REVIEW OF LITERATURE
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It has been a common observation that the incidence of poisoning has increased in last few years. This is attributed to rapid development of chemistry, leading to availability of newer compounds in trades, industries and medicine. Easy access to these substances, increasing stress, strain in modern time predispose to increased risk of suicidal poisoning, whereas accidental exposure occurs in industrial settings and in children consuming them out of curiosity. Homicidal poisoning is seen when revenge or personal gain is the factor. Organophosphorus and organochloride insecticides, rodenticide, ethyl alcohol, hypnotics and sedatives constitute majority of cases of poisoning. Region wise, organophosphorus poisoning is common in Maharashtra and Tamilnadu. Rodenticide (Rat poison) and copper sulphate poisoning is common in Uttar Pradesh and Madhya Pradesh (Anjaria, 1989).

On the basis of annual reports of chemical examination the common pattern of poison in state of Uttar Pradesh and Madhya Pradesh in the year of 1972, 1973 and 1974 is given table below (Tiwari, 1975).
<table>
<thead>
<tr>
<th>Name of poison</th>
<th>Percentage of total poisoning cases</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1972</td>
</tr>
<tr>
<td>Alcohol</td>
<td>28</td>
</tr>
<tr>
<td>Insecticide</td>
<td>25</td>
</tr>
<tr>
<td>Zinc phosphide (Rodenticide)</td>
<td>21</td>
</tr>
<tr>
<td>Dhatura</td>
<td>9</td>
</tr>
<tr>
<td>Sedatives (Barbiturate, Morphine, Psychotrophic drugs)</td>
<td>10</td>
</tr>
<tr>
<td>Copper sulphate</td>
<td>2</td>
</tr>
<tr>
<td>Others (Methylene, Methyl alcohol, Cynide)</td>
<td>5</td>
</tr>
</tbody>
</table>


<table>
<thead>
<tr>
<th>Name of poison</th>
<th>Percentage of poisoning</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1968</td>
</tr>
<tr>
<td>Organophosphorus</td>
<td>40</td>
</tr>
<tr>
<td>Endrin</td>
<td>32</td>
</tr>
<tr>
<td>Zinc phosphide (Rodenticide)</td>
<td>13</td>
</tr>
<tr>
<td>Dhatura</td>
<td>8</td>
</tr>
<tr>
<td>Alcohol</td>
<td>3</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td>4</td>
</tr>
</tbody>
</table>

According to Pohwala and Ghai (1956) accidental poisoning in children is a global problem. Accidental
poisoning is a 12th leading cause of admission in paediatric wards in India and account for about 1% of hospitalised patients. Most of cases of accidental poisoning were preventable. The pattern of accidental poisoning in children in central India is shown in table below.

<table>
<thead>
<tr>
<th>Accidental poisoning</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kerosene oil</td>
<td>30.2</td>
</tr>
<tr>
<td>Poison plants and seeds (Dhatura)</td>
<td>27.2</td>
</tr>
<tr>
<td>Household medicine (Aspirin etc.)</td>
<td>23.5</td>
</tr>
<tr>
<td>Insecticide, DDT, Naphthalene</td>
<td>8.1</td>
</tr>
<tr>
<td>Food poisoning</td>
<td>6.6</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td>4.4</td>
</tr>
</tbody>
</table>

In 1972, on the basis of theoretical model a WHO expert committee made the 1st global estimate for the number of cases of acute pesticide poisoning. This indicated that problem was large and required urgent attention. Data supporting the model were subsequently obtained through the study in Indonesia, Malasiya, Srilanka and Thailand. It is largely a problem of developing countries.

The extent of problem of acute pesticide poisoning has been highlighted as a major health concern in the Srilankan study. It was noted that in the year of study, 982 deaths from acute pesticide poisoning in State hospitals were reported. This figure strongly demonstrate public
health importance of the problem, as it was almost twice as high as the total number of deaths in same hospital because of Malaria, tetanus, the traditional killers in the developing countries. The study in developing country found that approximately 2/3rds of all acute pesticide poisoning were suicidal attempts and approximately 1/4th were accidental. In remaining cases, the cause was not defined. Pesticides are often used to commit suicide in these countries because these hazardous compounds are readily available to public (Jayaratnam, 1982).

Acute poisoning is an important medical emergency accounting for a significant proportion of admissions in the medical emergency wards. Three hundred sixty patients with acute poisoning comprising 2.15% of all medical admission in a year were analysed by Kumar et al (1988). Males out numbered females (283 : 77), age range was 13-70 years (mean age 28.7 years). Almost three fourths (73.3%) cases were in the 2nd and 3rd decades of life. Dhatura poisoning was the commonest (78, 21.66%) followed by alcohol poisoning (42, 11.66%), CNS depressant viz. sedative and psychotrophics (28), Barbiturate -9, dilantin-1 and opium - 2 (Total 40 cases, 11.11%), copper sulphate (26 cases, 7.22%), corrosive (16 cases, 4.4%), aluminium phosphide and rodenticide poisoning were seen in 2.5% and 2.7% cases respectively. Two cases of dog killer poisoning were also encountered, miscellaneous group (20 cases, 5.5%)
including poisoning by diverse substances viz. INH, Dapsone, Hg, potassium dichromate, carbon mono-oxide, ether, bhang and common household chemicals. Dettol, tincture iodine, camphor, phitkari, neel, naphthelene etc. exact poisons could not be identified in 66(18.33%) cases. The intention was suicidal in 177 (49.16%), accidental in 29 (8.05%) and homicidal in 6 (1.65%) cases. It was interesting that in 73 (20.27%) cases poison was used to stupify to rob the patients, 21 patients died and mortality rate was 5.7%.

Samaria et al (1988) carried out a study of poisoning in eastern UP and Western Bihar. They analyzed 225 patients of different poisonings attended the casualty department during a period of one year in Institution of Medical Sciences, Varanasi. Out of total 225 cases, 175 belonged to UP and 49 belonged to Bihar and one patient was from M.P. Of the total 120 (53.4%) were females and 105 (46.6%) cases were males. Maximum cases belonged to age group (11-20) years followed by age group 21-30 years (23.12%) and the age of 31-40 years (10.66%). Much less number of cases belonged to the age group of 10 years and 740 years. Majority of the cases (160, 71.2%) were suffering from organophosphorus poisoning followed by 20 (8.88%) cases of diazepam poisoning, 15(4.45%) of Barbiturate poisoning, 7(3.12%) cases of petroleum product like kerosene oil, petrol etc., 3(1.34%) of corrosive poisoning i.e. by acid and alkali and salicylate
phenothiazine poisoning was less common having only one patients each (0.44%).

From a decade, insecticides like aluminium phosphide and endosulphan are being frequently used for poisoning. Maltani et al (1989) conducted a study of changing spectrum of acute poisoning in 106 adults in period of 6 months in Medical College, Hospital, Patiala. Organophosphorus compound was the commonest agent(44.5%), aluminium phosphide (20%), endosulphan (16.36%), alcohol (7.27%), kerosene oil (2.3%), copper sulphate (1.82%), opium (1.82%), diazepam (0.91%) and mixed (3.64%). Ninety four (85.45%) cases were males and sixteen (14.55%) were females and peak age was between 21-30 years(43.18%). Acute poisoning was more common in lower income groups (56.36%) and in rural areas (64.54%). In addition to information supplied by the patients and/or attendants, clinical examinations helped in making the diagnosis. The mean hospital stay was 2-7 days and majority of patients stayed less than 24 hours (58.18%), over all mortality rate in this study was 25.5%. It was highest with aluminium phosphide poisoning (77.2%), followed by mixed poisoning and organophosphorus poisoning (25% and 18.32% respectively). Easy availability and quick lethal action of aluminium phosphide and endosulphan has prompted frustrated people to consume these agents, lack of awareness, regarding methods of spraying caused organophosphorus
compound poisoning in large number of cases.

The clinical examination from 121 patients of acute poisoning admitted through casualty department in Medical College, Hospital, Cuttuck within the period of 2 years, were analysed by Mahapatra et al (1991). They showed that there were 77 (63.6%) males and 44 (36.3%) females patients. The mean age was 27.2±10.4 years and it was commonest (40.49%) in age group of 21-30 years followed by 11-20 years (25.6%) and then progressively declining over the years. Organophosphorus compound poisoning was the commonest (28.9%) followed by Oleander (17.3%) and adulterated alcohol (17.3%) intoxication. There was self induced poisoning in 90 (74.3%) cases and accidental in 31 (25.6%) patients.

Chitalkar et al (1991) conducted a retrospective study of forty two patients with poisoning admitted to Military Hospital, Ambala Cantt during an eighteen months period. Their age ranged from eighteen months to forty eight years with mean age of 21.6 years. Twenty were males and 22 were females. They took a total of eighteen different substances. Organophosphorus in eight (18.9%) and aluminium phosphide in 6 (14.2%) were the commonest, 18 patients consumed poison with suicidal intent and ten consumed it accidentally. The cause remained unknown in twelve. Two cases were given dhatura homicidally.
Psychiatric evaluation showed at least six individuals with depression. Four of forty two (9.1%) died.

Clinical profile of 100 cases of poisoning admitted in acute medical care unit were studied in Vishakhapatnam from January, 1992 to 31st May, 1992. Fifty five percent cases were males and 45% cases were females. Majority of cases (74%) were under the age of 30 years. No case of poisoning was recorded after the age of 50 years. The commonest type of poisoning in this study was due to organophosphorus and allied compounds (35%), followed by drugs (22%), ant poison (Gamanaxene-20%), rat poison (zinc phosphide - 10%), alcohol - 5%, Nerium seeds - 4%, phenol - 3%, sulphuric acid - 1%,

ALUMINIUM PHOSPHIDE POISONING

Aluminium phosphide is widely used for grains and fruits preservations against rodents and insects (Roy and Bedi, 1979). Aluminium phosphide is a solid fumigate pesticide. It is marketed in India as a tablet of celphos and quickphos manufactured by Oexcel Laboratory and Inventa Corporation respectively. It was declared as an ideal pesticide because of its being cheap, most efficacious and easy to use (Thomas, 1973). Its effects were due to liberation of phosphine gas which is toxic to pests, insects and rodents. After fumigation the non toxic residue left in the grains are the
phosphite and hypophosphite of aluminium.

Human toxicity which is usually acute, occurs either due to inhalation of phosphine from fumigated grains (Wilson et al, 1980) or after ingestion of aluminium phosphide. The latter liberates phosphine in the stomach and this is widely absorbed through out the gastrointestinal tracts. Earlier acute intoxication was limited to inhalation of phosphine, but in last decade poisoning due to aluminium phosphide ingestion has been reported from different northern states (Jain et al, 1985; Agrawal et al, 1989; Khosla et al, 1990).

This acute ingestional toxicity has become a common cause of death among young generation in northern India. In India the first case was reported in 1981, during 1984-85 study of few cases appears in literature on its epidemiological and electrocardiographic aspects. Its prevalence rate calculated on the basis of hospital admission was 2-3 cases per thousand admissions and it surpassed every other poisoning in Haryana. The poisoning is more common in young adults, mostly in their teens. The mode of poisoning is usually intentional, occasionally accidental and rarely homicidal. The agricultural community irrespective of sex is more at risk due to illiteracy and easy availability of pesticide in household. The peak season is from May to September.
Organ damage is widespread, its pathogenesis is still not clear (Khosla et al, 1990). The damage is hypoxic in nature and due to binding of cytochrome oxidase at cellular level. The acute cardiotoxicity is also due to subcellular transmembrane exchange of ions (Na, K, Mg and Ca)$^+$ brought on by focal myocardial necrosis produced by phosphine. The high mortality is due to rapid onset of symptoms delay in arrival of hospital, delay in instituting treatment, lack of an antidote, non responsiveness of shock to resuscitative measure and consumption of large dose (Chug et al, 1989). Little attention has been paid to literature to management of these cases and even standard text book do not deal with the subject in details despite increasing awareness of this poisoning.

Each tablet of celphos or Quick phos (3.0 gm) contains 56% aluminium phosphide and 44% ammonium carbonate and had the capacity to liberate 1.0 gm of phosphine gas. The lethal dose for human is 150-500mg for a 70 kg person. (Sidney, 1980). The gases liberated during its reaction with water, humidity are phosphine and ammonia etc.

Aluminium phosphide + 3 $\text{H}_2\text{O}$ $\rightarrow$ $\text{Al(OH)}_3 + \text{PH}_3$ (Phosphine)

$(\text{NH}_4)_2 \text{CO}_3 + \text{H}_2\text{O}$ $\rightarrow$ $2\text{NH}_4\text{OH} + \text{CO}_2$ $\rightarrow$ $\text{NH}_3 + \text{CO}_2 + \text{H}_2\text{O}$ (Ammonia)
CLINICAL FEATURES

The clinical symptomatology is more or less same irrespective of the mode of toxicity, except that the initial symptoms pertain to the route of entry. The signs and symptoms depend on the dose and severity of poisoning. In mild ingestional intoxication severe systemic features do not occur except G.I.T. symptoms viz., nausea, vomiting, headache and abdominal pain or discomfort, and these patients usually recover. On the other hand systemic manifestations are early and progressive in moderate and severe poisoning and most of times prove fatal. Initial gastrointestinal symptoms may be followed by shock (Singh et al, 1985 and Chopra et al, 1986).

Chug et al (1991) reported the following symptoms and signs of moderate to severe aluminium phosphide poisoning:

a. Gastrointestinal symptoms are nausea, vomiting, diarrhoea and retrosternal pain.

b. Cardiovascular - hypotension or shock, changes in heart rate (bradycardia or tachycardia), myocarditis, pericarditis, acute congestive heart failure and ECG changes.

c. Respiratory - cough, dyspnoea, cyanosis, rales and rhonchi, respiratory failure (Type-I).

d. Hepatobiliary - Jaundice, tender hepatomegaly, raised transaminases, enzymes.
e. Renal - oliguric and non oliguric renal failure.
f. Central Nervous System - Headache, dizziness, restlessness without alteration of consciousness, acute hypoxic encephalopathy.

The clinical parameter of aluminium phosphide poisoning were studied by various authors in different states. The incidence of aluminium phosphide poisoning in Haryana was unknown before 1980 but now its incidence is progressively increasing not only in Haryana but in the whole of Northern India. The incidence of aluminium phosphide poisoning was 0.06 per thousand of hospital admissions in 1981 and steadily increased to 10 per thousand in 1989 in Medical College Hospital Rohtak (Chug et al, 1991). Majority of patients (92%) were belonging to age group of 15-40 years. The male : female ratio was 40 to 73% patients and male 27 to 60% females. Sixty to 85% patients were belonging to rural area and in 71 to 97% of cases the poisoning was intentional (suicidal) in nature Sepaha et al, 1985; Singh et al, 1985; Siwatch et al, 1988; Agarwal, 1989; Chug et al, 1991; Khaniju et al, 1990; and Khosla et al, 1992).

The clinical spectrum of aluminium phosphide poisoning gastrointestinal upsets such as nausea, vomiting, shock (61-90%), tachycardia (51-74%) patients, 30 to 50% patients had S3 and S4 gallop, tachypnoea, dyspnoea, crepts and rhonchi, 20-30% patients had altered sensorium, 8-10% had acute renal failure, 1-5% patients had tender
hepatomegaly and 3% patients had bradycardia (Katira et al, 1990; Khosla et al, 1990 and Chug, Dushyant and Santram, 1992).

In aluminium phosphide poisoning ECG changes were present in 75-95% of cases. They were ST-T changes (both ST elevation and depression) in 30-65%, atrial fibrillation in 50 to 60%, sinus tachycardia 20 to 40%, right bundle branch block 15 to 30%, sinus bradycardia 10 to 15%, ventricular ectopics 10 to 15%, T wave inversion 10 to 14%, cases, left bundle branch block 10%, idioventricular rhythm 10%, wandering pacemaker 3 to 5%, ventricular tachycardia 5%, tachybradycardia syndrome 5% (Trivedi et al, 1982; Jain et al, 1985; Raman and Dubey, 1985; Chopra et al, 1986; Chug et al, 1989 and Katira et al, 1989).

Aluminium phosphide poisoning is a life threatening emergency with a high mortality and no specific antidote (Khosla et al, 1992). Mortality has been highly variable (37-100%) (Chopra et al, 1986; Ram et al, 1988 and Bajaj et al, 1989). Mortality depend on the dose of poison, freshness of the compound (Katira et al, 1990), duration and severity of shock, presence or absence of bad prognostic factors, and complications. The bad prognostic indices included poor response of shock to dopamine infusion, anaemia, chest infections, metabolic acidosis, severe hypoxia, electrolyte disturbances, presence of arrhythmia and aspiration, pneumonia (Chug et al, 1990).
The complications included pericarditis, acute congestive cardiac failure, acute massive gastrointestinal bleeding and respiratory failure (Chug et al, 1991). The time interval between ingestion and death varied from 4 to 40 hours with a mean of 12.1 hour. The commonest cause of death was peripheral circulatory failure (76%), remaining died due to recurrent ventricular fibrillation (9%), acute pulmonary oedema (9%) and fulminant hepatic failure (6%), (Agarwal, Agarwal and Jain, 1989).

ORGANOPHOSPHORUS POISONING

Organophosphorus insecticides are chemical agents that have a powerful inhibitory action on enzyme acetylcholinesterase. This is due to the fact that their phosphate radicals firmly bind to action site of enzyme to form stable phosphorylated complexes. These substances thus acts as anticholinesterase agents and results in accumulation of acetylcholine at cholinergic nerve endings throughout the body. The overabundance of acetylcholine initially stimulates and then paralysis impulse transmission in cholinergic synapses. Cholinergic transmission occurs in widespread areas of the body including autonomic ganglia, parasympathetic nerve endings, somatic nerves, cells of the central nervous system and some special sympathetic nerve endings such as in sweet glands. It is this wide spread distribution of cholinergic nerve endings
in the body that accounts for diversity of clinical features in organophosphorus poisoning (Gupta et al, 1988).

Pharmacological organophosphorus compounds have three types of actions (a) Muscaranic (b) Nicotinic and (c) on the central nervous system.

As a result of the muscaranic like effect the following signs and symptoms are observed.

A. Bronchial Tree: Tightness of the chest with prolonged wheezing expiration suggestive of bronchoconstriction and increased secretion. Therefore there is discomfort or pain in chest, dyspnoea, cough, pulmonary oedema and cyanosis. The effect stimulates bronchial asthma.

B. Gastrointestinal: Anorexia, nausea, vomiting, abdominal cramps, epigastric and substernal tightness with heart burn and eructations, diarrhoea, tinismus and involuntary defaecation.

C. Increased sweating, salivation, lactation, tears, may be red due to porphyria in lacrimal glands.

D. Heart bradycardia.

E. Pupils - Slight miosis, occasionally unequal and later more marked miosis.

F. Urinary bladder frequency of micturition and involuntary micturition.

As a result of nicotine like effects, the following signs and symptoms are observed. Straight
muscle - easy fatigue, mild weakness, muscular twitching, cramps, fasciculations, generalize weakness of muscles' of respiration with diarrhoea and cyanosis.

As a result of action of central nervous system the following effects may appear approximately in the order mentioned below:

1. Irritability, apprehension, restlessness.
2. Fine fibrillary tremors of hands, lids, face and tongue.
3. Mental confusion progressing to stupor and muscular weakness with tremors and convulsions.

Incidence of suicidal attempts with consumption of organophosphorus compound is increasing in recent years because of frustration and unemployment (Bitchili et al., 1990).

The clinical spectrum of organophosphorus poisoning were studied by various authors in different states. There were 60-70% of male cases and 30-40% cases were females. Majority of patients (80%) belonged to rural area (Bhakat Ram et al., 1989; Ghosh et al., 1991).

Clinical finding of organophosphorus poisoning were nausea, and vomiting (80-100%), altered sensorium (60%), constricted pupil (60%), pulmonary oedema (60%), excessive sweating, frothings, lacrimation, salivation
(30-60%), abdominal pain (30%), abnormal behaviour, ghabrawat, restlessness, giddiness (10-15%) and pulse rate varied from 32 to 140/min (Vishwanathan et al, 1962; Gupta et al, 1968; Surjeet et al, 1969; Gupta et al, 1989). The overall mortality was found to be 15-45% (Bhakt Ram, et al, 1989; Chamundeswari, 1990; Ghosh et al, 1991).

Sennanayke and Kanaliedda (1987) described yet another manifestation of neurotoxicity following the cholinergic illness of organophosphorus compounds, a paralytic syndrome was observed in 5-10% of patients with organophosphorus poisoning. Muscles weakness developed 24 to 96 hours after the cholinergic illness, involving primarily the proximal limb muscles, neck flexors, certain cranial motor nerves and the muscles of respiration, because of weakness of muscles of respiration. Seventy percent patients had difficulty in breathing and 30% patients died from respiratory failure. This condition called intermediate syndrome and was not responsible for atropine and pralidoxime and required urgent respiratory support.

Bitchilli et al (1990) reported interesting neurological deficit besides muscaranic and nicotinic manifestations of organophosphorus poisoning. They were cerebellar syndrome, proximal muscle weakness and sluggish reflexes, neck muscles weakness, ptosis.
Twenty five to forty five percent of cases of organophosphorus poisoning had some ECG changes. ECG changes showed sinus bradycardia (10-20%), sinus tachycardia (15%), proximal atrial tachycardia (10%) and 5% cases had ventricular premature beats (Singh and Malnotra, 1991 and Bitchili et al, 1990).

**Zinc Phosphide (Rodenticide Poisoning)**

Zinc phosphide is an efficient rodenticide when moist the chemical slowly gives phosphine, whose garlic odour is repellent to man and domestic animals. But seems to have no adverse effects on rats. Zinc phosphide now extensively used in India. It is used in the ratio of 1 part to 10 part of wheat or rice flour and mixed with a few drops of edible oil in order to render it more attractive to rats. Because of its good safety records, low cost and reasonably high effectiveness, zinc phosphide is recommended for large scale use against rat (Park and Park, 1986).

Commercial zinc phosphide is one of the well known rat poisons, it is a mixture of different phosphides of zinc in addition to traces of zinc phosphate and zinc oxide. The chief symptoms after the administration of zinc phosphide are vacant look, frequent vomiting with retching, tremors and drowsiness followed by respiratory distress and death. Zinc phosphide acts in the stomach
with liberation of phosphine which acts as a respiratory poison (Modi, 1985).

In 1989 Subramaniam carried out a study of 712 cases of poisoning admitted in toxicology department in Madras during 2 years. Out of which 32 cases were of zinc phosphide (rat killer) poisoning. The death rate of rat killer poisoning was 6% when compared to north Indian States (25%).

**DHATURA POISONING**

Dhatura plant is known as thorn apple and commonly grows in waste places all over India. The fruits are spherical and have sharp spines giving the name thorn apple to plant. An average sized fruit contains 450 to 500 seeds and weight about 8 gms. Dhatura is poisonous in all its parts but the seeds and fruits are considered to be most noxious. The active substance is known as dhaturine and contains the alkaloids levohyoscymine, hyoscine or scopolamine and traces of atropine. The alkaloids of dhatura powerfully stimulate the higher and other centres of the brain and also exercise at same time a paralysing action on various nerves. They inhibit secretion of sweat and saliva, dilate the cutaneous blood vessels and pupill and stimulate the heat regulating centre situated in floor of third ventricle. The initial stimulation of various centres is followed by depression (Parikh, 1985).
Within half an hour of taking the poison gastric irritation starts. The patients complains of bitter taste, dry mouth and throat, burning pain in stomach and difficulty in swallowing and talking. This is followed by giddiness, ataxia, inco-ordination of muscles, a peculiar flushed appearance of face, dry hot skin, rise in temperature, diplopia, dilated pupil with loss of accommodation redening of conjunctiva and drowsiness. Some time erythematous rash appears all over body. There is usually full, bounding pulse which later becomes weak and irregular. The patient develops muttering delirium, tries to run away from beds, picks at bed cloths, tries to pull imaginary threads from the tips of his fingers and develops dreadful hallucination of sight and hearing. The condition may pass on to stupor, convulsions, coma and some times death from respiratory failure (Tembe, 1991).

The treatment of Dhatura poisoning is gastric lavage with weak potassium permangnate solution and specific antidote neostigmine given intramuscularly. Excitement and delirium is best controlled with phenobarbitone-sodium administered intramuscularly (Udwadia, 1989).

ALCOHOL POISONING

In general use, the word alcohol means any intoxicating drink of the various alcohols in common use. Only ethyl and methyl alcohols are found in drink, the later only in cheap adulterated spirits. The term alcohol is in popular use and is referred to ethyl alcohol (Ethanol).
Ethyl alcohol depresses the central nervous system irregularly in descending order from cortex to medulla. It first depress the higher centres which control judgement and behaviour (stage of excitement) than the motor centre (stage of incoordination) and finally vital centres in medulla (stage of narcosis). The range between a dose which produce narcosis and one which impairs vital functions is small (Parikh, 1985).

Acute poisoning may result from consumption of an alcoholic beverage in small doses at short intervals or in excessively large dose at a time. The symptoms of acute poisoning is at first a sense of well being, self confidence, flushing of skin and face, a care free behaviour and then gradual loss of self control, argumentativeness, rude behaviour, sentimentality and moresness or melancholic. These are followed by stage of confusion and dulling of perception, muscular incoordination, staggering gait, slurred and incoherent speech, blurred vision and stupor. After a time, recovery may occur accompanied by nausea and vomiting which are regarded as the early signs of recovery. These may be followed by sleeps, severe headache and gastric upset. If recovery does not occurs, the patients passes gradually into unconsciousness, some times hypoglycaemia and coma with slow, stertorous breathing and a full rapid pulse which then becomes slow in volume. The breath smells of alcohol (Modi, 1986).
Occasionally acute alcohol intoxication manifest in atypical patterns, these include in acute excitation which takes form of sudden and unprovoked out burst of anger with even assaultive and destructive behaviour and black outs in the form of episodes of transient amnesia that accompany heavy intoxicants (Gupta, 1988).

As most instances of poisoning in adults are deliberate acts of self poisoning, it is very important that all patients, whether suffering from apparent accidental or intensional poisoning should have a psychiatric assessment. Self poisoning is often important feature of depression in particular. The incidence of repeated acts of self poisoning have been shown clearly to be reduced by early psychiatric consultations (Alexander, 1986).
AIMS OF THE STUDY

1. To study the magnitude of problem of poisoning and its incidence in Bundelkhand region.

2. To study the pattern of poisoning in Bundelkhand region.

3. To study the clinical features of acute poisoning.

4. To study the socio-demographic problem related to poisoning.