Introduction
The use of alcohol for purposes of relaxation or socializing by mankind has been reported throughout history in most civilizations. The social approval to alcohol use has varied from strong disapproval to being actively encouraged. In the 20th Century Western World, the use of alcohol as well as the related disorders, has been increasing rapidly. The recognition of alcoholism as a disease occurred during the early 1950s by the World health Organization (WHO) Jellinek’s description of “disease concept of alcoholism” and the subtypes of alcoholism generated a lot of interest. It proved to be stimulus for systematic descriptions of alcohol related problems. The first description of “Alcohol Dependence Syndrome” in 1976 by Edwards and Gross emphasized inability to control consumption, salience of drink seeking behaviour, and narrowing of drinking repertoire as the characteristics besides the phenomena of tolerance and withdrawal. The concept of alcoholism can be well understood by the entymological origin of the term. Like all other “isms”, alcohol becomes a way of life in persons with alcoholism. As in All other “isms”, in this one too, alcohol becomes the “raison d’ et re” or the reason for existence. In India, although alcohol use in ancient times and cannabis and affim (raw opium) in more recent times have been known and reported for some time, substance use problems have been recognized to have a significant importance as a public health problems and in various other facets of life only very recently.

A large number of persons are involved in treating alcohol dependent individuals. They include, General Physicians, Psychiatrists, Psychologist, Social Worker, lay volunteer, spiritual leader and even recovered patient as a result, there is considerable difference of opinion on treatment issues. This is mainly due to their different conceptual models of treatment. Thus there is a need for a common and uniform treatment guideline which can help in comprehensive management of these patients.

II Current definitions and diagnostic guidelines
Alcohol consumption occurs along a continuum, with considerable variability in drinking patterns among individuals. There is no sharp demarcation between “social or “moderate”, drinking and “problem” or “harmful” drinking (Babor et al 1987a). It is clear, however, that as average alcohol consumption and frequency of intoxication increase, so does the incidence of medical and psychosocial problems (Kranzler et al. 1990). The most visible group of people affected by alcohol problems are those who have developed a syndrome of alcohol dependence and who are commonly referred to as alcoholics. A less prominent group consists of those persons who experience problems with their drinking but who are not dependent on alcohol. These individuals are variously termed alcohol abusers, problem drinkers, and harmful drinkers. These two “worlds” of alcohol problems may require different approaches to diagnosis and clinical management.
The definitions of alcoholism have been constantly in the process of change. The idea of considering dependence to alcohol as significantly different from the dependence on other drugs of abuse is not favored any longer. The conditions of alcohol abuse and alcohol dependence are seen as being very similar in their clinical profile to these conditions as in case of other substances. According to ICD-10 “Dependence Syndrome” it is defined as a cluster of physiological, behavioural and cognitive phenomena in which the use of a substance or a class of substances takes on a much higher priority for a given individual than other behaviours that once had great value. A central descriptive characteristic of the dependence syndrome is the desire to take psychoactive drugs, alcohol or tobacco. There may be evidence that return to substance use after a period of abstinence leads to a more rapid appearance of the syndrome than occurs with non dependent individuals. The diagnostic guidelines as per ICD-10 require that three or more of the six characteristics be established at some point during the previous year.

**Diagnostic guidelines for Dependence Syndrome in ICD-10 (WHO, 1992)**

A definite diagnosis of dependence should usually be made only if three or more of the following have been experienced or exhibited at some time during the previous year:

a) A strong desire or sense of compulsion to take the substance;

b) Difficulties in controlling substance-taking behaviour in terms of its onset, termination, or levels of use;

c) A physiological withdrawal state when substance use has ceased or has been reduced, as evidenced by the characteristic withdrawal syndrome for the substance; or use of the same (or a closely related) substance with the intention of relieving or avoiding withdrawal symptoms;

d) Evidence of tolerance, such that increased doses of the psychoactive substance are required in order to achieve effect originally produced by lower;

e) Progressive neglect of alternative pleasures or interests because of psychoactive substance use, increased amount of time necessary to obtain or take the substance or to recover from its effects;

f) Persisting with substance use despite clear evidence of overtly harmful consequence, such as harm to the liver through excessive drinking, depressive mood states consequent to periods of heavy substance use or drug related impairment of cognitive functioning; efforts should be made to determine that user was actually, or could be expected to be, aware of the nature of extent of the harm.

The ICD-10 has opted for the newer concept of “Harmful Use” to define those individuals who do not satisfy the definition of dependence syndrome and yet, do have problems due to substance use. Harmful Use has been described as “a pattern of psychoactive substance use that is causing damage to health, the diagnosis requiring that actual damage should have been caused to the mental or physical health of the user”.

**III Etiology and Pathophysiology**

Alcoholism is a complex, multifaceted disorder which has long been recognized to run in families. There is substantial evidence from twin research and adoption studies that a major genetic component is operative in the development of alcoholism. Nonetheless, the disorder is etiologically complex, with a variety of other vulnerability factors (Goldman 1993, Gelernter 1995). It has been estimated that
there is a sevenfold risk of alcoholism in first-degree relatives of alcohol-dependent individuals, with male relatives of male alcohol-dependent individuals having the greatest risk for the disorder (Merikangas 1990). However, the majority of alcohol dependent individuals do not have a first-degree relative who is alcohol dependent. This underscores the fact that the risk for alcohol dependence is also determined by environment factors, which may interact in complex ways with genetics.

**ETIOLOGICAL SUBTYPES OF ALCOHOLICS**

Another approach to understanding the etiology of alcoholism is to identify distinct subtypes of alcoholics. A variety of typologic approaches have been proposed to simplify the diverse phenomena associated with alcoholism (Nixon 1993, Bohn and Meyer 1994, Babor and Laureman 1986). These include unidimensional approaches based on drinking history, drinking pattern, severity of alcohol dependence, family history of alcoholism, gender, personality style, comorbid psychopathology, cognitive impairment, and sociopathy, as well as multidimensional approaches that combine these characteristics into meaningful clusters. The best known of these typologies is the Type1/Type2 distinction developed by Cloninger and collegues (1981) from studies of adopted sons of Swedish alcoholics.

**Comparison of Two Typologies of Alcoholism**

<table>
<thead>
<tr>
<th>Variables</th>
<th>Late onset</th>
<th>Early onset</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Type 1</td>
<td>Type A</td>
</tr>
<tr>
<td>Age at onset (yr)</td>
<td>After 25</td>
<td>Male&gt;30</td>
</tr>
<tr>
<td>Sex</td>
<td>Male or female</td>
<td>Male: female=0.8</td>
</tr>
<tr>
<td>Sociopathy Binge drinking</td>
<td>Low frequent</td>
<td>Low frequent</td>
</tr>
<tr>
<td>Inability to abstain</td>
<td>Uncommon</td>
<td>Uncommon</td>
</tr>
<tr>
<td>Comorbid depression</td>
<td>High</td>
<td>Low</td>
</tr>
<tr>
<td>Heritability</td>
<td>Low</td>
<td>Probably low</td>
</tr>
</tbody>
</table>

**EPIDEMIOLOGY OF ALCOHOL ABUSE/DEPENDENCE**

Around two billion people consume alcohol and around 76.3 million are diagnosed to have alcohol used disorders. 3.2% of deaths and 4% of Disability-Adjusted Life Years (DALY)'* are attributed to alcohol use. Alcohol consumption has been associated with more than 60 types of disease and injury (WHO,2002). In India prevalence (current use) of alcohol is 21.4% (NHS, 2004). Amongst treatment seekers prevalence is 43.9% (DAMS, 2004). The prevalence rates of alcohol use and abuse vary across countries and cultures, and since the genetic predisposition gets expression in the context of the socio-cultural milieu and practices. Life-time prevalence rates of alcohol abuse and dependence have varied from 0.5 per cent to 22 per cent. In India, such direct epidemiological indicators like hospital admission rates, deaths from cirrhosis and sales figures for alcohol clearly suggest increasing patterns of alcohol use and dependence. Rates of abuse and dependence have been found to be consistently higher in male than in women, young and adult age groups than in others, in specific ethnic groups, and in certain occupational groups such as drivers, chefs & barmen, executives & salesman with expense accounts, defence personnel and medical doctors.
Patterns of drinking and the types of problems associated with alcohol misuse differ markedly throughout the world (Room et al. 2002). Thirteen percent of the adult population in the United States has history of alcohol dependence or alcohol abuse and the 12 month prevalence of alcohol dependence in between 4% and 5% (Grant et al 1997).

Analyses of world trends in alcohol consumption indicates that levels of alcohol use have been stable or slightly decreasing in Western Europe and the US since 1960.

Despite increased availability of alcoholic beverages worldwide, cultural influences continue to exert a strong influence on national patterns of drinking. While the highest alcohol consumption rates are generally found among the industrialized countries of Europe and North America continent, the lowest consumption rate are found in developing countries that are dominated by Islam, which proscribe the use of alcohol. The percentage of the drinking adult population ranges from a high of 86% in North and Central European countries to less than 10% of adults in Islamic countries such as Pakistan and Iraq (Room et al 2002).

Several large-scale community studies conducted since 1980 have provided estimates of the lifetime and past year prevalence of alcohol use disorders in the general population. For example, the National Comorbidity Study (NCS), a representative household survey of 8,098 persons aged 15 to 54 years that was conducted between 1990 and 1992, assessed lifetime and past-year alcohol disorders using DSM-III-R criteria (American Psychiatry Association 1987). The NCS estimated that the lifetime prevalence of alcohol abuse and alcohol dependence for adults 18 to 54 years old were 9.4 and 14.1% respectively, indicating that more than one-in-five young to middle-aged adults in the US have had a pattern of alcohol use that met criteria for lifetime alcohol abuse and dependence during the 12 months preceding the interview were 2.5 and 4.4%, respectively (Kessler et al. 1997).

Summary of the Studies on Epidemiology of Alcohol Use in India (Singh, 1989)

<table>
<thead>
<tr>
<th></th>
<th>General population</th>
<th>Students</th>
<th>Medical Professionals</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ever use</td>
<td>25.6-74.2%</td>
<td>21.6-58.4%</td>
<td>85-66.7%</td>
</tr>
<tr>
<td>Use in last one year</td>
<td>19.0-82.5%</td>
<td></td>
<td>-</td>
</tr>
<tr>
<td>Use in last one month</td>
<td>-</td>
<td>9.3-12.7</td>
<td>-</td>
</tr>
</tbody>
</table>

**ii STEPS FOR IDENTIFICATION OF ALCOHOL DEPENDENCE**

**Step I – SUSPICION**

The suspicion about alcohol use disorders can arise by evidence for problems in the spheres of work, marriage, finances or with the law. Intoxications with alcohol or the occurrence of withdrawal features of tremors, irritability, nausea & vomiting, insomnia or hallucinations in any of their patients should alert clinicians. Report of excessive use of alcohol, even if it is in the form of a complaint or a criticism, by the patient’s spouse or any any other family member is well worth looking into. Some medical or psychological symptoms/conditions like Liver disease, acid peptic symptoms, neuropathy, anxiety, memory lapses and repeated accidents are well known to be more commonly associated with alcohol dependence. Tentative suspicion so aroused must be followed up by specific measures for identification.
Step II - SCREENING

Screening Instruments and Rating Scales

1. Screening
   - MAST (Michigan Alcoholism Screening Test) (Selzer et al, 1971)
   - CAGE Questionnaire (Mayfield et al 1974)
   - AUDIT (Alcohol Use Disorders Identification Test) (WHO 1989)

2. Withdrawal rating scale
   - CIWA-A (Clinical Institute Withdrawal Assessment-Alcohol Scale) (Sellers 1980)

3. Scales for assessment of severity
   - ASI (Addiction Severity Index) (Mclellan 1980)

Biological markers of alcoholism (Vaswani et al 1997)

<table>
<thead>
<tr>
<th>Test</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Asparate Aminotransferase (AST)</td>
<td>If raised indicates liver damages from any cause. Useful when combined with other tests.</td>
</tr>
<tr>
<td>And Alanine Aminotransferase (ALT)</td>
<td></td>
</tr>
<tr>
<td>Gammaglutamyl Transferase (GGT)</td>
<td>Most commonly used test. High levels are strongly suggestive of alcoholic liver damage, but can also be raised in liver damage due to other causes. Useful test in combination with other tests and also to monitor treatment results or in motivating patients to stop excess consumption.</td>
</tr>
<tr>
<td>Mean Corpuscular Volume (MCV)</td>
<td>Useful test in combination with other tests and commonly used with GGT.</td>
</tr>
<tr>
<td>Erythrocyte Aldehyde Dehydrogenase (ADH)</td>
<td>Levels are decreased in long term abusers of alcohol.</td>
</tr>
<tr>
<td>Carbohydrate Deficient Transferrin (CDT)</td>
<td>Increased levels in long-term abusers of alcohol. Can distinguish recent excessive consumption from abstinence or very light drinking.</td>
</tr>
</tbody>
</table>

Laboratory tests: Of the many tests used for screening, two routinely available tests viz. Mean Corpuscular Volume (MCV) and Gamma – Glutamyl-Transpeptidase (GGT) are raised in a majority of patients with alcohol dependence. Research on hospital and community based screening suggests that raised value of one or both of these measures, in the absence of other causes, is strongly linked to the ultimate diagnosis of alcohol dependence.

Screening instruments are used in epidemiological studies where large populations are to be screened for the presence of alcohol related problems. They are also useful in clinical settings like medical and surgical outpatient departments where a detailed history of substance use in every patient is difficult to obtain due to time constraints. During the past 25 years a number of self-report screening tests
have been developed to identify alcoholics as well as persons at risk of alcohol problems eg. The Michigan Alcohol Screening Test (MAST), developed by Selzer (1971). Perhaps the most widely used alcohol screening test is the CAGE (Ewing 1984), which contains only four questions:

**CAGE Questionnaire for screening for alcohol abuse/dependence**

1. Have you ever felt you ought to **cut** down on your drinking?
2. Have people **annoyed** you by criticizing your drinking?
3. Have you ever felt **guilty** about your drinking?
4. Have you ever had a drink first thing in the morning (an **eye opener**) to steady your nerves or get rid of a hangover?

**Note:** Two or more positive answers indicates alcohol abuse/dependence.

Another one is alcohol use disorders Identifications test (audit) (Saunders et al. 1993, Babor and Higgins-Biddle 2001a), a -10 item screening instrument, may be used as the first step in a comprehensive and sequential alcohol use history.

**Step III- CONFIRMATION**

The final confirmation of alcohol dependence in each individual patient will have to be made by one or more clinical interviews, exploring the details of alcohol consumption and its effects on the different aspects of life. A gentle, persuasive approach on part of the interviewer is necessary, avoiding confrontation or provocation. The criteria and guidelines require to be applied in order to arrive at an objective and scientific diagnosis.

**iii types of treatment settings & levels of care types of treatment settings**

1. **primary care physicians/NGO/individual chamber practice**
2. **District hospital psychiatric units /ngo with inpatient facility/private hospital with psychiatric unit**
3. **general hospital psychiatric unit with specialist mental health personnel, non psychiatric medical unit (gastroenterology)**
4. **specialized de addiction treatment settings (e.g alims, nimhans, pgi, ihbas)**

Most of these treatment settings play a **complementary** role. Most patients move through each of these different setting over a period of time and many patients avail their services in more than one setting during a particular treatment attempt. So these settings are not mutually exclusive.

**level of care**

1. **optimum care – reasonable level of care (I,II)**
2. **good paractice – comprehensive level of care (multidisciplinary team approach) (III, IV).**

The level of care depends on the **infrastructure** of the treatment setting, **expertise** of the personnel involve and other factors.

Each type of treatment setting would have specific components, variable duration and different technique of treatment and different types of personnel involved:
iv Phases of treatment for Alcohol Dependence

Pretreatment:
1. Identification
2. Motivational Interviewing
3. Role of family members and physicians

Detoxification
1. Diazepam (20-60 mgs. Per day) or Chlordiazepoxide (50-150 mgs. Per day)/Lorazepam
2. Thiamine (or as part of Vitamin B-Complex) 50 mgs. Thrice a day orally or 100 mgs.IM daily
3. Supportive Measures viz fluids, electrolytes etc

Intensive Treatment:
1. Brief Intervention/Simple advice
2. Disulfiram (only with consent)/Anti craving drugs (NTX, Acamprosate)
3. Group Therapy
4. Family Therapy
5. Behaviour Therapy

Posttreatment/ Aftercare:
1. Treatment contact
2. Relapse Prevention
3. Social Rehabilitation
4. Occupational Rehabilitation
5. Continued Supervision

Viii GOALS OF ALCOHOLISM TREATMENT
• Promote complete abstinence
• Stabilize acute medical (including alcohol withdrawal) and psychiatric conditions, as needed.
• Increase motivation for recovery
• Initiate treatment for chronic medical and psychiatric conditions, as needed.
• Assist the patient in locating suitable housing (e.g. moving form a setting in which drinking is widespread), as needed.
• Enlist social support for recovery.
• Enhance coping and relapse prevention skills.
• Improve occupational functioning.
• Promote maintenance of recovery through ongoing participations in structured treatment or self-help groups

The goals of treatment vary according to the time frame and the scope envisaged. They will also differ across individual patients and can be revised from time to time. Immediate goals can be
detoxification, treatment of acute medical sequel and crisis intervention; short term goals usually target treatment of comorbid medical or psychiatric conditions, maintaining abstinence, family reintegration and vocational placement; and the long term goals focus on the larger issues of relapse prevention, occupational rehabilitation, social reintegration, abstinent life style and improvement of quality of life.

ix GUIDELINES FOR INPATIENT AND OUTPATIENT TREATMENT

A. Indication for inpatient treatment
1. Heavily intoxicated patient
2. Patient having complicated withdrawal (delirium tremens, withdrawal seizures)
4. Patient with significant psychopathology
5. Unsuccessful outpatient treatment
6. Poly substance dependence
7. Crisis in social support
8. Geographical consideration
9. Academic and research reason

B. Indication for outpatient treatment
1. Mild or moderate level of dependence
2. Short duration for dependence
3. Minimal health damage
4. Good social support

C. Unsuitability for inpatient treatment
1. Anti social personality disorder or traits
2. Significant criminal record
3. Disciplinary problems during inpatient treatment
4. Several aborted treatment attempts in inpatient settings

X ALCOHOL RELATED DISORDERS

The use of alcohol starts as a social phenomenon, leading to abuse or dependent use in some of the individuals. The occurrence of alcohol related disorders is not necessarily associated with abuse or dependent use. In fact, the average persons with alcohol related problems is likely to be neatly dressed, to have no severe signs of alcohol withdrawals, to have a job and good family support, and may have physical or psychiatric complications. In alcohol abuse, there is impairment of social, legal, interpersonal, and occupational functioning. The amount of alcohol intake, frequency, pattern of alcohol intake, type of liquor and response of persons show great variability. One person may take alcohol occasionally or dressed neatly and yet show severe alcohol related complications, whereas another person may take alcohol daily, throughout the day and yet show no impairment.
1. Alcohol Intoxication

Alcohol depresses the CNS and the extent of depression corresponds to blood alcohol concentration (BAC).

CNS Effects at Different Blood Alcohol Concentrations (BAC) (Schuckit, 1995)

<table>
<thead>
<tr>
<th>Blood alcohol concentration (BAC)</th>
<th>CNS effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>20-30mg/dl</td>
<td>Slowed motor performance and decreased thinking ability</td>
</tr>
<tr>
<td>30-80mg/dl</td>
<td>Increases motor and cognitive problem.</td>
</tr>
<tr>
<td>80-200mg/dl</td>
<td>Increased in coordination and errors in judgment, mood lability, deterioration in cognition</td>
</tr>
<tr>
<td>200-300mg/dl</td>
<td>Nystagmus, marked slurring of speech and alcohol blackouts</td>
</tr>
<tr>
<td>&gt;300mg/dl</td>
<td>Impaired vital signs and possible death</td>
</tr>
</tbody>
</table>

Clinical signs indicative of alcohol intoxication include slurred speech, lack of coordination unsteady gait, nystagmus, impairment of attention and memory, and in the most severe cases, stupor and coma. Alcohol intoxication may also present with severe disturbances in consciousness and cognition (alcohol intoxication delirium), especially when large amounts of alcohol have been ingested or after alcoholic intoxication has been sustained for extended periods. Usually, this condition subsides shortly after alcohol intoxication ends. Physical and mental status examination accompanied by analysis of blood and urine allow the clinician to rule out general medical conditions or psychiatric disorders mimicking this condition. In this regard, urine toxicology is a valuable tool in ruling out intoxication with benzodiazepines, barbiturates, or other sedatives that can present with a similar clinical picture. Collateral information from relatives or friends confirming the ingestion of alcohol is also useful, and should be actively pursued by the clinician.

2. Alcohol Withdrawal

Alcohol withdrawal is a condition that follows a reduction in alcohol consumption or an abrupt cessation of drinking in alcohol-dependent individuals. In addition to significant distress, alcohol withdrawal is also associated with impairment of social, occupational, and other areas of functioning. Uncomplicated cases of alcohol withdrawal are characterized by signs and symptoms of autonomic hyperactivity, and may include increased heart rate, increased blood pressure, hyperthermia, diaphoresis, tremor, nausea, vomiting, insomnia, and anxiety. Onset of symptoms of uncomplicated alcohol withdrawal usually occurs between 4 and 12 hours following the last drink. Symptom severity tends to peak around the second day, usually subsiding by the fourth or fifth day of abstinence. After this period, less severe anxiety, insomnia, and autonomic symptoms may persist for a few weeks, with some individuals experiencing a protracted alcohol withdrawal syndrome up to 5 or 6 months after cessation of drinking. A small but significant number of alcohol dependent individuals (10%) can experience complicated alcohol withdrawal episodes. Alcohol withdrawal delirium (also known as delirium tremens) can occur in 5% of the cases, usually between 36 and 72 hours following alcohol cessation. In addition to signs of autonomic hyperactivity, this condition is characterized by illusions, auditory, visual, or tactile hallucinations,
psychomotor agitation, fluctuating cloudiness of consciousness, disorientation. Grand mal seizures associated with alcohol withdrawal occur in 3 to 5% of the cases, typically within the first 48 hours following reduction or cessation of drinking. In both instances of complicated alcohol withdrawal, lack or delay in instituting proper treatment is associated with an increased mortality rate. Prior history of delirium tremens and/or alcohol-withdrawal seizures, older age, poor nutritional status, comorbid medical conditions, and history of high tolerance to alcohol are predictors of increased severity of alcohol withdrawal.

3. Alcohol Induced Persisting Amnestic Disorder

Continuous heavy alcohol consumption can lead to several neurological deficits caused by thiamin deficiency. Among them, alcohol-induced persisting amnestic disorder (AIPAD, also known as a Korsakoff's psychosis, due to the fantastic confabulatory stories described by patients suffering this condition) is prominent. Profound deficits in anterograde memory and some deficits in retrograde or learn characterize this condition. Cessation of drinking can lead to an improvement in memory with approximately 20% of the cases demonstrating complete recovery. However, in most cases memory deficits remain unchanged, and in some instances long-term care is needed despite sobriety.

4. Alcohol-induced Persisting Dementia

Continuous heavy drinking is also associated with progressive and gradual development of multiple cognitive deficits characterized by memory impairment, apraxia, agnosia, or disturbance in executive functioning. These deficits cause serious impairment in social and occupational functioning and persist beyond the duration of alcohol intoxication and alcohol withdrawal. History, physical exam, and laboratory test should be utilized to determine whether these deficits are etiologically related to the toxic effects of alcohol use.

5. Alcohol-Induced Mood Disorder

Alcohol-induced mood disorder (AIMD), characterized by depressed mood and anhedonia, as well as elevated, expansive, or irritable mood, frequently develops as a consequence of heavy drinking. Onset of symptoms can occur during episodes of alcohol intoxication or withdrawal, and may resemble a primary major depressive, manic, hypomanic or mixed episode. Regardless of the primary or secondary nature of mood symptoms, given the high prevalence of suicide among alcoholics, clinicians should closely monitor the patient for emerging suicidal thoughts.

6. Alcohol-Induced Anxiety Disorder

Although alcohol has anxiolytic properties at low doses, heavy alcohol consumption can induce prominent anxiety symptoms. Alcohol-induced Anxiety (AIA) symptoms more commonly include generalized anxiety symptoms, panic attacks, and phobias. In order to establish this diagnosis, clinicians must rule out other general medical conditions or psychiatric disorders that can mimic this problem.
7. **Alcohol-Induced Psychotic Disorder**

This disorder is characterized by prominent hallucinations or delusions that are judged by the clinician to be due to the effects of alcohol. The psychotic symptoms usually occur within a month of an alcohol intoxication or withdrawal episode, and the patient is characteristically fully alert and oriented, lacking insight that these symptoms are alcohol-induced. Although onset of psychotic symptoms can occur during or shortly after alcohol intoxication, delirium or alcohol withdrawal delirium, alcohol-induced hallucinations, and/or delusions do not occur exclusively during the delusions of these conditions.

8. **Alcohol-Induced Sleep Disorder**

Heavy alcohol consumption can be associated with a prominent disturbance of sleep. At intoxication BALs, especially when blood alcohol levels are declining, sedation and sleepiness can be observed. Alcohol intoxication induces an increase in non rapid eye movement (NREM) sleep, whereas rapid eye movement (REM) sleep density decreases. Subsequently, there is an increase in wakefulness, restless sleep, and vivid dreams or nightmares related to a reduction in NREM sleep and rebound in REM sleep density. During alcohol withdrawal, sleep is fragmented and discontinuous with an increase in REM sleep. After withdrawal, patients frequently complain of sleep difficulties and may experience superficial and fragmented sleep for months or years.

9. **Alcohol-Induced Sexual Dysfunction**

Although small doses of alcohol in healthy individuals appear to enhance sexual receptivity in women and facilitate arousal to erotic stimuli in men, continuous and/or heavy drinking may cause significant sexual impairment. Alcohol-induced sexual dysfunction is characterized by impaired desire, impaired arousal, and impaired orgasm, or sexual pain. It is also associated with marked distress or interpersonal conflicts. Onset of these impairments usually occurs during alcohol intoxication but duration of symptoms exceeds the uncomplicated course of alcohol intoxication. Symptoms usually subside after 3 to 4 weeks of alcohol abstinence. Persistence of symptoms beyond this time may suggest a primary sexual dysfunction (PSD) or a sexual dysfunction due to the medical complications of alcoholism (e.g. neuropathy, alcoholic-liver disease).

**xi GOALS OF PHARMACOTHERAPY FOR ALCOHOL PROBLEMS**

The goals of pharmacotherapy for alcohol abuse or dependence include

1. The reversal of the pharmacologic effects of alcohol
2. Treatment and prevention of withdrawal symptoms and complications
3. Maintenance of abstinence and the prevention of relapse with agents that decreased craving for alcohol or the loss of control over drinking or make it unpleasant to ingest alcohol, and
4. Treatment of coexisting psychiatric disorders that complicate recovery.
ALGORITHM FOR THE IDENTIFICATION AND MANAGEMENT FOR PATIENT WITH ALCOHOL ABUSE AND DEPENDENCE

1. Initial assessment

   - **AUDIT SCORE 16-40**
     - Diagnostic evaluation and treatment intervention
     - Evaluate presence and severity of physical dependence
       - Physical dependence is absent or mild: Outpatient treatment
       - Physical dependence is moderate or severe: Management in psychiatrist's office or referral to outpatient or impatient rehabilitation
         - Aftercare including mutual help organizations

   - **AUDIT SCORE 8-15**
     - Brief intervention and periodic reevaluation

   - **AUDIT score<8**
     - No intervention needed
      - Inpatient rehabilitation
Once an individual is confirmed to be suffering from alcohol abuse, dependence or other alcohol-related problems, it is necessary that a detailed assessment of the case is done before planning treatment. Such an assessment should include:

- **Details of alcohol use:** its onset, duration, average daily consumption, presence of withdrawal symptoms if not used, over-intoxication with alcohol, abuse of any other drugs, recent changes in pattern of drinking.
- **Reasons for initiation and continued drinking.**
- **Health damage due to alcohol:** signs of medical consequences of alcohol use.
- **Behavioral problems associated with alcohol use:** depression, memory problems, suspiciousness, interpersonal problems.
- **Familial, social and legal consequences of alcohol use:**
- **Financial and occupational consequences:** includes current financial status and current occupational status.
- **Previous treatment attempts:** reasons for seeking help this time, motivation for getting changed.

**BARRIERS IN ASSESSMENT**

It is not always simple to get the sensitive information required for the assessment of alcohol use correctly and completely. Some specific barriers on the part of the subject in the assessment include:

- **Denial of the problem by the patient as well as ambivalence about the need for external help.**
- **Guilt about alcohol use and use related behavioral pattern which lead to withholding information.**
- **Feelings of shame** about having been ‘weak’ or ‘failure’ resulting in poor motivation.
- **Stigmatization** of the patient and the family that hinders active help-seeking process.
- **Dilemmas** about physician’s role and reactions may restrain a person form seeking help.
- **Apprehension** of possible legal and other punitive consequences may deter a person form approaching treatment agencies.

Even a clinician’s personal characteristics may serve as a barrier in the assessment of potential patients. Some important issues are:

- **Scientific knowledge** about the field of alcohol related problems, though expanding is still limited.
- **Judgmental attitude** to persons with alcohol related problems affects effective delivery of treatment.
- **False opinion** of therapeutic nihilism in cases of alcohol related problems leads to half hearted, non-proactive efforts on the part of treating teams.
- **Fear of inability** to manage alcohol related problems even at the most initial stages of assessment.

**REMEDIES FOR BARRIERS IN ASSESSMENT**

The barriers in assessment can be effectively overcome by remedial measures in the approach to the assessment of persons with alcohol related problems, for example:
1. Ensuring privacy and confidentiality
2. Remaining non-judgmental and non-moralistic.
3. Showing no-possessive warmth and concern.
4. Expressing empathy and optimism.
5. Readiness to listen and understand before reaching conclusions.
6. Avoiding ambiguous messages.
7. Being clear and firm regarding one's own role, functions, abilities and limitations.
8. Developing introspective ability by the helper (physician, health worker or any other)

XIV GOALS OF PHARMACOTHERAPY FOR ALCOHOL WITHDRAWAL

Goals for which substantial evidence of effectiveness exists

- Treatment of alcohol withdrawal symptoms
- Prevention of initial and recurrent seizures
- Prevention and treatment of delirium tremens

Other Goals

- Prevention of medical and psychiatric complications of alcohol withdrawal
- Prevention of kindling effect
- Prevention of Korsakoff's psychosis
- Improvement in the likelihood of abstinence
- Minimization of adverse drug effect
- Entry into ongoing medical and addictions treatment
- Cost-effective treatment

1. Selecting Patients for Outpatient Treatment for Alcohol Withdrawal

Contraindications to outpatient withdrawal management

- Severe alcohol withdrawal symptoms
- Delirium tremens
- Coexisting acute or chronic illness necessitating inpatient treatment
- Pregnancy
- Follow-up not feasible

Relative contraindications to outpatient withdrawal management

- History of seizures
- History of delirium tremens
- Significant craving

2. RISK FACTORS FOR MORE SEVERE ALCOHOL WITHDRAWAL SYMPTOMS, SEIZURES, AND DELIRIUM TREMENS

- Concomitant medical or surgical
• Moderate-to-severe withdrawal symptoms at baseline
• Older age
• Prior delirium tremens
• Prior detoxification(s)
• Prior seizures
• Time since last drink
• Severity of alcohol dependence
• Elevated aspartate aminotransferase
• Greater degree of craving
• Greater quantity and frequency of alcohol intake
• Higher blood or breath alcohol
• Longer duration of alcoholism
• More symptoms of alcohol dependence
• Presence of alcohol-associated gastrointestinal illness.

3. PHARMACOTHERAPIES FOR ALCOHOL WITHDRAWAL

Incidence of Symptomatic Alcohol Withdrawal

Between 13% and 71% of persons presenting for alcohol detoxification develop significant symptoms of alcohol withdrawal. This wide-ranging incidence depends on the population studied and the severity of dependence in the subjects. In patients hospitalized for alcohol withdrawal receiving no specific pharmacologic treatments, 15% developed seizures, and 15% developed delirium tremens.

A- Benzodiazepines:- are the drugs of choice for the management of alcohol withdrawal. The best evidence for efficacy exists for the long-acting benzodiazepines. Benzodiazepines have been shown not only to ameliorate the symptoms of alcohol withdrawal, but also to prevent seizures and delirium tremens. The best evidence for efficacy in preventing the serious complications of seizures and delirium tremens, however, lies with chlordiazepoxide (Librium) and diazepam.

Benzodiazepines have been compared with other medications for the management of alcohol withdrawal. Although phenothiazines, clonidine (Catapres), and carbamazepine (Tegretol) may reduce symptoms, no evidence supports their ability to prevent seizures or delirium tremens. In fact, seizures are more common when phenothiazines are used, and delirium is more common when β-blockers are used as monotherapy for alcohol withdrawal. There are no controlled data to guide pharmacotherapy of alcohol withdrawal in pregnancy. Although both chlordiazepoxide and diazepam are category D drugs, meaning that there is evidence of human fetal risk, the benefit of using a proven effective therapy for a brief course of therapy to prevent serious maternal and fetal complications likely outweighs the risks.

B. Other Drugs and Adjunctive Pharmacologic Therapies

Other drugs that have been used in the management of alcohol withdrawal include barbiturates, sympatholytics, carbamazepine, neuroleptics, magnesium, and thiamine.
Barbiturates: Barbiturates are the second most common drug class used to treat alcohol withdrawal in the United States. Although there is clinical experience suggesting efficacy, and anticonvulsant properties are well known, there is only one controlled trial of barbiturate use of for alcohol withdrawal. Although evidence is lacking, favorable clinical experience and consensus suggest that Phenobarbital is an acceptable alternative for the management of alcohol withdrawal in pregnant women.

Sympatholytics: Because the symptoms of alcohol withdrawal are at least in part due to increased sympathetic outflow, sympatholytics would appear to be logical treatments. Clonidine has been shown to be as effective as chlordiazepoxide and more effective than placebo in treating the signs and symptoms of alcohol withdrawal. But sympatholytics are not appropriate monotherapy for alcohol withdrawal. In fact, propranolol (Inderal) has been associated with a higher incidence of delirium.

Carbamazepine: This anticonvulsant is as effective as oxazepam and more effective than placebo in treating the minor signs and symptoms of alcohol withdrawal. Currently, however, there is no evidence regarding treatment or prevention of delirium and insufficient evidence in humans to permit safety and efficacy conclusions regarding the use of carbamazepine to treat alcohol withdrawal.

Neuroleptics: Neuroleptics can reduce the symptoms of alcohol withdrawal, but they (particularly the phenothiazines) are less efficacious in preventing delirium and may increase the risk of seizures during alcohol withdrawal. Neuroleptics, however, particularly the butyrophenones such as haloperidol (Haldol), are useful as adjunctive therapy to treat agitation and hallucinations.

Thiamine and Magnesium: Neither thiamine nor magnesium has been shown to have any impact on the signs or symptoms of alcohol withdrawal or the incidence of seizures or delirium during alcohol withdrawal. Both thiamine and magnesium, however, have a role in the pharmacologic management of alcohol withdrawal. Although relatively uncommon as part of the acute presentation of alcohol withdrawal, Wernicke's encephalopathy (confusion, ataxia and ophthalmoplegia) and Korsakoff's syndrome are disastrous complications if they develop. Acute Wernicke's encephalopathy may go undiagnosed initially but can be prevented by parenteral administration of thiamine followed by daily oral doses and should be administered to patients withdrawing from alcohol. Hypomagnesaemia is common in patients withdrawing from alcohol. Therefore, magnesium should be given to all persons with signs of deficiency and should be routinely considered for all patients withdrawing from alcohol. In severe magnesium deficiency, the deficit is approximately 1 to 2 mEq/kg of body weight; greater than 50% of dose given intravenously is excreted when renal function is normal. The goal is to replete half of the deficit on the first day, then the remaining deficit on the following 4 days. For example, for 70-kg persons, administer 32 to 48 mEq (4 to 6 ampoules) of magnesium sulfate in each of 2L intravenous fluid on the first day followed by half that amount daily for 4 days.
## Treatment Regimens for Alcohol Withdrawal

<table>
<thead>
<tr>
<th>Fixed-scheduled dosing</th>
<th>Chlordiazepoxide orally every 6h for 3d (50-100mg per dose day 1, then 25-50 mg per dose)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Front-loading</td>
<td>Diazepam 20 mg orally every 2h while symptomatic until resolution</td>
</tr>
<tr>
<td>Symptom-triggered therapy</td>
<td>Chlordiazepoxide 25-100 mg orally hourly whenever symptomatic (CIWA-Ar&gt;8)</td>
</tr>
<tr>
<td>For delirium tremens</td>
<td>Diazepam 10 mg intravenously, then 5 mg every 5 min until calm but awake.</td>
</tr>
<tr>
<td></td>
<td>If unable to take oral medication or in the presence of hepatic synthetic dysfunction (hypoalbuminemia, elevated prothrombin time), intramuscular, sublingual, oral, or intravenous (for delirium tremens only) lorazepam 1-4 mg may be substituted</td>
</tr>
<tr>
<td></td>
<td>Oral oxazepam 30-60 mg or lorazepam may be substituted in the elderly and those at risk of excessive sedation or its complications.</td>
</tr>
<tr>
<td></td>
<td>All patients receive thiamine 50-100 mg daily, first dose parentally. Consider magnesium administrations (intravenously) 2-4 mEq/kg on day 1, 0.5-1 mEq/kg daily on days 2-4</td>
</tr>
</tbody>
</table>

CIWA-Ar = Clinical Institute Withdrawal Assessment - Alcohol revised scale.

### Choice of Appropriate Regimens:

The main consideration in choosing a treatment regimen is whether medication must be delivered regardless of withdrawal symptoms (i.e., seizure history, acute medical illness) or if medication administration can be safely guided by symptoms. Regardless of regimen chosen, the key is frequent patient reevaluation, particularly early on, with attention to the symptoms and signs of alcohol withdrawal and to excessive sedation form medication. A cookbook regimen does not obviate this requirement; therapy should be individualized. Significant symptoms and signs should be treated with more medication. The tendency to declare a particular benzodiazepine as a treatment failure when symptoms persist should be resisted. This failure is usually due to inadequate dosing. Rather than switching specific drugs, larger additional doses usually treat all persistent alcohol withdrawal symptoms.

### Treatment of Alcohol Withdrawal Seizures

Alcohol withdrawal seizures are generalized, occur early in the course of withdrawal (first 24 to 48 hours), and usually are single or recur only once or twice. The seizure generally resolves spontaneously. Benzodiazepines, carbamazepine, and probably Phenobarbital prevent seizures, but phenytoin is ineffective and therefore not indicated for prophylaxis or treatment of seizures. (Alldredge at al 1989, Chance et al 1991) If the seizure is not typical for alcohol withdrawal (e.g. focal seizures, focal neurologic examination, head trauma, suspected intracranial bleeding) is reasonable. The mainstay of treatment as in the regimens previously described, preferably diazepam, chlordiazepoxide, or lorazepam, all shown to prevent initial and recurrent seizures.

### Treatment of Delirium Tremens:

It is a Medical emergency. 20-50% of patients die eventually if not treated, there is 5-10% mortality even with treatment it requires immediate hospitalization in untreated cases. Treatment of choice is intravenous diazepam (10 mg every 20 minutes till patient is sedated...
Supported treatment should continue. A safe and protective environment should be secured. With treatment it is usually controlled within 3 to 4 days.

XV MANAGEMENT OF ALCOHOL DEPENDENCE

Pharmacological Treatment

Non Pharmacological Treatment

a) **Long term pharmacological treatment:** It helps patients to maintain abstinence after completion of detoxification. As such ‘cure’ of dependence is improbable but successful treatment with pharmacological agents is possible (Desai 1992). Disulfiram, naltrexone and Acamprosate are the drugs approved by US Food and Drug Administration (FDA) for the pharmacotherapy of alcohol dependence.

(i) **Deterrent agents (Alcohol-Sensitizing Drugs):** Medications such as disulfiram or calcium carbamide cause an unpleasant reaction when combined with alcohol. The efficacy of such drugs in the prevention or limitation of relapse in alcoholics has not been demonstrated. However, these drugs may be of utility in selected samples of alcoholics with whom special efforts are made to ensure compliance.

**Disulfiram:** It is the most commonly used alcohol-sensitizing medication and the only one approved for use in the US. It was approved by US-FDA in 1948.

**Mechanism of Action:** It inhibits alcohol dehydrogenase enzyme which converts acetaldehyde to acetate in normal alcohol catabolism in the liver (Larson et al 1992). Following disulfiram treatment, ingestion of alcohol increases acetaldehyde levels 5 to 10 times more than the levels without disulfiram treatment. High concentrations of acetaldehyde are believed to mediate disulfiram-ethanol reaction, which is characterized by flushing, weakness, nausea, tachycardia, hypotension and in severe cases death (Chick et al 1999). Continuous treatment with disulfiram does not lead to the development of tolerance. Before disulfiram treatment is initiated, the patient should be abstinent from alcohol for at least 12 hours. Disulfiram effects are observed 3 to 12 hours after its oral administration. Disulfiram-alcohol reaction may occur as long as 1 to 2 weeks after the last dose of disulfiram. The standard dosage of disulfiram is 250 mg per day (range 125 to 500 mg daily). Goal is to achieve total abstinence. It is an aversive treatment that enhances motivation for continued abstinence by making the “high” unavailable, thus discouraging impulsive alcohol use. The cognitive awareness of occurrence of DER and/or any previous experience of DER with use of alcohol while on disulfiram therapy, prevents a drinking response when exposed to alcohol use related cues (like persuasion by friends, company of other users social gatherings and parties). Prevention of drinking response helps to maintain abstinence. It should be remembered that disulfiram does not reduce the craving but prevents response to craving. It should not be given surreptitiously. Patient to be taken in confidence and clinicians should never ever encourage the practice of giving this medicine without the informed consent of patient. Because of the hepatic toxicity and blood dyscrasias associated with disulfiram, the manufacturer recommends monitoring of liver function tests and complete blood count during treatment. Before starting treatment with disulfiram, patients should be warned to avoid alcohol containing products and foods.

**Side effects and adverse reactions:** The commonly experienced side effects are of drowsiness and gastric irritation. The adverse effects, which occur most commonly with use of disulfiram, are hepatic, neurological, skin reactions and psychosis. The safety of disulfiram, estimated on the amount
produced and the number of reactions reported, corresponds to an intermediate rate of adverse reaction (1 per 200-2000 treatment year).

**Clinical regimen:** Disulfiram should always be given after obtaining informed consent and baseline investigations for monitoring the side effects. The usual dose is 250mg/day; however due to metabolic differences, some patients may require a higher dose 500-750mg/day to experience DER. It is generally dispersed in a single daily dose in the morning but a bedtime single dose has also been used.

**Supervised Disulfiram therapy:** Disulfiram works well when its use is supervised as it ensures better compliance. Supervision can be most effective when done by spouse, though it can be done by an other family member or a supervisor at work place. Supervision also ensures that the possibility of DER is reduced. Some training of supervisor in the detection of the techniques of evasion, which the patients use, is desirable.

**Treatment of DER:** In case of a DER induced by a challenge test, the resuscitation should be and is usually available but the occurrence of a DER in uncontrolled situations is more likely to be fatal. Prompt treatment in such situation is necessary although it is mainly supportive and for controlling the fall in blood pressure. If DER is mild, assurance and oral fluids suffice. In patients with moderate or severe DER, intravenous fluids and in some patients dopamine infusion is necessary to control the severe hypotension. The use of antihistamines also has been recommended (Peterson et al 1992). The use of 4-methylpyrazole, which blocks formation at acetaldehyde has been found to be successful but is still not common in clinical practice. (Lindros et al 1981).

**Compliance and efficacy:** Although disulfiram has been in use for more than 50 years but it has been underused. (Brewer et al 1992) Azrin et al 1982 reported on a series of studies comparing supervised and unsupervised disulfiram therapy, with a package of essentially behavioral interventions termed as **community reinforcement therapy (CRT)**. The effectiveness of disulfiram therapy was demonstrated if it is properly supervised. The evidence for the effectiveness of supervised disulfiram therapy from American Studies has been confirmed in a multi-center British study by Chick et al 1992, in which supervised disulfiram therapy was compared with supervised vitamin C. The results have clearly favored the disulfiram group with significant reductions in several measures of drinking behavior including the level of GGT in the disulfiram group, compared to no reductions in these measures in the Vitamin C group. A review of controlled clinical trials with disulfiram concluded that there is unopposed evidence for its effectiveness.

**B) Anti craving agents:** A number of specific neurotransmitter systems have been implicated in the control of alcohol consumption, including endogenous opioids, catecholamines, especially dopamine, and serotonin. Although these systems appear to function interactively in their effects on drinking behavior efforts to use medications to treat excessive drinking have increasingly focused on agents that have selective effects on specific neurotransmitter systems.

1. **Naltrexone** – An extensive literature supports the role of opioidinergic neurotransmitter in the pathphysiology of alcohol consumption and related phenomena. An analog of naloxone, is a relatively pure opioid antagonist with highest affinity of for the mu-opiate receptor type. Naltrexone is absorbed better after oral administration and has longer duration of action than naloxone. Naltrexone reduced alcohol craving, days of drinking per week, and the rate of relapse among those who drank (Volpicelli et al 1992). It should be given to patients who categorically report craving and there are multiple instances of lapse or relapses because of craving. There is evidence that naltrexone leads to a
reduction in drinking quantity, maintain abstinence and helps prevent relapse to heavy drinking. The superior efficacy of naltrexone for alcohol relapse prevention is well documented (Volpicelli et al 1992). When administered at 50 mg/day for 3 months, this agent usually prevents relapse. It was approved by the Food and Drug Administration in 1994 for use in the treatment of alcohol dependence. Naltrexone is thought to attenuate the reinforcing effects but not the negative aspects of alcohol consumption, such as cognitive impairment and sedation. Among alcoholics in treatment, those who consumed alcohol reported feeling less intoxicated and less craving for alcohol. The efficacy of naltrexone for use in alcohol dependence has been investigated in two placebo-controlled clinical trials. In a combined analysis of the data from the 186 alcohol dependent patients in these two studies, patients randomized to receive 50 mg of naltrexone for 12 weeks were more likely to remain abstinent and to avoid relapse to heavy drinking 31% of placebo patients remained abstinent compared to 54% of patients on naltrexone 48% of placebo patients avoided heavy drinking, whereas 75% of naltrexone patients successfully avoided drinking to excess. Craving for alcohol was also significantly lower for patients on naltrexone. The decision regarding the continuation of naltrexone beyond 12 weeks is based on clinical judgement. In making this decision, the physician considers whether the patient has made changes that could support continued abstinence, the patient's previous history of response to treatment, and the patient's interest in continuing. Naltrexone may be particularly useful for subjects who present with high levels of craving and somatic symptoms (Volpicelli et al 1995). Another opioid antagonist, nalmefene, has also been evaluated in alcohol dependent subjects. In an initial pilot study (Mason et al. 1994) and a subsequent larger trial (Mason et al 1999), the medication was shown to be well tolerated and to be superior to placebo in reducing relapse.

In summary, naltrexone appears to produce a modest effect on drinking behavior among alcoholics (Kranzler and Van Kirk 2001). However, given the comparatively small overall effect of the medication, a variety of other factors, including medication compliance, the severity and chronicity of alcohol dependence, and the choice of concomitant psychotherapy, may determine whether an effect of the medication is observed. Common side effects are nausea, dizziness, headache.

**CONTRAINDICATIONS TO THE USE OF DISULFIRAM AND NALTREXONE**

**Conditions resulting in increased risk associated with the disulfiram ethanol reaction**
- Cerebrovascular disorders
- Cardiovascular disease
- Severe pulmonary disease
- Renal failure
- Cirrhosis with portal hypertension
- Occult atherosclerosis (i.e. >age 60, diabetes)

**Conditions that may be exacerbated by disulfiram**
- Psychosis
- Significant depressive illness
- Idiopathic seizure disorder
- Peripheral neuropathy
Other contraindications to disulfiram

- Organic brain syndrome: limited ability to understand risks of disulfiram ethanol reactions.
- Pregnancy: associated with birth defects

Precautions for the use of disulfiram

- Concurrent use of adrenergic receptor antagonists
- Concurrent use of vasodilators

Absolute contraindications to naltrexone

- Acute hepatitis or liver failure
- Current dependence on opiate or opiate withdrawal
- Need for opiate medication

Relative contraindications to naltrexone

- Pregnancy
- Adolescence

1. **Acamprosate**—Acamprosate an amino acid derivative, affects both gamma-aminobutyric acid (GABA) and excitatory amino acid (i.e. glutamate) neurotransmission (the latter effect most likely being the one that is important for its therapeutic effects in alcoholism). It inhibits the glutaminergic excitatory activity, acting, probably, on a subclass of glutamate (NMDA) receptors, especially when there is hyperactivity of these receptors. It has been approved by FDA in 2004. Acamprosate has been considered a partial co-agonist of the NMDA receptor (Naassila et al 1998). Acamprosate has a good oral absorption, which is, however, impaired by the concomitant ingestion of food. It is not metabolized, and completely eliminated by the kidneys. It has no protein link. All these characteristics suggest that this medication has no preoccupying interactions with drugs. It has been found efficacious for the treatment of alcoholism with long-term benefits. In fourteen of the sixteen double blind placebo-controlled trials it has shown favorable results (Mason and Ownby, 2000). Meta analysis of the studies done between 1990 to 2002 has shown that it increases abstinent days and also increases cumulative abstinence days (Carmen 2004). Acamprosate should be administered in alcohol dependent patients with more than 60 kg, in three daily doses, being two capsules with 333 mg in the three periods of the day, always before meals. For patients with less than 60 kg, most of the studies suggest the administration of lower doses i.e. a capsule with 333 mg in the three periods of the day. Maintenance time of the medication is variable. The clinical trials performed used the drug for 6 to 12 months (Wilde et al 1997). This new medication has great promise in alcohol relapse prevention (Seas et al 1996). Common side effects include diarrhea and headache. However, some authors proscribe the drug for patients with hepatic insufficiency CHILD-PUGH C, but not for CHILD-PUGH A or B. Pregnant women should not receive the medication, as there are no reliable data about its safeness for the fetus (Saivin et al 1998).

2. **Selective Serotonergic Reuptake Inhibitors**—Another major focus of research on medications to treat alcoholism has been the role of the indoleamine neurotransmitter serotonin (5-HT). 5-HT has been shown consistently to exert an influence over alcohol consumption in preclinical models of drinking behavior (McBridge et al. 1990, Le Marquand et al 1994a). In contrast to this
preclinical literature, data on the effects of serotonergic medication on human drinking behavior are more limited, and the results are less consistent (LeMarquand et al 1994b, Kranzler 2000). Naranjo and colleagues (1990) found that fluoxetine 60mg/day reduced average daily alcohol consumption by approximately 17% from baseline levels, while treatment with fluoxetine 40mg/day or placebo had no effect. Decreased brain serotonin has been linked with impulsiveness and low scores on the 'harm avoidance dimension of behaviour (Cloninger et al 1987). The largest study (Kranzler, Burleson, Korner et al 1995) involved 101 subjects, some of whom also had conorbid depression. Studies have failed to show significant reduction in drinking or other measures of drinking (Kabel and Petty 1996: Johonson et al 1996). Three months trial of Fluoxetine (n=51) with conorbid major depression found a significant effect on both condition compared with placebo (Cornelius et al 1997). It is not as effective as naltrexone or acamprosate.

3. Topiramate – It is an antiepiletic and adjuvant mood stabilizer. It inhibits release of dopamine in meso-cortico-limbic pathway by augmenting GABA function and inhibiting specific glutaminergic pathway. It also inhibits carbonic anhydrase enzyme (Gibbs et al 2000, Shank et al 2000). Dose is 25- to 300-mg/per day. Most common side effects are weight loss, parasthesiae, cognitive impairment. A 12-week study using topiramate (25-300mg/day) has shown that it is significantly better than placebo in decreasing craving (Johnson et al 2003). Abstinence is not required to start topiramate. It requires larger studies with control group and longer duration of the study.

4. Ondansetron – Ondansetron, a selective 5-HT3 receptor antagonist, has shown promise in reducing overall alcohol intake specifically among those with type I alcoholism (Cloninger et al 1988). It reduces urge to drink. Dose is 4 micro gram/kg bid. It produces better result in early onset alcohol dependence. (Johnson, 2000). It is not in common clinical use.

C Medications Used in Combination: - Drinking behavior is not dependent solely on a single neurotransmitter system. Rather, several systems have been implicated, including the opioidergic, glutamatergic, GABAergic, serotonergic, and dopaminergic systems as well as several neurohormones, such as thyrotropin releasing and adrenocorticotropic hormones (Litten et al 1996: Roberts and Koob 1997). In light of this multi-determination, many options in development of agents to reduce drinking merit exploration. One possibility is to use agents in combination, particularly to combine those that act on different receptors in the brain. This strategy has been supported by several animal studies, demonstrating that combinations of medications are more effective in reducing alcohol intake than the various drugs were used alone (Lankford and Myers 1996; Yu et al 1997). e.g. DS+NTX, ACAMPROSATE+NTX.

D Depot Preparation: - Implanted disulfiram has been tried but not on large scale. Sustain release formulation of depot Disulfiram provoked only mild Disulfiram ethanol reaction (DER). Depot Naltrexone is better in patients with poor compliance. It holds promise in patients with poor compliance.

For optimum benefits in the treatment of alcohol dependence pharmacological treatment has to be given along with psychosocial treatments.

B Psychosocial treatments in alcohol dependence

Psychosocial intervention for substance use disorders is a broad “umbrella” term that brings under its folds a diverse array of non-pharmacological interventions for effective and comprehensive management of drug abuse. A variety of treatment components are delivered
within the context of rehabilitation services. Therapeutic approaches most often employed in both residential and outpatient programs include behavior therapy, group therapy, family treatment, and pharmacotherapy. Regarding specific treatment modalities, the weight of evidence suggests that behavioral treatments are likely to be more effective than insight-oriented or family therapy (Miller and Hester 1986). Nevertheless, recent research (Project MATCH Research Group 1997a, 1997b 1998, Ouimette et al 1999) also indicates that Twelve Step Facilitation, which is based on the principles of AA, is as effective as more theory-based therapies. Controlled studies provide little support for the effectiveness of psychodynamic psychotherapy, although such treatment has been shown to be helpful in the treatment of drug abuse (Institute of Medicine 1989, Miller and Hester 1980).

GOALS OF PSYCHOSOCIAL TREATMENT

- Enhance efficacy of pharmacotherapy
- Achieving sustained drug free status
- Providing the relationship
- Change of life style
- Improved quality of life

Though some of psychosocial interventional skills require specially trained professionals, most of these skills can be acquired by general physicians, nurses, social workers and counselors making it possible to offer these interventions in a number of settings, including primary health care facilities and non governmental organizations working in the community.

MODALITIES OF PSYCHOSOCIAL INTERVENTIONS

1. Brief interventions – It is very popular because of its cost effectiveness. Brief interventions (studied mostly in relation to alcohol abuse so far) typically comprise of 1-4 session. Contrary to previous popular belief, relatively brief interventions have consistently been found to be effective in reducing alcohol consumption or achieving treatment referral or retention of problem drinkers, and a significant effect size was found in a meta analysis of 32 controled studies across 14 nations involving over 6000 problems drinkers (Bien et al 1993). The common elements of brief intervention were summarized as the acronym FRAMES.

   F. Feedback, R- Personal responsibility, A- Advice, M- Menu,E- Empathy,S- Self efficacy

   Quite effective in brief contact contexts such as primary health care settings and employee assistance programme. Although it requires much lesser time and cost, it was consistently better than no counseling and usually as good as other, more formal or long drawn therapies.

2. Extended interventions – utilizes 5-12 sessions with patient, either in a group or alone. Patients advanced well into substance using patients, either in a group or alone. Patients advance well into substance using career, or those with multiple relapses, hospitalizations etc., are suitable for this type of intervention.

3. RELAPSE PREVENTION

   - Recovery – Process of initiating and maintaining abstinence from the substance
   - Lapse - Isolated instances of substance use following a period of abstinence
• **Relapse** - Resumption of a previous pattern of substance abuse or dependence following a period of abstinence

**a. NEED AND SCOPE** – Numerous reviews of treatment outcome literature have documented high rates of relapse up to 65-80% in the first year of treatment among substance dependence disorder patients (Hunt et al 1971, Catalano 1988).

**b. STRATEGIES OF RP**
- Identification and management of specific relapse precipitant situations/events
- Maintaining abstinence
- Correction of psychosocial and physical consequences of drug use
- Sociooccupational Rehabilitation/reintegration
- Modifying or reverting underlying neuroadaptive process/changes

**c. DETERMINANTS OF RELAPSE (MARLATT AND GORDON, 1995)**

**INTERPERSONAL FACTORS**
- Negative emotional states (most common)
- Negative physical states
- Positive emotional states
- Testing of personal control over urges and temptations

**INTRAPERSONAL FACTORS** –
- Relationship conflict
- Social pressure to use substances
- Positive emotional states as a result of interpersonal intervention

**d. OBJECTIVES OF RELAPSE PREVENTION**
- To develop new coping skills for handling high risk situations and relapse
- Warning signs
- To make lifestyle changes to decrease the need for substances
- To increase healthy activities
- To prepare for interrupting lapses, so that they do not end in a full blown relapse and
- To prepare for managing relapses, so that adverse consequences can be minimized

**e. RELAPSE MANAGEMENT**
- Detoxification and assessment of consequences of substance use
- Assessment of the reasons and precipitants for current relapse and the past history of relapses
- Assessment of the reactions of the patient and the family members to the relapse
- Assessment of the recovery process, specifically the periods of abstinence and the factors contributing to abstinence
• Intervention for the relapse precipitants and the reaction
• Preparation for future relapses

3. COGNITIVE AND BEHAVIOUR THERAPY: - These therapies primarily focus on individual thoughts and behaviors. These structured, focused, collaborative approaches are based on the assumption that substance abuse is mediated by complex cognitive and behavioral processes, which can be modified to decrease the drug using and other related behaviors.

4. GROUP THERAPY

Group therapy for drug dependents can be defined as “an assembly of chemically dependent patients usually 5-10 in number, who meet regularly under guidance of a professional leader (usually a professional therapist or an addiction counselor), for the purpose of promoting abstinence from all mood altering chemicals and recovery from addiction (Washton 1997). Treatment goals in group therapy for substance dependent patients are enhancing motivation, establishing abstinence, psycho education, preventing relapse and addressing specific psycho social issues. Unfortunately in India, these therapies have not been followed much. Our cultural characteristics of sharing life intimately with large number of people can be utilized by adopting the available models to our needs (Desai, 1992).

5. FAMILY THERAPY

The role of family transcends genesis, maintenance, treatment seeking, recovery as well as relapse of substance used disorders. There is a vicious cycle in which the patient’s drug use adversely affects the family’s well-being and coping resources.

• Dysfunctional and burdened families have strongly negative responses towards patients, resulting in increased substance use by patients.
• This is much true in the Indian context because of the intact family systems and support, with family members having an important say in the matters of an individual and peculiar intra familial characteristics of the Indian families.
• “Family as a unit”, “family as a context”: - If the chemical dependence seems to be mainly owing to dysfunctional patterns in the family, Family as unit needs to be treated with intensive family or marital therapy. If chemical dependence has led to dysfunction in the family, then a less intensive but active and constant participation of family members shall be sought to treat patient while focus on family as context.

6. SELF help approach (Alcoholic Anonymous)

The 12 Steps of Alcoholics Anonymous
1. We admitted we were powerless over alcohol—that our lives had become unmanageable.
2. Came to believe that a Power greater than ourselves could restore us to sanity.
3. Made a decision to turn our will and our lives over to the care of God as we understood Him.
4. Made a searching and fearless moral inventory of ourselves.
5. Admitted to God, to ourselves, and to another human being the exact nature of our wrongs.
6. Were entirely ready to have God remove all these defects of character.
7. Humbly asked Him to remove our shortcomings.
8. Made a list of all persons we had harmed, and became willing to make amends to them all.
9. Made direct amends to such people wherever possible, except when to do so would injure them or others.
10. Continued to take personal inventory and when we were wrong, promptly admitted it.
11. Sought through prayer and medication to improve our conscious contact with God as we understood Him, praying only for knowledge of His will for us and the power to carry that out.
12. Having had a spiritual awakening as the result of these steps, we tried to carry this message to alcoholics, and to practice these principles in all our affairs.

It is a group of individuals with similar problems. Mutual assistance is required among members to satisfy a common need. It began in 1935 in Europe. The only requirement for membership is desire to stop drinking. Groups may or may not allow simultaneous treatment. AA meetings are of different type (closed meeting and open meeting). It follows 12 tradition and 12 steps as mentioned above. AA views alcoholism as a disease or spiritual illness. The central spiritual defect in alcoholic is described as an excessive preoccupation with self. Treatment of preoccupation with self is the core of AA approach. The theory of AA that addiction is not the property of the drug but a characteristics of the addict being powerless over drugs. Emotional sobriety rather than mere physical sobriety is the goal.

Innumerable individual accounts and collective impressions have been documented about the efficacy of these approaches. Efficacy depends on retention of members and extent of participation of AA meeting. Clinical experience does suggests that participation in self help groups can be an important adjunct to treatment for better outcome in many patients with substance use disorders. Patients, who have experienced loss of control, believe in the need to abstain, have no significant psychopathology, are socially stable and show signs of religiosity, should be referred to self help groups, besides the treatment they are receiving.

Limitations of AA: It neither provides motivation nor detoxification there is no follow up and they don't deal with the problems of multiple substance use.

Self help groups in India: It Started in 1957. Meetings are held mostly in churches. Mistaken impression is that these are Christianity based, but principle of faith is followed it is necessary to encourage more active referrals to AA and other self help groups

xv Special FEATURES Influencing treatment

There is considerable evidence that links the outcome of alcoholism treatment to comorbid psychopathology. General measures of psychopathology (McLellan et al. 1983), as well as the specific diagnoses of drug abuse, drug dependence, antisocial personality disorder and major depressive disorder have been shown to predict poorer outcomes in alcoholics (Rounsaville et al 1987, Kranzler et al. 1996b). The extent to which treatment of concomitant psychopathology enhances alcoholism treatment outcome is unclear.

Osher and Kofoed (1989), in advocating an integrated approach to patients with comorbid psychiatric and addictive disorders, have described four phases of treatment with this patient population: (1) engagement of dually-diagnosed patients in the treatment programs, (2) persuading these patients to accept long term abstinence as an appropriate goal of treatment, (3) active treatment, during which efforts are focused on the development of attitudes and the acquisition of skills necessary for sobriety, and (4) relapse prevention, which involves the long term maintenance of sobriety and extend beyond the acute treatment period.

(131)
Demographic Features

a) Adolescents:- Despite a paucity of controlled, age-specific treatment outcome studies of adolescents with alcohol use disorders, the need for prevention and specialized treatment for this group is clear. The literature indicates that in substance-abusing adolescents some treatment is better than no treatment, relapse rates are high, and there is no consistent support for the superiority of any single treatment modality (Brown et al 1989). However, several factors have been associated with better treatment outcome: later onset of problem drinking, pretreatment attendance at school, voluntary entrance into treatment, active parental input, and availability of ancillary adolescent-specific services, including those pertaining to school, recreation, vocational needs, and contraception (Kaminer 1994, Catalano et al. 1990-1991)

Because many adolescents have not yet fully developed formal operational thinking, treatment efforts should be concrete and goal-oriented. The use of age-appropriate support groups (e.g Alateen) may be particularly useful in this regard.

b) Geriatric Patients:- In addition to the high prevalence of medical problems, pharmacokinetic and pharmacodynamic variables can affect treatment outcome in elderly alcoholics. For example, Liskow and colleagues (1989) found that elderly alcoholics, despite having drunk less than younger patients during the month prior to admission, had more severe alcohol withdrawal symptomatology and required a higher dosage of chlordiazepoxide. An important question in treating elderly alcoholics is the extent to which specialized treatment services improve outcome. Kofoed and colleagues (1987) found that patients treated in special elderly peer groups remained in treatment longer and were likely to complete treatment than those treated in mixed age groups.

c) Gender (Including Pregnancy): - Epidemiological and clinical studies have shown that alcohol abuse and dependence have become quite common among women, as historical gender differences in drinking problems have diminished during the past 25 years. This trend has promoted greater awareness of the impact of alcohol use on women’s health and importance of gender as a potential determinant of treatment outcome. The recent requirement by the National Institutes of Health and the FDA that women and minorities be actively recruited to participate in health related research should help to increase knowledge about alcoholism treatment outcome in these groups. To guide the treatment of alcoholism in women, Blume (1992) suggests that evaluation should include special attention to the identification of physical abuse, sexual abuse, medical problems, psychiatric comorbidity, the presence of alcoholism and drug abuse in spouses, and alcohol-related birth defects in children. Blume (1992) also lists the following special treatment needs of women: information about the effects of substance use on the fetus, parenting skills, couples and family therapy, sober female role models, assertiveness training, and an awareness of sexism and its consequences.

d) Ethnic and Cultural Issues and Treatment: - Although socioeconomic and cultural issues should be addressed in alcoholism treatment (Franklin 1989), guidelines for such treatment are based largely on common sense, rather than systematic outcome evaluation. Obviously, where language barriers exist, special efforts must be made to ensure adequate communication. Treatment providers should also be aware of their patients traditional patterns of drinking, how drinking may be influenced by acculturation, differences among ethnic groups in their perception of alcohol related problems, the impact of sociocultural differences between patients and providers, and how prevailing social (e.g. family) relationships can affect treatment outcome.
A number of patients of alcohol dependence may have another psychiatric disorder on axis-I or axis-II. Management of these patients puts a tough challenge at all phases viz. evaluation, early treatment, long-term treatment for maintenance of abstinence and rehabilitation.

A. Problem in evaluation of patients with dual diagnosis
B. Problems in early treatment of patient with dual diagnosis
C. Problems in long-term treatment of patients with dual diagnosis
D. Treatment of specific comorbid conditions

a) TREATMENT OF SPECIFIC COMORBID CONDITIONS

1. Alcoholism patients with depressive symptoms: In the initial few days of detoxification many patients of alcoholism experience subjective symptoms and or demonstrate objective signs of depressive nature. These abate at rapid pace in majority of the patients. It is crucial that any decision of diagnosing these symptoms or initiating pharmacological treatment be deferred for a minimum of two weeks, preferably for four weeks. In small group of patients, who continue to have depressive symptoms, these have the potential of being relapse triggers. Appropriate psychosocial treatment methods, if possible group therapy, must be considered for the management of these symptoms as well as for relapse prevention.

2. Alcoholism patients with major depression: Although it is necessary to observe the depressive features in patients of alcoholism for a period of two to four weeks before arriving at a diagnosis, in some clinical situations, the imperative need for diagnosis and treatment is overriding, viz. in cases of major depression (DSM-IV) or depressive disorder-moderate/severe (ICD-10). Evidence from the past history as well as the family history can be helpful in reaching the diagnosis in such patients. Antidepressants must be considered in all these patients. The traditionally used tricyclic antidepressants have been recognized to have limitation in such clinical situation due to their sedation, cardiac toxicity and low safety profile. The antidepressants specifically the ones like fluoxetine and tianeptine acting through the serotonergic system, are more appropriate.

3. Alcoholism with dysthymic disorder: Dysthymic disorder (DSM-IV) or depressive disorder (mild) require careful evaluation need for use of antidepressants. Psychosocial therapies, particularly supportive psycho-therapy or cognitive behavior therapy must be considered according to the suitability and feasibility. The course in these patients is marked with frequent relapses and the prognosis of alcoholism is not too favorable.

4. Treatment of anxiety in alcoholism: Anxiety symptoms in alcoholism vary from anxiety states related to acute and protracted withdrawal to comorbid anxiety disorders. Prevalence rates for anxiety disorders in studies performed on patients in alcohol treatment programs range from 16-60%. (Powell et al 1982; Bowen et al 1984). A proper assessment of symptomatology and its temporal relationship with alcohol use is essential. It may be good to delay any definite diagnosis of an additional anxiety disorder till the patient has abstained from alcohol for 2 weeks. Anxiolytic agents should be administered to patients who experience anxiety disorders separately from any problems related to alcohol. Longer acting benzodiazepines are preferred because of
lower abuse liability, though daytime drowsiness and cumulative toxicity is problem. It has been proposed that there is no clear evidence that alcohol interacts with buspirone and this drug can be used in the treatment of anxiety syndromes in alcoholics. (Schckit et al 1987)

xvii Treatment issue in special groups

1. Physician patients: - Physicians and other health care professionals, who are dependent on substance(s), require different treatment strategies and their rehabilitation in the same profession poses difficult problems. No specialized facilities exist in India for such patients. The general attitudinal barriers in physicians and the other health professionals about their own health needs and the defenses specific to the issue of substance use makes the treatment of such patient a more challenging task. All the same, the attitude of the treating team needs to be empathic and rooted strongly in the disease model of use disorders.

2. Ex patients on treatment team: - The phenomenon, so common in the West, of ex-patient continuing to work on treatment teams or health care professionals recovering from dependence also continuing to function on treatment teams, is still very uncommon in India. Some voluntary agencies have ex-patients on their teams. Some problems of such an arrangement can be raised but clearly there are many advantages for the team and the individuals in particular. The phenomenon is likely to catch on and some precautions need to be planned and implemented to avoid counterproductive results.

3. Children of patients: - With the proliferation of substance abuse problem in general and the increased population of women patients in particular, there has been a recognition of distinct population, the children of substance abusers who are known to have unique characteristics, that require special attention. It is being increasingly documented that these children are at risk for serious educational, medical and emotional problems and have the potential for abusing substances as well as an increased risk for HIV infection. Comprehensive drug treatment services must include services for parents as patients and must address the special needs of the children of patients through services, like day care facilities, parenting skill training and school facilities.

XVIII CONCLUSION

During the past 25 years significant progress has been made in the scientific study of alcoholism and its treatment. A number of conclusions appear warranted at this time:

1. Alcoholism is heterogeneous with respect to demographic features (e.g. gender, race/ethnicity), age of onset of heavy drinking, severity of alcohol dependence, comorbid psychopathology, genetic vulnerability, and other prognostic factors.

2. The available evidence suggests that any treatment for alcoholism is better than no treatment. The majority of those treated demonstrate improvement, but many of these alcoholics may improve with minimal treatment.

3. The intensity of treatment has not been shown to produce pronounced differences in outcome (Moos and Moos in press). Similarly, medical inpatient treatment, while more costly, is not demonstrably more effective than nonmedical residential or outpatient treatment. For patients with serious comorbid medical and psychiatric disorders, medical inpatient treatment may, nonetheless be necessary. Some evidence indicates that continuing aftercare helps to maintain abstinence following short term intensive rehabilitation in patient settings.
There is little evidence that any one-treatment approach is superior. There is some support for certain kinds of behavior therapy, but the effectiveness of AA and disulfiram seem to depend on patient characteristics and compliance. Several kinds of carefully specified and theoretically derived therapeutic approaches show promise as a basis for a new generation of ambulatory treatments. These include the relapse prevention strategies that teach the alcoholic how to avoid high risk relapses situations, and new pharmacologic agents (e.g. naltrexone) that appear to reduce the alcoholic’s risk of relapse by dampening the reinforcement potential of alcohol. Continued improvements in treatments outcome will depend upon successfully matching treatment settings and modalities to the specific needs to the individual patient.

**XXI SOME IMPORTANT MYTHS AND FACTS ABOUT TREATMENT OF SUBSTANCE USE DISORDERS**

1. **Myth**: Detoxification is equivalent to treatment  
   **Fact**: Detoxification is only the initial phase in the long term process of treatment

2. **Myth**: Inpatient treatment is necessary and outpatient treatment does not work  
   **Fact**: Outpatient treatment is feasible and is effective in selected patients

3. **Myth**: More intensive treatment is more likely to succeed  
   **Fact**: Short-term, less intensive treatment or minimal treatment may be effective in some patients

4. **Myth**: Total abstinence is the only treatment goal for dependence disorder.  
   **Fact**: Other treatment goals like controlled use are feasible and worth pursuing in suitable patients

5. **Myth**: Relapse indicates that the treatment has failed  
   **Fact**: Relapse is a part of the recovery process and not failure of treatment

6. **Myth**: Anyone treatment method is superior to other treatment methods(s)  
   **Fact**: Different treatment methods suit different patients and non one method is superior to the others for all patients.

7. **Myth**: Combining more than one treatment method has no advantage.  
   **Fact**: Multimodal treatment is superior to any single treatment methods.

8. **Myth**: It is an individual’s problem and the treatment has to be focused on the individual  
   **Fact**: Involvement of a key family member, preferably the spouse helps in the treatment

9. **Myth**: Uniform structured programmes for all patients are more effective  
   **Fact**: Structured programmes with flexibility for individual needs and matching of patient with treatment methods are more effective.

10. **Myth**: Patients who join treatment do so either entirely voluntarily or due to coercion and those who join voluntarily fare better in treatment.  
    **Fact**: There is some element, in varying degrees, of voluntaries and coercion in all patients and there are no group differences in the outcome of voluntary and coerced patients.
XXII optimal & good practice depending on different level of treatment settings

Level 1 - Primary care physician, non psychiatric physician in practice/individual chamber practice

Optimal practice
1. To be conversant with "disease model" of addiction
2. To be able to recognize "problem drinking"
3. To be able to relate physical problems to alcohol consumption
4. To be able to communicate the alcohol related harm to the client and his/her family members.

Good practice
1. To be able to draw a balance sheet of harm and benefits of alcohol drinking in problem drinkers
2. To recognize specific alcohol related physical problems
3. To offer medical treatment for these problems
4. To recognize alcohol withdrawal features & communicate the significance of the same to the client
5. To prescribe benzodiazepine detoxification for those having withdrawal features.
6. To be able to refer those with heavy alcohol consumption patterns, those with past history of psychiatric problems, those with history or current evidence of fits, delirium, head injury, suspected metabolic disorders to GHPUs with inpatient facilities.

Level 2 District hospital psychiatrists, NGOs with inpatient facilities

Optimal practice
1. To be able to recognize dependence syndrome, especially by the presence of alcohol withdrawal features.
2. To initiate and plan OPD detoxification, by prescribing benzodiazepines (long acting) chlordiazepoxide 60-100 mg divided doses for 4-5 days and then tapering the dose 25 mg once in 2-3 days and withdrawing the dose by 10th to 12th day
3. To motivate the clients to remain abstinent by discussing the risk of developing dependence syndrome and physical problems.

Good Practice
1. To recognize the needs of inpatient and outpatient detoxification
2. To be able to accurately monitor the patient during detoxification
3. To differentiate simple and complicated withdrawal states
4. To initiate detoxification for complicated withdrawal states in emergency settings.
5. To plan post detoxification motivation enhancement involving patient and family
6. To offer option of DS therapy for 1 year to all patients who are well motivated and accepts the treatment and are apparently free from relative contraindications of DS therapy
7. To be able to monitor patients on DS therapy
8. To be able to refer those with mental disorders, comorbidity with heroin/benzodiazepine/cannabis/injection users, for GHPU/specialized deaddiction treatment settings.

**Level 3 GHPUs - Medical College department Optimal**

1. To be able to differentiate dependent and non dependent drinkers
2. To detoxify (in patient/outpatient) dependent drinker to use oxazepam/lorazepam for those with liver disease and chlordiazepoxide for those without to differentiate severity of withdrawal states and predict DT by evaluating risk factor (heavy consumption, past h/o DT head trauma, infection, metabolic disorder, dyselectrolyemia)
3. Post detoxification motivations for complete abstinence for dependent drinkers,
4. To offer drugs like DS, or anticraving agents like NLT, fluoxetine or acamprosate, or topiramate
5. To choose the drugs though a process of active discussion with the patient and family members, and evaluating their economic background
6. To monitor the patient during abstinence

**Good Practice**

In addition to above, to consider

1. Family education and support
2. Offer group educative sessions or individual educative session
3. Relapse prevention programme
4. Relapse management programme
5. Referrals – those with comorbidity, multiple relapses requiring long term inpatient stay (4 weeks).
6. To maintain consultation – liaison service for alcohol dependence in GHPUs
7. To run a programme for non dependent problem drinkers
   - education
   - appraisal of risk factors
   - harms/benefit
   - disease model
   - benefits of early detection/abstinence

**Level 4 Specialized treatment centers of apex institutes – complete MDC (Multidisciplinary care) facility**

**Optimal**

1. Comprehensive evaluation of the patient
2. To assess level of motivation according to Declement’s and Prochaska model (stages of motivation)
3. To enhance motivation (MET)
4. To plan detoxification as per the individual condition (severity, complicated state and associated physical problem)
5. To plan psychosocial treatment - education, support and guidance.

6. To offer specific CBT programme - Covert sensitization, assertive training, cue modification, social skills training, effective time management.

7. To plan various drug treatment options as discussed above.

8. To maintain the therapy - for lapse/relapse.

Good Practice - In addition to above:

1. To have parallel programmes for non dependent Problem drinkers: by a network with GHPUs/NGOs.

Education
- Harm benefits balance
- Disease model
- Chronic relapsing model
- Treatment options and outcome
- Outcome benefits
- Mortality risks in alcohol

2. Comprehensive relapse prevention programmes

- Cue management/assertive training/alternative methods of pleasure/efficient time management/social skills

To develop Indian model of relapse prevention.

3. To practice treatment matching programme

- Careful evaluation
- Cognitive styles, attitude, personality
- Assessment of Physical problems
- Assessment of brain damage
- Assessment of psychological problems
- Past h/o drinking pattern/relapse
- Changing motivation
- To recognize specific vulnerabilities for one individual

The treatment package to be determined

- Detox+drugs (DS) + family support
- Detox+drugs (anticraving) + family support
- Detox+CBT (RP) + family support
- Detox+drugs and CBT + family support
- Detox+educative session+ family support
- Detox+psychiatric treatment + family support
4. Rehabilitation

- Return to family and live in harmony with family members
- To change social circles – shift from drinking society to healthy, non drinking social network
- To develop healthy life styles
- Physical exercises
- Yoga, Transcendental Meditation
- Spiritual inclination (personal) – to be fostered /healthy recreation
- To avoid all addictive substances
- To return to productive work – avoid high risk work situations

xxiii REFERENCES


70. Sellers EM. Clinical Institute Withdrawal Assessment-Alcohol (CIWA-A) Scale Toronto: Alcoholism and Drug Addiction Research Foundation, 1980


