Sri Lakshmi Narayana Institute of Medical Sciences

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Date:02.1.2019

From
DR.V.R Sridhar
Professor and Head,
Department of Psychiatry,
Sri Lakshmi narayana institute of medical sciences,
Bharath Institute of Higher Education and Research,
Chennai.

To
The Dean,
Sri Lakshmi narayana institute of medical sciences,
Bharath Institute of Higher Education and Research,
Chennai.

Sub: Permission to conduct value-added course: ALCOHOL DEPENDANCE AND DEADDICTION

Dear Sir,

With reference to the subject mentioned above, the department proposes to conduct a value-added course titled:**ALCOHOL DEPENDANCE AND DEADDICTION** on 1/03/2019 . We solicit your kind permission for the same.

Kind Regards

Dr.V.R. Sridhar

FOR THE USE OF DEANS OFFICE

Names of Committee members for evaluating the course:

The Dean: **Dr. Jayalakshmi**

The HOD: **Dr. Sridhar**

The Expert: **Dr. Arun Seetharaman.**The committee has discussed about the course and is approved.

Dean (Sign & Seal)

Dr. G. JAYALAKSHMI, BSC., MBBS., DTCD., M.D., DEAN
Sri Lakshmi Narayana Institute of Medical Sciences
Osudu, Agaram, Kudapakkam Post,
Villianur Commune, Puducherry - 605502.

Subject Expert (Sign & Seal)

Dr. ARUN SEETHARAMAN, MD.,
Reg. No: 91440
Associate Professor, Psychiatry
Sri Lakshmi Narayana Institute of Medical Sciences
Osudu, Kudapakkam, Puducherry-605 502.

HOD (Sign & Seal)

Dr. V. R. SRIDHAR, MD.,D.P.M.,
Reg. No: 30995
Professor & HOD, Psychiatry
Sri Lakshmi Narayana Institute of Medical Sciences
Osudu, Kudapakkam, Puducherry-605 502.

OFFICE OF THE DEAN

Sri Lakshmi Narayana Institute of Medical Sciences

OSUDU, AGARAM VILLAGE, VILLIANUR COMMUNE, KUDAPAKKAM POST,
PUDUCHERRY - 605 502.
[Recognised by Medical Council of India, Ministry of Health letter No. U/12012/249/2005-ME (P -II) dt. 11/07/2011]
[Affiliated to Bharath University, Chennai - TN]

Circular

15/.01/.2019

Sub: Organising Value-added Course: ALCOHOL DEPENDANCE AND DEADDICTION.

With reference to the above mentioned subject, it is to bring to your notice that Sri Lakshmi Narayana Institute of Medical Sciences, **Bharath Institute of Higher Education and Research** is organizing **"ALCOHOL DEPENDANCE AND DEADDICTION"**. The course content and registration form is enclosed below."

The application must reach the institution along with all the necessary documents as mentioned. The hard copy of the application should be sent to the institution by registered/ speed post only so as to reach on or before 30/01/2019. Applications received after the mentioned date shall not be entertained under any circumstances.

Dean

Dr. G. JAYALAKSHMI, BSC., MBBS., DTCD., M.D.,
DEAN
Sri Lakshmi Narayana Institute of Medical Sciences
Osudu, Agaram, Kudapakkam Post,
Villianur Commune, Puducherry - 605502.

Encl: Copy of Course content

Course Proposal

Course Title: **Alcohol dependence and de-addiction**

Course Objective:

Awareness on the importance of Alcohol use disorders
Awareness On The Contributing Factors To Alcohol abuse and Bio-psychosocial Model Of Approach
Identify, classify and diagnostic criteria and management of Alcohol dependance

Course Outcome:

Course Audience: FINAL YEAR STUDENTS of 2019 Batch
Course Coordinator: Dr.V.R. Shridhar
Course Faculties with Qualification and Designation:
1.Dr.V.R.SHRIDHAR, Professor & HOD
2.Dr.Arun, Assistant Professor
Course Curriculum/Topics with schedule (Min of 30 hours)

SlNo	Date	Topic	Resource persons	Time	Hours
1.	14.05.2019	SUBSTANCE USE -ALCOHOL USE	Dr.Arun	4-5p.m	1
2.	15.05.2019	INTRODUCTION	Dr.Arun	2-3p.m	1
3.	16.05.2019	PREVALENCE	Dr.Arun	4-6p.m	2
4.	18.05.2019	NEUROBIOLOGY AND NEURAL CIRCUITS	Dr.Arun	4-6p.m	2
5.	19.05.2019	BIOPSYCHOSOCIAL APPROACH	Dr.Arun	4-6p.m	2
6.	20.05.2019	CLINICAL SUBTYPES OF ALCOHOL USE	Dr.Arun	4-5p.m	2
7.	21.05.2019	ALCOHOL DEPENDANCE	Dr.Arun	4-5P.M	1
8.	22.05.2019	ALCOHOL WITHDRAWAL	Dr.Arun	4-5p.m	1
9.	23.05.2019	ALCOHOL AND CO-MORBIDITIES	Dr. Shridhar	4-5p.m	1
10.	27.05.2019	ALCOHOL AND PHYSICAL CONDITIONS	Dr.Arun	4-6p.m	2
11.	28.05.2019	MANAGEMENT	Dr.Arun	4-6p.m	1
12.	29.05.2019	REHBALITATION AND GROUPS	Dr.Arun	4-6p.m	2
13.	30.05.2019	Pre course and Post Course evaluation, Feedback analysis from Likert scale	Dr.Arun	2-5p.m	3

		Practical Class I	Dr. Shridhar		
13.	02.06.2019	History Taking	Dr. Shridhar	2-3 PM	1
14.	03.06.2019	Mental Status Examination	Dr. Shridhar	2-3 PM	1
15.	04.06.2019	Alcohol Breath Analyzer-Demo	Dr. Shridhar	2-4 PM	2
16.	05.06.2019	Alcohol Withdrawal Case Presentation	Dr. Shridhar	2-4 PM	2
17.	06.06.2019	Alcoholic Anonymous	Dr. Shridhar	2-4p.m	2
		Total			30 hrs

REFERENCE BOOKS:

1.Comprehensive textbook of PSYCHIATRY by Kaplan and Saddocks.

2. OXFORD TEXTBOOK OF PSYCHIATRY

3. DSM 5 AND ICD 10

4.SYNOPSIS OF PSYCHIATRY BY PSYCHIATRY

VALUE ADDED COURSE

1. Name of the programme & Code

Alcohol dependence and de-addiction

2. Duration & Period

April –June 2019

3. Information Brochure and Course Content of Value Added Courses

Enclosed as Annexure- I

4. List of students enrolled

Enclosed as Annexure- II

5. Assessment procedures:

Alcohol dependence and de-addiction - *Enclosed as Annexure- III*

6. Certificate model

Enclosed as Annexure- IV

7. No. of times offered during the same year:

1 Time - April –June 2019

8. Year of discontinuation: 2019

9. Summary report of each program year-wise

Value Added Course- APRIL –JUNE 2019					
Sl. No	**Course Code**	**Course Name**	**Resource Persons**	**Target Students**	**Strength & Year**
1	PSYC09	**Alcohol dependence and de-addiction**	Dr. SRIDHAR Dr. Arun	FINAL YEAR	15 students APRIL - JUNE 2019

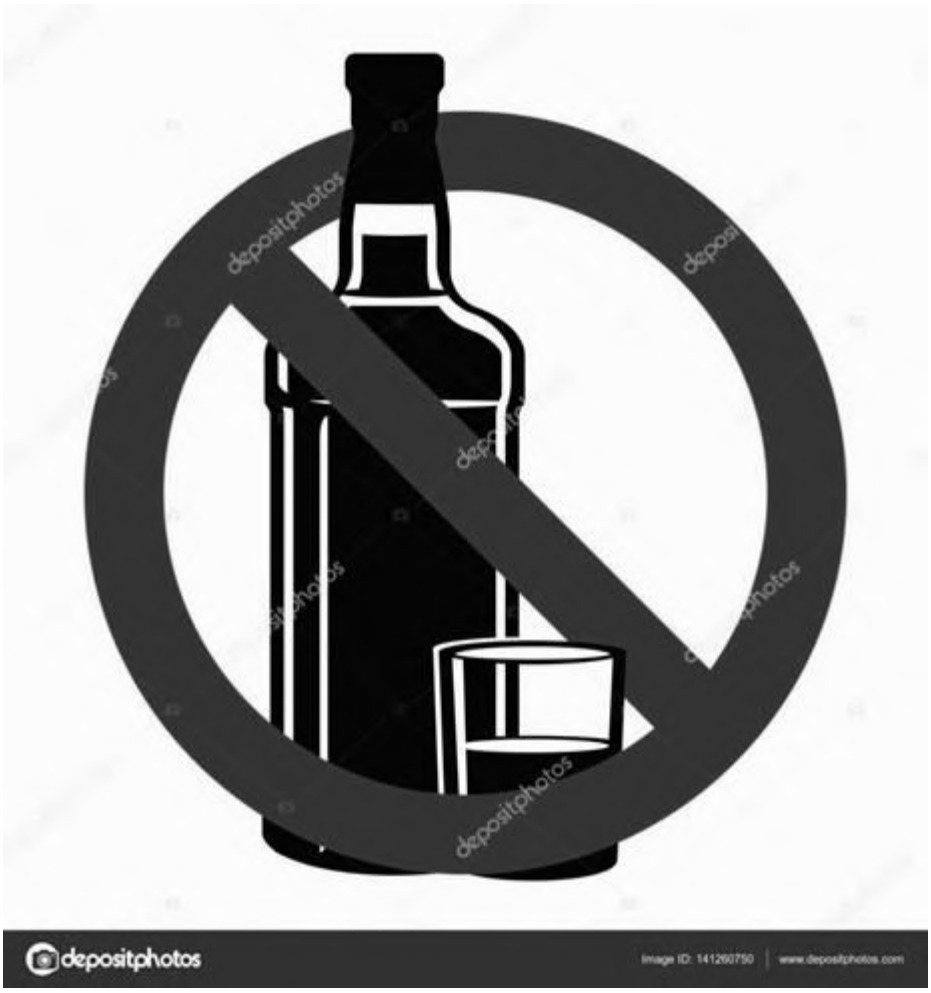
10. Course Feed Back

Enclosed as Annexure- V

RESOURCE PERSON
1. **Dr..SRIDHAR**
2. **Dr.Arun**

COORDINATOR
Dr.G. Jayalakshmi

ALCOHOL DEADDICTION



PARTICIPANT HAND BOOK

BIHER SLIMS

COURSE DETAILS

Particulars	**Description**
Course Title	Alcohol deaddiction
Course Code	PSYC09
Objective	1. Introduction 2. Prevalence 3. Diagnostic Criteria 4. Subtypes 5. Co-Morbidities 6. Dependence 7. Intoxication 8. Withdrawal 9. Delirium 10. Treatment And Rehabilitation 11. Alcoholic Anonymous
Further learning opportunities	Alcohol deaddiction
Key Competencies	On successful completion of the course the students will have knowledge on deaddiction
Target Student	Final year MBBS Students
Duration	30hrs Every February – May 2019
Theory Session	10hrs
Practical Session	20hrs
Assessment Procedure	Multiple choice questions

Men and women with higher education and income are most likely to imbibe, and, among religious denominations, Jews have the highest proportion who consume alcohol but among the lowest rates of alcohol dependence. Other ethnicities, such as the Irish, have higher rates of severe alcohol problems, but they also have significantly higher rates of abstentions. Some estimates show that more than 60 percent of men and women in some Native American and Inuit tribes have been alcohol dependent at some time. In the United States, the average adult consumes 2.2 gallons of absolute alcohol a year, a decrease from 2.7 gallons per capita in 1981.

Drinking alcohol-containing beverages is generally considered an acceptable habit in the United States. About 90 percent of all US residents have had an alcohol-containing drink at least once in their lives, and about 51 percent of all US adults are current users of alcohol.

After heart disease and cancer, alcohol-related disorders constitute the third largest health problem in the United States today. Beer accounts for about one half of all alcohol consumption, liquor for about one third, and wine for about one sixth. About 30 to 45 percent of all adults in the United States have had at least one transient episode of an alcohol-related problem, usually an alcohol-induced amnestic episode (e.g., a blackout), driving a motor vehicle while intoxicated, or missing school or work because of excessive drinking. About 10 percent of women and 20 percent of men have met the diagnostic criteria for alcohol abuse during their lifetimes, and 3 to 5 percent of women and 10 percent of men have met the diagnostic criteria for the more serious diagnosis of alcohol dependence during their lifetimes. About 200,000 deaths each year are directly related to alcohol abuse. The common causes of death among persons with the alcohol-related disorders are suicide, cancer, heart disease, and hepatic disease. Although persons involved in automotive fatalities do not always meet the diagnostic criteria for an alcohol-related disorder, drunk drivers are involved in about 50 percent of all automotive fatalities, and this percentage increases to about 75 percent when only accidents occurring in the late evening are considered. Alcohol use and alcohol-related disorders are associated with about 50 percent of all homicides and 25 percent of all suicides. Alcohol abuse reduces life expectancy by about 10 years, and alcohol leads all other substances in substance-related deaths. Table 20.2-2 lists other epidemiological data about alcohol use.

Table 20.2-2
Epidemiological Data for Alcohol-Related Disorders
COMORBIDITY
The psychiatric diagnoses most commonly associated with the alcohol-related disorders are other substance-related disorders, antisocial personality disorder, mood disorders, and anxiety disorders. Although the data are somewhat controversial, most suggest that persons with alcohol-related disorders have a markedly higher suicide rate than the general population.

Antisocial Personality Disorder
A relation between antisocial personality disorder and alcohol-related disorders has frequently been reported. Some studies suggest that antisocial personality disorder is particularly common in men with an alcohol-related disorder and can precede the development of the alcohol-related disorder. Other studies, however, suggest that antisocial personality disorder and alcohol-related disorders are completely distinct entities that are not causally related.

Mood Disorders
About 30 to 40 percent of persons with an alcohol-related disorder meet the diagnostic criteria for major depressive disorder sometime during their lifetimes. Depression is more common in women than in men with these disorders. Several studies reported that depression is likely to occur in patients with alcohol-related disorders who have a high daily consumption of alcohol and a family history of alcohol abuse. Persons with alcohol-related disorders and major depressive disorder are at great risk for attempting suicide and are likely to have other substance-related disorder diagnoses. Some clinicians recommend antidepressant drug therapy for depressive symptoms that remain after 2 to 3 weeks of sobriety. Patients with bipolar I disorder are thought to be at risk for developing an alcohol-related disorder; they may use alcohol to self-medicate their manic episodes. Some studies have shown that persons with both alcohol-related disorder and depressive disorder diagnoses have concentrations of dopamine metabolites (homovanillic acid) and γ-aminobutyric acid (GABA) in their cerebrospinal fluid (CSF).

Anxiety Disorders
Many persons use alcohol for its e□cacy in alleviating anxiety. Although the comorbidity between alcohol-related disorders and mood disorders is fairly widely recognized, it is less

well known that perhaps 25 to 50 percent of all persons with alcohol-related disorders also meet the diagnostic criteria for an anxiety disorder. Phobias and panic disorder are
particularly frequent comorbid diagnoses in these patients. Some data indicate that alcohol may be used in an attempt to self-medicate symptoms of agoraphobia or social phobia,
but an alcohol-related disorder is likely to precede the development of panic disorder or generalized anxiety disorder.

Suicide

Most estimates of the prevalence of suicide among persons with alcohol-related disorders range from 10 to 15 percent, although alcohol use itself may be involved in a much higher
percentage of suicides. Some investigators have questioned whether the suicide rate among persons with alcohol-related disorders is as high as the numbers suggest. Factors that
have been associated with suicide among persons with alcohol-related disorders include the presence of a major depressive episode, weak psychosocial support systems, a serious
coexisting medical condition, unemployment, and living alone.

ETIOLOGY

Many factors a□ect the decision to drink, the development of temporary alcohol-related di□culties in the teenage years and the 20s, and the
development of alcohol dependence. The initiation of alcohol intake probably depends largely on social, religious, and psychological
factors, although genetic characteristics might also contribute. The factors that in□uence the decision to drink or those that contribute to
temporary problems might differ, however, from those that add to the risk for the severe, recurring problems of alcohol dependence.

A similar interplay between genetic and environmental in□uences contributes to many medical and psychiatric conditions, and, thus, a
review of these factors in alcoholism o□ers information about complex genetic disorders overall. Dominant or recessive genes, although
important, explain only relatively rare conditions. Most disorders have some level of genetic predisposition that usually relates to a series of
different genetically influenced characteristics, each of which increases or decreases the risk for the disorder.

It is likely that a series of genetic in□uences combine to explain approximately 60 percent of the proportion of risk for alcoholism, with
environment responsible for the remaining proportion of the variance. The divisions o□ered in this section, therefore, are more heuristic
than real, because it is the combination of a series of psychological, sociocultural, biological, and other factors that are responsible for the
development of severe, repetitive alcohol-related life problems.

Psychological Theories

A variety of theories relate to the use of alcohol to reduce tension, increase feelings of power, and decrease the e□ects of psychological pain. Perhaps the greatest interest has been paid to the observation that people with alcohol-related problems often report that alcohol decreases their feelings of nervousness and helps them cope with the day-to-day stresses of life. The psychological theories are built, in part, on the observation among nonalcoholic people that the intake of low doses of alcohol in a tense social setting or after a di□cult day can be associated with an enhanced feeling of well-being and an improved ease of interactions. In high doses, especially at falling blood alcohol levels, however, most measures of muscle tension and psychological feelings of nervousness and tension are increased. Thus, tension-reducing e□ects of this drug might have an impact most on light to moderate drinkers or add to the relief of withdrawal symptoms, but play a minor role in causing alcoholism. The theories that focus on alcohol's potential to enhance feelings of being powerful and sexually attractive and to decrease the effects of psychological pain are difficult to evaluate definitively.

Psychodynamic Theories

Perhaps related to the disinhibiting or anxiety-lowering e□ects of lower doses of alcohol is the hypothesis that some people may use this drug to help them deal with self-punitive harsh superegos and to decrease unconscious stress levels. In addition, classic psychoanalytical theory hypothesizes that at least some alcoholic people may have become □xated at the oral stage of development and use alcohol to relieve their frustrations by taking the substance by mouth. Hypotheses regarding arrested phases of psychosexual development, although heuristically useful, have had little e□ect on the usual treatment approaches and are not the focus of extensive ongoing research. Similarly, most studies have not been able to document an "addictive personality" present in most alcoholics and associated with a propensity to lack control of intake of a wide range of substances and foods. Although pathological scores on personality tests are often seen during intoxication, withdrawal, and early recovery, many of these characteristics are not found to predate alcoholism, and most disappear with abstinence. Similarly, prospective studies of children of alcoholics who themselves have no co-occurring disorders usually document high risks mostly for

alcoholism. As is described later in this text, one partial exception occurs with the extreme levels of impulsivity seen in the 15 to 20 percent of alcoholic men with antisocial
personality disorder, because they have high risks for criminality, violence, and multiple substance dependencies.

Behavioral Theories

Expectations about the rewarding e□ects of drinking, cognitive attitudes toward responsibility for one's behavior, and subsequent reinforcement after alcohol intake all contribute to
the decision to drink again after the □rst experience with alcohol and to continue to imbibe despite problems. These issues are important in e□orts to modify drinking behaviors in
the general population, and they contribute to some important aspects of alcoholic rehabilitation.

Sociocultural Theories

Sociocultural theories are often based on extrapolations from social groups that have high and low rates of alcoholism. Theorists hypothesize that ethnic groups, such as Jews, who
introduce children to modest levels of drinking in a family atmosphere and eschew drunkenness have low rates of alcoholism. Some other groups, such as Irish men or some
American Indian tribes with high rates of abstention but a tradition of drinking to the point of drunkenness among drinkers, are believed to have high rates of alcoholism. These
theories, however, often depend on stereotypes that tend to be erroneous, and prominent exceptions to these rules exist. For example, some theories based on observations of the
Irish and the French have incorrectly predicted high rates of alcoholism among the Italians.

Yet, environmental events, presumably including cultural factors, account for as much as 40 percent of the alcoholism risk. Thus, although these are di□cult to study, it is likely
that cultural attitudes toward drinking, drunkenness, and personal responsibility for consequences are important contributors to the rates of alcohol-related problems in a society. In
the □nal analysis, social and psychological theories are probably highly relevant, because they outline factors that contribute to the onset of drinking, the development of temporary
alcohol-related life difficulties, and even alcoholism. The problem is how to gather relatively definitive data to support or refute the theories.

Childhood History

Researchers have identi□ed several factors in the childhood histories of persons with later alcohol-related disorders and in children at high

risk for having an alcohol-related disorder because one or both of their parents are a□ected. In experimental studies, children at high risk for alcohol-related disorders have been found to possess, on average, a range of de□cits on neurocognitive testing, low amplitude of the P300 wave on evoked potential testing, and a variety of abnormalities on electroencephalography (EEG) recordings. Studies of high-risk o□spring in their 20s have also shown a generally blunted e□ect of alcohol compared with that seen in persons whose parents have not been diagnosed with alcohol-related disorder. These □ndings suggest that a heritable biological brain function may predispose a person to an alcohol-related disorder. A childhood history of attention-deficit/hyperactivity disorder (ADHD), conduct disorder, or both, increases a child's risk for an alcohol-related disorder as an adult. Personality disorders, especially antisocial personality disorder, as noted earlier, also predispose a person to an alcohol-related disorder.

Genetic Theories

Importance of Genetic Influences. Four lines of evidence support the conclusion that alcoholism is genetically influenced. First, a threefold to fourfold increased risk for severe alcohol problems is seen in close relatives of alcoholic people. The rate of alcohol problems increases with the number of alcoholic relatives, the severity of their illness, and the closeness of their genetic relationship to the person under study. The family investigations do little to separate the importance of genetics and environment, and the second approach, twin studies, takes the data a step further. The rate of similarity, or concordance, for severe alcohol-related problems is significantly higher in identical twins of alcoholic individuals than in fraternal twins in most investigations, which estimate that genes explain 60 percent of the variance, with the remainder relating to nonshared, probably adult environmental influences. Third, the adoption-type studies have all revealed a significantly enhanced risk for alcoholism in the offspring of alcoholic parents, even when the children had been separated from their biological parents close to birth and raised without any knowledge of the problems within the biological family. The risk for severe alcohol-related difficulties is not further enhanced by being raised by an alcoholic adoptive family. Finally, studies in animals support the importance of a variety of yet-to-be-identified genes in the free-choice use of alcohol, subsequent levels of intoxication, and some consequences.

EFFECTS OF ALCOHOL

The term *alcohol* refers to a large group of organic molecules that have a hydroxyl group (-OH) attached to a saturated carbon atom. Ethyl

alcohol, also called *ethanol,* is the common form of alcohol; sometimes referred to as *beverage alcohol,* ethyl alcohol is used for drinking.
The chemical formula for ethanol is CH3-CH2-OH.
The characteristic tastes and □avors of alcohol-containing beverages result from their methods of production, which produce various congeners in the □nal product, including
methanol, butanol, aldehydes, phenols, tannins, and trace amounts of various metals. Although the congeners may confer some di□erential psychoactive e□ects on the various
alcohol-containing beverages, these di□erences are minimal compared with the e□ects of ethanol itself. A single drink is usually considered to contain about 12 g of ethanol, which
is the content of 12 ounces of beer (7.2 proof, 3.6 percent ethanol in the United States), one 4-ounce glass of nonforti□ed wine, or 1 to 1.5 ounces of an 80-proof (40 percent
ethanol) liquor (e.g., whiskey or gin). In calculating patients' alcohol intake, however, clinicians should be aware that beers vary in their alcohol content, that beers are available in
small and large cans and mugs, that glasses of wine range from 2 to 6 ounces, and that mixed drinks at some bars and in most homes contain 2 to 3 ounces of liquor. Nonetheless,
using the moderate sizes of drinks, clinicians can estimate that a single drink increases the blood alcohol level of a 150-pound man by 15 to 20 mg/dL, which is about the
concentration of alcohol that an average person can metabolize in 1 hour.
The possible bene□cial e□ects of alcohol have been publicized, especially by the makers and the distributors of alcohol. Most attention has been focused on some epidemiological
data that suggest that one or two glasses of red wine each day lower the incidence of cardiovascular disease; these findings, however, are highly controversial.

Absorption

About 10 percent of consumed alcohol is absorbed from the stomach, and the remainder from the small intestine. Peak blood concentration of alcohol is reached in 30 to 90
minutes and usually in 45 to 60 minutes, depending on whether the alcohol was ingested on an empty stomach (which enhances absorption) or with food (which delays absorption).
The time to peak blood concentration also depends on the time during which the alcohol was consumed; rapid drinking reduces the time to peak concentration, slower drinking

increases it. Absorption is most rapid with beverages containing 15 to 30 percent alcohol (30 to 60 proof). There is some dispute about whether carbonation (e.g., in champagne
and in drinks mixed with seltzer) enhances the absorption of alcohol.
The body has protective devices against inundation by alcohol. For example, if the concentration of alcohol in the stomach becomes too high, mucus is secreted and the pyloric
valve closes. These actions slow the absorption and keep the alcohol from passing into the small intestine, where there are no signi□cant restraints on absorption. Thus, a large
amount of alcohol can remain unabsorbed in the stomach for hours. Furthermore, pylorospasm often results in nausea and vomiting.
Once alcohol is absorbed into the bloodstream, it is distributed to all body tissues. Because alcohol is uniformly dissolved in the body's water, tissues containing a high
proportion of water receive a high concentration of alcohol. The intoxicating e□ects are greater when the blood alcohol concentration is rising than when it is falling (the Mellanby
effects). For this reason, the rate of absorption bears directly on the intoxication response.

Metabolism

About 90 percent of absorbed alcohol is metabolized through oxidation in the liver; the remaining 10 percent is excreted unchanged by the kidneys and lungs. The rate of oxidation
by the liver is constant and independent of the body's energy requirements. The body can metabolize about 15 mg/dL per hour, with a range of 10 to 34 mg/dL per hour. That is, the
average person oxidizes three fourths of an ounce of 40 percent (80 proof) alcohol in an hour. In persons with a history of excessive alcohol consumption, upregulation of the
necessary enzymes results in rapid alcohol metabolism.
Alcohol is metabolized by two enzymes: alcohol dehydrogenase (ADH) and aldehyde dehydrogenase. ADH catalyzes the conversion of alcohol into acetaldehyde, which is a toxic
compound; aldehyde dehydrogenase catalyzes the conversion of acetaldehyde into acetic acid. Aldehyde dehydrogenase is inhibited by disul□ram (Antabuse), often used in the
treatment of alcohol-related disorders. Some studies have shown that women have a lower ADH blood content than men; this fact may account for woman's tendency to become
more intoxicated than men after drinking the same amount of alcohol. The decreased function of alcohol-metabolizing enzymes in some Asian persons can also lead to easy

intoxication and toxic symptoms.

Effects on the Brain

Biochemistry. In contrast to most other substances of abuse with identified receptor targets—such as the *N*-methyl-D-aspartate (NMDA) receptor of phencyclidine (PCP)—no single molecular target has been identified as the mediator for the effects of alcohol. The longstanding theory about the biochemical effects of alcohol concerns its effects on the membranes of neurons. Data support the hypothesis that alcohol produces its effects by intercalating itself into membranes and, thus, increasing fluidity of the membranes with short-term use. With longterm use, however, the theory hypothesizes that the membranes become rigid or stiff. The fluidity of the membranes is critical to normal functioning of receptors, ion channels, and other membrane-bound functional proteins. In recent studies, researchers have attempted to identify specific molecular targets for the effects of alcohol. Most attention has been focused on the effects of alcohol at ion channels.

Specifically, studies have found that alcohol ion channel activities associated with the nicotinic acetylcholine, serotonin 5-hydroxytryptamine3 (5-HT3), and GABA type A (GABAA) receptors are enhanced by alcohol, whereas ion channel activities associated with glutamate receptors and voltage-gated calcium channels are inhibited.

Behavioral Effects. As the net result of the molecular activities, alcohol functions as a depressant much like the barbiturates and the benzodiazepines, with which alcohol has some cross-tolerance and cross-dependence. At a level of 0.05 percent alcohol in the blood, thought, judgment, and restraint are loosened and sometimes disrupted. At a concentration of 0.1 percent, voluntary motor actions usually become perceptibly clumsy. In most states, legal intoxication ranges from 0.1 to 0.15 percent blood alcohol level. At 0.2 percent, the function of the entire motor area of the brain is measurably depressed, and the parts of the brain that control emotional behavior are also affected. At 0.3 percent, a person is commonly confused or may become stuporous; at 0.4 to 0.5 percent, the person falls into a coma. At higher levels, the primitive centers of the brain that control breathing and heart rate are affected, and death ensues secondary to direct respiratory depression or the aspiration of vomitus. Persons with long-term histories of alcohol abuse, however, can tolerate much higher concentrations of alcohol than can alcohol-naïve persons; their alcohol tolerance may cause them to falsely appear less intoxicated than they really are.

Sleep Effects. Although alcohol consumed in the evening usually increases the ease of falling asleep (decreased sleep latency), alcohol

also has adverse effects on sleep architecture. Specifically, alcohol use is associated with a decrease in rapid eye movement sleep (REM or dream sleep) and deep sleep (stage 4) and more sleep fragmentation, with more and longer episodes of awakening. Therefore, the idea that drinking alcohol helps persons fall asleep is a myth.

Other Physiological Effects

Liver. The major adverse effects of alcohol use are related to liver damage. Alcohol use, even as short as week-long episodes of increased drinking, can result in an accumulation of fats and proteins, which produce the appearance of a fatty liver, sometimes found on physical examination as an enlarged liver. The association between fatty infiltration of the liver and serious liver damage remains unclear. Alcohol use, however, is associated with the development of alcoholic hepatitis and hepatic cirrhosis.

Gastrointestinal System. Long-term heavy drinking is associated with developing esophagitis, gastritis, achlorhydria, and gastric ulcers. The development of esophageal varices can accompany particularly heavy alcohol abuse; the rupture of the varices is a medical emergency often resulting in death by exsanguination. Disorders of the small intestine occasionally occur, and pancreatitis, pancreatic insufficiency, and pancreatic cancer are also associated with heavy alcohol use. Heavy alcohol intake can interfere with the normal processes of food digestion and absorption; as a result, consumed food is inadequately digested. Alcohol abuse also appears to inhibit the intestine's capacity to absorb various nutrients, such as vitamins and amino acids. This effect, coupled with the often poor dietary habits of those with alcohol-related disorders, can cause serious vitamin deficiencies, particularly of the B vitamins.

Other Bodily Systems. Significant intake of alcohol has been associated with increased blood pressure, dysregulation of lipoprotein and triglyceride metabolism, and increased risk for myocardial infarction and cerebrovascular disease. Alcohol has been shown to affect the hearts of nonalcoholic persons who do not usually drink, increasing the resting cardiac output, the heart rate, and the myocardial oxygen consumption. Evidence indicates that alcohol intake can adversely affect the hematopoietic system and can increase the incidence of cancer, particularly head, neck, esophageal, stomach, hepatic, colonic, and lung cancer. Acute intoxication may also be associated with hypoglycemia, which, when unrecognized, may be responsible for some of the sudden deaths of persons who are intoxicated. Muscle weakness is another side effect of alcoholism. Recent evidence shows that alcohol intake raises the blood concentration of estradiol in women. The increase in estradiol correlates with the blood alcohol level.

Laboratory Tests. The adverse effects of alcohol appear in common laboratory tests, which can be useful diagnostic aids in identifying persons with alcohol-related disorders. The γ-glutamyl transpeptidase levels are high in about 80 percent of those with alcohol-related disorders, and the mean corpuscular volume (MCV) is high in about 60 percent, more so in women than in men. Other laboratory test values that may be high in association with alcohol abuse are those of uric acid, triglycerides, aspartate aminotransferase (AST), and alanine aminotransferase (ALT).

Drug Interactions

The interaction between alcohol and other substances can be dangerous, even fatal. Certain substances, such as alcohol and phenobarbital (Luminal), are metabolized by the liver, and their prolonged use can lead to acceleration of their metabolism. When persons with alcoholrelated disorders are sober, this accelerated metabolism makes them unusually tolerant to many drugs such as sedatives and hypnotics; when they are intoxicated, however, these drugs compete with the alcohol for the same detoxi□cation mechanisms, and potentially toxic concentrations of all involved substances can accumulate in the blood.

The e□ects of alcohol and other central nervous system (CNS) depressants are usually synergistic. Sedatives, hypnotics, and drugs that relieve pain, motion sickness, head colds, and allergy symptoms must be used with caution by persons with alcohol-related disorders.

Narcotics depress the sensory areas of the cerebral cortex and can produce pain relief, sedation, apathy, drowsiness, and sleep; high doses can result in respiratory failure and death. Increasing the dosages of sedative-hypnotic drugs, such as chloral hydrate (Noctec) and benzodiazepines, especially when they are combined with alcohol, produces a range of e□ects from sedation to motor and intellectual impairment to stupor, coma, and death. Because sedatives and other psychotropic drugs can potentiate the e□ects of alcohol, patients should be instructed about the dangers of combining CNS depressants and alcohol, particularly when they are driving or operating machinery.

DISORDERS

Alcohol Use Disorder

Diagnosis and Clinical Features. In the fifth edition of *Diagnostic and Statistical Manual of Mental* Disorders (DSM-5), all substance use disorders use the same general criteria for dependence and abuse (see Section 20.1). A need for daily use of large amounts of alcohol for adequate functioning, a regular pattern of heavy drinking limited to weekends, and long periods of sobriety interspersed with binges of heavy alcohol intake lasting for weeks or months strongly suggest alcohol dependence and alcohol abuse. The drinking patterns are often

associated with certain behaviors: the inability to cut down or stop drinking; repeated efforts to control or reduce excessive drinking by
"going on the wagon" (periods of temporary abstinence) or by restricting drinking to certain times of the day; binges (remaining intoxicated
throughout the day for at least 2 days); occasional consumption of a fifth of spirits (or its equivalent in wine or beer); amnestic periods for
events occurring while intoxicated (blackouts); the continuation of drinking despite a serious physical disorder that the person knows is
exacerbated by alcohol use; and drinking nonbeverage alcohol, such as fuel and commercial products containing alcohol. In addition,
persons with alcohol dependence and alcohol abuse show impaired social or occupational functioning because of alcohol use (e.g., violence
while intoxicated, absence from work, job loss), legal difficulties (e.g., arrest for intoxicated behavior and traffic accidents while intoxicated),
and arguments or difficulties with family members or friends about excessive alcohol consumption.
Mark, a 45-year-old divorced man, was examined in a hospital emergency room because he had been confused and unable to care for
himself of the preceding 3 days. His brother, who brought him to the hospital, reported that the patient has consumed large quantities of
beer and wine daily for more than 5 years. His home and job lives were reasonably stable until his divorce 5 years prior. The brother
indicated that Mark's drinking pattern since the divorce has been approximately 5 beers and a fourth of wine a day. Mark often
experienced blackouts from drinking and missed days of work frequently. As a result, Mark has lost several jobs in the past 5 years.
Although he usually provides for himself marginally with small jobs, 3 days earlier he ran out of money and alcohol and resorted to
panhandling on the streets for cash to buy food. Mark had been poorly nourished, having one meal per day at best and was evidently
relying on beer as his prime source of nourishment.
On examination, Mark alternates between apprehension and chatty, super□cial warmth. He is pretty keyed up and talks constantly in a
rambling and unfocused manner. His recognition of the physician varies; at times he recognizes him and other times he becomes confused
and believes the doctor to be his other brother who lives in another state. On two occasions he referred to the physician by said brother's
name and asked when he arrived in town, evidently having lost track of the interview up to that point. He has a gross hand tremor at rest
and is disoriented to time. He believes he's in a parking lot rather than a hospital. E□orts at memory and calculation testing fail because
Mark's attention shifts so rapidly.

Subtypes of Alcohol Dependence. Various researchers have attempted to divide alcohol dependence into subtypes based primarily on phenomenological characteristics. One recent classification notes that type A alcohol dependence is characterized by late onset, few childhood risk factors, relatively mild dependence, few alcohol-related problems, and little psychopathology. Type B alcohol dependence is characterized by many childhood risk factors, severe dependence, an early onset of alcohol-related problems, much psychopathology, a strong family history of alcohol abuse, frequent polysubstance abuse, a long history of alcohol treatment, and a lot of severe life stresses.

Some researchers have found that type A persons who are alcohol dependent may respond to interactional psychotherapies, whereas type B persons who are alcohol dependent may respond to training in coping skills.

Other subtyping schemes of alcohol dependence have received fairly wide recognition in the literature. One group of investigators proposed three subtypes: earlystage problem drinkers, who do not yet have complete alcohol dependence syndromes; a□liative drinkers, who tend to drink daily in moderate amounts in social settings; and schizoid-isolated drinkers, who have severe dependence and tend to drink in binges and often alone.

Another investigator described gamma alcohol dependence, which is thought to be common in the United States and represents the alcohol dependence seen in those who are active in Alcoholics Anonymous (AA). This variant concerns control problems in which persons are unable to stop drinking once they start. When drinking is terminated as a result of ill health or lack of money, these persons can abstain for varying periods. In delta alcohol dependence, perhaps more common in Europe than in the United States, persons who are alcohol dependent must drink a certain amount each day but are unaware of a lack of control. The alcohol use disorder may not be discovered until a person who must stop drinking for some reason exhibits withdrawal symptoms.

Another researcher has suggested a *type I, male-limited* variety of alcohol dependence, characterized by late onset, more evidence of psychological than of physical dependence, and the presence of guilt feelings. *Type II, male-limited* alcohol dependence is characterized by onset at an early age, spontaneous seeking of alcohol for consumption, and a socially disruptive set of behaviors when intoxicated.

Four subtypes of alcoholism were postulated by still another investigator. The □rst is *antisocial alcoholism*, typically with a predominance in men, a poor prognosis, early onset of alcohol-related problems, and a close association with antisocial personality disorder. The second is

developmentally cumulative alcoholism, with a primary tendency for alcohol abuse that is exacerbated with time as cultural expectations foster increased opportunities to drink. The third is *negative-a□ect alcoholism,* which is more common in women than in men; according to this hypothesis, women are likely to use alcohol for mood regulation and to help ease social relationships. The fourth is *developmentally limited alcoholism,* with frequent bouts of consuming large amounts of alcohol; the bouts become less frequent as persons age and respond to the increased expectations of society about their jobs and families.

Alcohol Intoxication

The DSM-5 diagnostic criteria for alcohol intoxication (also called simple drunkenness) are based on evidence of recent ingestion of ethanol, maladaptive behavior, and at least one of several possible physiological correlates of intoxication (Table 20.2-3). As a conservative approach to identifying blood levels that are likely to have major e□ects on driving abilities, the legal de□nition of intoxication in most states in the United States requires a blood concentration of 80 or 100 mg ethanol per deciliter of blood (mg/dL), which is the same as 0.08 to 0.10 g/dL. The following is an outline of the rough estimates of the levels of impairment likely to be seen at various blood alcohol concentrations, for most people. Evidence of behavioral changes, a slowing in motor performance, and a decrease in the ability to think clearly occurs at doses as low as 20 to 30 mg/dL, as shown in Table 20.2-4. Blood concentrations between 100 and 200 mg/dL are likely to increase the impairment in coordination and judgment to severe problems with coordination (ataxia), increasing lability of mood, and progressively greater levels of cognitive deterioration. Anyone who does not show signi□cant levels of impairment in motor and mental performance at approximately 150 mg/dL probably has signi□cant pharmacodynamic tolerance. In that range, most people without signi□cant tolerance also experience relatively severe nausea and vomiting. With blood alcohol concentrations in the 200 to 300 mg/dL range, the slurring of speech is likely to become more intense, and memory impairment (*anterograde amnesia* or *alcoholic blackouts*) becomes pronounced. Further increases in blood alcohol concentration result in the □rst level of anesthesia, and the nontolerant person who reaches 400 mg/dL or more risks respiratory failure, coma, and death.

Table 20.2-3

Signs of Alcohol Intoxication

Table 20.2-4

Impairment Likely to be Seen at Different Blood Alcohol Concentrations

Alcohol Withdrawal

Alcohol withdrawal, even without delirium, can be serious; it can include seizures and autonomic hyperactivity. Conditions that may predispose to, or aggravate, withdrawal symptoms include fatigue, malnutrition, physical illness, and depression. The DSM-5 criteria for alcohol withdrawal require the cessation or reduction of alcohol use that was heavy and prolonged as well as the presence of speci□c physical or neuropsychiatric symptoms. The diagnosis also allows for the speci□cation "with perceptual disturbances." One positron emission tomography (PET) study of blood □ow during alcohol withdrawal in otherwise healthy persons with alcohol dependence reported a globally low rate of metabolic activity, although, with further inspection of the data, the authors concluded that activity was especially low in the left parietal and right frontal areas.

The classic sign of alcohol withdrawal is tremulousness, although the spectrum of symptoms can expand to include psychotic and perceptual symptoms (e.g., delusions and hallucinations), seizures, and the symptoms of delirium tremens (DTs), called alcohol delirium in DSM-5. Tremulousness (commonly called the "shakes" or the "jitters") develops 6 to 8 hours after the cessation of drinking, the psychotic and perceptual symptoms begin in 8 to 12 hours, seizures in 12 to 24 hours, and DTs anytime during the □rst 72 hours, although physicians should watch for the development of DTs for the □rst week of withdrawal. The syndrome of withdrawal sometimes skips the usual progression and, for example, goes directly to DTs.

The tremor of alcohol withdrawal can be similar to either physiological tremor, with a continuous tremor of great amplitude and of more than 8 Hz, or familial tremor, with bursts of tremor activity slower than 8 Hz. Other symptoms of withdrawal include general irritability, gastrointestinal symptoms (e.g., nausea and vomiting), and sympathetic autonomic hyperactivity, including anxiety, arousal, sweating, facial flushing, mydriasis, tachycardia, and mild hypertension. Patients experiencing alcohol withdrawal are generally alert but may startle easily.

Twenty-nine-year-old Mr. F had been a heavy drinker for 8 years. One evening after work, he started drinking with friends and drank throughout the evening. He fell asleep in the early morning hours and upon awakening had a strong desire to drink and decided not to attend work. He had several Bloody Marys instead of food because food did not appeal to him. He went to a local bar in the afternoon and consumed large quantities of beer. That evening he met with some friends and continued to drink.

This drinking pattern continued for the next week. The beginning of the following week he attempted to have a cup of co□ee and

found that he hands were shaking so much that he could not get the cup to his mouth to drink. He eventually managed to pour himself some wine in a glass and drank as much as he could. His hands then became less shaky, but he now felt nauseous and began having dry heaves. He tried to drink repeatedly but he could not keep the alcohol down. He felt very ill and anxious so he contacted his physician who recommended he report to a hospital.

Upon evaluation, Mr. F was alert. He had a marked resting and intention tremor of the hands, and his tongue and eyelids were tremulous. He was oriented and had no memory impairment. When inquired about his drinking, Mr. F admits to drinking several drinks each day for the past 8 years, but claims that his drinking never interfered with his work or his relations with colleagues or friends. He denies having any aftere□ects from his drinking other than mild hangovers. He denies ever having a binge such as this before and denies ever needing to drink daily in order to function adequately. He admits, however, that he has never tried to reduce or stop drinking.

Withdrawal Seizures. Seizures associated with alcohol withdrawal are stereotyped, generalized, and tonic–clonic in character. Patients often have more than one seizure 3 to 6 hours after the first seizure. Status epilepticus is relatively rare and occurs in less than 3 percent of patients. Although anticonvulsant medications are not required in the management of alcohol withdrawal seizures, the cause of the seizures is difficult to establish when a patient is first assessed in the emergency room; thus, many patients with withdrawal seizures receive anticonvulsant medications, which are then discontinued once the cause of the seizures is recognized. Seizure activity in patients with known alcohol abuse histories should still prompt clinicians to consider other causative factors, such as head injuries, CNS infections, CNS neoplasms, and other cerebrovascular diseases; long-term severe alcohol abuse can result in hypoglycemia, hyponatremia, and hypomagnesemia—all of which can also be associated with seizures.

Treatment. The primary medications to control alcohol withdrawal symptoms are the benzodiazepines (Table 20.2-5). Many studies have found that benzodiazepines help control seizure activity, delirium, anxiety, tachycardia, hypertension, diaphoresis, and tremor associated with alcohol withdrawal. Benzodiazepines can be given either orally or parenterally; neither diazepam (Valium) nor chlordiazepoxide (Librium), however, should be given intramuscularly (IM) because of their erratic absorption by this route. Clinicians must titrate the dosage of the benzodiazepine, starting with a high dosage and lowering the dosage as the patient recovers. Sufficient

benzodiazepines should be given to keep patients calm and sedated but not so sedated that they cannot be aroused for clinicians to perform appropriate procedures, including neurological examinations.

Table 20.2-5

Drug Therapy for Alcohol Intoxication and Withdrawal

Although benzodiazepines are the standard treatment for alcohol withdrawal, studies have shown that carbamazepine (Tegretol) in daily doses of 800 mg is as e□ective as benzodiazepines and has the added bene□t of minimal abuse liability. Carbamazepine use is gradually becoming common in the United States and Europe. The β-adrenergic receptor antagonists and clonidine (Catapres) have also been used to block the symptoms of sympathetic hyperactivity, but neither drug is an effective treatment for seizures or delirium.

Delirium

Diagnosis and Clinical Features. Patients with recognized alcohol withdrawal symptoms should be carefully monitored to prevent progression to alcohol withdrawal delirium, the most severe form of the withdrawal syndrome, also known as DTs. Alcohol withdrawal delirium is a medical emergency that can result in significant morbidity and mortality. Patients with delirium are a danger to themselves and to others. Because of the unpredictability of their behavior, patients with delirium may be assaultive or suicidal or may act on hallucinations or delusional thoughts as if they were genuine dangers. Untreated, DTs has a mortality rate of 20 percent, usually as a result of an intercurrent medical illness such as pneumonia, renal disease, hepatic insufficiency, or heart failure. Although withdrawal seizures commonly precede the development of alcohol withdrawal delirium, delirium can also appear unheralded. The essential feature of the syndrome is delirium occurring within 1 week after a person stops drinking or reduces the intake of alcohol. In addition to the symptoms of delirium, the features of alcohol intoxication delirium include autonomic hyperactivity such as tachycardia, diaphoresis, fever, anxiety, insomnia, and hypertension; perceptual distortions, most frequently visual or tactile hallucinations; and fluctuating levels of psychomotor activity, ranging from hyperexcitability to lethargy.

About 5 percent of persons with alcohol-related disorders who are hospitalized have DTs. Because the syndrome usually develops on the third hospital day, a patient admitted for an unrelated condition may unexpectedly have an episode of delirium, the □rst sign of a previously undiagnosed alcohol-related disorder. Episodes of DTs usually begin in a patient's 30s or 40s after 5 to 15 years of heavy drinking, typically of the binge type. Physical illness (e.g., hepatitis or pancreatitis) predisposes to the syndrome; a person in good physical

health rarely has DTs during alcohol withdrawal.

Mr. R, a 40-year-old man, was admitted to the orthopedic department of a general hospital after experiencing a fall down stairs and breaking his leg. On the third day of his hospital stay, he became increasingly nervous and started to tremble. He was unable to sleep at night, talked incoherently, and was obviously very anxious. Mr. R, when asked, denied an alcohol problem other than an occasional glass of wine.

When asked directly, his wife admitted that Mr. R drank large quantities of wine for over 4 years. During the previous year, his drinking would begin every evening when he came home from work and would not end until he fell asleep. On the evening of admittance, the fall occurred before he was able to consume any alcohol.

During the few weeks prior to his admittance, Mr. R had eaten very little. On several occasions, Mrs. R noticed that Mr. R was unable to recall even important events from the previous day. He had a car accident 3 years prior but without major injury. Mr. R had no other major health problems. His relationship with Mrs. R became very di□cult after he began drinking and Mrs. R was seriously contemplating divorce. Mr. R had a tense relationship with his four children and he often argued with them. Recently, the children tried to avoid Mr. R as much as possible.

On examination, Mr. R's speech was rambling and incoherent. He believed that he was still at work and that he had a job to □nish. At times he thought the physicians and nurses were his co-workers. At times he picked at bugs that he could see on his bed sheets. He was disoriented in time and was startled easily by sounds from outside the room. He sweat profusely and could not hold a glass without spilling some of the contents.

Treatment. The best treatment for DTs is prevention. Patients withdrawing from alcohol who exhibit withdrawal phenomena should receive a benzodiazepine, such as 25 to 50 mg of chlordiazepoxide every 2 to 4 hours until they seem to be out of danger. Once the delirium appears, however, 50 to 100 mg of chlordiazepoxide should be given every 4 hours orally, or lorazepam (Ativan) should be given intravenously (IV) if oral medication is not possible (Table 20.2-5). Antipsychotic medications that may reduce the seizure threshold in patients should be avoided. A high-calorie, high-carbohydrate diet supplemented by multivitamins is also important.

Physically restraining patients with the DTs is risky; they may □ght against the restraints to a dangerous level of exhaustion. When patients are disorderly and uncontrollable, a seclusion room can be used. Dehydration, often exacerbated by diaphoresis and fever, can be corrected

with □uids given by mouth or IV. Anorexia, vomiting, and diarrhea often occur during withdrawal. Antipsychotic medications should be avoided because they can reduce the seizure threshold in the patient. The emergence of focal neurological symptoms, lateralizing seizures, increased intracranial pressure, or evidence of skull fractures or other indications of CNS pathology should prompt clinicians to examine a patient for additional neurological disease. Nonbenzodiazepine anticonvulsant medication is not useful in preventing or treating alcohol withdrawal convulsions, although benzodiazepines are generally effective.

Warm, supportive psychotherapy in the treatment of DTs is essential. Patients are often bewildered, frightened, and anxious because of their tumultuous symptoms, and skillful verbal support is imperative.

Alcohol-Induced Persisting Dementia

Alcohol-induced persisting dementia is a poorly studied, heterogeneous long-term cognitive problem that can develop in the course of alcoholism. Global decreases in intellectual functioning, cognitive abilities, and memory are observed, but recent memory di□culties are consistent with the global cognitive impairment, an observation that helps to distinguish this from alcohol-induced persisting amnestic disorder. Brain functioning tends to improve with abstinence, but perhaps half of all a□ected patients have long-term and even permanent disabilities in memory and thinking. Approximately 50 to 70 percent of these patients evidence increased size of the brain ventricles and shrinkage of the cerebral sulci, although these changes appear to be partially or completely reversible during the □rst year of complete abstinence.

Alcohol-Induced Persisting Amnestic Disorder

Diagnosis and Clinical Features. The essential feature of alcohol-induced persisting amnestic disorder is a disturbance in shortterm memory caused by prolonged heavy use of alcohol. Because the disorder usually occurs in persons who have been drinking heavily for many years, the disorder is rare in persons younger than age 35.

Wernicke-Korsakoff Syndrome. The classic names for alcohol-induced persisting amnestic disorder are Wernicke's encephalopathy (a set of acute symptoms) and Korsakoff's syndrome (a chronic condition). Whereas Wernicke's encephalopathy is completely reversible with treatment, only about 20 percent of patients with Korsakoff's syndrome recover. The pathophysiological connection between the two syndromes is thiamine deficiency, caused either by poor nutritional habits or by malabsorption problems. Thiamine is a cofactor for several important enzymes and may also be involved in conduction of the axon potential along the axon and in synaptic transmission. The

neuropathological lesions are symmetrical and paraventricular, involving the mammillary bodies, the thalamus, the hypothalamus, the midbrain, the pons, the medulla, the fornix, and the cerebellum.

Wernicke's encephalopathy, also called *alcoholic encephalopathy,* is an acute neurological disorder characterized by ataxia (a□ecting primarily the gait), vestibular dysfunction, confusion, and a variety of ocular motility abnormalities, including horizontal nystagmus, lateral orbital palsy, and gaze palsy. These eye signs are usually bilateral but not necessarily symmetrical. Other eye signs may include a sluggish reaction to light and anisocoria. Wernicke's encephalopathy may clear spontaneously in a few days or weeks or may progress into Korsakoff's syndrome.

Treatment. In the early stages, Wernicke's encephalopathy responds rapidly to large doses of parenteral thiamine, which is believed to be effective in preventing the progression into Korsakoff's syndrome. The dosage of thiamine is usually initiated at 100 mg by mouth two to three times daily and is continued for 1 to 2 weeks. In patients with alcohol-related disorders who are receiving IV administration of glucose solution, it is good practice to include 100 mg of thiamine in each liter of the glucose solution.

Korsako□'s syndrome is the chronic amnestic syndrome that can follow Wernicke's encephalopathy, and the two syndromes are believed to be pathophysiologically related. The cardinal features of Korsako□'s syndrome are impaired mental syndrome (especially recent memory) and anterograde amnesia in an alert and responsive patient. The patient may or may not have the symptom of confabulation. Treatment of Korsako□'s syndrome is also thiamine given 100 mg by mouth two to three times daily; the treatment regimen should continue for 3 to 12 months. Few patients who progress to Korsako□'s syndrome ever fully recover, although many have some improvement in their cognitive abilities with thiamine and nutritional support.

Blackouts. Blackouts are similar to episodes of transient global amnesia in that they are discrete episodes of anterograde amnesia that occur in association with alcohol intoxication. The periods of amnesia can be particularly distressing when persons fear that they have unknowingly harmed someone or behaved imprudently while intoxicated. During a blackout, persons have relatively intact remote memory but experience a specific short-term memory deficit in which they are unable to recall events that happened in the previous 5 or 10 minutes. Because their other intellectual faculties are well preserved, they can perform complicated tasks and appear normal to casual observers. The neurobiological mechanisms for alcoholic blackouts are now known at the molecular level; alcohol blocks the consolidation

of new memories into old memories, a process that is thought to involve the hippocampus and related temporal lobe structures.

Alcohol-Induced Psychotic Disorder

Diagnosis and Clinical Features. Approximately 3 percent of alcoholic persons experience auditory hallucinations or paranoid delusions in the context of heavy drinking or withdrawal. The most common auditory hallucinations are voices, but they are often unstructured. The voices are characteristically maligning, reproachful, or threatening, although some patients report that the voices are pleasant and nondisruptive. The hallucinations usually last less than a week, but during that week impaired reality testing is common. After the episode, most patients realize the hallucinatory nature of the symptoms.

Hallucinations after alcohol withdrawal are considered rare, and the syndrome is distinct from alcohol withdrawal delirium. The hallucinations can occur at any age, but usually appear in persons abusing alcohol for a long time. Although the hallucinations usually resolve within a week, some linger; in these cases, clinicians must consider other psychotic disorders in the di□erential diagnosis. Alcohol withdrawal-related hallucinations are di□erentiated from the hallucinations of schizophrenia by the temporal association with alcohol withdrawal, the absence of a classic history of schizophrenia, and their usually short-lived duration. Alcohol withdrawal-related hallucinations are differentiated from the DTs by the presence of a clear sensorium in patients.

Mr. G was a 40-year-old unemployed man living alone in a studio apartment and was brought to the hospital by the police. He contacted them complaining that he heard voices of men on the street below his window talking about him and threatening to kill him.

He stated that every time he looked out the window the men had always disappeared.

Mr. G had a 15-year history of almost daily alcohol use. He was intoxicated each day and often experienced shakes upon awakening in the morning. On the previous day, he had only one glass of beer instead of his usual four because of gastrointestinal problems. He was fully alert and oriented.

Treatment. The treatment of alcohol withdrawal-related hallucinations is much like the treatment of DTs—benzodiazepines, adequate nutrition, and fluids, if necessary. If this regimen fails or for long-term cases, antipsychotics may be used.

Alcohol-Induced Mood Disorder

Heavy intake of alcohol over several days results in many of the symptoms observed in major depressive disorder, but the intense sadness

markedly improves within several days to 1 month of abstinence. Eighty percent of people with alcoholism report histories of intense depression, including 30 to 40 percent who were depressed for 2 or more weeks at a time. However, only 10 to 15 percent of alcoholic persons have ever had depression that meets the criteria for major depressive disorder when they have not been drinking heavily.

Even severe substance-induced depressions are likely to improve fairly rapidly with abstinence, without medication or intensive psychotherapy aimed at the depressive symptoms. A logical approach for these substance-induced conditions is to teach the patient how to best view and deal with the temporary sadness through education and cognitive-behavioral treatment, and to watch and wait at least 2 to 4 weeks before starting antidepressant medications.

A consultation was requested on a 42-year-old woman with alcohol dependence who complained of persisting severe depressive symptoms despite 5 days of abstinence. In the initial stage of the interview, she noted that she had "always been depressed" and felt that she "drank to cope with the depressive symptoms." Her current complaint included a prominent sadness that had persisted for several weeks, di□culties concentrating, initial and terminal insomnia, and a feeling of hopelessness and guilt. In an e□ort to distinguish between an alcohol-induced mood disorder and an independent major depressive episode, a time-line–based history was obtained. This focused on the age of onset of alcohol dependence, periods of abstinence that extended for several months or more since the onset of dependence, and the ages of occurrence of clear major depressive episodes lasting several weeks or more at a time. Despite this patient's original complaints, it became clear that there had been no major depressive episodes prior to her mid-20s when alcohol dependence began, and that during a 1-year period of abstinence related to the gestation and neonatal period of her son, her mood had signi□cantly improved. A provisional diagnosis of an alcohol-induced mood disorder was made. The patient was o□ered education, reassurance, and cognitive therapy to help her to deal with the depressive symptoms, but no antidepressant medications were prescribed. The depressive symptoms remained at their original intensity for several additional days and then began to improve. By approximately 3 weeks abstinent the patient no longer met criteria for a major depressive episode, although she demonstrated mood swings similar to dysphemia for several additional weeks. This case is a fairly typical example of an alcohol-induced mood disorder in an individual with alcohol dependence.

(Courtesy of Marc A. Shuckit, M.D.)

Alcohol-Induced Anxiety Disorder

Anxiety symptoms ful□lling the diagnostic criteria for alcohol-induced anxiety disorder are also common in the context of acute and protracted alcohol withdrawal. Almost 80 percent of alcoholic persons report panic attacks during at least one acute withdrawal episode; their complaints can be su□ciently intense for the clinician to consider diagnosing panic disorder. Similarly, during the □rst 4 weeks or so of abstinence, people with severe alcohol problems are likely to avoid some social situations for fear of being overwhelmed by anxiety (i.e., they have symptoms resembling social phobia); their problems can at times be severe enough to resemble agoraphobia. However, when psychological or physiological symptoms of anxiety are observed in alcoholic persons only in the context of heavy drinking or within the first several weeks or month of abstinence, the symptoms are likely to diminish and subsequently disappear with time alone.

A 48-year-old woman was referred for evaluation and treatment of her recent onset of panic attacks. These episodes occurred two to three times per week over the preceding 6 months, with each lasting typically between 10 and 20 minutes. Panic symptoms occurred regardless of levels of life stress and could not be explained by current medications or medical conditions. The workup included an evaluation of her laboratory test values, which revealed a carbohydrate-de□cient transferrin (CDT) level of 28 U/L, a uric acid level of 7.1 mg, and a γ-glutamyltransferase value of 47. All other blood tests were within normal limits.

The atypical age of onset of the panic attacks, along with the blood results, encouraged the clinician to probe further regarding the pattern of alcohol-related life problems with both the patient and, separately, her spouse. This step documented a history of alcohol dependence with an onset at approximately 35 years of age, with no evidence of panic disorder before that date. Nor did the patient have repetitive panic attacks beyond 2 weeks of abstinence during her frequent periods of nondrinking, which often lasted for 3 or 4 months. A working diagnosis of alcohol dependence with an alcohol-induced anxiety disorder characterized by panic attacks was made, and the patient was encouraged to abstain and was appropriately treated for possible withdrawal symptoms. Over the subsequent 3 weeks after a taper of benzodiazepines used for the treatment of withdrawal, the panic symptoms diminished in intensity and subsequently disappeared. (Courtesy of Marc A. Schuckit, M.D.)

Alcohol-Induced Sexual Dysfunction

The formal diagnosis of symptoms of sexual dysfunction associated with alcohol intoxication is alcohol-induced sexual dysfunction (*see* Section 17.2).

Alcohol-Induced Sleep Disorder

The diagnostic criteria for alcohol-induced sleep disorders with an onset during either alcohol intoxication or alcohol withdrawal are found in the sleep disorders section (see Section 16.2).

Unspecified Alcohol-Related Disorder

The diagnosis of unspeci□ed alcohol-related disorder is used for alcohol-related disorders that do not meet the diagnostic criteria for any of the other diagnoses.

Idiosyncratic Alcohol Intoxication

Whether there is such a diagnostic entity as idiosyncratic alcohol intoxication is under debate. Several well-controlled studies of persons who supposedly have the disorder have raised questions about the validity of the designation. The condition has been variously called pathologic, complicated, atypical, and paranoid alcohol intoxication; all these terms indicate that a severe behavioral syndrome develops rapidly after a person consumes a small amount of alcohol that would have minimal behavioral e□ects on most persons. The diagnosis is important in the forensic arena because alcohol intoxication is not generally accepted as a reason for judging persons not responsible for their activities.

Idiosyncratic alcohol intoxication, however, can be used in a person's defense if a defense lawyer can argue successfully that the defendant has an unexpected, idiosyncratic, pathological reaction to a minimal amount of alcohol.

In anecdotal reports, persons with idiosyncratic alcohol intoxication have been described as confused and disoriented and as experiencing illusions, transitory delusions, and visual hallucinations. Persons may display greatly increased psychomotor activity and impulsive, aggressive behavior. They can be dangerous to others and they may also exhibit suicidal ideation and make suicide attempts. The disorder, usually described as lasting for a few hours, terminates in prolonged sleep, and those a□ected cannot recall the episodes on awakening. The cause of the condition is unknown, but it is reported to be most common in persons with high levels of anxiety. According to one hypothesis, alcohol causes su□cient disorganization and loss of control to release aggressive impulses. Another suggestion is that brain damage, particularly encephalitic or traumatic damage, predisposes some persons to an intolerance for alcohol and thus to abnormal behavior after they ingest only small amounts. Other predisposing factors may include advancing age, using sedative-hypnotic drugs, and feeling fatigued. A person's behavior while intoxicated tends to be atypical; after one weak drink, a quiet, shy person becomes belligerent and aggressive.

In treating idiosyncratic alcohol intoxication, clinicians must help protect patients from harming themselves and others. Physical restraint may be necessary, but is difficult because of the abrupt onset of the condition. Once a patient has been restrained, injection of an antipsychotic drug, such as haloperidol (Haldol), is useful for controlling assaultiveness. This condition must be differentiated from other causes of abrupt behavioral change, such as complex partial epilepsy. Some persons with the disorder reportedly showed temporal lobe spiking on an EEG after ingesting small amounts of alcohol.

Other Alcohol-Related Neurological Disorders

Only the major neuropsychiatric syndromes associated with alcohol use have been discussed here. The complete list of neurological syndromes is lengthy (Table 20.2-6). Alcoholic pellagra encephalopathy is one diagnosis of potential interest to psychiatrists presented with a patient who appears to have Wernicke-Korsakoff syndrome but who does not respond to thiamine treatment. The symptoms of alcoholic pellagra encephalopathy include confusion, clouding of consciousness, myoclonus, oppositional hypertonias, fatigue, apathy, irritability, anorexia, insomnia, and sometimes delirium. Patients have a niacin (nicotinic acid) deficiency, and the specific treatment is 50 mg of niacin by mouth four times daily or 25 mg parenterally two to three times daily.

Table 20.2-6

Neurological and Medical Complications of Alcohol Use

Fetal Alcohol Syndrome. Data indicate that women who are pregnant or are breast-feeding should not drink alcohol. Fetal alcohol syndrome, the leading cause of intellectual disability in the United States, occurs when mothers who drink alcohol expose fetuses to alcohol in utero. The alcohol inhibits intrauterine growth and postnatal development. Microcephaly, craniofacial malformations, and limb and heart defects are common in affected infants. Short adult stature and development of a range of adult maladaptive behaviors have also been associated with fetal alcohol syndrome.

Women with alcohol-related disorders have a 35 percent risk of having a child with defects. Although the precise mechanism of the damage to the fetus is unknown, the damage seems to result from exposure in utero to ethanol or to its metabolites; alcohol may also cause hormone imbalances that increase the risk of abnormalities.

PROGNOSIS

Between 10 and 40 percent of alcoholic persons enter some kind of formal treatment program during the course of their alcohol problems.

A number of prognostic signs are favorable. First is the absence of preexisting antisocial personality disorder or a diagnosis of other

substance abuse or dependence. Second, evidence of general life stability with a job, continuing close family contacts, and the absence of severe legal problems also bodes well for the patient. Third, if the patient stays for the full course of the initial rehabilitation (perhaps 2 to 4 weeks), the chances of maintaining abstinence are good. The combination of these three attributes predicts at least a 60 percent chance for 1 or more years of abstinence. Few studies have documented the long-term course, but researchers agree that 1 year of abstinence is associated with a good chance for continued abstinence over an extended period. Alcoholic persons with severe drug problems (especially intravenous drug use or cocaine or amphetamine dependence) and those who are homeless may have only a 10 to 15 percent chance of achieving 1 year of abstinence, however.

Accurately predicting whether any speci□c person will achieve or maintain abstinence is impossible, but the prognostic factors listed earlier are associated with an increased likelihood of abstinence. The factors re□ecting life stability, however, probably explain only 20 percent or less of the course of alcohol use disorders. Many forces that are di□cult to measure a□ect the clinical course signi□cantly; they are likely to include such intangibles as motivational level and the quality of the patient's social support system.

In general, alcoholic persons with preexisting independent major psychiatric disorders—such as antisocial personality disorder, schizophrenia, and bipolar I disorder—are likely to run the course of their independent psychiatric illness. Thus, for example, clinicians must treat the patient with bipolar I disorder who has secondary alcoholism with appropriate psychotherapy and lithium (Eskalith), use relevant psychological and behavioral techniques for the patient with antisocial personality disorder, and o□er appropriate antipsychotic medications on a long-term basis to the patient with schizophrenia. The goal is to minimize the symptoms of the independent psychiatric disorder in the hope that greater life stability will be associated with a better prognosis for the patient's alcohol problems.

TREATMENT AND REHABILITATION

Three general steps are involved in treating the alcoholic person after the disorder has been diagnosed: intervention, detoxi□cation, and rehabilitation. These approaches assume that all possible e□orts have been made to optimize medical functioning and to address psychiatric emergencies. Thus, for example, an alcoholic person with symptoms of depression su□ciently severe to be suicidal requires inpatient hospitalization for at least several days until the suicidal ideation disappears. Similarly, a person presenting with cardiomyopathy, liver

difficulties, or gastrointestinal bleeding first needs adequate treatment of the medical emergency.

The patient with alcohol abuse or dependence must then be brought face-to-face with the reality of the disorder (intervention), be detoxi□ed if needed, and begin rehabilitation. The essentials of these three steps for an alcoholic person with independent psychiatric syndromes closely resemble the approaches used for the primary alcoholic person without independent psychiatric syndromes. In the former case, however, the treatments are applied after the psychiatric disorder has been stabilized to the extent possible.

Intervention

The goal in the intervention step, which has also been called *confrontation*, is to break through feelings of denial and help the patient recognize the adverse consequences likely to occur if the disorder is not treated. Intervention is a process aimed at maximizing the motivation for treatment and continued abstinence.

This step often involves convincing patients that they are responsible for their own actions while reminding them of how alcohol has created signi□cant life impairments. The psychiatrist often □nds it useful to take advantage of the person's chief presenting complaint, whether it is insomnia, di□culties with sexual performance, an inability to cope with life stresses, depression, anxiety, or psychotic symptoms. The psychiatrist can then explain how alcohol has either created or contributed to these problems and can reassure the patient that abstinence can be achieved with a minimum of discomfort.

JP, a 47-year-old physician, was confronted regarding his alcohol-related behaviors by his wife and 21-year-old daughter. They told him about his slurred speech on several recent occasions when the daughter called home, as well as a large number of wine bottles in the trash each week. JP's wife complained of the hours he spent alone in his study and his practice of staying up after she went to bed, retiring later with alcohol on his breath. She also related her concern about his consumption of about 10 or 12 drinks at a recent party, with the resulting tendency to isolate himself from the other guests. She then reminded him of his need to pack liquor when they go on trips where alcohol may not be readily available, and the tremor of his hands some mornings after being drunk the night before. The family shared their concern directly with JP at a time when he was not actively intoxicated, emphasizing speci□c times and events when his impairment with alcohol occurred. They had also made an appointment with the clinician at an alcohol and drug treatment program so that a next step could be established if the intervention was successful. (Adapted from Marc A. Schuckit, M.D.)

A physician intervening with a patient can use the same nonjudgmental but persistent approach each time an alcohol-related impairment is identi□ed. It is the persistence rather than exceptional interpersonal skills that usually gets results. A single intervention is rarely su□cient.

Most alcoholic persons need a series of reminders of how alcohol contributed to each developing crisis before they seriously consider abstinence as a long-term option.

Family

The family can be of great help in the intervention. Family members must learn not to protect the patient from the problems caused by alcohol; otherwise, the patient may not be able to gather the energy and the motivation necessary to stop drinking. In addition, during the intervention stage, the family can suggest that the patient meet with persons who are recovering from alcoholism, perhaps through AA, and family members can meet with groups, such as Al-Anon, that reach out to family members. Those support groups for families meet many times a week and help family members and friends see that they are not alone in their fears, worry, and feelings of guilt. Participants share coping strategies and help each other □nd community resources. The groups can be most useful in helping family members rebuild their lives, even if the alcoholic person refuses to seek help.

Detoxification

Most persons with alcohol dependence have relatively mild symptoms when they stop drinking. If the patient is in relatively good health, is adequately nourished, and has a good social support system, the depressant withdrawal syndrome usually resembles a mild case of the □u.

Even intense withdrawal syndromes rarely approach the severity of symptoms described by some early textbooks in the field.

The essential □rst step in detoxification is a thorough physical examination. In the absence of a serious medical disorder or combined drug abuse, severe alcohol withdrawal is unlikely. The second step is to o□er rest, adequate nutrition, and multiple vitamins, especially those containing thiamine.

Mild or Moderate Withdrawal. Withdrawal develops because the brain has physically adapted to the presence of a brain depressant and cannot function adequately in the absence of the drug. Giving sufficient brain depressant on the first day to diminish symptoms and then weaning the patient off the drug over the next 5 days offers most patients optimal relief and minimizes the possibility that severe withdrawal will develop. Any depressant—including alcohol, barbiturates, or any of the benzodiazepines—can work, but most clinicians choose a benzodiazepine for its relative safety. Adequate treatment can be given with either short-acting drugs (e.g., lorazepam), or longacting

substances (e.g., chlordiazepoxide and diazepam).
An example of treatment is the administration of 25 mg of chlordiazepoxide by mouth three or four times a day on the □rst day, with a notation to skip a dose if the patient is
asleep or feeling sleepy. An additional one or two 25-mg doses can be given during the □rst 24 hours if the patient is jittery or shows signs of increasing tremor or autonomic
dysfunction. Whatever benzodiazepine dosage is required on the □rst day can be decreased by 20 percent each subsequent day, with a resulting need for no further medication after
4 or 5 days. When giving a long-acting agent, such as chlordiazepoxide, the clinician must avoid producing excessive sleepiness through overmedication; if the patient is sleepy, the
next scheduled dose should be omitted. When taking a short-acting drug, such as lorazepam, the patient must not miss any dose because rapid changes in benzodiazepine
concentrations in the blood can precipitate severe withdrawal.
A social model program of detoxi□cation saves money by avoiding medications while using social supports. This less-expensive regimen can be helpful for mild or moderate
withdrawal syndromes. Some clinicians have also recommended β-adrenergic receptor antagonists (e.g., propranolol [Inderal]) or α-adrenergic receptor agonists (e.g., clonidine),
although these medications do not appear to be superior to the benzodiazepines. Unlike the brain depressants, these other agents do little to decrease the risk of seizures or
delirium.
Severe Withdrawal. For the approximately 1 percent of alcoholic patients with extreme autonomic dysfunction, agitation, and
confusion—that is, those with alcoholic withdrawal delirium, or DTs—no optimal treatment has yet been developed. The first step is to ask
why such a severe and relatively uncommon withdrawal syndrome has occurred; the answer often relates to a severe concomitant medical
problem that needs immediate treatment. The withdrawal symptoms can then be minimized through the use of either benzodiazepines (in
which case high doses are sometimes required) or antipsychotic agents, such as haloperidol. Once again, on the first or second day, doses are
used to control behavior, and the patient can be weaned off the medication by about the fifth day.
Another 1 percent of patients may have a single grand mal convulsion; the rare person has multiple □ts, with the peak incidence on the second day of withdrawal. Such patients

require neurological evaluation, but in the absence of evidence of a seizure disorder, they do not benefit from anticonvulsant drugs.

Protracted Withdrawal. Symptoms of anxiety, insomnia, and mild autonomic overactivity are likely to continue for 2 to 6 months after the acute withdrawal has disappeared. Although no pharmacological treatment for this syndrome appears appropriate, it is possible that some of the medications used for the rehabilitation phase, especially acamprosate (Campral), may work by diminishing some of these symptoms. It is important that the clinician warn the patient that some level of sleep problems or feelings of nervousness might remain after acute withdrawal and discuss cognitive and behavioral approaches that might be appropriate to helping the patient feel more comfortable.

These protracted withdrawal symptoms may enhance the probability of relapse.

Rehabilitation

For most patients, rehabilitation includes three major components: (1) continued e□orts to increase and maintain high levels of motivation for abstinence; (2) work to help the patient readjust to a lifestyle free of alcohol; and (3) relapse prevention. Because these steps are carried out in the context of acute and protracted withdrawal syndromes and life crises, treatment requires repeated presentations of similar materials that remind the patient how important abstinence is and that help the patient develop new day-to-day support systems and coping styles.

No single major life event, traumatic life period, or identi□able psychiatric disorder is known to be a unique cause of alcoholism. In addition, the e□ects of any causes of alcoholism are likely to have been diluted by the e□ects of alcohol on the brain and the years of an altered lifestyle, so that the alcoholism has developed a life of its own. This is true even though many alcoholic persons believe that the cause was depression, anxiety, life stress, or pain syndromes. Research, data from records, and resource persons usually reveal that alcohol contributed to the mood disorder, accident, or life stress, not vice versa.

The same general treatment approach is used in inpatient and outpatient settings. Selection of the more expensive and intensive inpatient mode often depends on evidence of additional severe medical or psychiatric syndromes, the absence of appropriate nearby outpatient groups and facilities, and the patient's history of having failed in outpatient care. The treatment process in either setting involves intervention, optimizing physical and psychological functioning, enhancing motivation, reaching out to family, and using the □rst 2 to 4 weeks of care as an intensive period of help. Those e□orts must be followed by at least 3 to 6 months of less frequent outpatient care.

Outpatient care uses a combination of individual and group counseling, judicious avoidance of psychotropic medications unless needed for independent disorders, and involvement in such self-help groups as AA.

Counseling. Counseling efforts in the first several months should focus on day-to-day life issues to help patients maintain a high level of motivation for abstinence and to enhance their functioning. Psychotherapy techniques that provoke anxiety or that require deep insights have not been shown to be of benefit during the early months of recovery and, at least theoretically, may actually impair efforts at maintaining abstinence. Thus, this discussion focuses on the efforts likely to characterize the first 3 to 6 months of care.

Counseling or therapy can be carried out in an individual or group setting; few data indicate that either approach is superior. The technique used is not likely to matter greatly and usually boils down to simple day-to-day counseling or almost any behavioral or psychotherapeutic approach focusing on the here and now. To optimize motivation, treatment sessions should explore the consequences of drinking, the likely future course of alcohol-related life problems, and the marked improvement that can be expected with abstinence.

Whether in an inpatient or an outpatient setting, individual or group counseling is usually o□ered a minimum of three times a week for the first 2 to 4 weeks, followed by less intense efforts, perhaps once a week, for the subsequent 3 to 6 months.

Much time in counseling deals with how to build a lifestyle free of alcohol. Discussions cover the need for a sober peer group, a plan for social and recreational events without drinking, and approaches for reestablishing communication with family members and friends.

The third major component, relapse prevention, □rst identi□es situations in which the risk for relapse is high. The counselor must help the patient develop modes of coping to be used when the craving for alcohol increases or when any event or emotional state makes a return to drinking likely. An important part of relapse prevention is reminding the patient about the appropriate attitude toward slips. Short-term experiences with alcohol can never be used as an excuse for returning to regular drinking. The e□orts to achieve and maintain a sober lifestyle are not a game in which all bene□ts are lost with that □rst sip. Rather, recovery is a process of trial and error; patients use slips that occur to identify high-risk situations and to develop more appropriate coping techniques.

Most treatment e□orts recognize the e□ects that alcoholism has on the signi□cant persons in the patient's life, and an important aspect of recovery involves helping family members and close friends understand alcoholism and realize that rehabilitation is an ongoing process that

lasts for 6 to 12 or more months. Couples and family counseling and support groups for relatives and friends help the persons involved to rebuild relationships, to learn how to avoid protecting the patient from the consequences of any drinking in the future, and to be as supportive as possible of the alcoholic patient's recovery program.

Medications. If detoxification has been completed and the patient is not one of the 10 to 15 percent of alcoholic persons who have an independent mood disorder, schizophrenia, or anxiety disorder, little evidence favors prescribing psychotropic medications for the treatment of alcoholism. Lingering levels of anxiety and insomnia as part of a reaction to life stresses and protracted abstinence should be treated with behavior modification approaches and reassurance. Medications for these symptoms (including benzodiazepines) are likely to lose their effectiveness much faster than the insomnia disappears; thus, the patient may increase the dose and have subsequent problems. Similarly, sadness and mood swings can linger at low levels for several months. Controlled clinical trials, however, indicate no benefit in prescribing antidepressant medications or lithium to treat the average alcoholic person who has no independent or long-lasting psychiatric disorder. The mood disorder will clear before the medications can take effect, and patients who resume drinking while on the medications face significant potential dangers. With little or no evidence that the medications are effective, the dangers significantly outweigh any potential benefits from their routine use.

One possible exception to the proscription against the use of medications is the alcohol-sensitizing agent disul□ram. Disul□ram is given in daily doses of 250 mg before the patient is discharged from the intensive □rst phase of outpatient rehabilitation or from inpatient care. The goal is to place the patient in a condition in which drinking alcohol precipitates an uncomfortable physical reaction, including nausea, vomiting, and a burning sensation in the face and stomach. Few data prove that disul□ram is more e□ective than a placebo, however, probably because most persons stop taking the disul□ram when they resume drinking. Many clinicians have stopped routinely prescribing the agent, partly in recognition of the dangers associated with the drug itself: mood swings, rare instances of psychosis, the possibility of increased peripheral neuropathies, the relatively rare occurrence of other signi□cant neuropathies, and potentially fatal hepatitis.

Two additional promising pharmacological interventions have recently been studied. The □rst involves the opioid antagonist naltrexone (ReVia), which at least theoretically is believed possibly to decrease the craving for alcohol or blunt the rewarding e□ects of drinking. In any

event, two relatively small (approximately 90 patients on the active drug across the studies) and short-term (3 months of active treatment)
investigations using 50 mg per day of this drug had potentially promising results.
The second medication of interest, acamprosate (Campral), has been tested in more than 5,000 alcohol-dependent patients in Europe.
This drug is not yet available in the United States. Used in dosages of approximately 2,000 mg per day, this medication was associated with
approximately 10 to 20 percent more positive outcomes than placebo when used in the context of the usual psychological and behavioral
treatment regimens for alcoholism. The mechanism of action of acamprosate is not known, but it may act directly or indirectly at GABA
receptors or at NMDA sites, the e□ects of which alter the development of tolerance or physical dependence on alcohol.

Table 20.2-7
Medications for Treating Alcohol Dependence

Another medication with potential promise in the treatment of alcoholism is the non-benzodiazepine antianxiety drug buspirone (BuSpar), although the effect of this drug on alcohol rehabilitation is inconsistent between studies. No evidence exists that antidepressant medications,
such as the selective serotonin reuptake inhibitors (SSRIs), lithium, or antipsychotic medications, are significantly effective in the treatment
of alcoholism.

Alcoholics Anonymous. Clinicians must recognize the potential importance of self-help groups such as AA. Members of AA have
help available 24 hours a day, associate with a sober peer group, learn that it is possible to participate in social functions without drinking,
and are given a model of recovery by observing the accomplishments of sober members of the group.
Learning about AA usually begins during inpatient or outpatient rehabilitation. The clinician can play a major role in helping patients
understand the di□erences between speci□c groups. Some are composed only of men or women, and others are mixed; some meetings are
composed mostly of blue collar men and women, whereas others are mostly for professionals; some groups place great emphasis on
religion, and others are eclectic. Patients with coexisting psychiatric disorders may need some additional education about AA.
should remind them that some members of AA may not understand their special need for medications and should arm the patients with
ways

SRI LAKSHMI NARAYANA INSTITUTE OF MEDICAL SCIENCE

VALUE ADDED COURSE STUDENT DETAILS

ALCOHOL DEADDICTION AND ITS MANAGEMENT FOR FINAL YEAR STUDENTS

S.No	Register No	Students List	Department	Signature
1	U15MB360	SELVASRINIVASAN. B	Psychiatry	
2	U15MB361	SENTHILKUMARAN. A	Psychiatry	
3	U15MB362	SHAHARA ZAD .S	Psychiatry	
4	U15MB363	SHAKTHI. K	Psychiatry	
5	U15MB364	SHALINI. A	Psychiatry	
6	U15MB369	SIVA SAKTHI VELAN .A.V	Psychiatry	
7	U15MB370	SIVAKUMAR. S	Psychiatry	
8	U15MB371	SIVANAMBI .S	Psychiatry	
9	U15MB372	SOPHIYA .L	Psychiatry	
10	U15MB373	SOUNDARIYA.M	Psychiatry	
11	U15MB374	SOWMYA LAKSHMI .I	Psychiatry	

12	U15MB375	SREERAM.M	Psychiatry	
13	U15MB376	SRINIVASAN.R	Psychiatry	
14	U15MB377	SRUTHI .S	Psychiatry	
15	U15MB378	SUBALAKSHMI .C	Psychiatry	

SRI LAKSHMI NARAYANA INSTITUE OF HIGHER EDUCATON AND RESEARCH

Alcohol dependence and de-addiction

Candidate Name		Assessor Name	
Date of Assessment		Assessor Position	

MULTIPLE CHOICE QUESTIONS

Course Code: PSYC09

I. ANSWER ALL THE QUESTIONS

1. Consumption of this causes increase in the fat synthesis, dilation of blood vessels, low blood sugar and stomach-inflammation

(a) drug addiction and tobacco

(b) alcohol

(c) tobacco

(d) drug addiction

2. This causes tunnel vision

(a) Smoking

(b) Alcohol

(c) Barbiturates

(d) Vitamin A deficiency

3. After drinking alcohol, consumption of this leads to death

(a) Morphine

(b) Opium

(c) Barbiturate

(d) All of these

4. This drug along with alcohol generates marked drowsiness otherwise does not produce sedative effect

(a) Marijuana

(b) Valium

(c) Antihistamine

(d) Barbiturate

5. Constituent of alcoholic beverage is

(a) Propyl alcohol

(b) Methyl alcohol

(c) Ethyl alcohol

(d) Mix of all of these

6. This is a result of the appearance of the fatty liver syndrome

(a) synthesis of fatty acids, fat and glycerols

(b) synthesis of fat from alcohol

(c) synthesis of fat from amino acids

(d) excessive synthesis of fat from fatty acids

7. Alcoholism causing fatty acid syndrome is called

(a) Cirrhosis

(b) Neuritis

(c) Gastritis

(d) Nephritis

8. This is a withdrawal symptom of alcohol consumption

(a) Delirium

(b) Nausea and vomiting

(c) swollen and patchy face

(d) all of these

9. This causes the maximum accumulation of fat in the liver

(a) meat and egg

(b) alcohol

(c) saturated fat

(d) starch

10. This is a consequence of alcohol addiction

(a) psychosis, hypertension and fatty liver syndrome

(b) cardiovascular diseases, hypertension and fatty liver syndrome

(c) ulcers, all types of mental illness, vitamin deficiency, cardiovascular diseases

(d) all of these

SRI LAKSHMI NARAYANA INSTITUE OF HIGHER EDUCATON AND RESEARCH

Alcohol dependence and de-addiction

Candidate Name	Selvasrinivasan.V	Assessor Name	DR. ARUN
Date of Assessment	15.4.2019	Assessor Position	ASSOCIATE PROFESSOR.

UNIVERSITY REG NO:- U1SMB360

10/10

MULTIPLE CHOICE QUESTIONS

Course Code: PSYC09

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-1-

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AND RESEARCH

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SRI LAKSHMI NARAYANA INSTITUE OF HIGHER EDUCATON AND RESEARCH

Alcohol dependence and de-addiction

Candidate Name	Senthilkumaran A	Assessor Name	Dr. ARUN
Date of Assessment	15.4.2019	Assessor Position	ASSOCIATE PROFESSOR

UNIVERSITY REG NO: U15MB361

2
10

MULTIPLE CHOICE QUESTIONS

Course Code: PSYC09

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Sri Lakshmi Narayana Institute of Medical Sciences

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CERTIFICATE OF MERIT

This is to certify that SRUTHI .S has actively participated in the Value Added Course on Alcohol dependence and de-addiction held during April - June 2019 Organized by Sri Lakshmi Narayana Institute of Medical Sciences, Pondicherry- 605 502, India.

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Sri Lakshmi Narayana Institute of Medical Sciences

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CERTIFICATE OF MERIT

This is to certify that SUBALAKSHMI .C has actively participated in the Value Added Course on **Alcohol dependence and de-addiction** held during April - June 2019 Organized by Sri Lakshmi Narayana Institute of Medical Sciences, Pondicherry- 605 502, India.

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SRI LAKSHMI NARAYANA INSTITUE OF HIGHER EDUCATON AND RESEARCH

Student Feedback Form

Course Name: **Alcohol deaddiction**
Subject Code: **PSYC09**

Name of Student: _____ Roll No.: _____

We are constantly looking to improve our classes and deliver the best training to you. Your evaluations, comments and suggestions will help us to improve our performance

Sl. NO	Particulars	1	2	3	4	5
1	Objective of the course is clear					
2	Course contents met with your expectations					
3	Lecturer sequence was well planned					
4	Lectures were clear and easy to understand					
5	Teaching aids were effective					
6	Instructors encourage interaction and were helpful					
7	The level of the course					
8	Overall rating of the course	1	2	3	4	5

Rating: 5 – Outstanding; 4 - Excellent; 3 – Good; 2– Satisfactory; 1 - Not-Satisfactory

Suggestions if any:

Signature

Date:

SRI LAKSHMI NARAYANA INSTITUE OF HIGHER EDUCATON AND RESEARCH

Student Feedback Form

Course Name: ALCOHOL DEADDICTION

Subject Code: PSYC09

Name of Student: SRINIVASAN. R Roll No.: U15MB376

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Sl. NO	Particulars	1	2	3	4	5
1	Objective of the course is clear			✓		
2	Course contents met with your expectations				✓	
3	Lecturer sequence was well planned					✓
4	Lectures were clear and easy to understand				✓	
5	Teaching aids were effective			✓		
6	Instructors encourage interaction and were helpful					✓
7	The level of the course				✓	
8	Overall rating of the course	1	2	3	4	5

** Rating: 5 – Outstanding; 4 - Excellent; 3 – Good; 2– Satisfactory; 1 - Not-Satisfactory*

Suggestions if any:

R. Srinivasan

Student Feedback Form

Course Name: **ALCOHOL DEADDICTION**

Subject Code: **PSYC09**

Name of Student: SREERAM-M Roll No.: U15MB375

We are constantly looking to improve our classes and deliver the best training to you. Your evaluations, comments and suggestions will help us to improve our performance

Sl. NO	Particulars	1	2	3	4	5
1	Objective of the course is clear					✓
2	Course contents met with your expectations				✓	
3	Lecturer sequence was well planned					✓
4	Lectures were clear and easy to understand				✓	
5	Teaching aids were effective					✓
6	Instructors encourage interaction and were helpful			✓		
7	The level of the course				✓	
8	Overall rating of the course	1	2	3 ✓	4	5

** Rating: 5 – Outstanding; 4 - Excellent; 3 – Good; 2– Satisfactory; 1 - Not-Satisfactory*

Suggestions if any:

M. Sreeram
Signature

SRI LAKSHMI NARAYANA INSTITUE OF HIGHER EDUCATON AND RESEARCH

Date: 30-03-2019

From

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Department of Psychiatry,
Sri Lakshmi Narayana Institute of Medical Sciences
Bharath Institute of Higher Education and Research,
Chennai.

Through Proper Channel

To

The Dean,
Sri Lakshmi Narayana Institute of Medical Sciences
Bharath Institute of Higher Education and Research,
Chennai.

Sub: Completion of value-added course:, Alcohol dependence and de-addiction

Dear Sir,

With reference to the subject mentioned above, the department has conducted the value-added course titled: : **Alcohol dependence and de-addiction**. We solicit your kind action to send certificates for the participants, that is attached with this letter. Also, I am attaching the photographs captured during the conduct of the course.

Kind Regards,

Dr. V. R. SRIDHAR, MD.,D.P.M.,
Reg. No: 30995
Professor & HOD, Psychiatry
Sri Lakshmi Narayana Institute of Medical Sciences
Osudu, Kudapakkam, Puducherry-605 502.

Encl: Certificates

Photographs

SRI LAKSHMI NARAYANA INSTITUE OF HIGHER EDUCATON AND RESEARCH

PSYC09: **Alcohol dependence and de-addiction**

APRIL TO JUNE 2019