respiratory alkalosis, yield clinical syndromes similar to those of alcohol withdrawal. Attempts to treat the alcohol-withdrawal syndrome by administering carbon dioxide to reduce the respiratory alkalosis were rather promising, despite technical difficulties in administering carbon dioxide. The efficacy of pharmacological agents to correct the respiratory alkalosis is currently being evaluated.

Psychological Dependence

Definitions of drug dependence are often subdivided into physical dependence and psychological dependence. Psychological dependence is usually inferred when drug use takes priority over other coping mechanisms and assumes a central focus in the organization of daily behavior. The term "psychological dependence" is imprecisely defined and usually used to refer to the motivational factors which underlie drug-seeking behavior *and* to the psychological effects of drug use. This semantic confusion between motivational and drug effect aspects of dependence reflects our current limited understanding of the factors which initiate and maintain drinking episodes. The conditions which initiate and perpetuate heavy drinking behavior are unknown. The resumption of addictive drinking after a period of abstinence probably reflects many psychogenic and stress factors. The possible contribution of the condition of physical dependence upon alcohol to reinitiation of drinking after sobriety is undetermined. It is unlikely that the condition of physical dependence in the sober alcoholic influences drinking behavior in the same way that the presence or absence of food-deprivation affects food seeking behavior. There are no satisfactory explanations for the repetition of excessive drinking and withdrawal sequences, despite the totally predictable adverse medical, social, economic, and often legal consequences. It is the immediate rather than the long-range consequences of alcohol abuse which appear to control drinking behavior.

In trying to account for the persistence of addictive drinking, it is tempting to extrapolate from one's own enjoyment of alcohol and to imagine that the immediate pleasures of drinking negate either the awesome prospect or the concurrent awareness of its many aversive consequences. However, it has been shown repeatedly that alcoholics become progressively more dysphoric, anxious, agitated, and depressed during a chronic drinking sequence. A comparable dysphoria during chronic drug use has been observed in morphine addicts and it has been suggested that increased narcotics intake may be motivated by an effort to regain an initial euphoria. It has also been shown that drinking tends to confirm and aggravate feelings of inadequacy and low self-esteem in alcoholics. Although the voluminous psychiatric literature on alcoholism has tended to present "the alcoholic" as an impulsive hedonist who drinks to dissolve his anxieties and achieve a diffuse sense of omnipotence, direct observations of intoxicated alcoholics reveal a pathetic failure to attain such goals.

The somewhat paradoxical increase in disturbing affect observed in alcoholics during studies of experimentally induced intoxication is difficult to reconcile with information that most alcoholics provide about their drinking experiences during sobriety. It appears that the sober alcoholic does not recall the seemingly aversive aspects of his drinking experience during a subsequent period of sobriety and therefore these aversive consequences cannot effectively modify his future behavior. There are considerable data

which converge to suggest that there is a substantial dissociation of experience during drinking, and subsequent recall and expectancy during sobriety. It is possible that psychotherapeutic techniques which involve efforts to integrate experiences during inebriation with awareness during sobriety through the use of videotaped interviews may provide an effective tool for bridging this alcohol-induced dissociation.

A second factor often advanced to account for the perpetuation of drinking is the notion of "craving" which implies that once an alcoholic starts to drink, "he is compelled to continue until he reaches a state of severe intoxication." The circularity inherent in this reasoning is evident, i.e., the concept of "craving" is defined by the behavior that it is evoked to explain. The concept of "craving" with its implication of "loss of control" over drinking has been the source of considerable confusion in the literature on alcoholism and has stimulated continuing debate. There has been no empirical support for the notion of "craving" on the basis of direct experimental observations of alcoholics given alcohol and clinical material. Although this construct appears to be of limited utility, it has long formed the basis for the usual therapeutic goal of total abstinence in the treatment of the alcoholic patient. (See also discussion on Treatment of Alcoholism, pp. 394-395).

A third factor assumed to be related to the maintenance of addictive drinking is the avoidance of withdrawal signs and symptoms which follow an

abrupt reduction in blood-alcohol concentration. If the avoidance of withdrawal signs is the factor which motivates an alcoholic to continue drinking, it would be expected that during a drinking spree, he should drink quite consistently and maintain stable blood-alcohol levels. However, recent data have shown a considerable cyclicity of alcohol self-administration both in primate and human alcohol addicts. Despite the attendant discomfort, the alcoholic does not invariably respond to these partial withdrawal signs by increased drinking. This situation may resemble that of the narcotics addict in which it has been suggested that incipient withdrawal signs may add both to the gratification and perpetuation of drug use. The relationship between physical dependence and subsequent drug self-administration is, at best, ambiguous. The assumption that physical dependence is one aspect of an addict's motivation for drug use is a complex and elusive issue to approach experimentally. Logically, it is difficult to account for the reinitiation of an addictive drinking sequence, after a prolonged period of sobriety, in terms of physical dependence alone. Presumably, whatever factors first prompted excessive drinking before the development of physical dependence, continue to affect resumption of alcohol abuse.

At the core of the many dynamic formulations concerning the basis of alcohol abuse are two related notions: (1) the alcoholic drinks to achieve a pleasurable subjective state; and

the alcoholic drinks to avoid or reduce the impact of current problems. Until progress in behavioral science permits more precise specification of the factors which initiate and perpetuate addictive drinking, the efficacy of attempts to intervene and avert repetition of destructive drinking sequences will be greatly limited. At present, we can only speculate about the possible psychological and social determinants of alcohol dependence.

Clinical Disorders Associated with Alcohol Abuse and Alcoholism

Factors in the Development of Alcoholism

Alcoholism can develop in any man or woman, irrespective of individual personality characteristics, educational, cultural, religious, ethnic, or socioeconomic background. No single psychological, social, or biological variable has yet been shown to predict the development of problem drinking or to uniquely differentiate alcoholics from nonalcoholics. The differences between alcoholic individuals far outweigh the commonalities of repetitive excessive drinking and tolerance for and physical dependence upon alcohol.

Many individuals develop alcohol problems independently of any family history of alcoholism. However, recent evidence suggests the possibility of a genetic component associated with the genesis of alcohol problems. Dissociation of familial learning factors from genetic variables is a difficult methodological problem. However, a study of fifty-five adult males, separated during infancy from their biological parents (one of whom had been hospitalized for alcoholism) had a significantly higher incidence of alcoholism than matched adoptee controls. Another study of 164 adoptees showed a significantly greater tendency to develop alcohol problems if the biological parent was an alcoholic than if the adopted parent was alcoholic.

The psychiatric literature on alcoholism contains countless theories which attempt to conceptualize alcoholism in terms of a psychodynamic formulation, a psychosocial developmental model, or as the outgrowth of specific personality characteristics such as depression, dependency, immaturity, hostility, and social isolation. An excellent comprehensive review of psychodynamic and personality factors in alcoholism appears in the first edition of this Handbook. This chapter does not attempt to recapitulate this material, in part because there has been little in the way of novel or substantive revision of the various psychological theories of alcoholism since the Handbook was first published in 1959. Some more recent conceptualizations seem little more than relabeling exercises with terms currently in fashion, e.g., "systems theory" etc. More important, the most plausible and ingenious theories concerning the psychological determinants of alcoholism have contributed little to the development of effective treatment and, for the most part, have been difficult to subject to rigorous experimental scrutiny.

Motivation for drinking varies greatly between individuals as well as within an individual from occasion to occasion. Drinking patterns are idiosyncratic; no consistent behavioral or biological correlates have yet been identified. Anxiety and depression may be far less at the beginning of a drinking episode than during or following prolonged intoxication. Stressful incidents may correlate with either the initiation or the cessation of drinking

in alcohol addicts. Alcohol intoxication does not appear to produce any reproducible pattern of social interactions. Often it appears that alcohol may accentuate a person's characteristic mode of coping with his environment and social world. Efforts at objective behavioral analysis of drinking patterns are very new, and the findings are gradually dispelling some basic misconceptions about alcoholism derived from retrospective, self-report data from the sober alcoholic.

The Alcohol-Withdrawal Syndromes

Hippocrates was the first to report an association between alcohol abuse and tremulousness, delirium, and seizure disorders. However, it was not until the late eighteenth and early nineteenth century that the alcohol-abstinence syndrome was accurately described in the medical literature. Despite a long history of clinical observation of the association between cessation of drinking and signs and symptoms characterizing the withdrawal state, the basis of the alcohol-abstinence syndrome was not determined until the mid twentieth century. Studies carried out by Victor and Adams in 1953 provided the first careful clinical documentation of the alcohol-withdrawal states and an accurate classification of the withdrawal syndromes. Based upon these observations three unique, but not mutually exclusive withdrawal states were differentiated: the tremulous syndrome, alcohol-related seizure disorders, and delirium tremens.

The critical determinants of the onset of withdrawal symptoms are unclear, since either a relative decrease in blood-alcohol levels *or* the abrupt cessation of drinking may precipitate the syndrome. The severity and duration of withdrawal symptoms also do not appear to be directly related either to the volume of alcohol consumed or to the duration of a drinking spree. It appears that the pattern of drinking may be more important than the duration of drinking in accounting for the expression of the alcohol-abstinence syndrome. (See also p. 375 and pp. 380-381.)

Tremulousness is the most common withdrawal sign observed following cessation of drinking. Tremulousness may be relatively mild with involvement of only the distal upper extremities, or it may be severe, involving upper and lower extremities as well as tongue and trunk. The onset of tremulousness may occur as early as six hours following cessation of alcohol intake but the peak of intensity of the syndrome usually occurs after 24 to 48 hours of abstinence. Tremor is usually relatively coarse and may be exaggerated or exacerbated when the patient is asked to extend his arms with palms either separated or pronated. Duration of the tremulous state rarely extends beyond seventy-two hours following cessation of drinking but in some cases it may last as long as four or five days.

Tremulousness is often associated with a subjective sensation of moderate to severe anxiety. Hallucinations may also accompany the acute

alcohol withdrawal state. Acute alcoholic hallucinosis is usually auditory and there is little support for the notion that these hallucinatory events are related to an underlying schizophrenia. It is also important to emphasize that hallucinosis is not necessarily an index of the severity of the abstinence syndrome. Recent data, obtained under experimental research ward conditions, have shown that hallucinations may occur when alcohol addicts are severely intoxicated as well as following cessation of drinking.

Seizure disorders due to alcohol withdrawal are often associated with antecedent and consequent states of tremulousness, although isolated seizure disorders without tremulousness do occur. Seizure disorders associated with alcohol withdrawal may occur as early as several hours following the last intake of alcohol, but the peak incidence of this syndrome is usually twelve to twenty-four hours following cessation of drinking. Seizures are usually grand mal in nature and are rarely preceded by auras but usually followed by a post-ictal state. Seizures may occur during alcohol withdrawal in patients who have no other evidence of neurological disease and have normal EEG's during sobriety. The occurrence of seizure disorders may herald subsequent development of overt delirium tremens. According to Victor and Adams, approximately one third of all patients who have seizure disorders during alcohol withdrawal eventually develop delirium tremens.

Seizure disorders are not unique to alcohol-withdrawal states and have

been observed following withdrawal of many centrally acting drugs. Seizure disorders are commonly seen during barbiturate withdrawal and following cessation of prolonged use of high dosages of meprobamate, chloral hydrate, and paraldehyde. In contrast, the heroin abstinence syndrome is rarely associated with seizure disorders. Although it has been suggested that seizure disorders observed during the alcohol abstinence state represent a latent form of epilepsy which is precipitated either by heavy alcohol use or withdrawal, there is no evidence to support this hypothesis. At present the underlying mechanisms which produce seizure states during alcohol withdrawal are not known.

The term “delirium tremens” is often erroneously used as a generic description of most alcohol-withdrawal states. However, delirium tremens is a distinct and specific disorder which occurs with relative infrequency. In contrast to the common alcohol-withdrawal syndrome, delirium tremens usually occurs late, i.e., seventy-two to ninety-six hours following cessation of drinking. It may be preceded by tremulousness and is often preceded by seizure disorders.

Delirium tremens is characterized by profound confusion, disorientation, delusional states, and hallucinatory episodes as well as motor and autonomic dysfunction. Confusional states and delirium are rarely observed in the common tremulousness syndrome of alcohol abstinence.

Hallucinations associated with delirium tremens tend to be visual rather than auditory. However, there is little empirical support for the popular notion of a "typical" hallucinatory pattern. Although frightening and dysphoric hallucinations may occur, comforting, pleasurable and euphoric hallucinations are also observed, and it is the sensory and perceptual richness of the hallucinatory event that is distinctive. Hallucinations associated with delirium tremens are distinguished from hallucinations of schizophrenia by the intense visual nonideational quality, increased frequency of occurrence at night, and reports of subjective intensification when the patients' eyes are closed.

In addition to disorders of the sensorium, patients with delirium tremens frequently show profuse sweating, tachycardia, hyper-reflexia, mild to severe tremulousness, hypertension, and fever. Intercurrent illness, particularly pulmonary and gastrointestinal disorders, are also common in these patients. Delirium tremens is a potentially lethal condition in contrast to the relatively benign common tremulous syndrome.

In the process of establishing a differential diagnosis between acute alcoholic tremulousness, seizure disorders, and delirium tremens, it is essential to consider the time course as well as the presence or absence of confusion, disorientation, and autonomic dysfunction. It is also important to remember that alcohol withdrawal is not an all-or-none phenomenon. During

the course of chronic drinking, alcohol addicts may experience wide fluctuations in blood alcohol levels as a function of periodicity of alcohol intake plus alterations in the rate of absorption and metabolism of alcohol. A number of clinical and laboratory studies have demonstrated that onset of the withdrawal syndrome, particularly tremulousness, may occur when alcoholics are consuming ethanol, but have a relative decrease in their blood-alcohol levels. It is impossible to arbitrarily define a critical blood alcohol level for any given individual which either initiates or suppresses the withdrawal state. For example, alcohol addicts who have blood alcohol levels of 300 mg./100 ml. may experience severe abstinence syndromes when their blood alcohol levels fall 100 or only 50 mg./100 ml.

In addition to symptoms which appear to be directly related to alcohol (or partial alcohol) withdrawal, a number of associated disorders may contribute to the severity and duration of the withdrawal syndromes. Alcohol addicts may have disturbances in acid-base, water, and electrolyte balance during alcohol withdrawal. Hyperventilation is frequently observed in the early hours following cessation of drinking and this phenomenon may be associated with induction of respiratory alkalosis and elevated blood pH. Hypomagnecemia may also be found in patients in alcohol withdrawal.

Low serum magnesium levels are probably the result of two factors: poor dietary intake and malabsorption of magnesium associated with heavy

drinking, and a shift of magnesium from the intravascular to intracellular fluid compartments. Although a state of dehydration may be present in some patients during withdrawal, overhydration may also be a problem.

Somatic Disorders

Prolonged alcohol abuse has been shown to have toxic effects on a number of organ systems as well as secondary effects on metabolic processes. Since the liver is the major drug-metabolizing organ in the body, perhaps it is not surprising that *liver damage* is both the earliest and most profound consequence of excessive alcohol use. Alcohol has been shown to induce a transient development of fatty liver in normal drinkers in as little as two days. In chronic alcoholism, there may be a temporal progression from alcohol-induced fatty liver to alcoholic hepatitis or cirrhosis, a potentially fatal condition. The extent to which alcoholic hepatitis, characterized by extensive necrosis and inflammation, may in turn initiate scarring, fibrosis, and finally cirrhosis is unclear. Moreover, although excessive alcohol use almost invariably induces fatty liver, not all alcoholics develop cirrhosis. Recent data suggest that the development of alcohol-induced hyperlipidemia may be related to genetic factors, since alcoholics with primary hypertriglyceridemia develop striking lipid abnormalities during experimentally induced chronic intoxication. Normal drinkers with primary hypertriglyceridemia also develop marked increases in triglyceride levels after acute administration of

low doses of alcohol.

Gastrointestinal disorders associated with alcohol abuse are very common. Gastritis and pancreatitis are probably the most frequently encountered gastrointestinal disorders in alcohol abusers. *Gastritis* may progress to gastric or duodenal ulcers which, in turn, may lead to potentially fatal gastrointestinal bleeding. *Pancreatitis* is also a potentially lethal consequence of alcohol ingestion. The mechanism of pancreatitis production in relation to alcoholism is unknown. Excessive alcohol use may also interfere with the absorption of essential nutrients and vitamins from the small intestine into the blood stream. The nutritional malabsorption syndrome, so frequently observed secondary to alcoholism, may also contribute to the compromised nutritional status often seen in the alcohol addict.

The neurological disorders frequently associated with alcohol addiction are also related to malabsorption of critical nutrients. The most frequently observed disorder of the peripheral nervous system in alcoholics usually involves motor and sensory nerves in the arms and legs. These *peripheral neuropathies* are characterized by pain, impaired movement and coordination, and eventually by muscle wasting. This condition is usually reversible with an adequate diet and cessation of drinking.

Disorders of the *central nervous system* may also be associated with

chronic alcohol abuse. Until recently, it has not been clear whether these disorders were caused by the direct action of alcohol or a combination of alcohol abuse and poor nutrition. It is now generally believed that nutritional deficiencies are the most important etiological factor in most neurological diseases associated with chronic alcoholism. The incidence of CNS diseases associated with nutritional deficiency and alcohol abuse is very low. Even in 1953, neurological disease in hospitalized alcoholics ranged only between 1 and 3 percent. There has been a persistent mistaken impression that certain CNS diseases are uniquely associated with alcoholism. Table 15-1 summarizes some other conditions in which CNS diseases traditionally associated with alcoholism are also seen. The clinical neurology and neuropathology associated with amblyopia, cerebellar cortical degeneration, central pontine myelinolysis, myelopathy, Marchiafava-Bignami disease, and Wernicke-Korsakoff syndrome has recently been reviewed by Dreyfus.

The latter two syndromes require some special emphasis because of their historical association with the condition of alcoholism. Marchiafava-Bignami disease is a rare condition of unknown origin, which presents a clinical picture rather similar to delirium tremens, and is characterized by demyelination of the central portion of the corpus collasum. Only about sixty-four cases have been reported in the world literature. The disorder was once thought to occur only in Italian males who drank large amounts of crude red wine. This syndrome has subsequently been observed in non-Italian alcoholic

patients after abuse of various types of alcohol. The pathological changes associated with Marchiafava-Bignami disease have also been found in patients with Wernicke-Korsakoff syndrome.

Table 15-1. Disorders of the Central Nervous System Associated with Alcoholism and Other Specific Conditions[4]

	DISEASES OF THE NERVOUS SYSTEM				
ASSOCIATED CONDITIONS	WERNICKE-KORSAKOFF SYNDROME	AMBLYOPIA	CEREBELLAR CORTICAL DEGENERATION	CENTRAL PONTINE MYELINOLYSIS	MYELOPATHY
Liver Disease					✓133, 192
Thyro Toxicosis	✓28				
Pernicious Vomiting of Pregnancy	✓18				
Strachan's Disease					✓35
Nutritional Factors					
Chronic Malnutrition		✓35,76,153			

Chronic Malnutrition Associated with Disease			✓6, 17,80, 98,151	
Nutritional Depletion			✓94	
Thiamine Deficiencies	✓171			
Vitamin B Deficiencies	✓171	✓54		
Pellagra				✓157
		Treatment Variables		
Chronic Hemodialysis	✓89			
Isonicotinic Acid Hydrazide		✓73		

Wernicke's syndrome is an encephalopathy frequently associated with alcohol abuse. Prolonged alcohol intake, even in the presence of an adequate diet, may result in impairment of eye muscle control due to nerve dysfunction. Although this condition is usually benign and readily reversible following cessation of alcohol intake, it may herald a potentially serious

disorder which could lead to a permanent incapacity. This more serious disorder has been termed Wernicke's disease and Korsakoff's psychosis and is characterized by opthalmoplegia, ataxia, weakness, profound disorders of memory and deranged process of thinking.

This syndrome results primarily from vitamin deficiency and, in particular, a deficiency of thiamin. Alcohol abusers are at a high risk for the development of vitamin deficiencies because they eat poorly and alcohol may inhibit absorption and transport of vitamins from the GI (gastrointestinal) tract. Remission of opthalmoplegia usually occurs promptly after parenteral administration of vitamin B complex. Ataxia and confusional states clear more slowly. The prolonged derangements of memory (Korsakoff's psychosis) are discussed under Disorders of Memory Function (p. 389).

The possible contribution of alcoholism to *heart* disease is, as yet, unknown. Alcohol is a myocardial depressant and may play some role in the development of cardiomyopathies. A comparison of clinically normal alcoholic patients with cardiomyopathy and normal groups showed abnormalities of cardiac function similar to but quantitatively less than in the cardiomyopathy group.

Less frequent but not uncommon conditions associated with chronic alcohol abuse include disturbances in *blood-cell* formation and anemia, *muscle*

disease, and enhanced susceptibility to infection. An interaction between poor nutrition and excessive drinking is probably most important in the genesis of these disorders. It is known that alcoholics have a very high incidence of tuberculosis and this infectious disease is most common in individuals who are debilitated and have poor dietary intake.

Affective Disorders and Psychosis

Depression as an antecedent or consequent phenomenon in patients with alcohol-abuse problems has been well documented in the clinical literature (see references). However, the causation of alcohol problems as unique sequela of affective disorders has never been substantiated. Some individuals with neurotic and psychotic affective disorders may abuse alcohol, but many individuals with these symptoms do not drink heavily and may even abstain from using alcoholic beverages. Although it is tempting to postulate that alcoholics drink to alleviate depression, there is some evidence that chronic drinking enhances depression. A number of studies have shown that depression and dysphoric states are precipitated by or perhaps increased during chronic intoxication. It is therefore important for the physician to recognize that depression is not a necessary "underlying" basis for alcohol abuse and to avoid simplistic formulations for "treatment of the depression" rather than employing a more comprehensive therapeutic approach.

Alcoholism is a common finding in individuals who attempt or commit suicide. It has been estimated that one third of all reported suicides are associated with chronic alcohol abuse. However, the available statistics indicate that this figure may be valid only for white middle-aged males. Suicide rates appear to be relatively low for black males over thirty-five, although problem drinking in this group is more likely to occur at an earlier age than for white males. Until more comprehensive data are available, the precise interaction between alcoholism and suicide will remain obscure. On the other hand, there is very good evidence that alcohol consumption frequently occurs prior to suicidal behavior, since about 25 percent of suicide victims have detectable amounts of alcohol in tissue on necropsy examination. It is tempting to postulate that this finding supports the observation that alcohol may enhance dysphoric mood states. Since there are no data available concerning the number of individuals who contemplate suicide, drink, but do not attempt the act, or who are even dissuaded from suicide as a consequence of drinking, the relationship between suicidal behavior and drinking is unclear.

The causal relationship often assumed to exist between alcoholism and a number of psychiatric states is also tenuous at best. The “alcoholic paranoid state” has been listed in the Diagnostic and Statistical Manual (DSM-II), published in 1968 by the American Psychiatric Association. There is no evidence that this behavior disorder is a unique psychosis caused or even

aggravated by drinking. An examination of the clinical literature reveals no convincing data which demonstrate either acquisition or remission of a psychiatric disorder as a function of alcohol intake. However, it is not unlikely that patients may initiate drinking or abuse alcohol as one of a series of behaviors associated with recrudescence of a psychotic episode. Assignment of a causal rather than an associational relationship between alcohol abuse and psychotic disorders is naive and impedes development of the basis for a comprehensive differential diagnosis.

"Pathological intoxication" is a term often found in the literature and it is most commonly characterized as uncontrolled and usually violent aggressive behavior and rage following alcohol intake. Rage states have been associated with even very small volume alcohol intake, and a causal basis has been assigned to alcohol in producing the disordered behavior. No such causal basis has been verified in controlled studies, nor has an association between blood alcohol levels, abnormal brain-wave activity, and violence been experimentally validated. As noted previously, some patients may exhibit increased aggressive behavior when intoxicated. However other individuals may become more friendly, while others show no change in behavior. So-called "pathological intoxication" is probably more closely related to host and environmental determinants than to a specific pharmacological effect of alcohol in the CNS.

During recent years, it has been reported that a large number of new admissions to state mental hospitals have alcoholism as a primary diagnosis. Among those patients a significant number have “organic brain dysfunction” which has been linked to alcohol abuse. It should be kept in mind that these patients have a number of similar features in addition to a history of alcohol abuse, e.g., most are poor; most have a past history of marginal nutritional status; most have family backgrounds of low socioeconomic status and poor educational achievement. The apparent increase in the number of admissions of these patients does not necessarily reflect a true increase in prevalence of alcoholism. Rather it may reflect the increased willingness of state institutions to admit these patients as a consequence of decreased duration of hospitalization for other psychiatric patients who are more amenable to psychopharmacological interventions.

The entire category of the organic brain disorders is poorly understood. More rigorous and obsessive relabeling will not provide better insight into the causal determinants of the disorders of cognition, perception, and memory function. Much more knowledge is necessary before the role of alcohol abuse, alone or in combination with other disorders of behavior, can be determined in the genesis and progression of deranged intellectual function and aberrant thought processes.

Disorders-of-Memory Function

Excessive alcohol use has been associated with several types of impaired memory function. These range from a severe persistent amnestic disorder, Wernicke's Korsakoff syndrome, to the transient total memory loss which may accompany heavy intoxication, i.e., the alcoholic "blackout" to the fragmentary absence of recall of events during drinking which has been described as a dissociative state. The mechanisms which account for these alcohol-induced memory impairments are unknown.

Wernicke's Korsakoff syndrome is usually associated with pathological changes of the mammillary bodies and the medial thalamus and hypothalamus. The patient presents with anterograde and retrograde amnesia. Although memory of early life experiences is intact, there is usually an inability to recall more recent life circumstances. Patients are unable to acquire new information and often cannot recall, for example, the route to the hospital dining room, the food served at lunch, the name of their doctor, etc. The most comprehensive analysis of the memory deficits associated with the Wernicke's Korsakoff syndrome has been completed by Talland. The extent to which this complex memory disorder is a function of impairment in information storage, or in retrieval of that information is the subject of continuing study.

The alcoholic "blackout" is a general term used to describe a total loss of memory for events during an episode of heavy intoxication. In some

instances, intoxicated individuals may drive long distances, fight, be arrested, and subsequently awaken without any recollection of their activities for the past several hours or days. The incidence of “blackouts” is unknown. In a sample of one hundred alcoholics about two-thirds reported experiencing “blackouts”. The degree of intoxication does not reliably predict the occurrence of this profound memory loss. Moreover, alcohol-induced periods of amnesia may occur in moderate or heavy drinkers as well as in alcoholics. The frequency of blackout occurrence does appear to correlate with severity of alcoholism, a history of head trauma, malnutrition, and fatigue. It is unlikely that deliberate malingering often accounts for these clinical phenomena. Total loss of memory for significant events is a frightening experience for most patients and “blackouts” do not appear to have any motivational consequences for the maintenance of drinking behavior. The occurrence of blackouts is not necessarily correlated with a history of head trauma. No information is available to rule out the possibility that alcohol-induced blackouts reflect an underlying neuropathological process such as impaired temporal-lobe function which is accentuated by intoxication. Comparable reports of hours or days of amnesia are not a prominent feature of heroin or barbiturate intoxication.

It has been suggested that the “blackout” may be explained in terms of a disruption of “short-term” memory[5] consolidation by alcohol. It is argued that information lost to “short-term” memory would not be available subsequently

from “long-term” storage and so could account for a period of global memory loss. The validity of this hypothesis has been the subject of controversy. An alcohol-induced decrement in “short-term” memory function was observed in alcoholics with a clinical history of “blackouts” but not in alcoholics without such a history. However, these findings are inconclusive since no measures of memory function were taken during sobriety and, therefore, it is impossible to establish that memory function did not differ between these two groups before alcohol administration. Another report indicated that alcoholics with a clinical history of blackouts showed “short term” memory impairment during intoxication but not during sobriety. Since no comparison of the effect of alcohol on memory function in alcoholics *without* a clinical history of blackouts was made, the extent to which blackout history and alcohol may interact uniquely to affect memory function was not determined. A third study compared the performance of alcoholics with and without a clinical history of blackouts, during sobriety, and acute and chronic alcohol intoxication and found no impairment of short-term memory in either group under any condition. It appears that when alcoholic subjects are adequately motivated to perform a task, their behavioral tolerance for alcohol permits accurate performance, even at very high blood alcohol levels. Visual short-term memory can be disrupted by relatively low doses of alcohol in normal drinkers who lack behavioral tolerance. Data collection on alcoholics with no history of blackouts and rhesus monkeys also failed to reveal any direct effect

of alcohol on “short-term” memory.

A third form of memory dysfunction associated with alcohol intoxication is the fragmentary absence of recall of some events during a subsequent period of sobriety. In some instances, events during intoxication (e.g., hiding money or alcohol) are only recalled during a subsequent period of intoxication, and therefore fall into the framework of state-dependent effects. The observation that behavior learned during a drug state may not occur during a nondrug state, then reoccur during reinstitution of a drug state, has been demonstrated in experimental animals. Simply stated, responses established under specific conditions are most easily elicited once these conditions are re-established. State-dependent dissociation of recall for verbal materials learned during alcohol intoxication has also been shown in normal college students.

Clinically, the dissociative effect of alcohol is most often expressed by a failure to recall aversive consequences of drinking (e.g., depression, anxiety, illness) during a subsequent period of sobriety. Alcohol addicts form positive expectancies about a forthcoming drinking experience, and, following a period of chronic intoxication, they tend to recall their experience in terms of the predrinking expectancy, rather than the events that actually transpired. The implications of this dissociative phenomena for treatment of the alcoholic were first noted in 1962 by Diethelm and Barr. Patients interviewed under

conditions of acute intoxication talked more freely and described emotions, especially guilt and hostility, which they had not discussed during sobriety. Usually these patients forgot the content of these interviews once they became sober again. Traditionally, physicians have tended to refuse treatment to intoxicated alcoholics. A reevaluation of this principle and an effort to assist patients in integrating feelings expressed during intoxication with self-perception during sobriety could result in more effective psychotherapy for alcoholic individuals.

Sleep Disturbances

There is a prevailing impression that alcohol facilitates sleep. Although this notion is consistent with the usual classification of alcohol as a CNS depressant, it is not supported by electroencephalographic analysis of the effects of alcohol on sleep. It has been generally agreed that "poor sleep" is characterized by a decrease in REM activity and a decrease in stage 4 sleep. It is now well established that acute administration of alcohol does produce a relative suppression of REM activity in normal individuals, alcohol addicts, and experimental animals. The effect of alcohol on stage 4 sleep is a controversial issue, but it is agreed that stage 4 is decreased during alcohol withdrawal. Sleep of alcoholics is generally light and somewhat fragmented. Such sleep disturbances and insomnia seem to be characteristic symptoms of chronic alcoholism.

There has been considerable interest in the possibility that hallucinations often seen in alcohol addicts during alcohol withdrawal may reflect a REM "overshoot" following a period of alcohol-induced REM suppression. Hughlings Jackson and William James were among the first to suggest the possibility of a neuro-physiological continuity between dreaming sleep and waking hallucinatory activity. Hallucinosis is sometimes associated with difficulty in discriminating between sleeping and waking states. The idea that REM sleep and dreams are isomorphic is no longer accepted since dreamlike activity has also been reported during non-REM awakenings. However, the hypothesis that REM suppression as a function of chronic alcohol intoxication may facilitate the eruption of hallucinations during withdrawal has received confirmation in several studies of alcohol addicts during a period of *acute* alcohol withdrawal. Interpretation of reports of EEG correlates of sleep activity during alcohol withdrawal must be made with caution since it has usually been impossible to obtain adequate baseline sleep measures. Consequently, the high levels of REM activity observed may reflect a combined effect of acute hospitalization and alcohol withdrawal. It is seldom possible to determine the number of hours since the last drink with any degree of accuracy in acute hospital admissions, and this can confuse differential diagnosis of alcoholic hallucinosis versus delirium tremens (see discussion on p. 385).

A recent study of alcohol addicts before, during, and after a period of

chronic alcohol consumption did not reveal a consistent REM hyperactivity during alcohol withdrawal. Although hallucinations were frequently associated with an antecedent REM suppression or insomnia, there were no invariant relationships between hallucinosis and REM activity. Also, hallucinations were reported during intoxication as well as during alcohol withdrawal. There were no consistent dose-response relationships between alcohol intake and the subjective intensity of reported dreams or hallucinations. It may be somewhat premature to assume an invariant association between dream-hallucinatory episodes and REM activity, and the drug-suppression-withdrawal overshoot REM hypothesis is currently being reevaluated in several laboratories. Attempts to discern relationships between the presumed physiological accompaniments of the behavioral expressions of intoxication and withdrawal are necessarily restricted by limitations inherent in existing techniques, and the notion of a physiology of dreaming remains a provocative but hypothetical construct.

Treatment of Acute and Long-Term Alcohol Problems

Treatment for the alcohol-related disorders will be described separately for the acute effects of alcohol intoxication and withdrawal and the chronic phase of the illness. The implications of problems in establishing outcome criteria for current treatment approaches will also be considered in the discussion of treatment evaluation and prevention.

The Treatment Setting

Most physicians treat patients with alcohol-related problems in two clinical situations, each of which involves substantially different problems. In hospital practice, patients are rarely seeking assistance for alcohol problems per se, but are usually under treatment for intercurrent illness associated with alcohol abuse. Therapy is often directed towards solving medical problems of acute illness, with minimal attention to the underlying alcohol problem. Many such patients return to problem drinking once they are discharged from the hospital and then are frequently readmitted with identical or similar disorders. This unfortunate recidivism often promotes an attitude of despair in the patient and disdain in the physician and other hospital personnel.

In office practice, despair and disdain are fostered by other factors.

Patients seeking aid in this situation generally initiate therapy because of some degree of external coercion. Such coercion may range from threats of an employer to terminate employment to threats of a spouse to end a marriage unless the patient seeks and obtains assistance. In this situation, the patient is usually both frightened and angry and the sum of both conditions is often interpreted by the physician as evidence of lack of motivation to do something about his or her drinking problem. Motivational factors have become so emphasized in diagnosis and therapy that they have frequently been assigned predictive value in determining efficacy of treatment. It is therefore possible for a physician to prematurely assume a poor prognosis for a patient who appears "poorly motivated" and then to employ a "poor-motivation" vs. "good-motivation" dichotomy to account for either success or failure of the treatment provided. Since motivational states are rarely static in patients treated for any disorder, it is obvious that this criterion is not of great value.

Differential Diagnosis

Although there have been significant advances in public perception of alcoholism as a disease rather than evidence of "moral weakness", a severe stigma continues to be associated with this disorder. In most instances, patients with alcohol-related illness recognize this stigma and are reluctant to fully discuss the duration or severity of their drinking problems. Successful

case finding and elucidation of past and current problem history taking involve techniques which are common to all psychiatric interviewing procedures. Since these are discussed in other portions of this *Handbook* (see Volume 1, Chapters 53 and 54), they will not be repeated here.

Much attention has been paid to the role of attitudes and values held by physicians in determining their diagnostic approach to patients with alcohol problems. Similar contingencies probably apply to all categories of mental disorders and perpetuation of emphasis on the importance of this issue provides a rationale for accepting or rejecting patients. At the present time, there are no data which specify the optimal qualities, attitudes and approaches in the treating physician as a determinant of treatment outcome.

The process of differential diagnosis for alcohol related problems requires a conceptual approach which has been employed by physicians for many decades in general medicine. In psychiatric practice, this approach often suffers because of lack of basic information about causation and natural history of mental illness. An attempt has recently been made to systematize the diagnostic criteria for alcohol abuse and alcoholism. These criteria include behavioral, physiological, and attitudinal factors, with particular attention to the major illnesses associated with alcoholism and the related patterns of clinical laboratory test abnormalities. Although this system is imperfect, it represents a considerable advance which deserves attention and critical

appraisal by physicians.

Treatment of Alcohol Intoxication

There are no effective, readily available means of rapidly reducing the blood-alcohol concentration of a severely intoxicated individual. Although the rate of ethanol metabolism can be increased by fructose administration, this technique has found little successful clinical application since its discovery thirty-five years ago. Recent explorations of hemodialysis procedures to rapidly reduce blood-alcohol concentrations have limited general applicability because of their expense and potential risk of infection.

Fatal alcohol poisoning is very rare. There are occasional reports of children or adolescents who die of respiratory depression following an overdose of alcohol. However, in view of the many people who drink and the large volumes of alcohol consumed, it must be concluded that alcohol is a relatively safe drug in comparison to the opiate narcotics. It is virtually impossible to drink an acutely toxic amount of alcohol before vomiting or unconsciousness occurs.

The comparative safety of alcohol may also be related to its dual properties as a drug and a food. Alcohol does contain calories and is metabolized like other carbohydrates. The principal enzyme responsible for alcohol metabolism, alcohol dehydrogenase, is ubiquitous in body organs and

most highly concentrated in liver. Consequently, the lethal toxic potential of alcohol is counteracted by nature's provision for its rapid degradation.

The recent increase in polydrug abuse requires added caution in treatment of acute intoxication. The concurrent use of several drugs which may act synergistically can result in overdose. Improved techniques for determining the blood concentrations of, e.g., heroin and barbiturates as well as ethanol, can aid the physician in accurate diagnosis. Fast acting pharmacological antagonists are currently available for heroin but not for barbiturates or alcohol.

Since acute intoxication in the chronic inebriate is usually complicated by other medical problems (see pp. 385-388) it is essential that the care of afflicted individuals occur in the context of good medical management. Until recently, it has been difficult, especially for impoverished alcoholics, to obtain adequate medical treatment for the acute effects of alcohol. However, it is likely that most of these patients are admitted to hospitals for treatment of acute intoxication under the guise of some other diagnostic criteria.

Following the recent change in the legal status of intoxication and alcoholism in 1966, a number of detoxification centers were established to treat the acute inebriate. While these facilities do provide some resource for patient care, they were seldom established within the mainstream of medical

care. Consequently, there is great danger that these centers may become nothing more than a respectable version of the traditional "drying-out" facilities, i.e., the jail or the drunk tank.

Treatment of Alcohol Withdrawal

Rather good progress has been made by biomedical scientists in devising new methods for the treatment of the alcohol withdrawal syndrome. Improvements in treatment have occurred primarily because of general advances in medicine which provide more accurate diagnosis and better patient management, i.e., treatment of metabolic disturbances and infections which frequently accompany the abstinence syndrome. Less than twenty years ago, the mortality associated with delirium tremens was reported as about 15 percent in various hospitals and institutions. Today, the incidence of death associated with delirium tremens has fallen to less than 1 percent.

The development of new psychopharmacological agents, particularly the minor tranquilizers, has provided a means of mildly sedating patients and reducing severity of agitation and tremor without compromising the patient's ability to eat well and receive other medical care. Chlordiazepoxide (Librium) has been reported to be an effective anticonvulsant in the treatment of withdrawal seizures (see also pp. 383-385).

Treatment of Alcoholism

At present, there is no specific and uniformly efficacious treatment either for the disease of alcoholism or for problem drinking. The treatment techniques that have been used include individual and group psychotherapy, Alcoholics Anonymous, aversive conditioning therapies, Antabuse, vitamin therapies, and LSD treatment, singly or in various combinations. In the few relatively controlled therapy evaluation studies, the rate of improvement or alcohol abstinence following therapy was very low. Since the spontaneous recovery rate for alcoholics has been estimated at about 20 percent, the efficacy of the existing therapies is discouragingly low. These figures compare rather poorly with the improvement rate of heroin addicts treated with methadone, an estimated 70 percent.

The complexities and difficulties involved in treating the chronic alcohol abuser have been thoroughly reviewed by many concerned investigators. Since all variants of alcoholism are multiply determined, the treatment of alcohol problems presents the challenge of any complex behavioral disorder. The dynamic conceptualizations of an “alcoholic personality” have received no empirical support, and there is considerable heterogeneity on many dimensions, even among the end stage, “skid-row” alcoholics. Although there is no evidence to indicate that alcoholism is invariably associated with a predisposing psychiatric illness, the development of problem drinking rarely

occurs in isolation from emotional, interpersonal, and job-related problems. Whatever its origins, alcoholism is characterized by a vast diversity of related difficulties.

The treatment of alcoholism is further complicated by the fact that most people with alcohol problems tend to deny the reality of their illness and to reject treatment. It has been shown that patient acceptance of treatment can be greatly improved if initial hospital contacts are sympathetic and positive. However, physicians have tended to reject alcoholic patients and to avoid diagnosing the problem unless it was glaringly apparent in the terminal phase. Frustration with relapsing patients who deny the significance of their alcohol problems, and limitations of available treatments have contributed to physicians' negative attitudes. The point has often been made that treatment goals for the alcoholic should have limited objectives and a multimodality therapy suited to the needs of the individual and his resources should be offered. The logic of this position is obvious and can be extended to argue for treatment of the alcoholic within the mainstream of medical practice, where the greatest range of medical, psychiatric, and social services is potentially available. However, the question of which treatment will most benefit the patient with alcohol problems remains unanswered. Until there is a better understanding of the disease process of alcoholism, and better treatments available, it is unlikely that the incidence of alcoholism will be greatly reduced, despite the recent improvements in the delivery of health-care

services to alcoholics.

The low success rate of current treatment approaches seems to point to the need for an effective pharmacotherapy for alcohol addiction. The recent advances in the treatment of heroin addiction have occurred largely because of the availability of blocking agents or antagonists. However, the use of blocking agents for heroin addiction has been criticized because these agents have high addictive potential. An ideal blocking agent for any centrally acting drug of abuse, including alcohol, would have the following characteristics: (1) low addictive potency; (2) little or no synergistic action with other drugs; (3) no central nervous depression or excitatory effects. In essence, the drug of choice for treating alcoholics would be more closely analogous to a narcotic antagonist than a blocking agent. It should be emphasized that the rationale for the use of blocking agents which reduce the subjective effect of a drug is very different from that for the use of Antabuse which produces severe discomfort, and potentially lethal consequences if taken in combination with alcohol.

In view of the wide spectrum of alcohol-related problems, drug therapy alone would not suffice. A variety of other psychological and social interventions would probably be necessary to produce the greatest change in alcoholics with diverse behavioral, biological, and social problems. However, an effective drug therapy would permit other types of therapeutic

intervention to occur under conditions where confounding effects of perpetuation of alcohol intake were significantly reduced.

Treatment Evaluation

One of the most important and frequently ignored issues related to the treatment of alcoholism is the problem of evaluation. Unless there are adequate evaluation and follow-up data, it is not possible to demonstrate the efficacy of any treatment approach. One fundamental issue in evaluation is the establishment of valid outcome criteria. The establishment of comprehensive criteria of efficacy which can be uniformly applied to all treatment programs is critical for adequate evaluation. There has been no consensus that the traditional goal of absolute abstinence is the optimal therapeutic goal for the alcoholic. Indeed, it appears that for many alcohol abusers, giving up drinking completely can also result in severe social and psychological dysfunction. There is accumulating evidence that some alcohol addicts may be able to return to social drinking. The persistent rationale for the treatment criterion of absolute abstinence is based on the erroneous concept of "craving", which is discussed under Psychological Dependence, p. 382.

Once outcome criteria have been formulated, construction of an adequate clinical research design assumes paramount importance.

Traditionally, it has been argued that clinical research cannot yield "hard data" because of the complexity of the dependent variables, the difficulties in controlling relevant factors as well as ethical constraints. It is curious that the expectancy for objectivity, sophistication, and accuracy in basic research has not been extended to treatment research. A lack of these qualities in the laboratory would have severe consequences for research development, and it could be argued that casual evaluation of treatment programs could have even more profound consequences for longevity and quality of human life. It does not seem unreasonable to require an even more precise specification of methods and outcome criteria for treatment programs which involve human beings than for isolated physical and chemical studies involving *in vitro* biochemical constituents. Enthusiastic testimonials by proponents of a particular treatment approach are too often substituted for adequate data. Awareness of the difficulties attendant on clinical treatment research should not constitute an excuse for neglecting or evading basic tenets of experimental design. A lucid summary and discussion of basic requirements for the design of clinical research has recently been prepared by Ludwig.

Prevention

There is little question that the best treatment for any disorder is prevention, and significant emphasis has been placed on public education concerning use hazards in virtually all drug-abuse areas. Evaluation of the

impact of such public education and prevention efforts is extremely difficult. The problem is complicated by the lack of good data on the incidence and prevalence of alcohol abuse and alcoholism, and the many difficulties associated with adequate case finding (see discussion on pp. 374-376). In the absence of firm incidence data, it is difficult to demonstrate conclusively that public-education programs or attempts to shape attitudes have had a significant impact on alcohol-abuse problems. Some preliminary evidence suggests that naive programs of public education and attitude shaping may sometimes prompt exploration and thereby increase the incidence of drug-abuse-related problems. Unfortunately, problem drinking usually occurs in situations where behavior is not determined by logical thinking, but rather by internal and external stress-contingent factors which are not highly amenable to rational persuasion. People with well-established alcohol related problems may be unresponsive to reminders that a certain pattern of drinking can be dangerous to their health. There is probably no substitute for the early development of responsible attitudes about alcohol use and awareness of the nature of alcohol problems.

Prevention techniques designed to change attitudes are quite distinct from coercive efforts to control distribution of alcohol or to prevent some individuals from drinking through imposition of age limits, etc. Attempts to control alcoholism through prohibition were a dramatic and unequivocal failure. There has been little systematic study of the effects of increased

taxation or restricted hours for bars and liquor stores in areas where these techniques have been applied. It is often argued that consumption of low-alcohol-content beverages such as beer may prevent alcoholism. This argument is somewhat misleading in that consumption of large enough quantities of a 6-percent alcohol beverage can yield an alcohol intake equivalent to that of the distilled spirits drinker. Physical dependence upon alcohol has been seen in individuals who consume large quantities of beer and wine.

Until there is a better understanding of the many factors which contribute to the development and maintenance of alcohol abuse, it will be difficult to formulate more effective approaches to the prevention and treatment of alcoholism.

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Notes

1 Portions of this chapter are taken from an administrative report on Alcohol Use and Alcoholism prepared for the Special Action Office for Drug Abuse Prevention of the Executive Office of the President by the senior author.

2 Recent reviews of the legal status of intoxication and alcoholism may be found in Chapter 7 of Legal Issues in Alcoholism and Alcohol Usage

3 The effects of alcohol intoxication on memory function and sleep will be discussed under Clinical Disorders.

4 This material is abstracted from Dreyfus. Numbers refer to bibliographic entries.

5 Most concepts of memory function differentiate between a “short-term” registration phase and a “longterm” consolidation phase, with the implication that these are sequential processes required for subsequent information retrieval. A recurrent source of confusion in the short-term memory literature has been the inconsistency in definition of this term. Short-term memory has been variously defined as l sec., 5 sec., 1 min., 5 min., and 30 min.