# "STUDY OF PATTERN OF POISONINGS AND THEIR OUTCOME AMONG IN PATIENTS ADMITTED IN A RURAL TERTIARY CARE CENTRE"


**By**

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**DOCTOR OF MEDICINE**

**IN**

**GENERAL MEDICINE**

**Under the Guidance of**

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**Dr. DINESH REDDY ANAPALLI**

# ABSTRACT

## STUDY OF PATTERN OF POISONINGS AND THEIR OUTCOME AMONG IN-PATIENTS ADMITTED IN A RURAL TERTIARY CARE CENTRE

**Objectives of the study:**

1. To study the clinical profile, investigations of patients presenting with toxicological emergencies.
2. To identify the factors, which help in predicting the severity on admission in patients presenting with toxicological emergencies
3. To assess utility of POISONING SEVERITY SCORE in predicting outcome at admission in these patients
4. Psychiatric evaluation of patients to asses' cause of poisoning.

**Study design:**

The study will be carried out among patients who present to the emergency department of R.L.Jalappa Hospital, Tamaka, Kolar, over a period of one year (Jan2013-Dec2013).

Ours will be a prospective observational study carried out on adult patients presenting with acute poisoning or drug overdose.

**Materials and Methods:**

A prospective, observational study. Informed consent from the patient or the relatives will be taken prior to inclusion in the study. Thorough clinical examination will be done in all patients. Standard proforma was used. Patients will be arbitrarily divided into 5 groups- 1) Drug overdose,2) Insecticide and pesticide poisonings.

3) Plant poisons. 4) Acids and alkalis. 5) Miscellaneous. Patients who fit into the inclusion criteria will be included in the study from the time of presentation at casualty till the time of discharge / death. Autopsy will be done for all patients who succumb due to poisoning or secondary complications. Severity on admission will be predicted using scoring systems – Poisoning Severity Score

**RESULTS**

A total of 310 cases were studied in one year. The Mean age among the subjects was 34.69 ± 13.01 yrs. Majority 37.7% were in the age group 21 to 30 years. Majority of subjects were females 53.2% and Males were 46.8%. It was observed that most common poison used among the subjects was Insecticides and Pesticides in 41.3%, followed by drug overdose 26.5%. In the study death occurred in 22.7% of insecticide poisoning, 15% of miscellaneous poisoning, 4% in acid poisoning and 1.2% in tablet poisoning. Majority of poisoning had high recovery rate. Poison severity score showed Majority i.e. 36% had normal score, 29% had minor score, 15.5% had moderate score, 12.3% had mortality, and 7.1% had severe score. There was significant association between PSS score and Outcome in Poisoning. I.e. among patients with PSS score of death 94.7% died in the study. 40.9% of Severe PSS score had complications. In our study out of 37 deaths, 32 (86.5%) of cases detected by chemical analysis, (70.3%) were positive for organophosphorus, (70.3%),phosphide (10.8%), lastly phenol and oleander (2.7%) each. Out of 29 cases (100%) of insecticides and pesticide poisoning stomach mucosa was congested for 15(51.8%) patients, hemorrhagic for 4 (13.8%), erosive for 5(17.2%) and edematous for 5 (17.2%). Depression is one of the major causes for suicidal deaths. Farmers and unemployed persons were more prone to death by poisoning in the present study

**CONCLUSION:**

The most common poison group is insecticide and pesticide, but there is also increase in consumptions of other group of poisons such as drugs, miscellaneous products.

In the study death occurred in 22.7% of insecticide poisoning, 15% of miscellaneous poisoning, 4% in acid poisoning and 1.2% in tablet poisoning. Equal importance should be given newer group of poisons as there is increase in mortality. There was significant association between PSS score and Outcome in Poisoning. I.e. among patients with PSS score of death 94.7% died in the study. 40.9% of Severe PSS score had complications. Hence PSS can be a useful tool for predicting the outcome in Poisoning subjects. Psychiatric evaluation done for poisoned patients and poverty is most common followed by unemployment in males, in females also poverty is primary cause followed by marital problems and family issues.

## LI ST OF ABBREVIATIONS

| | |
|---|---|
| HB | haemoglobin |
| TC | Total count |
| DC | Differential count |
| ESR | Erythrocyte sedimentation rate |
| HCT | Hematocrit |
| T.PROTEIN | total protein |
| BT | bleeding time |
| CT | clotting time |
| SGOT | serum glutamic oxaloacetic transaminase |
| SGPT | serum glutamate pyruvate transaminase |
| ABG | arterial blood gas |
| PT | prothrombin time |
| INR | international normalized ratio |
| APTT | activated partial thromboplastin time |
| RBS | random blood sugar |
| HBsAG; | surface antigen of hepatits b virus |
| HIV | human immunodeficiency virus |
| ECG | electrocardiogram |
| CPK | creatine phosphokinase |
| CKMB | creatinekinase MB |
| TROP T | troponin T |
| PCHE | pseudocholinesterase |
| $PCO_2$ | partial pressure of carbon di oxide |
| $PO_2$ | partial pressure of oxygen |
| $HCO_3$ | bicarbonate |

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# INTRODUCTION

Poisoning is a significant global public health problem. According to world health organization (WHO), in the year 2004, it is estimated that 346,000 people died worldwide from unintentional poisoning. Of 5,00,000 deaths occurring every year 2,00,000 are due to self poisoning with op compound in south east Asia.

Organophosphate poisoning is the most common cause of poisoning worldwide and intended to use as suicide in agrarian area.[2]Also across Asia Aluminum phosphide poisoning is very common, with mortality as high as 31% in Iran.[3]

Paraquat is the leading single agent causing death from pesticide poisoning in many countries including Sri Lanka.[4] In India, mortality rate from poisoning varies between 15 – 30 % and is the fourth most common cause of mortality , especially in rural India. Pesticides and insecticides are most common poisons in a rural set up worldwide. India is a predominantly agrarian country with about 60-80% of rural population. . As per the rough estimate, in India, around 10 million cases of poisoning are reported annually, of which about 10 thousand happen to die. The incidence of poisonings is increasing day by day because of its low cost, easily availability without any check on their sales and irregularity in distribution

Pesticides are routinely used for advanced farming and they are readily available over the counter. Therefore, a pesticide is an easy access source for the suicidal purpose.[5]Nowadays, it has been observed that patients are presenting with acute poisoning of newer plant like Asparagus, atropa belladonna, aquilegia, drugs, pesticides and fungicides on whom no published data of human toxicity is available. This study is being undertaken to study the prevalence of poisoning in our hospital and the impact it has on society

# OBJECTIVES OF THE STUDY

1. To study the clinical profile, investigations of patients presenting with toxicological emergencies.
2. To identify the factors, which help in predicting the severity on admission in patients presenting with toxicological emergencies
3. To assess utility of POISONING SEVERITY SCORE in predicting outcome at admission in these patients
4. Psychiatric evaluation of patients to assess the cause of poisoning.

# REVIEW OF LITERATURE

Poisoning is the harmful effect that occurs when a toxic substance is swallowed, inhaled, or comes in contact with the skin, eyes, or mucous membranes, such as those of the mouth or nose. The term poison first appeared in the English literature around the year 1230 A.D. to describe a potion or draught that was prepared with deadly ingredients. Poisons are as old as mankind or perhaps even older. Their description can be found in the ancient Egyptian, Babylonian, Hebrew and Greek literature. Poisons have been described in Atharva Veda (1500 BC), Kalpasthana. Chikitsaathana and Uttarasthana of the Shastras have described symptoms and antidotes of poisons in detail. Susruta (350 BC) described the procedures for incorporating poisons into foods, drinks, perfumes, medicine, bathing water, snuffer sprinklers (The ancient Indian's had mastered the art of turning dazzling damsels into beings capable of delivering death kisses, known as the Vishkanya)[6]. The word **'poison'** has been evolved from the Latin word **'potion'** i.e. **'to drink for health'**, but in the due course of time the definition of 'poison' has changed reversibly to its present form i.e. any substance which when administered, inhaled or ingested is capable of acting deleteriously on the human body.[7] Thus, almost anything is a poison and there is really no boundary between a medicine and a poison, for medicine in a toxic dose may be a poison and a poison in a small dose may be medicine. A pesticide poisoning occurs when chemicals intended to control a pest affect non-target organisms such as humans.

There are two types of pesticide poisoning.

1. *A single and short-term very high-level of exposure* - by individuals who commit suicide, as well as pesticide formulators. A long-term high-level exposure - pesticide formulators and manufacturers.
2. *A long-term low-level exposure* - Individuals are exposed to from sources such as pesticide residues in food as well as contact with pesticide residues in the air, water, soil, sediment, food materials, plants and animals.

   In INDIA, pesticide poisonings from short-term very high-level of exposure (acute poisoning) is the most worrisome type of poisoning.

   The term drug overdose (or simply overdose or OD) describes the ingestion or application of a drug or other substance in quantities greater than are recommended or generally practiced.[8]An overdose may result in a toxic state or death. Drug overdoses are sometimes caused intentionally to commit suicide or as self-harm, but many drug overdoses are accidental, the result of intentional or unintentional misuse of medication. Studies from across India have also shown drug overdose to be one of the cause of suicidal deaths.[9] Usage of illicit drugs of unexpected purity, in large quantities, or after a period of drug abstinence can also induce overdose.M.Akhlaghi, et al (2009) conducted the study on a pattern of acute poisoning in Shahrekord (Western Iran). Altogether 638 patients with acute self poisoning were identified and hospitalized over the study period. Multiple drug poisoning was the main reason of intoxication (89.34) Followed by organophosphates (5.33), opiates (3.14), detergents (1.25) and alcohol (0.94).[10]Hair dye consumption is not an uncommon means of deliberate self harmis in trend as a major source of suicidal poisoning following pesticides because of its easy availability and low cost. Numerous case reports have

been published from India, many of which are from Andhra Pradesh.[11]Accidental or intentional poisoning results in systemic toxicity in a dose-dependent manner.[12]

Among poisonings with household items in one study most cases (66.7%) were due to phenol poisoning while the rest were due to petroleum products (20%) and camphor (13.3%). Pesticides were the second commonest cause of poisoning seen in 11 cases (14.9%) that compromised of baygon, organophosphorus compounds, rat poison, and insect repellents.[13]

Deliberate self-poisoning has reached epidemic proportions in parts of the developing world where the toxicity of available poisons and sparse medical facilities ensure a high fatality rate.[14]. Reactive depression was seen in 80 (41%) patients secondary to failure in academic, social and financial areas and crisis in interpersonal adjustment.. Other contributory factors were financial stress, psychotic disorder, impulsive disorder and anxiety. Consultation by the psychiatrist is one of the important modalities of treatment in a case of suicidal poisoning as it helps an individual to assess himself and prevent such episodes in future.

Poisoning being invariably medico legal in nature among fatal cases, postmortem examination is done to establish the exact cause and manner of death. Manner of death in these cases is predominately Suicidal because of the general belief that it terminates life with minimal sufferings or accidental but however homicidal cases are also reported and alleged which was more prevalent in the past as there were no well established means of detecting poison from the viscera, etc. With the advent of modern techniques of sample analysis, this method of committing homicide has lost its grounds. Many authors have quoted in literature about the stomach mucosa. Colour changes in cases of poisoning, like in fatal Arsenic poisoning, the stomach mucosa has a red velvet appearance. It’s leathery in Phenol (carbolic acid) poisoning

and corroded & blackened in Corrosive poisonings.[15]Fatal poisons like oraganophosphorus, organochlorines, carbamates, aluminum phosphide, and alcohol were the commonest poisons in the area. Appearance of stomach mucosa in a particular kind of poisoning might be of immense help in making a provisional diagnosis. The analytical methods employed at Forensic Science Laboratory, for the detection of organophosphorus, alcohol with organophosphorus, organchlorine, pyrethroid group, oleander, phosphide, and barbiturates were color test and Thin Layer Chromatography. Color test and volumetric methods responded for the presence of Alcohol and the highest percentage of alcohol detected is 126.5mg/100ml of blood and the least percentage of alcohol is 86.5mg/100ml of blood. The reason for more number of victims to choose organophosphorus were due to low cost, easy availability of highly toxic pesticide, agriculture based economics and to the fact that poisoning by agro-chemical is practically inevitable because modern farming is unthinkable without the use of these and especially for a developing country like ours. Most of the deaths were due to OPcompounds.[16]

Alcohol dependence is a substance related disorder in which an individual is addicted to alcohol either physically or mentally and continues to use alcohol despite significant areas of dysfunction, evidence of physical dependence or hardship.

According to the DSM-IV criteria for alcohol dependence, at least 3 out of 7 of the following criteria must be manifest during a 12 month period:

1. Tolerance
2. Withdrawal symptoms or clinically defined Alcohol Withdrawal Syndrome
3. Use in larger amounts or for longer periods than intended
4. Persistent desire or unsuccessful efforts to cut down on alocol use
5. Time is spent obtaining alcohol or recovering from effects

6. Social, occupational and recreational pursuits are given up or reduced because of alcohol use
7. Use is continued despite knowledge of alcohol-related harm (physical or physiological)

**Toxic kinetics**

The median lethal dose (LD 50) is the smallest dose of given chemical that will kill 50% of a test group of animals. LD 50 is used to extrapolate the toxic potential of compound to the human. It is used with all the routes of poisoning except inhalation[17].The median lethal concentration (LC 50) is the smallest concentration of a given chemical that will kill 50% of a test group of animals. It is applicable to chemicals that are inhaled. The LD 50 is expressed relative to the duration of exposure. The threshold limit value (TLV) is the maximum amount a chemical that is considered safe.

**Pharmacokinetics**

The route of entry of poison into the body can be by any route inhalation, ingestion or external application, on the wound or unbroken skin, ocular exposure by sublingual route[17].

Action of poison may be local or combined generalized one after absorption. Local action results from direct action on the tissues as in the case of corrosives or kerosene after aspiration in the lungs. Remote action is due to absorption of poison into the system. After absorption it may cause specific action or nonspecific action. The specific action depends upon the effect of poison on certain organs with which it

has specific affinity. Nonspecific action like shock may occur similar to that which occur after mechanical injury.

**FACTORS MODIFYING THE ACTION OF POISON**

I. **Dose:** There is a direct relationship between the dose and toxic effect. But sometimes even a small amount can cause severe toxic effects due to personal hypersensitivity to the drug, Eg. Aspirin, Sulphonamides, Penicillin, Phenothiazine, or to the certain foods. Eg. Fish, eggs, mushrooms[17]

II. **Form of Poison:** The physical state, chemical combination and mechanical combination influence the toxicity of poison.

Gases of vapors act more quickly than liquid poisons because they absorbed immediately. Liquid poisons act more quickly than solid poisons. The toxic effect of substances may vary greatly according to their solubility or insolubility resulting from chemical combination. Some subset the action of poison is altered when combined mechanically with inert substances. Poisons act slowly when the stomach is full and contains fatty foods.

I. **Route of administration:** A poison acts most rapidly when inhaled in gaseous or vaporous form or when injected IV. More rapidly when given IM or SC, least rapidly when swallowed[17].

II. **Condition of the body:** The action of poison depends upon the age of a person. A substance nontoxic to adult is toxic to the child. Children cannot tolerate adult doses. As a rule children are more susceptible to poisons than adults.

III. **State of health and nutrition:** Children who are malnourished are more susceptible to poison probably due to absence of glycogen[17].ances such as metallic copper and arsenic are nonpoisonous but their chemical compounds manifests toxicity.

**FATE OF A POISON IN THE BODY**

Most part of a poison may be lost by vomiting or diarrhea unless the poison is liquid or given in small quantities. Once it has been absorbed, it is dealt with the body in several ways[17].

1. It may resist all attempts by body to alter its chemical composition and may be excreted unchanged in urine.
2. It may undergo biotransformation either partially or completely metabolized.

Most of the poisons are detoxified in the liver. The main route of excretion of poisons and its products is the urinary tract, but the other routes are the intestine, usually the mucosa of colon, bile ducts, sweat glands, saliva, lungs and mucous or serous outflows.

The excretion of poison in urine and faeces is most important. Certain tissues such as epidermis, nails and hairs retain the poison such as arsenic even after the other tissues have eliminated. Bone holds poisons such as lead and radioactive metals for longer periods [17].

## EVALAUATION OF CASES OF POISONING

This can be done by:

1. History.
2. Physical Examination
3. Laboratory Evaluation

**History:** Obtaining an accurate problem oriented history is of paramount importance if a poison has occurred or is suspected. In majority of the cases there will be direct history of having consumed some poisonous substances or drugs. The following information should be obtained during the initial assessment[18]

1. Description of toxin
2. Time of exposure
3. Magnitude of exposure
4. Progression of symptoms and
5. Medical history for underlying disease.

When there is no direct history, the diagnosis of poisoning must be considered, when a child presents with acute decrease in level of consciousness, abnormal behavior, seizures or coma, shock, cardiac arrhythmias, respiratory distress, severe vomiting and diarrhea, unexplained metabolic acidosis and any unexplained multi-system disorder. Information about medication in the home or toxic chemical agents stored in household is important in guiding the physician in the right path to initiate treatment[17,16].

**Physical examination:** When the history is unreliable and inadequate, the physical examination assumes greater importance. The physician should pay close attention to look for various signs based on which underlying poison can be identified.

They physical examination starts with vital signs, which determines the immediate treatment. If immediate threat to life is present, emergency management should be immediately started and the patient is stabilized. Once the stabilization is assumed, clues be sought to aid in the determination of confirmation of what was ingested and the rest of the examination is carried out[18].

The Physical examination consists of

1. Vital signs – pulse, respiration, BP, temperature
2. Examination of skin and mucous membrane
3. Odor of breath and body
4. Neurological examination
5. Cardio-vascular and respiratory system examination

The patient should be assessed at appropriate intervals, since many toxins have a delayed onset of action.

**Laboratory Evaluation:** Specialized toxicology laboratories now carry out techniques that allow identification of most poisons within two hrs. Often it is much easier and quicker to analyze the remains of poison in the contained than the body fluids because of greater dilution of the later. In general, for determining the presence of a substance urine screens are used, when determination of quantity is important blood is the preferred sample[18,16].

Additional information may be gathered by doing general laboratory tests like, serum electrolytes, arterial blood gas analysis and hematological investigations. Finally hepatic and renal function tests may be desirable to estimate the patient's capacity to metabolise toxins and / or to determine whether the toxin has damaged these organs[18].

## GENERAL PLAN FOR THE MANAGEMENT OF POISIONING

The corner stone of managing patients suspected of poisoning is attention to the airway, breathing and circulation, the ABCs. Even with ready availability of antidotes, basic resuscitation skills are essential before gastro-intestinal decontamination and further medical toxicological assessment can be of value [19,18]. Majority of the poisonous cases, however have rarely serious symptoms or serious outcome. Hence attempts are being made for accurate risk assessment to avoid unnecessary medication or procedures.

### Current concept in management of poisoning

### Gastric lavage

Although gastric lavage has been widely used for many years. The procedure is time consuming and even under the best of circumstances, removes only a fraction of gastric contents. It should only be used in selected situations[20,19].

### Activated charcoal

The use of activated charcoal to prevent absorption of toxins has increase dramatically in the past 2 decades. Activated charcoal is specially prepared to have a very large

adsorptive surface area. Some toxins including heavy metals, lithium, hydrocarbons and low molecular weight alcohols are not significantly bound to charcoal[18,].

Activated charcoal in a dose 10 times the amount of poison ingested, when given orally absorbs most toxins and decrease the absorption. The dose can be calculated as 0.5 to 1 gm / kg dispersed in 60 to 90 ml of water. In some serious poisoning when life threatening symptoms are present, repeated doses of charcoal may be useful to adsorb either toxin not bound by the first dose, or toxin may be recirculated through the gut. Overall activated charcoal appears superior both to syrup ipecac and gastric lavage[19].

**Whole bowel Irrigation**

As a gastrointestinal decontamination procedure whole bowel irrigation is theoretically used to rinse toxin from the entire gastrointestinal tract to prevent absorption. However, it may also create a concentration gradient to allow previously absorbed toxin to diffuse back into the GI tract. The solution most commonly used is sodium sulphate and polyethylene glycol electrolyte solution, which is not absorbed from GI tract. The usual dose 2L/hour for teenager, continuing until the rectal effluent is clear, usually 4-6 hour[21].

**Enhancing elimination**

In practice, enhancing excretion is useful for only a minority of toxins. Dialytic techniques are not useful for drugs that are either highly protein bound or have a large volume distribution. These techniques are also invasive and associated with risk [18].

Increased poison excretion may be achieved by forced alkaline or acid diuresis, hemoperfusion, hemodialysis, peritoneal dialysis and exchange transfusion. Each of these methods have specific indications and may be attempted in intensive care unit.

**Diuresis**

Diuretics producing diuresis along with increasing the pH of urine with intravenously administered bicarbonate increases the elimination of weak acids. Alternatively acidifying the urine to increase the elimination of weak bases is not clinically usefully. This technique is termed "lon trapping". Drugs which are excreted unchanged by the kidney may have better chanced of rapid elimination through increased urinary flow[18].

**Dialysis**

Hemodialysis is 2-3 times more effective than peritoneal dialysis as amount of toxin in peritoneum is dependent on blood flow rate and is affected its presence of hypotension. Few drugs or toxins are removed by dialysis in amount sufficient to justify the risks and difficulty of dialysis[18].

**Hemo perfusion**

It is a dialytic technique in which blood is passed through a column of activated charcoal or resin. It is rarely used in small children because of the risks associated with its use[22].

**Pharmacological antagonism and chelation**

The use of pharmacological antagonism or diluting agents should be considered though this will be possible in very few poisonings. Antagonists can specifically displace poisons from site of action or act in competition. The use of chelating agents should always be considered in moderate to severe poisoning with metals[19].

**POISONING IS DIVIDED INTO 5 GROUPS**

i. Insecticide and pesticide poisonings
ii. Drug overdose
iii. Plant poisons
iv. Acids and alkalis
v. Miscellaneous

## 1. INSECTICIDES AND PESTICIDES

### Organo Phosphorous compound poisoning




They were first used as an agricultural insecticide and later as potential chemical warfare agents.[2] Organophosphorous (OP) compounds are used as pesticides, herbicides, and chemical warfare agents in the form of nerve gases.[23] Its widespread use and easy availability has increased the likelihood of poisoning with these compounds. India is a predominantly agrarian country with about 60-80% of rural population. Pesticides are routinely used for advanced farming and they are readily available over the counter. Therefore, a pesticide is an easy access source for the suicidal purpose.[24]

**CLASSIFICATION**

| GROUP | X | EXAMPLES |
|---|---|---|
| A | Halogen, Cyanide & Thiocyanate | Disopropylophosphate flourdate (DFP) , ISO propyl methyl phosphoflouridate (SARIN),Pinacolyl Methyl phosphoflouridate (SOMAN) |
| B | Alkyl, alkoxy, aryloxy | Forstenon, DDVP, Pyrazoxon |
| C | Thiol or Thiophosphorous Compound | Parathion, Malathion, Azethion, Diazinon, Systox, and Demeton |
| D | Pyrophosphates and related compounds | TEPP, DPDA, OMPA |
| E | Quaternary Ammonium Compound | Phospholin |

**Pathophysiology and clinical features**

Acetyl choline acts through two receptors:[25,26]

1. **MUSCARINIC RECEPTORS**: *Muscarine* is a poison from toad stools that activates only muscarinic receptors. Effector cells are stimulated by post ganglionic neurons of the parasympathetic nervous system and also postganglionic cholinergic neurons of the sympathetic nervous system.

2. **NICOTINIC RECEPTORS**: Nicotine will activate the nicotinic receptors in pre and post ganglionic neurons of both the sympathetic and parasympathetic nervous systems and also in the membranes of skeletal muscle fibers at NMJ.

**CLINICAL FEATURES**

The clinical manifestations of Organophosphorous poisoning are a result of cholinergic over activity and can be divided into the effects of over stimulation of the muscarinic, nicotinic and CNS receptors.[27]

| Muscarinic | Nicotinic | Central |
|---|---|---|
| Miosis | Muscle Fasciculations | Unconsciousness |
| Blurred vision | Paralysis | Confusion |
| Nausea | Pallor | Toxic psychosis |
| Vomiting | Muscle weakness | Seizures |
| Diarrhoea | Hypertension | Fatigue |
| Salivation | Tachycardia | Respiratory Depression |
| Lacrimation | Mydriasis (rare) | Dysarthria |
| Bradycardia | | Ataxia |
| Abdominal pain | | Anxiety |
| Diaphoresis | | |
| Wheezing | | |
| Urinary Incontinence | | |
| Fecal Incontinence | | |

Following Organophosphorous poisoning three well-defined clinical phases are seen:

- Initial acute cholinergic crisis
- The intermediate syndrome
- Delayed Polyneuropathy(OPIDN-Organophosphorous Induced Delayed Neuropathy)
- Chronic Organophosphate Induced Neuropsychiatric Disorder (COPIND).

1. **Acute cholinergic phase**

   This is the initial phase of acute poisoning resulting in muscarinic and nicotinic effects.

   - The accumulation of acetylcholine at the muscarinic site produces an increase in secretions. Bronchorrhea, salivation, sweating, bradycardia, vomiting and an increase in gastro-intestinal motility (abdominal tightness and cramps) are

commonly seen. In the eye, Organophosphorous agents cause the diagnostic miosis which results in blurring of vision.

- The effects of increased acetylcholine at nicotinic sites [E.g.: The neuromuscular junction] cause muscle fasciculation. Inhibition of acetylcholinesterase in the brain leads to headache, insomnia, giddiness, confusion and drowsiness. After severe exposure, slurred speech, convulsions, respiratory depression and coma occur.
- Death is likely during this initial cholinergic phase due to effects on the heart like bradycardia, arrhythmias; respiratory failure and depression of vital centers in the brain. Bradycardia may be severe and may progress to heart block.

The cholinergic phase usually lasts 24 to 48 hours and constitutes a medical emergency that required treatment in an ICU.[28]

1. **Intermediate syndrome** After recovery from the cholinergic crisis, but before the expected onset of delayed polyneuropathy, some patients develop a muscle paralysis, which is described as Intermediate syndrome.

The cardinal feature of this syndrome is muscle weakness affecting predominantly the proximal limb muscle and neck flexors. Motor cranial nerve palsies (III to VII and X) also occur. Respiratory muscle weakness leading to respiratory failure could lead to a fatal outcome. Deep tendon reflexes are usually depressed. The intermediate syndrome occurs after recovery from the cholinergic crisis within 24 hours to 96 hours but before the expected onset of the delayed neuropathy, which occurs 2 to 3 weeks after the poisoning.[29]

Complete recovery occurs within 4 to 18 days, if adequate ventilator support is provided. But, altered function at the NMJ may persist upto 2 years after its occurrence.

### 3. Organophosphorous Induced Delayed Polyneuropathy (OPIDP)

The neuropathy develops following latent periods of 2-4 weeks after the cholinergic crisis. The cardinal symptoms are distal muscle weakness, calf pain preceding the weakness and in some cases paraesthesia in the distal parts of the limbs. Weakness initially appears in the leg muscles causing foot drop, followed by small muscles of the hands. Later it may extend proximally and may even involve the truncal muscles. Deep tendon jerks are absent. The prognosis of patients with mild neuropathy is good but those with severe neuropathy are usually left with persistent deficits.

The occurrence of Delayed Polyneuropathy appears to follow phosphorylaion and subsequent aging of an enzyme in axons called as *Neuropathy Target Esterase* (NTE). It is present in the brain, spinal cord and the peripheral nervous system. NTE is a membrane bound protein with high esterase catalytic activity. This phosphorylation enzyme also undergoes ageing.[30]

## MANAGEMENT

All patients should be managed as emergencies in hospital.

### Acute Cholinergic Crisis

First aid measures should include:

- Removal of patient from the contaminated environment.
- Removal of contaminated clothes and washing of the skin and eyes.

Respiratory failure is the usual cause of death in the acute phase. Resuscitation and artificial respiration may be required immediately.

Cardiac arrhythmias include various degrees of heart block and should be managed accordingly.

Gastric lavage is most effective within 30 minutes of ingestion. If the patient is semiconscious/unconscious Ryle's tube aspiration can be done

## ATROPINE

Treatment with anticholinergic medication is still the mainstay of treatment and should be started as soon as the airway has been secure. Atropine acts as a physiological antidote, effectively antagonizing the muscarinic-receptor-mediated action. It has virtually no effect against the peripheral neuromuscular dysfunction and subsequent paralysis induced by organophosphorous agent. A recommended dose is 2-4 mg intravenous, repeated at interval of 5-10 minutes initially and continued until signs of atropinisation (dry mucous membrane, dilated pupils, flushing of skin and a heart rate of > 100 beats/minute) appear. Atropine therapy should be maintained until there is complete recovery.

Infusion of atropine is used in some centers in dose of 0.02-0.08 mg/kg/hr. Infusion of atropine has produced significant reduction in mortality in some centers when compared to conventionally intermittent therapy.[31] A heart rate exceeding 140 beats/minute should be avoided. ST-segment abnormalities in the ECG may be induced by large doses of atropine. These may be corrected with *Propronalol*, eliminating any need to reduce the rate of administration of atropine

**GLYCOPYRROLATE**

This is a quaternary ammonium compound can be used as an alternative to atropine. The advantages of Glycopyrrolate over atropine are:

- Better control of secretions[32]
- Less tachycardia[33]
- Fewer CNS side effects.[34]

**OXIMES**

The reactivating action of Pralidoxime is most marked at the skeletal neuromuscular junction. It acts by reactivation of the inhibited phosphorylated enzyme to free the active form.

Initial adult dose of 2-PAM Cl is 1.0 g intravenously. Loading dose of20-50mg/kg based on symptoms severity (dissolved in 0.9% NS infused over 30 minutes), followed by a continuous infusion of 10-20mg/kg/hr. The maximum recommended dose in adults is 12gm in 24 hours. PAM should be administered as early as possible, at least within 4-36 hours as regeneration of AchE. but beneficial response is seen upto 24 hours of poisoning.

The major pharmacological action of oximes is to reactivate AchE by removal of phosphate group bound to the esteritic site.[27]

**DIAZEPAM**

Some reports have indicated that benzodiazepines are useful as antidotes in poisoning by anticholinesterases.[35]

**OTHER MEASURES**

- Dialysis of blood against activated charcoal (hemoperfusion) is effective in Demeton-S-Methyl Sulphoxide; Dimethoate & Parathion poisoning.[36]

**ROLE OF ANTIOXIDANTS IN OP POISONING**

The toxicity of OP compounds is mediated by generation of nitric oxide and other free radicals. These toxic molecules can be counteracted by antioxidants such as vitamins C and E, spin traps, melatonin and low molecular weight thiols. The latter compounds can also increase the synthesis of glutathione, which can both ameliorate the OP induced oxidative stress and enhance OP detoxification.[37] Vitamin E is also a family of lipid – soluble vitamins, of which α-tocopherol is the most active form and is powerful biological antioxidant. Vitamin E may effectively minimize oxidative stress, lipid peroxidation and toxic effects of reactive oxygen species in biological systems. Selenium appears to function as an anti-mutagenic agent, preventing the malignant transformation of normal cells. These protective effects of selenium (as co-antioxidant) seem to be primarily associated with its presence in the seleno-enzymes, which are known to protect DNA and other cellular components from oxidative damage.[38]

## MANAGEMENT OF INTERMEDIATE SYNDROME[39]

Prompt and effective management of respiratory insufficiency is the cornerstone of treatment of Intermediate syndrome. Patients should be observed for early signs of respiratory failure and facilities for ventilator care should be made available. Frequent blood-gas analyses are useful in monitoring and weaning from ventilator support.

## MANAGEMENT OF DELAYED NEUROPATHY[39]

No specific drug therapy has proved useful. The muscle weakness benefits from regular exercise and physiotherapy.

## RODENTICIDE POISONING :[40 (p 148-150)]

Coumarin derivatives are used both therapeutically and as rodenticides. Warfarin (Coumadin) is widely used as a therapeutic anticoagulant, but is no longer popular as a rodenticide because rats and mice have become resistant. The most common anticoagulant rodenticides available today contain long-acting **"superwarfarins"** which have profound and prolonged anticoagulant effects.[40]

**I. Mechanism of toxicity.** All these compounds inhibit hepatic synthesis of the vitamin K-dependent coagulation factors II, VII, IX, and X. Only the synthesis of new factors is affected, and the anticoagulant effect is delayed until currently circulating factors have been degraded.

**II. Toxic dose.** The toxic dose is highly variable.

**A.** Generally, a single small ingestion of **warfarin** (eg, 10–20 mg) will not cause serious intoxication (most warfarin-based rodenticides contain 0.05% warfarin). In contrast, chronic or repeated ingestion of even small amounts can produce significant anticoagulation.

**B. Superwarfarins** are extremely potent and have prolonged effects even after a single small ingestion (ie, as little as 1 mg in an adult).

**III. Clinical presentation.** Excessive anticoagulation may cause ecchymoses, subconjunctival hemorrhage, bleeding gums, or evidence of internal hemorrhage (eg, hematemesis, melena, or hematuria). The most immediately life-threatening complications are massive gastrointestinal bleeding and intracranial hemorrhage.

**IV. Diagnosis** is based on the history and evidence of anticoagulant effects. It is important to identify the exact product ingested to ascertain whether a superwarfarin is involved.

**A. Specific levels.** Blood levels of anticoagulants are not available, nor are they helpful.

An anticoagulant effect is best quantified by baseline and daily repeated measurement of the **prothrombin time** (PT) and calculation of the International Normalized Ratio (INR), which may not be elevated until 1–2 days after ingestion. A normal PT 48 hours after exposure rules out significant ingestion.

**B. Other useful laboratory studies** include CBC and blood type and crossmatch.

The partial thromboplastin time (PTT), bleeding time, and platelet count may be used to rule out other causes of bleeding.

**V. Treatment.** [40]

**A. Emergency and supportive measures.** If significant bleeding occurs, be prepared to treat shock with transfusions and fresh-frozen plasma, and obtain immediate neurosurgical consultation if intracranial bleeding is suspected.

1. Take care not to precipitate hemorrhage in severely anticoagulated patients; prevent falls and other trauma. If possible, avoid use of nasogastric or endotracheal tubes or central intravenous lines.
2. Avoid drugs that may enhance bleeding or decrease metabolism of the anticoagulant

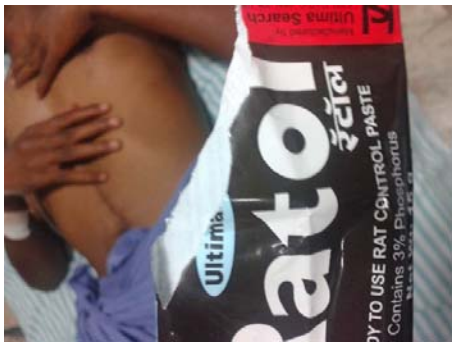
**B. Specific drugs and antidotes. Vitamin K1** (phytonadione), effectively restores the production of clotting factors. It should be given if there is evidence of significant anticoagulation.

1. Give 5–10 mg of **vitamin K1** very slowly IV or subcutaneously (SC) .**Repeated doses** of vitamin K may be required, especially in patients who have ingested a long-acting superwarfarin product. Doses as high as 200 mg/d have been used.
2. Because vitamin K will not begin to restore clotting factors for 6 or more hours (peak effect 24 hours), patients with active hemorrhage may require **fresh-frozen plasma** or **fresh whole blood**.

**C. Decontamination**

Administer activated charcoal. Gastric emptying is not necessary if activated charcoal can be given promptly, and should be avoided in the person who is already anticoagulated.

**PHOSPHINE**[40 ( p 263-264)]




Phosphine is a colorless gas for fumigation, and it is a serious potential hazard in operations producing metal phosphides, where phosphine can be released in the chemical reaction of water and metal alloys. Workers at risk include metal refiners, acetylene workers, fire fighters, pest-control operators, and those in the semiconductor industry. Zinc phosphide and aluminum phosphide are used as fumigants and rodenticides.

I. **Mechanism of toxicity**. Phosphine is a highly toxic gas, especially to organs of high oxygen flow and demand such as the lungs, brain, kidneys, heart, and liver. The pathophysiologic action of phosphine is not clearly understood, but may be related to inhibition of electron transport in mitochondria.

II. **Toxic dose**

Phosphides. Ingestion of as little as 500 mg *aluminum phosphide* has caused death in an adult. The LD50 for *zinc phosphide* in rats is 40 mg/kg; the lowest reported lethal dose in humans is 4 g.

III. **Clinical presentation**.

Inhalation of phosphine gas is associated with cough, dyspnea, headache, dizziness, and vomiting. Phosphide ingestion may cause nausea, vomiting, diarrhea, hypotension unresponsive to pressors, and a rottenfish or garlicky odor. [40]In both exposures, pulmonary edema, myocardial necrosis, convulsions, and coma may occur. Renal and hepatic toxicity are also reported. The onset of symptoms is usually rapid, although delayed onset of pulmonary edema has been described. Most of the patients have vomiting; and systolic blood pressure (SBP) less than 100 mmHg . Also, ECG abnormality was found in some cases at the time of admission. The evaluation of ABG showed that the pH ranged between 6.7 and 7.55.[41]

IV. **Diagnosis** is based on a history of exposure to the agent.

A. Specific levels. Body fluid phosphine levels are not clinically useful.

B. Other useful laboratory studies include BUN, creatinine, electrolytes, liver transaminases, arterial blood gases or oximetry, and chest x-ray.

**V. Treatment**.

A. Emergency and supportive measures

1. Maintain an open airway and assist ventilation. Administer supplemental oxygen, and treat non cardiogenic pulmonary edema if it occurs.
2. Treat seizures and hypotension if they occur.
3. Patients with a history of significant phosphine inhalation or phosphide ingestion should be admitted and observed for 48–72 hours for delayed onset of pulmonary edema.

B. Specific drugs and antidotes. There is no specific antidote.

C. **Decontamination**

Administer activated charcoal, although studies have not determined its binding affinity for phosphides. Consider gastric lavage for large recent ingestion. Use of 3–5% sodium bicarbonate in the lavage has been proposed (to reduce stomach acid and resulting production of phosphine)but is not of proven benefit.

D. Enhanced elimination. Dialysis and hemoperfusion have not been shown to be useful in hastening elimination of phosphine.

**PHOSPHORUS**[40] (p 264-265)

There are two naturally occurring types of elemental phosphorus: red and yellow.

Red phosphorus is not absorbed and is essentially nontoxic. In contrast, white or yellow phosphorus is a highly toxic cellular poison. Although no longer a component of matches, white phosphorus is still used in the manufacture of fireworks and fertilizer and as a rodenticide.

I. **Mechanism of toxicity**.

A. Phosphorus is highly corrosive and is also a general cellular poison. Cardiovascular collapse occurring after ingestion probably results not only from fluid loss owing to vomiting and diarrhea but also from direct toxicity on the hearand vascular tone.

II. **Toxic dose**.

A. Ingestion. The fatal oral dose of white-yellow phosphorus is approximately 1 mg/kg. Deaths have been reported after ingestion of as little as 15 mg.

III. **Clinical presentation**.

A. Acute inhalation may cause conjunctivitis, mucous, cough, wheezing, chemical pneumonitis, and noncardiogenic pulmonary edema. Chronic inhalation of phosphorus may result in mandibular necrosis ("phossy jaw").

B. Skin or eye contact may cause severe dermal or ocular burns.

C. Acute ingestion may cause gastrointestinal burns, severe vomiting, and diarrhea with "smoking" stools. Systemic effects include headache, confusion, seizures, coma, arrhythmias, and shock. Metabolic derangements may occur, including hypocalcemia and hypophosphatemia (or hypophosphatemia). If the victim survives, hepatic or renal failure may occur after 4–8 days.

IV. **Diagnosis** is based on a history of exposure and the clinical presentation. Smoking stools caused by spontaneous combustion of elemental phosphorus suggest phosphorus ingestion.

A. Specific levels. Because serum phosphorus may be elevated, depressed, or normal, it is not a useful test for diagnosis or estimation of severity.

B. Other useful laboratory studies include BUN, creatinine, liver transaminases, urinalysis, arterial blood gases or oximetry, and chest x-ray (acute inhalation).

V. **Treatment.**

**A. Emergency and supportive measures**

1. Observe the victim of inhalation closely for signs of upper-airway injury and perform endotracheal intubation and assist ventilation if necessary. Administer supplemental oxygen. Treat bronchospasm and pulmonary edema if they occur.

2. Treat fluid losses from gastroenteritis with aggressive intravenous crystalloid fluid replacement.
3. Consider endoscopy if oral, esophageal, or gastric burns are suspect

B. **Decontamination** .

1. Inhalation. Remove the victim from exposure and give supplemental oxygen if available.
2. Skin and eyes. Remove contaminated clothing and wash exposed areas with soap and water. Irrigate exposed eyes with copious tepid water or saline. Covering exposed areas may help prevent spontaneous combustion of white-yellow phosphorus.
3. Ingestion

a. Prehospital. Administer activated charcoal if available (although there is no evidence that it adsorbs phosphorus). Do *not* induce vomiting because of the potential for corrosive injury.
b. Hospital. Perform careful gastric lavage. Administer activated charcoal (although there is no evidence that it adsorbs phosphorus).

## PYRETHRINS AND PYRETHROIDS[40] (P 276-277)


Pyrethrins are naturally occurring insecticides derived from the chrysanthemum plant. Acute human poisoning from exposure to these insecticides is rare; however, they can cause skin and upper-airway irritation and hypersensitivity reactions.

**I. Mechanism of toxicity.** In insects, pyrethrins and pyrethroids rapidly cause death by paralyzing the nervous system through disruption of the membrane ion transport system in nerve axons, and pyrethroids prolong sodium influx and also may block inhibitory pathways. Mammals are generally able to metabolize these compounds rapidly and thereby render them harmless.

**II. Toxic dose.** The toxic oral dose in mammals is greater than 100–1000 mg/kg, and the potentially lethal acute oral dose is 10–100 g.

**Chinese chalk** (CockroachWipeout Chalk) contains up to 37.6 mg of deltamethrin per stick of chalk. Ingestion of a single chalk is generally considered nontoxic..

**III. Clinical presentation. .** Toxicity to humans is primarily associated with hypersensitivity reactions.

**A.** Anaphylactic reactions including bronchospasm, oropharyngeal edema, and shock may occur in hypersensitive individuals.

**B.** Inhalation of these compounds may precipitate wheezing in asthmatics. Inhalation or pulmonary aspiration may also cause a hypersensitivity pneumonitis.

**C.** Skin exposure may cause burning, tingling, numbness, and erythema.

**D.** Ingestion**.** With large ingestions (200–500 mL of concentrated solution), the central nervous system (CNS) may be affected, resulting in seizures, coma, or respiratory arrest.

**IV. Diagnosis** is based on a history of exposure. There are no characteristic clinical symptoms or laboratory tests that are specific for identifying these compounds.

**A.** Specific levels**.** These compounds are rapidly metabolized in the body, and methods for determining the parent compound are not routinely available.

**B.** Other useful laboratory studies include electrolytes, glucose, and arterial blood gases or oximetry.

**V. Treatment.**

**A. Emergency and supportive measures**

**1.** Treat bronchospasm or anaphylaxis if they occur.

**2.** Observe patients with a history of large ingestions for at least 4–6 hours for any signs of CNS depression or seizures.

**B. Specific drugs and antidotes.** There is no specific antidote.

**C. Decontamination**

**1. Inhalation.** Remove victims from exposure and give supplemental oxygen.

**2. Skin.** Wash with copious soap and water. Topical application of vitamin E in vegetable oil was reported anecdotally to relieve paresthesias.

**3. Eyes.** Irrigate with copious water. After irrigation, perform a fluorescein examination and refer the victim to an ophthalmologist if there is evidence of corneal injury.

**4. Ingestion** (see p 45). In the majority of cases, a subtoxic dose has been ingested and no decontamination is necessary. However, after a large ingestion of Chinese chalk or a concentrated solution:

Administer activated charcoal.

## II. DRUG OVER DOSE




Drug over dosage were one of the most common poisonings that required critical care management, the most commonly consumed drugs being Sedatives (Barbiturates and benzodiazepines) and Paracetamol.[42] which is again a common pattern similar to the Indian study by Dash *et al.*[43]

**ACETAMINOPHEN[40 (p 62-65)]**

Acetaminophen (Anacin-3, Panadol, Paracetamol, Tylenol, many others) is a widely used drug found in many over-the-counter and prescription analgesics and cold remedies.

**Mechanism of toxicity.**

A. Hepatic injury. One of the minor products of normal metabolism of acetaminophen by the cytochrome P-450 mixed-function oxidase system is highly toxic; normally this reactive metabolite is rapidly detoxified by glutathione in liver cells. However, in an overdose, production of the toxic metabolite exceeds glutathione capacity and the metabolite reacts directly with hepatic macromolecules, causing liver injury.

B. Renal damage may occur by the same mechanism, owing to renal metabolism.

**Toxic dose**.

A. 6–7 g in adults is potentially hepatotoxic.

On the other hand, the margin of safety is lower in patients with induced cytochrome P-450 microsomal enzymes, because more of the toxic metabolite may be produced. High-risk patients include alcoholics and patients taking anticonvulsant medications or isoniazid. Fasting and malnutrition also increase the risk of hepatotoxicity, presumably by lowering cellular glutathione stores.

B. Chronic toxicity has been reported after daily consumption of high therapeutic doses (4–6 g/day) by alcoholic patients.

III **Clinical presentation**. Clinical manifestations depend on the time after ingestion.

A. Early after acute acetaminophen overdose, there are usually no symptoms other than anorexia, nausea, or vomiting. Rarely, a massive overdose may cause altered mental status and metabolic acidosis.

B. After 24–48 hours, when transaminase levels (AST and ALT) rise, hepatic necrosis becomes evident. If acute fulminant hepatic failure occurs, encephalopathy and death may ensue. Encephalopathy, metabolic acidosis, and a continuing rise in the prothrombin time (PT) indicate a poor prognosis. Acute renal failure occasionally occurs, with or without concomitant liver failure.

IV. **Diagnosis**. Prompt diagnosis by serum acetaminophen level. However, patients may fail to provide the history of acetaminophen ingestion, because they are unable (eg, comatose from another ingestion), unwilling, or unaware of its importance. Therefore, many clinicians routinely order acetaminophen levels in all overdose patients, regardless of the history of substances ingested.

A. Specific levels.

After an acute overdose, obtain a 4-hour-postingestion acetaminophen level and use the nomogram to predict the likelihood of toxicity.

B. Other useful laboratory studies include electrolytes, glucose, BUN, creatinine, liver transaminases, and prothrombin time.

**V**. **Treatment.**

**A. Emergency and supportive measures**

1. Spontaneous vomiting may delay the administration of antidote and charcoal and should be treated with metoclopramide or ondansetron.

2. Provide general supportive care for hepatic or renal failure if it occurs.

Emergency liver transplant may be necessary for fulminant hepatic failure. Encephalopathy, metabolic acidosis, hypoglycemia, and progressive rise in the prothrombin time are indications of severe liver injury.

**Specific drugs and antidotes.** ***N*-acetylcysteine** , with a loading dose of 140 mg/kg orally. The effectiveness of NAC depends on **early treatment,** before the metabolite accumulates; it is of maximal benefit if started within 8–10 hours and of diminishing value after 12–16 hours (however, treatment should not be withheld, even if the delay is 24 hours or more). If vomiting interferes with oral acetylcysteine administration, give it by gastric tube and use high-dose metoclopramide (1–2 mg/kg intravenously (IV) or ondansetron , or give the NAC intravenously if necessary.

**3**. **Duration of NAC treatment.** The current widely-used U.S. protocol for treatment of acetaminophen poisoning calls for 17 doses of oral NAC given over approximately 72 hours. We give NAC orally until 36 hours have passed since the time of ingestion. Then, if the serum acetaminophen level is below the limits of detection and liver transaminase levels are normal, NAC can be stopped. If there is evidence of hepatic toxicity, then NAC should be continued until liver function tests are improving.

**4. Chronic** acetaminophen ingestions: patients may give a history of several doses taken over 24 hours or more, in which case the nomogram cannot accurately estimate the risk of hepatotoxicity. In such cases, advise NAC treatment if the amount ingested was more than 150–200 mg/kg or 6–7 g within a 24-hour period, or if liver enzymes are elevated, or if the patient falls within a high-risk group (see above). Treatment may be stopped 36 hours after the last dose of acetaminophen if the liver enzymes are normal.

**C. Decontamination**

Administer activated charcoal. Although activated charcoal adsorbs some of the orally administered antidote *N*-acetylcysteine, this effect is not considered clinically important. Gastric emptying is not necessary if charcoal can be given promptly. Do not administer charcoal if more than 3–4 hours have passed.

**D. Enhanced elimination.** Hemoperfusion effectively removes acetaminophen from the blood but is not generally indicated because antidotal therapy is so effective.

**BENZODIAZEPINES (DIAZEPAM, LORAZEPAM, AND MIDAZOLAM etc)**[40]

(p 106-107)

All benzodiazepines in clinical use have the capacity to promote the binding of the major inhibitory neurotransmitter gabba aminobutyric acid (GABA) to the $GABA_A$ subtype of GABA receptors, which exist as multi subunit, ligand-gated chloride channels, thereby enhancing the GABA-induced ionic currents through these channels. Virtually all effects of the benzodiazepines result from their actions on the CNS. The most prominent of these effects are sedation, hypnosis, decreased anxiety,

muscle relaxation, anterograde amnesia, and anticonvulsant activity. Only two effects of these drugs result from peripheral actions: coronary vasodilation, seen after intravenous administration of therapeutic doses of certain benzodiazepines, and neuromuscular blockade, seen only with very high doses.

**1. Diazepam.** Onset of action is fast after intravenous (IV) injection, but slow to intermediate after oral or rectal administration. The half-life is greater than 24 hours, although anticonvulsant effects and sedation are often shorter as a result of redistribution from the central nervous system.

**2. Lorazepam.** Onset is intermediate after intramuscular (IM) dosing. The elimination half-life is 10–20 hours, and anticonvulsant effects are generally longer than for diazepam.

**3. Midazolam.** Onset is rapid after intramuscular or intravenous injection and intermediate after nasal application or ingestion. The half-life is 1.5–3 hours and the duration of effects is very short due to rapid redistribution from the brain. However, sedation may persist for 10 hours or longer after prolonged infusions due to saturation of peripheral sites and slowed redistribution.

**Mechanism of toxicity.** Benzodiazepines enhance the action of the inhibitory neurotransmitter gabba aminobutyric acid (GABA). They also inhibit other neuronal systems by poorly defined mechanisms. The result is generalized depression of spinal reflexes and the reticular activating system. This may cause coma and respiratory arrest.

**A.** Respiratory arrest is more likely with newer short-acting triazolo benzodiazepines such as triazolam ,alprazolam , and midazolam .

**B.** Cardiopulmonary arrest has occurred after rapid injection of diazepam, possibly because of CNS-depressant effects or because of the toxic effects of the diluent propylene glycol.

**C. Pharmacokinetics.** Most agents are highly protein bound (90–100%).

**II. Toxic dose.** In general, the toxic: therapeutic ratio for benzodiazepines is very high. For example, oral overdoses of diazepam have been reported in excess of 15–20 times the therapeutic dose without serious depression of consciousness. On the other hand, respiratory arrest has been reported after ingestion of 5 mg of triazolam and after rapid intravenous injection of diazepam, midazolam, and many other benzodiazepines. Also, ingestion of another drug with CNS-depressant properties (eg, ethanol, barbiturates, opioids, etc) will likely produce additive effects.

**Clinical presentation.** Onset of CNS depression may be observed within 30–120 minutes of ingestion, depending on the compound. Lethargy, slurred speech, ataxia, coma, and respiratory arrest may occur. Generally, patients with benzodiazepine-induced coma have hyporeflexia and mid-position or small pupils. Hypothermia may occur. Serious complications are more likely when newer short-acting agents are involved or when other depressant drugs have been ingested.

**IV. Diagnosis** is usually based on the history of ingestion or recent injection Coma and small pupils do not respond to naloxone but will reverse with administration of flumazenil.

**A. Specific levels.** Urine and blood qualitative screening may provide rapid confirmation of exposure. Certain immunoassays may not detect newer benzodiazepines or those in low concentrations. Triazolam and prazepam are rarely detectable.

**B. Other useful laboratory studies** include glucose, arterial blood gases, or pulse oximetry.

**V. Treatment.**

**A. Emergency and supportive measures**

**1.** Protect the airway and assist ventilation if necessary.

**2.** Treat coma,hypotension , and hypothermia if they occur. Hypotension usually responds promptly to supine position and intravenous fluids.

**B. Specific drugs and antidotes. Flumazenil** is a specific benzodiazepine receptor antagonist that can rapidly reverse coma. However, because benzodiazepine overdose by itself is rarely fatal, the role of flumazenil in routine management has yet to be established. It is administered intravenously with a starting dose of 0.1–0.2 mg, repeated as needed up to a total of no more than 3 mg. It has some important potential drawbacks:

1. It may induce seizures in patients with tricyclic antidepressant overdose.
2. It may induce acute withdrawal, including seizures and autonomic instability, in patients who are addicted to benzodiazepines.
3. Re sedation is common when the drug wears off after 1–2 hours, and repeated dosing is usually required.

**C. Decontamination**

Administer activated charcoal.

**BARBITURATES**[40 (p 101-103)]

Barbiturates are used as hypnotic and sedative agents, for the induction of anesthesia, and for the treatment of epilepsy and status epilepticus. They are often divided into four major groups by their pharmacologic activity and clinical use: **ultrashortacting (thiopental), short-acting (pentobarbital), intermediate-acting (butabarbitol),** and **long-acting (Phenobarbital).**

**Mechanism of toxicity.**

**A.** All barbiturates cause generalized **depression of neuronal activity** in the brain. Interaction with a barbiturate receptor leads to enhanced gabba aminobutyric acid (GABA)-mediated chloride currents and results in synaptic inhibition. Hypotension that occurs with large doses is caused by depression of central sympathetic tone as well as by direct depression of cardiac contractility.

**II. Toxic dose.** The toxic dose of barbiturates varies widely and depends on the drug, the route and rate of administration, and individual patient tolerance. In general, toxicity is likely when the dose exceeds 5–10 times the hypnotic dose. Chronic users or abusers may have striking tolerance to depressant effects

The potentially fatal **oral dose** of the shorter-acting agents is 2–3 g, compared with 6–10 g for phenobarbital.

**III. Clinical presentation.** The onset of symptoms depends on the drug and the route of administration.

**A.** Lethargy, slurred speech, nystagmus, and ataxia are common with mild to moderate intoxication. With higher doses, hypotension, coma, and respiratory arrest commonly occur. With deep coma, the pupils are usually small or mid position; the patient may lose all reflex activity and appear to be dead.

**B. Hypothermia** is common in patients with deep coma, especially if the victim has suffered exposure to a cool environment. Hypotension and bradycardia commonly accompany hypothermia.

**IV. Diagnosis** is usually based on a history of ingestion and should be suspected in any epileptic patient with stupor or coma. Although skin bullae are sometimes seen with barbiturate overdose, these are not specific for barbiturates. Other causes of coma should also be considered.

**A. Specific levels** concentrations greater than 60–80 mg/L are usually associated with coma and those greater than 150–200 mg/L with severe hypotension. For short- and intermediate-acting barbiturates, coma is likely when the serum concentration exceeds 20–30 mg/L. Barbiturates are easily detected in routine urine toxicologic screening.

**B. Other useful laboratory studies** include electrolytes, glucose, BUN, creatinine, arterial blood gases or pulse oximetry, and chest x-ray.

**Treatment.**

**A. Emergency and supportive measures**

**1.** Protect the airway and assist ventilation if necessary.

**2.** Treat coma, hypothermia , and hypotension if they occur.

**B. Specific drugs and antidotes.** There is no specific antidote.

**C. Decontamination**

**1.** Administer activated charcoal. Consider gastric lavage for massive ingestion.

**D. Enhanced elimination**

**1. Alkalinization** of the urine increases the urinary elimination of phenobarbital. Its value in acute overdose is unproved, and it may potentially contribute to fluid overload and pulmonary edema.

**2. Hemoperfusion** or hemodialysis may be necessary for severely intoxicated patients not responding to supportive care (ie, with intractable hypotension).

**PHENYTOIN**[40] **(P 261-262)**

Phenytoin is used orally for the prevention of generalized (grand mal) and psychomotor seizures. Intravenous phenytoin is used to treat status epilepticus and occasionally as an antiarrhythmic agent. Oral formulations include suspensions, capsules and tablet preparations.

**I. Mechanism of toxicity.**

**Phenytoin** alters neuronal ion fluxes, increasing refractory periods and decreasing repetitive neuronal firing. It is also known to increase brain concentrations of □-aminobutyric acid (GABA). Toxic levels usually cause central nervous system depression.

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**II. Toxic dose.** The minimum acute toxic oral overdose is approximately 20 mg/kg. Because phenytoin exhibits dose-dependent elimination kinetics, accidental intoxication can easily occur in patients on chronic therapy owing to drug interactions or slight dosage adjustments.

**III. Clinical presentation.**

**A. Mild to moderate intoxication** commonly causes nystagmus, ataxia, and dysarthria. Nausea, vomiting, diplopia, hyperglycemia, agitation, and irritability have also been reported.

**B. Severe intoxication** can cause stupor, coma, and respiratory arrest.

**C. Rapid intravenous injection**, usually at rates exceeding 50 mg/min, can cause profound hypotension, bradycardia, or cardiac arrest. Cardiac toxicity does not occur with oral overdose.

**IV. Diagnosis** is based on a history of ingestion or is suspected in any epileptic patient with altered mental status or ataxia.

**A. Specific levels.** Serum phenytoin concentrations are generally available in all hospital clinical laboratories. Obtain repeated blood samples because slow absorption may result in delayed peak levels. The therapeutic concentration range is 10–20 mg/L.

**1.** Above 20 mg/L, nystagmus is common. Above 30 mg/L, ataxia, slurred speech, and tremor are common. With levels higher than 40 mg/L, lethargy, confusion, and stupor ensue.

**2.** Because phenytoin is protein bound, patients with renal failure or hypoalbuminemia may experience toxicity at lower serum levels. Free (unbound) serum phenytoin levels are not routinely available.

**B. Other useful laboratory studies** include electrolytes, glucose, BUN, creatinine, serum albumin, and ECG monitoring (during intravenous infusion).

**V. Treatment.**

**A. Emergency and supportive measures**

1. Maintain an open airway and assist ventilation if necessary. Administer supplemental oxygen.
2. Treat stupor and coma if they occur. Protect the patient from self injury caused by ataxia.
3. If hypotension occurs with intravenous phenytoin administration, immediately stop the infusion and administer intravenous fluids and pressors if necessary.

**B. Specific drugs and antidotes.** There is no specific antidote.

**C. Decontamination**

Administer activated charcoal. Gastric emptying is not necessary if activated charcoal can be given promptly.

## .ANTIDIABETIC AGENTS[40 (p 81-84)]

Agents used to lower blood glucose are divided into two main groups: oral drugs and insulin products. The oral agents include sulfonylureas, biguanides, acarbose, and troglitazone, All **insulin** products are given by the parenteral route, and all produce effects similar to those of endogenous insulin; they differ by antigenicity and by onset and duration of effect.

**Mechanism of toxicity.**

**A. Oral agents**

**1. Sulfonylureas** lower blood glucose primarily by stimulating endogenous pancreatic insulin secretion and secondarily by enhancing peripheral insulin receptor sensitivity and reducing glycogenolysis.

**2. Biguanides.** Metformin decreases hepatic glucose production and intestinal absorption of glucose, while increasing peripheral glucose uptake and utilization. It does not stimulate insulin release, and is not likely to produce acute hypoglycemia. Severe **lactic acidosis** is a rare but potentially fatal side effect of metformin. It occurs mainly in patients with renal insufficiency, alcoholism, and advanced age, and has occurred after injection of iodinated contrast agents resulted in acute renal failure.

**3. Acarbose** is an alpha-glucosidase inhibitor that delays the digestion of ingested carbohydrates, reducing postprandial blood glucose concentrations.

**4. Troglitazone** decreases hepatic glucose output and improves target cell response to insulin.

**Insulin.** Blood glucose is lowered directly by the stimulation of cellular uptake and metabolism of glucose. Cellular glucose uptake is accompanied by an intracellular shift of potassium and magnesium. Insulin also promotes glycogen formation and lipogenesis

**Toxic dose.**

**A. Sulfonylureas.** Toxicity depends on the agent and the total amount ingested.

Toxicity may also occur owing to drug interactions, resulting in impaired elimination of the oral agent.

1. **Acetohexamide.** Two 500-mg tablets have caused hypoglycemic coma in an adult.
2. **Chlorpropamide**, 500–750 mg/d for 2 weeks has caused hypoglycemia in adults.
3. **Glyburide.** Acute overdose (10–15 mg) in a child produced profound hypoglycemic coma.
4. **Interactions** with the following drugs may increase the risk of hypoglycemia: other hypoglycemics, sulfonamides, propranolol, salicylates, clofibrate, probenecid, pentamidine, valproic acid, dicumarol, cimetidine, MAO inhibitors, and alcohol. In addition, co-ingestion of alcohol may occasionally produce a disulfiram like interaction.
5. **Hepatic** or **renal insufficiency** may impair drug elimination and result in hypoglycemia.

**C. Insulin.** Severe hypoglycemic coma and permanent neurologic sequelae have occurred after injections of 800–3200 units of insulin. Orally administered insulin is not absorbed and is not toxic.

**III. Clinical presentation.**

**A. Hypoglycemia** may be delayed in onset depending on the agent used and the route of administration (i.e., subcutaneous versus intravenous). Manifestations of hypoglycemia include agitation, confusion, coma, seizures, tachycardia, and diaphoresis.

**B. Lactic acidosis** from metformin or phenformin may begin with nonspecific symptoms such as malaise, vomiting, myalgia's, and respiratory distress. The mortality rate for severe lactic acidosis is reportedly as high as 50%.

**IV. Diagnosis.** Overdose involving a sulfonylurea or insulin should be suspected in any patient with hypoglycemia. Other causes of hypoglycemia that should be considered include alcohol ingestion (especially in children) and fulminant hepatic failure.

**A. Specific levels**

**1.** Serum concentrations of many agents can be determined in regional commercial toxicology laboratories, but have little utility in acute clinical management.

**2.** Exogenously administered animal insulin can be distinguished from endogenous insulin (ie, in a patient with hypoglycemia caused by insulinoma) by determination of C peptide (present with endogenous insulin secretion).

**B. Other useful laboratory studies** include glucose, electrolytes, magnesium, and ethanol, blood lactate levels.

**V. Treatment.**

**A. Specific drugs and antidotes**

**1.** In adults, give 50% dextrose (D50W), 1–2 mL/kg; in children, use 25% dextrose (D25W), 2–4 mL/kg.

**2.** Follow serum glucose levels closely for several hours after the last dose of dextrose. Give repeated glucose boluses and administer 5–10% dextrose (D5–D10) as needed to maintain the serum glucose level at or above 100mg/dL.

**3.** For patients with a **sulfonylurea overdose**, consider intravenous **octreotide** or **diazoxide** if 5% dextrose infusions do not maintain satisfactory glucose concentrations.

**4.** Lactic acidosis may be treated with judicious doses of sodium bicarbonate. Excessive bicarbonate administration may worsen intracellular acidosis.

**C. Decontamination**

Administer activated charcoal. Gastric emptying is not necessary if activated charcoal can be given promptly. Because there is limited experience with the effects of acute overdoses of these agents, ingestion of a large or unknown amount should be treated with oral activated charcoal.

**Insulin**

- Orally ingested insulin is not absorbed and produces no toxicity, so gut decontamination is not necessary.
- Local excision of tissue at the site of massive intradermal injection has been performed, but the general utility of this procedure has not been established.

**D. Enhanced elimination**

**1. Sulfonylureas.** Alkalization of the urine (pH 8 or greater) increases the renal elimination of chlorpropamide. Forced diuresis and dialysis procedures are of no known value for other hypoglycemic agents. The high degree of protein binding of the sulfonylureas suggests that dialysis procedures would not generally be effective. However, charcoal hemoperfusion reduced the serum half-life of chlorpropamide in a patient with renal failure.

**2. Metformin** is effectively removed by hemodialysis, which can also help correct severe lactic acidosis.

## BETA-ADRENERGIC BLOCKERS[40 (p 107-109)]

Beta-adrenergic blocking agents are widely used for the treatment of hypertension, arrhythmias, angina pectoris, migraine headaches, and glaucoma. Many patients with beta-blocker overdose will have underlying cardiovascular diseases or will be taking other cardioactive medications, both of which may aggravate beta-blocker overdose. A variety of beta blockers are available, with various pharmacologic effects and clinical uses.

**I. Mechanism of toxicity.** Excessive beta-adrenergic blockade is common to overdose with all drugs in this category. Although beta-receptor specificity is seen at low doses, it is lost in overdose.

**A. Propranolol** and other agents with membrane-depressant (quinidine like) effects further depress myocardial contractility and conduction. Propranolol is also lipid soluble, which enhances brain penetration and can cause seizures and coma.

**B. Pindolol** and other agents with partial beta-agonist activity may cause hypertension.

**C. Sotalol**, which also has type III antiarrhythmic activity, prolongs the QT interval in a dose-dependent manner and may cause torsades de pointes and ventricular fibrillation.

**II. Toxic dose.** The response to beta-blocker overdose is highly variable depending on underlying medical disease or other medications. Susceptible patients may have severe or even fatal reactions to therapeutic doses. There are no clear guidelines, but ingestion of only 2–3 times the therapeutic dose should be considered potentially life-threatening in all patients.

**III. Clinical presentation.** The pharmacokinetics of beta blockers vary considerably, and duration of poisoning may range from minutes to days.

**A. Cardiac disturbances**, including hypotension and bradycardia, are the most common manifestations of poisoning. Atrioventricular block, intraventricular conduction disturbances, cardiogenic shock, and asystole may occur with severe overdose, especially with membrane-depressant drugs such as propranolol.

**B. CNS toxicity**, including convulsions, coma, and respiratory arrest, is commonly seen with propranolol and other membrane-depressant and lipid soluble drugs.

**C. Bronchospasm** is most common in patients with preexisting asthma or chronic bronchospastic disease.

**D. Hypoglycemia** and **hyperkalemia** may occur.

**IV. Diagnosis** is based on the history of ingestion, accompanied by bradycardia and hypotension. Other drugs that may cause a similar presentation after overdose include sympatholytic and antihypertensive drugs, digitalis, and calcium channel blockers.

**A. Specific levels.** Measurement of beta-blocker serum levels may confirm the diagnosis but does not contribute to emergency management. Metoprolol and propranolol may be detected in comprehensive urine toxicology screening.

**B. Other useful laboratory studies** include electrolytes, glucose, BUN, creatinine, arterial blood gases, and 12-lead ECG and ECG monitoring.

**V. Treatment.**

**A. Emergency and supportive measures**

**1.** Maintain an open airway and assist ventilation if necessary.

**2.** Treat coma, seizures, hypotension, hyperkalemia ,and hypoglycemia if they occur.

**3.** Treat bradycardia with atropine, 0.01–0.03 mg/kg IV or cardiac pacing.

**4.** Treat bronchospasm with nebulized bronchodilators.

**5.** Continuously monitor the vital signs and ECG for at least 6 hours after ingestion.

**B. Specific drugs and antidotes**

1. Bradycardia and hypotension resistant to the above measures should be treated with **glucagon**, 5–10 mg IV bolus, repeated as needed and followed by an infusion of 1–5 mg/h .

   **Epinephrine** intravenous infusion may also be useful.

2. Wide complex conduction defects caused by membrane-depressant poisoning may respond to **sodium bicarbonate**, 1–2 meq/kg, as given for tricyclic antidepressant overdose.

3. Torsades de pointes polymorphous ventricular tachycardia can be treated with **isoproterenol** infusion, **magnesium**, or **overdrive pacing**. Correction of hypokalemia may also be useful.

**C. Decontamination**

Administer activated charcoal. Consider gastric lavage for large ingestions, especially involving propranolol.

**D. Enhanced elimination.** Most beta blockers, especially the more toxic drugs such as propranolol, are highly lipophilic and have a large volume of distribution (Vd). For those with a relatively small volume of distribution coupled with a long half-life or low intrinsic clearance (eg, acebutolol, atenolol, nadolol, or sotalol), hemoperfusion, hemodialysis, or repeat-dose charcoal may be effective.

**CALCIUM ANTAGONISTS**[40 (p 119-121)]

Calcium antagonists (also known as calcium channel blockers or calcium blockers) are widely used to treat angina pectoris, coronary spasm, hypertension, hypertrophic cardiomyopathy, supraventricular cardiac arrhythmias, and migraine headache. Toxicity from calcium antagonists may occur with therapeutic use (often owing to drug interactions) or as a result of accidental or intentional overdose. Overdoses of calcium antagonists are frequently life-threatening and represent an increasingly important source of drug-induced mortality.

**I. Mechanism of toxicity.** Calcium antagonists slow the influx of calcium through cellular calcium channels. Currently marketed agents act primarily on vascular

smooth muscle and the heart. They result in coronary and peripheral vasodilation, reduced cardiac contractility, slowed (AV) nodal conduction, and depressed sinus node activity. Lowering of blood pressure through a fall in peripheral vascular resistance may be offset by reflex tachycardia, although this reflex response may be blunted by depressant effects on contractility and sinus node activity.

**A.** In usual therapeutic doses, amlodipine, felodipine, isradipine, nicardipine, nifedipine, and nitrendipine act primarily on blood vessels, whereas verapamil, diltiazem, and mibefradil act on both the heart and blood vessels. In overdose, however, this selectivity may be lost.

**B. Nimodipine** has a greater action on cerebral arteries and is used to reduce vasospasm after recent subarachnoid hemorrhage.

**III. Clinical presentation.**

**A.** The primary features of calcium antagonist intoxication are **hypotension** and **bradycardia**.

**1.** Hypotension may be caused by peripheral vasodilation, reduced cardiac contractility, slowed heart rate, or a combination of all of these.

**2.** Bradycardia may result from sinus bradycardia, second- or third-degree AV block, or sinus arrest with junctional rhythm.

**3.** Most calcium antagonists do not affect intraventricular conduction, so the QRS duration is usually unaffected. The PR interval is prolonged even with therapeutic doses of verapamil. Bepridil prolongs the QT interval and may cause ventricular arrhythmias, including torsades de pointes. Mibefradil also causes QT prolongation, but has not been associated with arrhythmias.

**B. Non cardiac manifestations** of intoxication include nausea and vomiting, abnormal mental status (stupor and confusion), metabolic acidosis (probably resulting from hypotension), and hyperglycemia (owing to blockade of insulin release).

**IV. Diagnosis.** The findings of hypotension and bradycardia, particularly with sinus arrest or AV block, in the absence of QRS interval prolongation should suggest calcium antagonist intoxication. The differential diagnosis should include beta blockers and other sympatholytic drugs.

**A. Specific levels.** Serum or blood drug levels are not widely available. Diltiazem and verapamil may be detectable in comprehensive urine toxicology screening.

**B. Other useful laboratory studies** include electrolytes, glucose, BUN, creatinine, arterial blood gases or oximetry, and ECG and ECG monitoring.

**V. Treatment.**

**A. Emergency and supportive measures**

**1.** Maintain an open airway and assist ventilation if necessary.

**2.** Treat coma, hypotension, and Bradyarrhythmias if they occur. The use of cardiopulmonary bypass to allow time for liver metabolism has been reported in a patient with massive verapamil poisoning. Cardiac pacing should be considered for Bradyarrhythmias that are contributing to hypotension.

**3.** Monitor the vital signs and ECG for at least 6 hours after alleged ingestion. Admit symptomatic patients for at least 24 hours.

**B. Specific drugs and antidotes**

**1. Calcium** usually promptly reverses the depression of cardiac contractility, but it does not affect sinus node depression or peripheral vasodilation and has variable effects on AV nodal conduction. Administer **calcium chloride** 10%, 10 mL (0.1–0.2 mL/kg) IV, or **calcium gluconate**10%, 20 mL (0.3–0.4 mL/kg) IV. Repeat every 5–10 minutes as needed.

**2. Glucagon**, **epinephrine,** and have been reported to increase blood pressure in patients with refractory hypotension. Glucagon and epinephrine can also increase the heart rate.

**3.** Outside the United States, **4-aminopyridine** may be available as an antidote for calcium antagonist intoxication.

**C. Decontamination**

Administer activated charcoal. Consider gastric lavage for all but the most trivial ingestions. For large ingestions of a sustained-release preparation, consider whole bowel irrigation in addition to repeated doses of charcoal. Continue charcoal administration for up to 48–72 hours.

## III. ACIDS AND ALKALIS [40 (p 129-131)]


A wide variety of chemical and physical agents may cause corrosive injury. These include mineral and organic acids, alkalies, oxidizing agents.

**I. Mechanism of toxicity.**

**A. Acids** cause an immediate coagulation-type necrosis that creates an eschar, which tends to self-limit further damage.

**B.** In contrast, **alkalies** (eg, Drano) cause a liquefactive necrosis with saponification and continued penetration into deeper tissues, resulting in extensive damage.

**II. Toxic dose.** There is no specific toxic dose or level, because the concentration of corrosive solutions and the potency of caustic effects vary widely. The concentration or the pH of the solution may indicate the potential for serious injury. For alkalies, the titratable alkalinity (concentration of the base) is a better predictor of corrosive effect than the pH.

**III. Clinical presentation.**

**Eye or skin** exposure to corrosive agents usually results in immediate pain and redness, followed by blistering. Conjunctivitis and lacrimation are common. Serious full-thickness burns and blindness can occur.

**Ingestion** of corrosives can cause oral pain, dysphagia, drooling, and pain in the throat, chest, or abdomen. Esophageal or gastric perforation may occur, manifested by severe chest or abdominal pain, signs of peritoneal irritation, or pancreatitis. Free air may be visible in the mediastinum or abdomen on x-ray. Hematemesis and shock may occur. Systemic acidosis has been reported after acid ingestion and may be partly caused by absorption of hydrogen ions. Scarring of the esophagus or stomach may result in permanent stricture formation and chronic dysphagia.

Presence of shock, large volume haematemesis, peritonitis, Hamman's sign, and subcutaneous emphysema suggest severe injuries or perforation, and require urgent surgical intervention[44].

**Systemic toxicity** can occur after inhalation, skin exposure, or ingestion of a variety of agents

**Table 1: Common home use corrosives.**[45]

| **Acids** | **Use** |
|---|---|
| Sulphuric acid | Car batteries |
| Sodium hypochlorite | Disinfectant (bleach) |
| Nitric acid | Metal cleaners |
| Sodium hydroxide | Laundry detergent |
| Hydrochloric acid De scalers Sodium carbonate | Dish washing agent |
| Hydrofluoric acid | Rust remover |
| Oxalic acid | Rust remover |
| Phenol | Disinfectant |

**Diagnosis** is based on a history of exposure to a corrosive agent and characteristic findings of skin, eye, or mucosal irritation or redness and the presence of injury to the gastrointestinal tract. Victims with oral or esophageal injury nearly always have drooling or pain on swallowing.

**A. Endoscopy.** Esophageal or gastric injury is unlikely after ingestion if the patient is completely asymptomatic, but studies have repeatedly shown that a small number of patients will have injury in the absence of oral burns or obvious dysphagia. For this reason, many authorities recommend endoscopy for all patients regardless of symptoms.

**B. X-rays**

X-rays may air in the mediastinum from esophageal perforation or free abdominal air from gastric perforation.

**C. Specific levels.** See specific chemical. Urine mercury levels have been reported to be elevated after button battery ingestion.

**D. Other useful laboratory studies** include CBC, electrolytes, glucose, arterial blood gases, chest x-ray, and upright abdominal x-ray.

**V. Treatment.**

**Emergency and supportive measures**

**Ingestion**

- Immediately give water or milk to drink.
- If esophageal or gastric perforation is suspected, obtain immediate surgical or endoscopic consultation.

**1. Inhalation.** Remove from exposure; give supplemental oxygen if available.

**2. Skin and eyes.** Remove all clothing; wash skin and irrigate eyes with copious water or saline.

**3. Ingestion**

**Gastric lavage** to remove the corrosive material is controversial but is probably beneficial in acute liquid corrosive ingestion, and it will be required before endoscopy anyway. Use a soft flexible tube and lavage with repeated aliquots of water or saline, frequently checking the pH of the washings.

**PHENOL**[40 (P 255-257)]

**Phenol** (carbolic acid) was introduced into household use as a potent germicidal agent but has limited use today because less toxic compounds have replaced it.

**Mechanism of toxicity.** Phenol denatures protein, disrupts the cell wall, and produces coagulative necrosis. It may cause corrosive injury to the eyes, skin, and respiratory tract. Some phenolic compounds (eg, dinitrophenoln and hydroquinone) may induce **methemoglobinemia**

**Clinical presentation**. Toxicity may result from inhalation, skin or eye exposure, or ingestion.

A. **Inhalation**. Vapors of phenol may cause respiratory tract irritation and chemical pneumonia. Smoking of clove cigarettes (clove oil contains the phenol derivative eugenol) may cause severe tracheobronchitis.

B. **Skin and eyes**. Topical exposure to the skin may produce a deep white patch that turns red, then stains the skin brown. This lesion is often relatively painless. Eye

irritation and severe corneal damage may occur if concentrated liquids are spilled into the eye.

C. **Ingestion** usually causes vomiting and diarrhea, and diffuse corrosive gastrointestinal tract injury may occur. Systemic absorption may cause agitation confusion, seizures, coma, hypotension, arrhythmias, and respiratory arrest.

**Diagnosis** based on a history of exposure, the presence of a characteristic odor, and painless white skin burns.

**A. Specific levels.** Normal urine phenol levels are less than 20 mg/L.

**B. Other useful laboratory studies** include CBC, electrolytes, glucose, ABG, BUN, creatinine, and ECG. After hydroquinone exposure, obtain a methemoglobin level

**Treatment:**

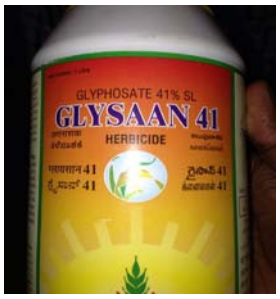
**Decontamination**

**1. Inhalation.** Remove victims from exposure and administer supplemental oxygen if available.

**2. Skin and eyes.** Remove contaminated clothing and wash exposed skin with soapy water or, if available, mineral oil, olive oil, or petroleum jelly. Immediately flush exposed eyes with copious tepid water or saline.

4. **Ingestion:** Administer activated charcoal, orally or by gastric tube. Consider gastric lavage for large recent ingestions.

## IV. PLANT AND HERBAL POISON[40] (p 265-274)





Plant ingestions serious poisoning or death is extremely rare because the quantity of toxin ingested is small. Unfortunately, there is little consumer awareness of the potential harm from some herbal preparations. Besides toxicity from natural products contained in some medicinal plants, "herbal" or "traditional" preparations may sometimes actually contain allopathic drugs such as phenylbutazone, corticosteroids, salicylates, ephedrine, or toxic metal salts such as mercury or lead.

**Group 1** plants contain systemically active poisons that may cause serious intoxication. eg: Nightshade deadly- *Atropa belladonna* , Jimsonweed; thornapple - *Datura stramonium* ( Anticholinergic alkaloids), Oleander

**B. Group 2a** plants contain insoluble calcium oxalate crystals that cause burning pain and swelling of mucous membranes. Eg: Pothos; yellow pothos, Swiss cheese plant, Skunk cabbage

**C. Group 2b** plants contain soluble oxalate salts (sodium or potassium) that can produce acute hypocalcemia, renal injury, and other organ damage secondary to precipitation of calcium oxalate crystals in various organs. Mucous membrane irritation and gastroenteritis may also occur. Eg: American ivy

**D. Group 3** plants contain various chemical agents that generally produce only mild to moderate gastrointestinal irritation after ingestion or dermatitis after skin contact. Eg: Eucalyptus, Ficus (sap)

**Toxic dose.**

The amount of toxin ingested is usually unknown. Concentrations of the toxic agent may vary depending on the plant part, the season, and soil conditions. In general, childhood ingestion of a single leaf or a few petals from even Group 1 plants results in little or no toxicity because of the small amount of toxin absorbed.

**Clinical presentation.**

**A. Group 1.** The presentation depends upon the active toxic agent. In most cases, vomiting, abdominal pain, and diarrhea occur within 60–90 minutes of a significant ingestion. With some toxins (eg, ricin), severe gastroenteritis may result in massive fluid and electrolyte loss.

**B. Group 2a.** Insoluble calcium oxalate crystals cause immediate burning, prickly pain upon contact with mucous membranes. Swelling of the lips, tongue, and pharynx may occur, and in rare cases glottic edema may result in airway obstruction. Symptoms usually resolve within a few hours.

**C. Group 2b.** Soluble oxalates may be absorbed into the circulation, where they precipitate with calcium, resulting in acute hypocalcemia and multiple-organ injury, including renal tubular necrosis.

**D. Group 3.** Skin or mucous membrane irritation may occur, although it is less severe than with Group 2 plants. Vomiting and diarrhea are common but are usually mild and self-limited. Fluid and electrolyte imbalances caused by severe gastroenteritis are rare.

**IV. Diagnosis** is usually based on a history of exposure and is suspected when plant material is seen in vomitus. Identification of the plant is essential to proper treatment. Because common names sometimes refer to more than one plant, it is preferable to confirm the botanical name. If in doubt about the plant identification, take the specimen to a local nursery, florist, or college botany department.

**A. Specific levels.** Serum toxin levels are not available for most plant toxins. In selected cases, laboratory analyses for therapeutic drugs may be used (eg,digoxin assay for oleander glycosides).

**B. Other useful laboratory studies** include, for patients with gastroenteritis, CBC, electrolytes, glucose, BUN, creatinine, and urinalysis. If hepatotoxicity is suspected, also obtain liver transaminases and prothrombin time (PT).

**V. Treatment.** Most ingestions cause no symptoms or only mild gastroenteritis, and patients recover quickly with supportive care.

**A. Emergency and supportive measures**

**1.** Maintain an open airway and assist ventilation if necessary .Administer supplemental oxygen.

**2.** Treat coma, seizures, arrhythmias, and hypotension if they occur.

**3.** Replace fluid losses caused by gastroenteritis with intravenous crystalloid solutions.

**B. Decontamination** Administer activated charcoal. Gastric emptying is not necessary if activated charcoal can be given promptly.

**V. MISCELLANEOUS**



**Paraquat**

Paraquat is a commonly used herbicide which, on contact with green foliage and in the presence of sunlight, kills plant tissue. It is rendered harmless on contact with soil.

**Clinical features**. These include, nausea, vomiting, abdominal pain and diarrhoea, and, if concentrated formulations are swallowed, oral and throat ulcerations can occur. Signs of renal and hepatic dysfunction develop within 1 - 3 days, and are usually reversible when treated by conventional means. Pulmonary fibrosis and respiratory failure, on the other hand, is nonreversible and is the common cause of death. The

respiratory complications usually appear with pulmonary oedema within 24 hr of ingestion, followed after 1 - 2 weeks by a progressive pulmonary fibrosis. The pulmonary tissue is thought to be particularly susceptible because both type I and II alveolar cells actively accumulate paraquat,[46] even against a concentration gradient.[47]

**Mechanism of toxicity** is due to an inhibition of superoxide dismutase, generation of free radicals, and NADH depletion.[48] Plasma paraquat levels peak at 0.5 - 2 hours after ingestion, with the probability of survival predicted for the 4 - 12 hr plasma level predicted using a graph[49] or at any specified time using the equation: exp (logit)/[1 + exp(logit)], where logit = 0.58 - 2.33 x log (plasma paraquat) - 1.15 x log (hr since ingestion).[50] However, when plasma levels cannot be performed a urinary sodium dithionate test with a 'navy blue' or 'dark blue' reaction generally indicates significant paraquat poisoning with a subsequent poor prognosis.[36,]

**Treatment** Without appropriate treatment, the mortality after ingestion of paraquat varies from 87 - 100%.[51] The estimated fatal dose in humans may be as low as 4 mg/kg, although the $LD_{50}$ in an adult human is normally about 3 - 5 g (i.e. 10 - 15 mL of the 20% concentrate).[46] Apart from gastric lavage and supportive management, the specific binder 'Fuller's earth' (calcium montmorillonite) should be administered. As only 5 - 10% of paraquat is absorbed in 24 h, Fuller's earth is given orally as soon as possible (e.g. 1 litre of a 30% solution - 300 g suspended in 1 litre of water - followed by 200 mL of 20% mannitol). This is followed 2-hourly by a 15% solution (1000 mL of water with 150 g of Fuller's earth), followed by 200 mg of 20% mannitol, every 4 hr to induce a catharsis, and it is repeated until the stools are seen to contain Fuller's

earth. If Fuller's earth is unavailable, experimentally activated charcoal appears to be just as effective.[52] If purgation is not achieved within 4-6 hours then gastrointestinal decontamination should be discontinued.

During purgation the patients fluid and electrolyte status needs to be carefully monitored (Fuller's earth may also cause hypercalcaemia and faecoliths with bowel obstruction or perforation).Haemodialysis, peritoneal dialysis and haemoperfusion are all ineffective for paraquat removal,[53,54] although haemodialysis may be required to manage renal failure. Also large doses of vitamin C and vitamin E as antioxidants have not yet been confirmed to be helpful[48] and other antidotes including superoxide dismutase, selenium, niacin, N-acetylcysteine, corticosteroids, immunosuppressive agents and radiotherapy, have not yet been shown to be effective in limiting the lung injury.[46] Lung transplantation has been used successfully to manage a patient with progressive respiratory failure 6 weeks after paraquat poisoning.[55]

**METHEMOGLOBINEMIA [40 (p 220-222)]**

Methemoglobin is an oxidized form of hemoglobin. Many oxidant chemicals and drugs are capable of inducing methemoglobinemia: nitrites and nitrates, bromatesand chlorates, aniline derivatives, antimalarial agents, dapsone, sulfonamides.

**I**. **Mechanism of toxicity**.

Methemoglobin inducers act by oxidizing ferrous (Fe2+) to ferric (Fe3+) hemoglobin. This abnormal hemoglobin is incapable of carrying oxygen, inducing a functional anemia. In addition, the shape of the oxygen-hemoglobin dissociation curve is altered, aggravating cellular hypoxia.

**II**. **Toxic dose**. The ingested dose or inhaled air level of toxin required to induce methemoglobinemia is highly variable Concomitant hemolysis suggests either heavy oxidant exposure or increased cell vulnerability.

**III**. **Clinical presentation**. The severity of symptoms usually correlates with measured methemoglobin levels.

A. Symptoms and signs are caused by decreased blood oxygen content and cellular hypoxia and include headache, dizziness, and nausea, progressing to dyspnea, confusion, seizures, and coma. Even at low levels, skin discoloration ("chocolate cyanosis"), especially of the nails, lips, and ears, is striking.

**IV**. **Diagnosis**. The patient with mild to moderate methemoglobinemia appears markedly cyanotic, yet may be relatively asymptomatic. The arterial oxygen partial pressure (pO2) is normal. The diagnosis is suggested by the finding of "chocolate brown" blood (dry a drop of blood on filter paper and compare with normal blood), which is usually apparent when the methemoglobin level exceeds 15%. Differential diagnosis includes other causes of cellular hypoxia (eg, carbonmonoxide, cyanide, and hydrogen sulfide) and sulf hemoglobinemia.

A**. Specific levels**. The co-oximeter type of arterial blood gas analyzer will directly measure oxygen saturation and methemoglobin percentages (measure as soon as possible, because levels fall rapidly in vitro).

1. Sulfhemoglobin and the antidote methylene blue both produce erroneously high levels on the co-oximeter: a dose of 2 mL/kg methylene blue gives a false-positive methemoglobin of approximately 15%.

2. The routine arterial blood gas machine measures the serum pO2 (which is normal) and calculates a falsely normal oxygen saturation.

3. Pulse oximetry is *not* reliable; it will appear deceptively near-normal in a patient with severe methemoglobinemia, and falsely desaturated in a patient who has been given methylene blue.

B. Other useful laboratory studies include electrolytes and glucose. If hemolysis is suspected, add CBC, haptoglobin, peripheral smear, and urinalysis dipstick for occult blood (free hemoglobin is positive).

**V**. **Treatment**.

A. Emergency and supportive measures

1. Maintain an open airway and assist ventilation if necessary. Administer supplemental oxygen.

2. Usually, mild methemoglobinemia (<15 – 20%) will resolve spontaneously and requires no intervention.

**B**. **Specific drugs and antidotes**

1. Methylene blue is indicated in the symptomatic patient with methemoglobin levels higher than 20% or when even minimal compromise of oxygen-carrying capacity is potentially harmful (eg, pre-existing anemia,

Methemoglobin Level*a* Typical Symptoms

< 15% usually asymptomatic

15–20% Cyanosis, mild symptoms

20–45% Marked cyanosis, moderate symptoms

45–70% severe cyanosis, severe symptoms

> 70% usually lethal

*a*-These percentages assume normal range total hemoglobin concentrations. Concomitant anemia may lead to more severe symptoms at lower proportional methemoglobinemia. congestive heart failure, pneumocystis pneumonia, angina pectoris, etc).

Give methylene blue, 1–2 mg/kg (0.1–0.2 mL/kg of 1% solution) over several minutes.

2. Ascorbic acid, which can reverse methemoglobin by an alternate metabolic pathway, is of minimal use acutely because of its slow action.

C**. Decontamination** depends on the specific agent involve

D. **Enhanced elimination**

- If methylene blue is contraindicated (e.g., G6PD deficiency) or has not been effective, exchange transfusion may rarely be necessary in patients with severe methemoglobinemia.
- Hyperbaric oxygen is theoretically capable of supplying sufficient oxygen independent of hemoglobin, and may be useful in extremely serious cases that do not respond rapidly to antidotal treatment

**Kerosene Oil**[56 (p 1986)]

Kerosene oil, a petroleum product, is a mixture of hydrocarbons contaminated with organic sulphur. Petrol, gasoline, vaselin and paraffin are other related hydrocarbons. Besides household cooking and burning, kerosene is used as paint thinner. Lethal dose of kerosene oil is 30 to 100 mL.

***Clinical features***

Kerosene is toxic both through inhalational and oral routes. Inhalation results in dizziness, nausea, vomiting, burning sensation in chest, dry cough, headache and ataxia. Severe poisoning produces pulmonary oedema, hemoptysis, mental confusion, hallucinations, stupor, cyanosis, convulsion and coma. Death is mainly due to ventricular fibrillation and respiratory failure. Ingestion of kerosene oil produces burning pain in the mouth, throat, dry irritating cough, nausea, vomiting, colicky abdominal pain and diarrhea. Large quantity of oil can produce neurological dysfunction similar to the one described for inhalational toxicity. The pupils are initially constricted and later dilated. Aspiration of as little as 0.2 mL of kerosene oil can produce chemical pneumonia and pulmonary oedema. The breath, vomitus and urine gives off the peculiar smell of kerosene. The chest radiograph may reveal perihilar densities, basal pneumonia, and atelectasis.

***Treatment***

In case of inhalational toxicity, remove the victim to open air, ensure patent airway, keep the body warm and on hospitalization administer oxygen. Following ingestion, induction of vomiting or gastric lavage is contraindicated due to increased risk of aspiration. Activated charcoal is not useful. Antibiotic and steroids are not indicated.

Victims with severe respiratory symptoms and abnormal radiography should be observed for complications and managed appropriately for at least 2 to 3 days.

**Detergents**[56(p 1987)]

These are products that contain anionic surfactants and detergent builders, such as sodium phosphates, sodium carbonate and sodium aluminosilicates, which are highly alkaline. Detergents fall into three main categories—nonionic, anionic, and cationic. Nonionic and anionic detergents are of low toxicity. Cationic detergents, such as benzalkonium chloride and cetrimide, are less frequently encountered in domestic cleaners and produce corrosive effects if a concentrated solution is consumed. The route of exposure is almost always oral ingestion but inhalational, ocular and dermal exposures and even intravenous route have been reported. Corrosive injury to the gastrointestinal, respiratory and corneal mucosa (depending on route of exposure), cardiac dysfunction, acute renal failure with rhabdomyolysis, ARDS (early and late onset), haemolysis, frequent ventricular tachycardia and coagulation abnormalities have been reported secondary to exposure to household detergents.

There is no standardized treatment protocol for detergent poisoning. Endotracheal intubation and supportive ventilation are required frequently. Syrup of ipecac is not advised due to the risk of aspiration and further caustic injury to the oesophagus.

**Bleaches**[56(p 1987)]

These household solutions contain approximately 3% to 10% sodium hypochlorite or less commonly 3% hydrogen peroxide. These are extremely

unpalatable and unlikely to cause serious damage. Symptoms are same as that of caustic ingestion but respiratory symptoms may result when bleach is mixed with ammonia (producing chloramine gas) or with toilet cleaners, (forming chlorine gas).

***Treatment***

Removing the patient from the source and minimizing the period of contact between the corrosive and the tissue will prevent tissue damage. Irrigation with copious amount of water is paramount as prompt dilution reduces the exposure. Patients with respiratory distress may have significant oral, pharyngeal, and/or laryngotracheal injury necessitating emergency airway management. Prior to intubation, extent of airway injury should be assessed by fiberoptic endoscopy

**Essential Oils**[56(p 1987)]

These are volatile mixtures of esters, alcohols and ketones. Some substances, e.g. camphor, an active ingredient of common OTC products such as Vicks Vapo Rub are very toxic. Mucosal irritation, vomiting, epigastric pain may be followed by secondary hepatic and renal failure in severe toxicity. Clinical observation and supportive treatment is needed.

**Nail Care/Nail Varnish Removers**[56(p 1988)]

These contain acetone or ethyl acetate. Irritation of mucous membranes, vomiting, CNS depression, ketosis, acidosis and hyperglycaemia are commonly observed. Presence of other solvents may lead to different complications like caustic injury (methanol/methacrylic acid), methaemoglobinaemia (nitroethane) and cyanide

poisoning (acetonitrile). Hospital admission is needed in symptomatic individuals. Nitroethane and acetonitrile ingestions require referral to the emergency.

**Naphthalene**[56(p 1988)]

It is a hydrocarbon contained in the middle oil distillation of coal tar. It is chiefly used in the manufacturing of certain azo dyes, moth repellent, wood preservative and deodorant. Ingestion of naphthalene produces nausea, vomiting, loose motions, headache, gait disturbances, drowsiness, muscle twitching, cyanosis and profuse perspiration. Severe toxicity produces jaundice, haemolytic anaemia in G6PD deficient individuals, albuminuria, haematuria and acute renal failure. CNS manifestations include encephalopathy, seizures, coma and death. There is no specific antidote; hence the treatment consists of gastric lavage, saline purgative, intravenous fluids, sodium bicarbonate, blood transfusion and diuretics as supportive therapy.

**DISINFECTANT AND ANTISEPTIC**[40 (p 93-95)]

They are widely used in the household, food industry, and hospitals. All these agents are generally used as dilute solutions and cause little or no toxicity.

**Mechanism of toxicity**

**Hydrogen peroxide** is an oxidizing agent, but it is very unstable and readily breaks down to oxygen and water. Generation of oxygen gas in closed body cavities can potentially cause mechanical distention resulting in gastric or intestinal perforation, as well as venous or arterial gas embolization.

**Potassium permanganate** is an oxidant, and the crystalline form or concentrated solutions are corrosive.

**Toxic dose.**

**A. Hydrogen peroxide** for household use is available in 3–5% solutions and it causes only mild throat and gastric irritation with ingestion of less than 1 oz. Concentrations above 10% are found in some hair-bleaching solutions and are potentially corrosive.

**B.Potassium permanganate** solutions of greater than 1:5000 strength may cause corrosive burns.

**Clinical presentation.** Most antiseptic ingestions are benign, and mild irritation is self-limited. Spontaneous vomiting and diarrhea may occur, especially after a large-volume ingestion.

**A.** Exposure to **concentrated** solutions may cause corrosive burns on the skin and mucous membranes, and oropharyngeal, esophageal, or gastric injury may occur. Glottic edema has been reported after ingestion of concentrated potassium permanganate.

**B.** Permanganate may also cause **methemoglobinemia**

**C.** Hydrogen peroxide ingestion may cause gastric distention and, rarely, perforation. Severe corrosive injury and air emboli have been reported with the concentrated forms and may be caused by the entry of gas through damaged gastric mucosa or gas production within the venous or arterial circulation.

**IV. Diagnosis** is based on a history of exposure and the presence of mild gastrointestinal upset or frank corrosive injury. Solutions of potassium permanganate are dark purple, and skin and mucous membranes are often characteristically stained.

**Specific levels.** Drug levels in body fluids are not generally useful or available.

**B. Other useful laboratory studies** include electrolytes, glucose, methemoglobin level (for potassium permanganate exposure), and upright chest x-ray (for suspected gastric perforation).

**V. Treatment.**

**A. Emergency and supportive measures**

**1.** In patients who have ingested concentrated solutions, monitor the airway for swelling and intubate if necessary.

**2.** Consult a gastroenterologist for possible endoscopy after ingestions of corrosive agents such as concentrated hydrogen peroxide or potassium permanganate. Most ingestions are benign, and mild irritation is self-limited.

**3.** Consider **hyperbaric oxygen** treatment for gas emboli associated with concentrated peroxide ingestion.

**B. Specific drugs and antidotes.** No specific antidotes are available for irritant or corrosive effects. If **methemoglobinemia** occurs, administer methylene blue

**C. Decontamination**

**1. Ingestion** of concentrated corrosive agents

**a.** Dilute immediately with water or milk.

**b.** Do ***not*** induce vomiting because of the risk of corrosive injury. Perform gastric lavage cautiously.

**c.** Activated charcoal and cathartics are not effective.

**2. Eyes and skin.** Irrigate the eyes and skin with copious amounts of tepid water. Remove contaminated clothing

## SCORING SYSTEM

## POISONING SEVERITY SCORE

A standardized scale for grading the severity of poisoning allows qualitative evaluation of morbidity caused by poisoning, better identification of real risks and comparability of data. The

PSS has been published externally.[57]

The PSS is a classification scheme for cases of poisoning in adults and children. This scheme should be used for the classification of acute poisonings regardless of the type and number of agents involved. However, modified schemes may eventually be required for certain poisonings and this scheme may then serve as a model. The PSS should take into account the overall clinical course and be applied according to the most severe symptomatology (including both subjective symptoms and objective signs). Therefore it is normally a retrospective process, requiring follow-up of cases. If the grading is undertaken at any other time (e.g. on admission) this must be clearly stated when the data are presented.

The use of the score is simple. The occurrence of a particular symptom is checked against the chart and the severity grading assigned to a case is determined by the most severe symptom(s) or sign(s) observed. Severity grading should take into account only the observed clinical symptoms and signs and it should not estimate risks or hazards on the basis of parameters such as amounts ingested or serum/plasma concentrations. The signs and symptoms given in the scheme for each grade serve as examples to assist in grading severity.

Treatment measures employed are not graded themselves, but the type of symptomatic and/or supportive treatment applied (e.g. assisted ventilation, inotropic support, haemodialysis for renal failure) may indirectly help in the evaluation of

severity. However, preventive use of antidotes should not influence the grading, but should instead be mentioned when the data are presented.

Although the scheme is, in principle, intended for grading of acute stages of poisoning, if disabling sequelae and disfigurement occur, they would justify a high severity grade and should be commented on when the data are presented. If a patient's past medical history is considered to influence the severity of poisoning this should also be commented on.

**Severity Grades**

- NONE (0): No symptoms or signs related to poisoning.
- MINOR (1): Mild, transient and spontaneously resolving symptoms.
- MODERATE (2): Pronounced or prolonged symptoms.
- SEVERE (3): Severe or life-threatening symptoms.
- FATAL (4): Death.

## PSYCHIATRIC COUNCELLING

Deliberate self-poisoning among adolescents is a major health problem. Most suicide attempts are a response to depression.[58] Drugs are the most frequent agent of self-poisoning [59], and that acetaminophen is the drug most frequently consumed.[58,59,60]

# MATERIALS AND METHODS

## OBJECTIVES OF THE STUDY

1. To study the clinical profile, investigations of patients presenting with toxicological emergencies.

2. To identify the factors, which help in predicting the severity on admission in patients presenting with toxicological emergencies

3. To assess utility of POISONING SEVERITY SCORE in predicting outcome at admission in these patients

4. Psychiatric evaluation of patients to asses cause of poisoning

**STUDY GROUP:**

The study will be carried out among patients who present to the emergency department of R.L .Jalappa Hospital, Tamaka, Kolar, over a period of one year (Jan2013-Dec2013).

Ours will be a prospective observational study carried out on adult patients presenting with acute poisoning or drug overdose.

**Inclusion Criteria**

- Patients with acute poisonings – drug overdoses, insecticide poisonings, plant and other poisonings admitted within 24 hrs of consumption of the poison.
- Alcohol dependence according to DSM-iv criteria
- Adult patients with age > 18 years

**Exclusion criteria**

- Unknown compound poisonings
- Patients who were treated at an outside hospital previous to admission.
- Patients who consume alcohol along with a toxic substance or drugs are excluded.

**Methodology**

1. A prospective , observational study
2. Informed consent from the patient or the relatives will be taken prior to inclusion in the study
3. Thorough clinical examination will be done in all patients
4. Patients will be arbitrarily divided into 5 groups-
    i. Drug overdose
    ii. Insecticide and pesticide poisonings
    iii. Plant poisons
    iv. Acids and alkalis
    v. miscellaneous
5. Severity on admission will be predicted using scoring systems – Poisoning Severity Score

**METHOD OF COLLECTION OF DATA:**

Patients who fit into the inclusion criteria will be included in the study from the time of presentation at casualty till the time of discharge / death.

Autopsy will be done for all patients who succumb due to poisoning or secondary complications. Data will be entered in previously prepared proforma.

**SAMPLE SIZE :**

310 Patients admitted in R.LJalappa Hospital over a period of one year ( Jan 2013-Dec 2013 ).

## Statistical Analysis :

- Data was entered in to Microsoft excel data sheet and was analyzed using EPI info 7 version software. Categorical data was presented in the form of frequencies and proportions. Bar charts and pie diagrams was used to represent graphically. Chi-square test was the test of significance. Continuous data was represented in the form of Mean and Standard deviation. p value $<0.05$ was considered as statistically significant.

**INVESTIGATIONS**

1. Haemogram for Haematocrit, Haemoglobin, Whole Blood Cell counts, platelet counts
2. Renal function Tests – Blood Urea, Serum Creatinine, Serum electrolytes.
3. Arterial Blood Gas Analysis
4. Random Blood Sugar
5. Liver Function Tests – Albumin, Bilirubin, liver enzymes
6. Chest X ray
7. Routine Urine analysis
8. Serum pseudocholinesterase levels when indicated
9. Electrocardiography
10. CKMB levels and CPK levels
11. Toxic blood screens when indicated.

## Statistical analysis:

Data was entered in to Microsoft excel data sheet and was analyzed using EPI info 7 version software. Categorical data was presented in the form of frequencies and proportions. Bar charts and pie diagrams was used to represent graphically. Chi-square test was the test of significance. Continuous data was represented in the form of Mean and Standard deviation. p value $<0.05$ was considered as statistically significant.

## RESULTS:

**Table 1: Age distribution of Subjects**

| | | Frequency | Percent |
|---|---|---|---|
| **Age** | **<20 yrs** | 27 | 8.7 |
| | **21 to 30 yrs** | 117 | 37.7 |
| | **31 to 40 yrs** | 80 | 25.8 |
| | **41 to 50 yrs** | 46 | 14.8 |
| | **>50 yrs** | 40 | 12.9 |
| | **Total** | 310 | 100.0 |

The Mean age among the subjects was 34.69 ± 13.01 yrs. Majority 37.7% were in the age group 21 to 30 years, 26% in the age group 31 to 40 yrs. Hence it can be said that poisoning was common in younger age group.

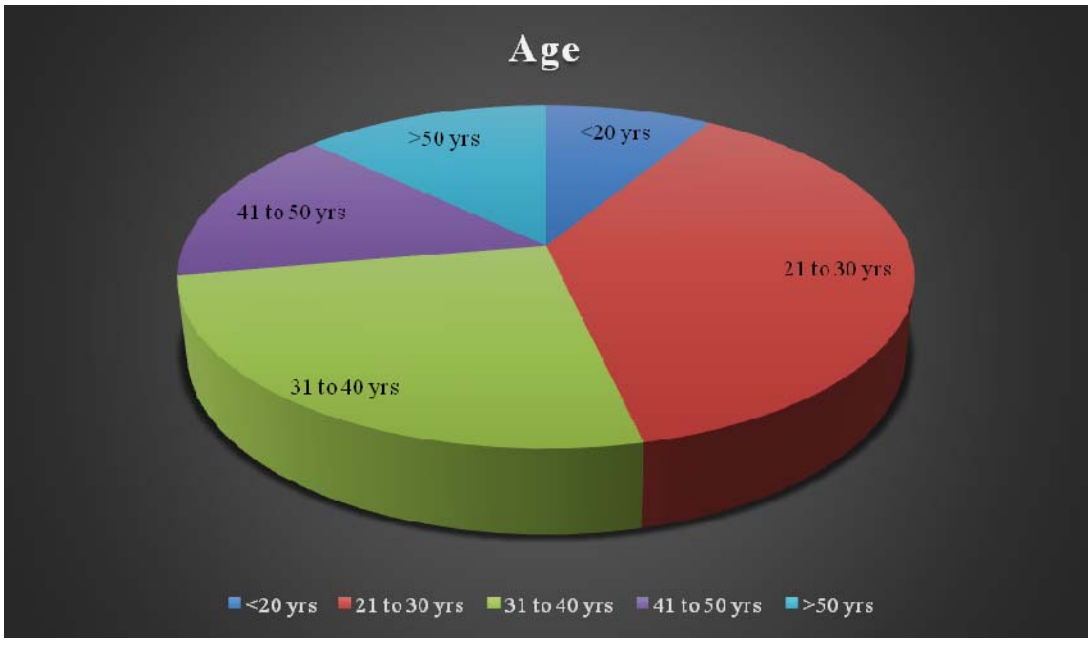



***Figure 1: Pie diagram showing age distribution***

**Table 2: Gender distribution of subjects**

| | | Frequency | Percent |
|---|---|---|---|
| Sex | Female | 165 | 53.2 |
| | Male | 145 | 46.8 |
| | Total | 310 | 100.0 |

Majority of subjects were females 53.2% and Males were 46.8%.

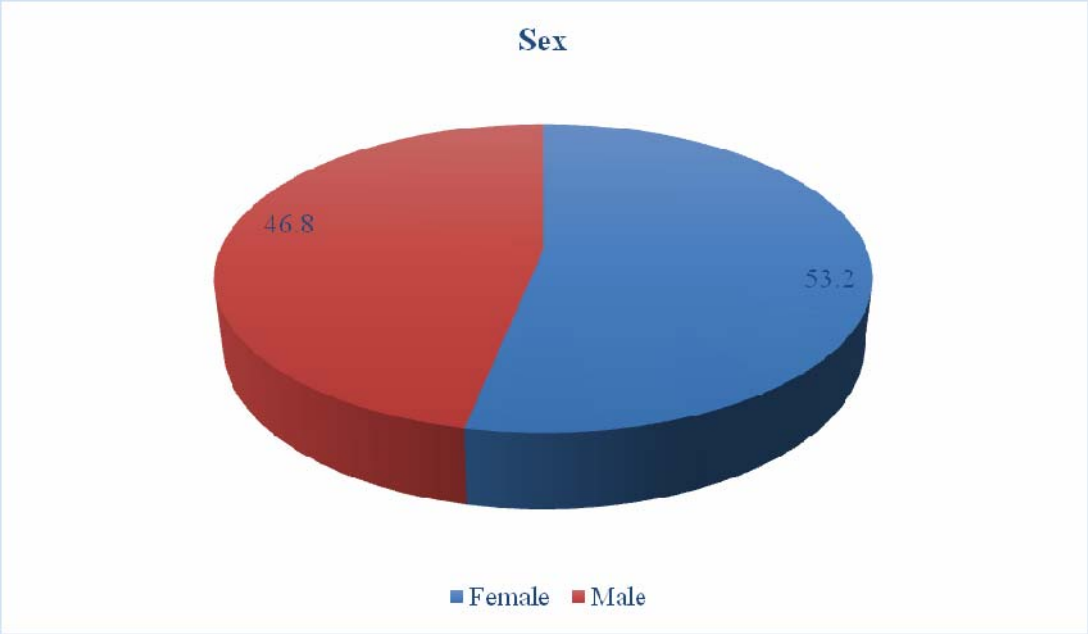



***Figure 2: Sex distribution of the subjects***

**Table 3: Type of Poisoning among subjects**

| | | Frequency | Percent |
|---|---|---|---|
| **Type of Poisoning** | **Acids** | 25 | 8.1 |
| | **Alkalis** | 15 | 4.8 |
| | **Insecticides and Pesticides** | 128 | 41.3 |
| | **Plant poison** | 20 | 6.5 |
| | **Tablets** | 82 | 26.5 |
| | **Miscellaneous** | 40 | 12.9 |
| | **Total** | 310 | 100.0 |

It was observed that most common poison used among the subjects was Insecticides and Pesticides in 41.3%, followed by tablets 26.5% and others as shown in the table.

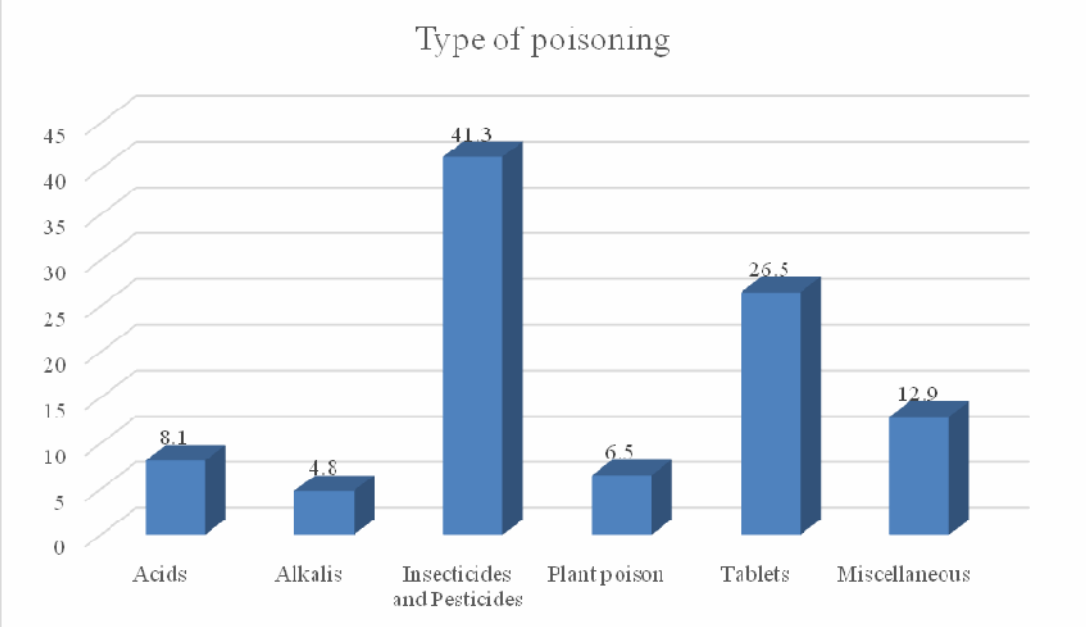



***Figure 3: Bar diagram showing type of poison used among subjects***

**Table 4: Association between Type of Poisoning and symptoms among subjects**

| Type of poison | SYMPTOMS | | | | | | Total |
|---|---|---|---|---|---|---|---|
| | CNS | CVS | GI | RS | Multi System | NO | |
| **Acids** | 0 | 0 | 12 | 0 | 0 | 13 | 25 |
| **Alkalis** | 0 | 0 | 6 | 0 | 0 | 9 | 15 |
| **Insecticides and Pesticides** | 15 | 12 | 24 | 18 | 9 | 50 | 128 |
| **Plant poison** | 1 | 0 | 9 | 0 | 0 | 10 | 20 |
| **Tablets** | 15 | 0 | 32 | 5 | 0 | 30 | 82 |
| **Miscellaneous** | 2 | 2 | 13 | 6 | 0 | 17 | 40 |
| **Total** | 33 | 14 | 96 | 29 | 9 | 129 | 310 |

**$\chi$ 2 = 62.39, df = 25, p <0.0001**

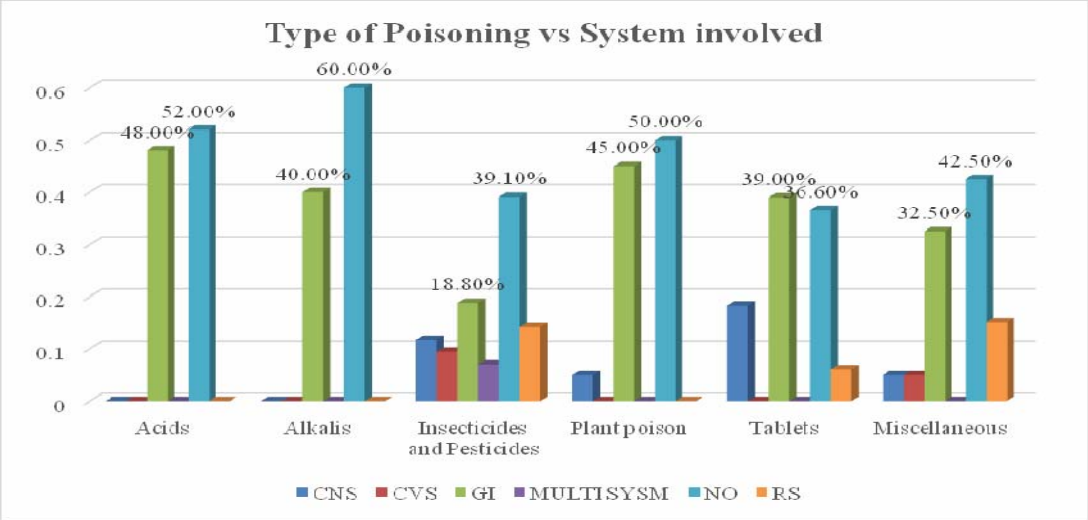



***Figure 4: Bar diagram showing association between type of poisoning and system involved***

It was observed that 52% of acid poisoning, 60% of Alkali poisoning, 40% of Insecticide and pesticide poisoning, 50% of plant poisoning, 37% of Tablet poisoning and 43% of miscellaneous poisoning had no symptoms.

GI system was the most common system involved in all types of poisoning, CVS was involved in Insecticide and pesticide poisoning and miscellaneous poisoning. CNS, RS and Multisystem are involved mainly in Insecticide and pesticide poisoning.

**Table 5: Association between Type of Poisoning and symptoms among subjects**

| Type of poison | ABG | | | | Total |
|---|---|---|---|---|---|
| | Acute respiratory acidosis | Metabolic acidosis | Mixed | Normal | |
| **Acids** | 0 | 4 | 1 | 20 | 25 |
| **Alkalis** | 1 | 3 | 1 | 10 | 15 |
| **Insecticides and Pesticides** | 18 | 33 | 19 | 58 | 128 |
| **Plant poison** | 2 | 1 | 0 | 17 | 20 |
| **Tablets** | 5 | 8 | 2 | 67 | 82 |
| **Miscellaneous** | 0 | 1 | 2 | 37 | 40 |
| **Total** | 26 | 50 | 25 | 209 | 310 |

**Mixed (Respiratory alkalosis and metabolic acidosis)**

**$\chi$ 2 = 56.87, df = 15, p <0.0001**

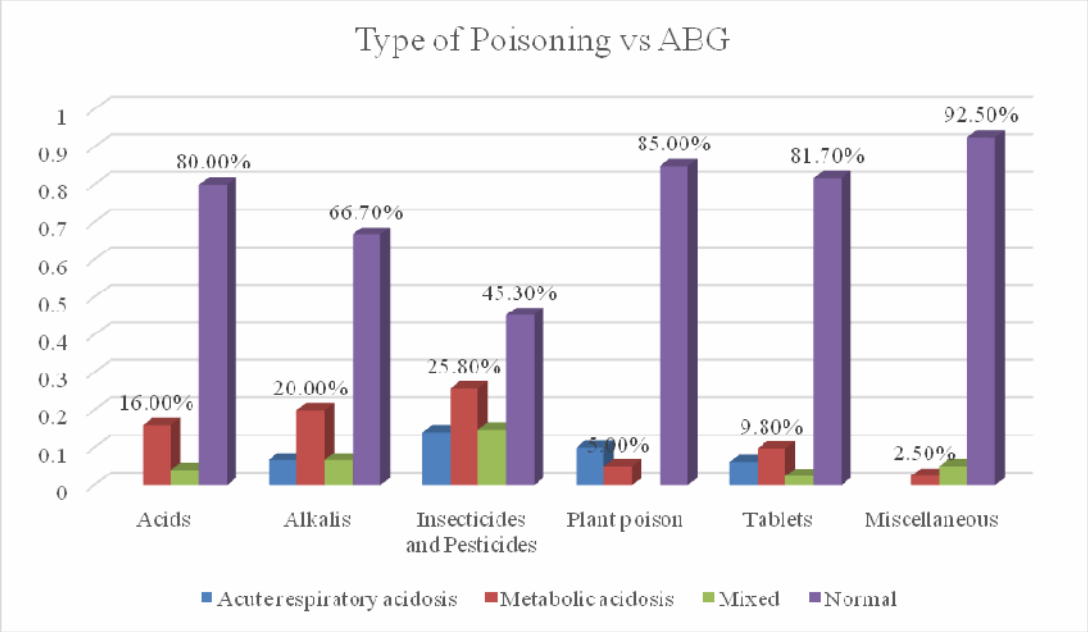



***Figure 5: Bar diagram showing association between type of poisoning and ABG***

In the study it was observed that in all the groups among majority of subjects ABG was normal. Metabolic acidosis, Mixed ABG and Acute respiratory acidosis was commonly seen in Insecticide and pesticide poisoning. This association was statistically significant.

**Table 6: Association between Type of Poisoning and symptoms among subjects**

| Type of poison | Troponin T | | Total |
|---|---|---|---|
| | Negative | Positive | |
| **Acids** | 21 | 4 | 25 |
| **Alkalis** | 12 | 3 | 15 |
| **Insecticides and Pesticides** | 109 | 19 | 128 |
| **Plant poison** | 18 | 2 | 20 |
| **Tablets** | 65 | 17 | 82 |
| **Miscellaneous** | 33 | 7 | 40 |
| **Total** | 258 | 52 | 310 |

**$\chi$ 2** = 2.057, **df = 5, p = 0.841**

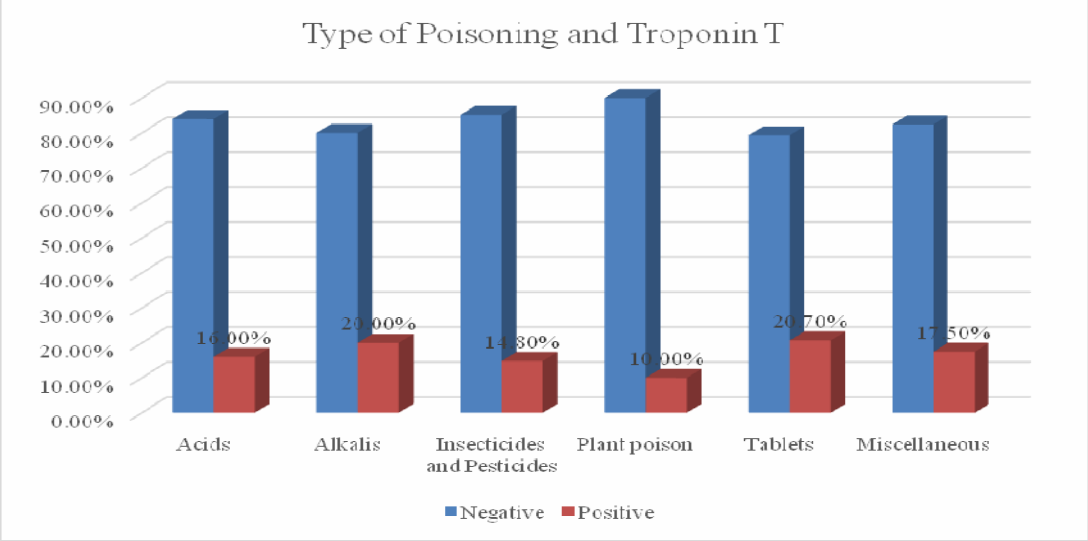



***Figure 6: Bar diagram showing association between type of poisoning and Troponin T levels***

Troponin T was positive among 21% of tablets, 20% of alkali, 17.5% of miscellaneous, 16% of acid, 15% of insecticides and 10% of plant poisoning. There was no significant association between type of poisoning and Troponin T levels.

**Table 7: Association between Type of Poisoning and ECG among subjects**

| Type of poison | ECG Findings | | | | | Total |
|---|---|---|---|---|---|---|
| | Bundle branch blocks | Extra systole | NSR | Sinus Tachycardia | Sinus Bradycardia | |
| **Acids** | 3 | 3 | 8 | 9 | 2 | 25 |
| **Alkalis** | 1 | 2 | 5 | 5 | 2 | 15 |
| **Insecticides and Pesticides** | 11 | 2 | 55 | 39 | 21 | 128 |
| **Plant poison** | 6 | 1 | 6 | 4 | 3 | 20 |
| **Tablets** | 13 | 5 | 14 | 33 | 17 | 82 |
| **Miscellaneous** | 6 | 2 | 3 | 24 | 5 | 40 |
| **Total** | 40 | 15 | 91 | 114 | 50 | 310 |

**$\chi$ 2 =** 46.1**, df = 20, p = 0.001****

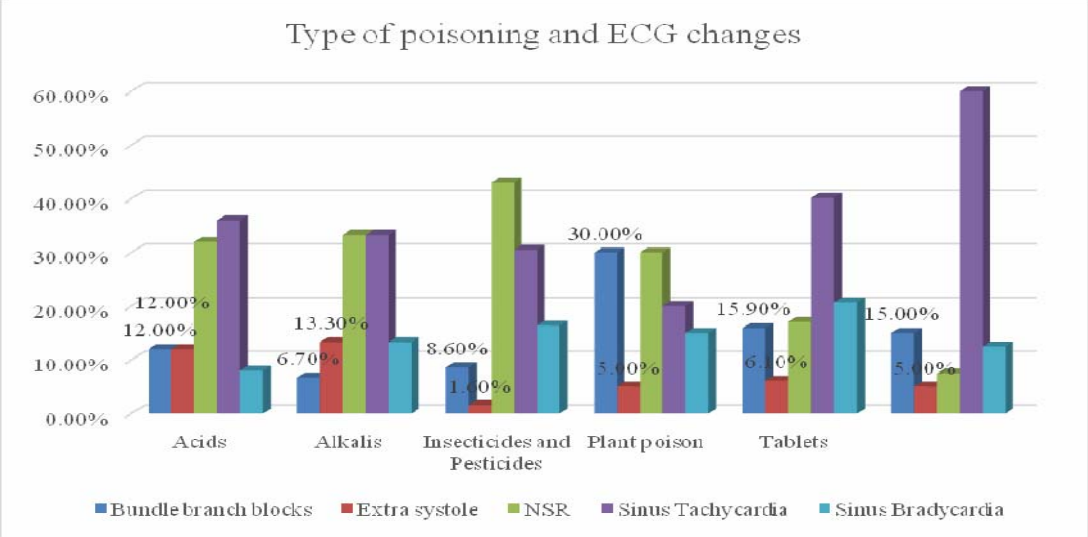



***Figure 7: Bar diagram showing association between type of poisoning and ECG changes***

In the study NSR and Sinus tachycardia were the common ECG findings in all poisoning. Bundle branch blocks was commonly seen in plant and tablet poisoning. Extra systole was seen in acid and alkali poisoning commonly. Sinus bradycardia was common in Insecticides and Plant poisoning.

**Table 8: Association between Type of Poisoning and RBS among subjects**

| | | RBS | | Total |
|---|---|---|---|---|
| | | <140 Normal | >140 Increased | |
| Type of poison | Acids | 7 | 18 | 25 |
| | Alkalis | 2 | 13 | 15 |
| | Insecticides and Pesticides | 59 | 69 | 128 |
| | Plant poison | 4 | 16 | 20 |
| | Tablets | 25 | 57 | 82 |
| | Miscellaneous | 10 | 30 | 40 |
| Total | | 107 | 203 | 310 |

**$\chi$ 2** = 15.094[a], **df = 5, p = 0.010****

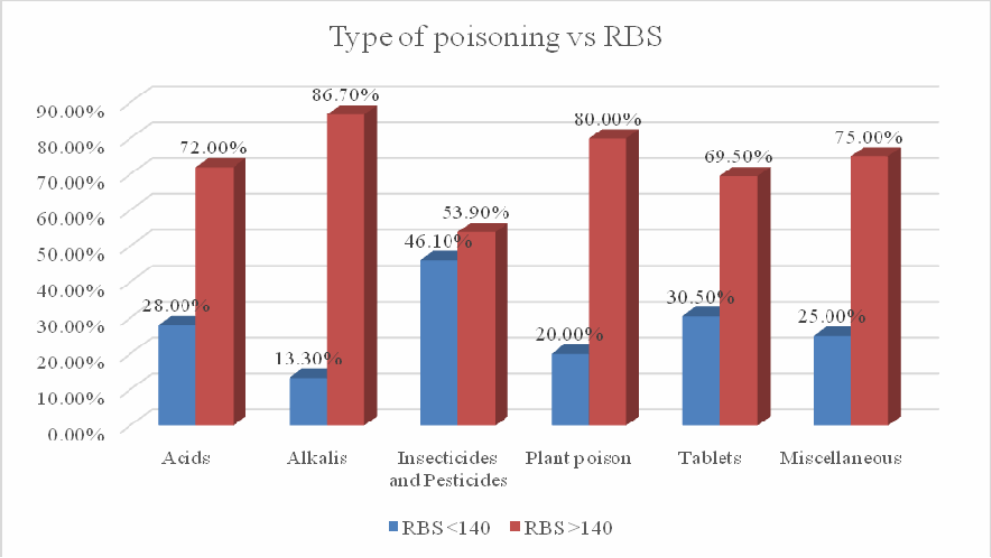



***Figure 8: Bar diagram showing association between type of poisoning and RBS***

In the study RBS was increased in all most all the poisoning and majority of them were in alkali poisoning. There was significant association between RBS levels and type of poisoning.

**Table 9: Association between Type of Poisoning and CPK among subjects**

| | | CPK | | Total |
|---|---|---|---|---|
| | | <195 Normal | >195 Increased | |
| Type of poison | Acids | 20 | 5 | 25 |
| | Alkalis | 11 | 4 | 15 |
| | Insecticides and Pesticides | 77 | 51 | 128 |
| | Plant poison | 13 | 7 | 20 |
| | Tablets | 57 | 25 | 82 |
| | Miscellaneous | 31 | 9 | 40 |
| Total | | 209 | 101 | 310 |

**$\chi$ 2 = 7.18, df = 5, p = 0.207**

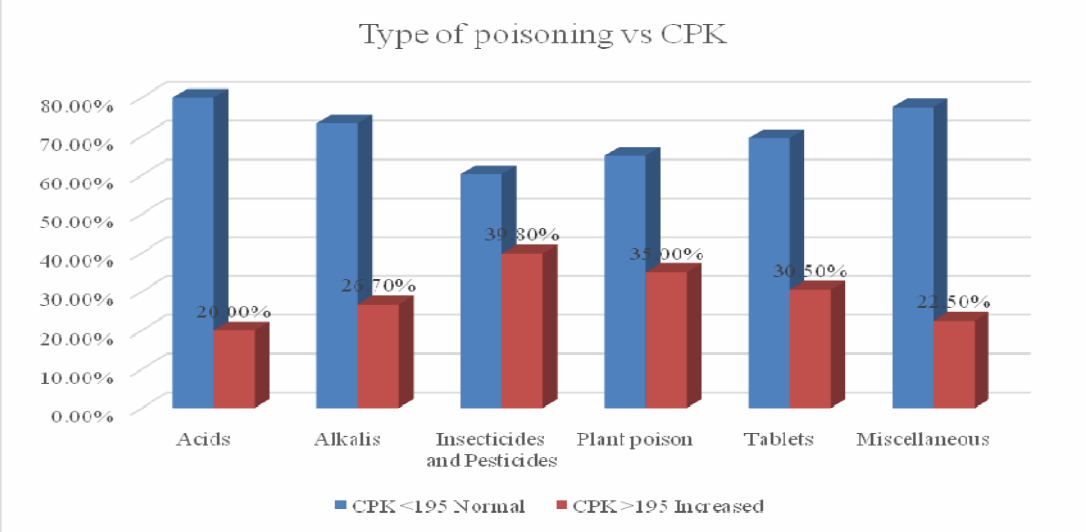



***Figure 9: Bar diagram showing association between type of poisoning and CPK***

In the study CPK was normal among majority in all the poisoning. CPK was raised in 40% of Insecticide poisoning. There was no significant association between CPK levels and type of poisoning.

**Table 10: Association between Type of Poisoning and CKMB among subjects**

| | | CKMB | | Total |
|---|---|---|---|---|
| | | <24 Normal | >24 Increased | |
| Type of poison | Acids | 25 | 0 | 25 |
| | Alkalis | 12 | 3 | 15 |
| | Insecticides and Pesticides | 70 | 58 | 128 |
| | Plant poison | 14 | 6 | 20 |
| | Tablets | 64 | 18 | 82 |
| | Miscellaneous | 31 | 9 | 40 |
| Total | | 216 | 94 | 310 |

**$\chi$ 2 = 29.12, df = 5, p = 0.0001*****

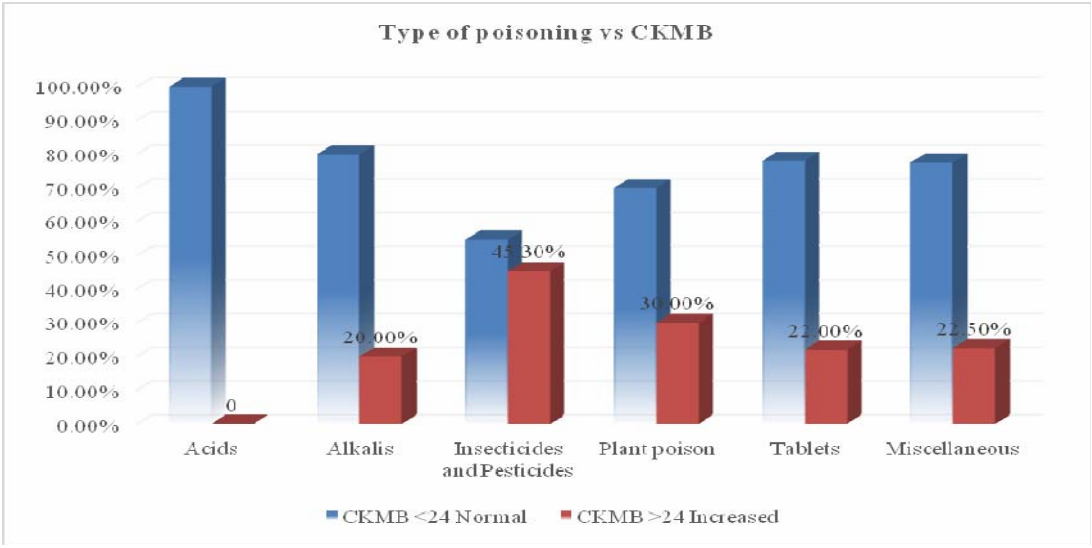



***Figure 10: Bar diagram showing association between type of poisoning and CKMB***

In the study it was observed that 45.3% of insecticide poisoning had increased CKMB, followed by 30% in plant poisoning, 22.5% in miscellaneous, 22% in tablets poisoning and 20% in alkali poisoning. No increase in CKMB was found in acid poisoning. This observation was statistically significant.

**Table 11: Association between Type of Poisoning and PCHE (Pseudo cholinesterase enzyme) among subjects**

| | | PCHE | | Total |
|---|---|---|---|---|
| | | >4000 Normal | <4000 Decreased | |
| Type of poison | Insecticides and Pesticides | 65 | 62 | 127 |

**$\chi$ 2 = 1.04, df = 1, p = 0.308**

In the study PCHE was estimated only in 128 subjects. It was observed that PCHE was reduced in 48.8% of Insecticide poisoning. There was no significant association for increase in PCHE with respect to type of poisoning.

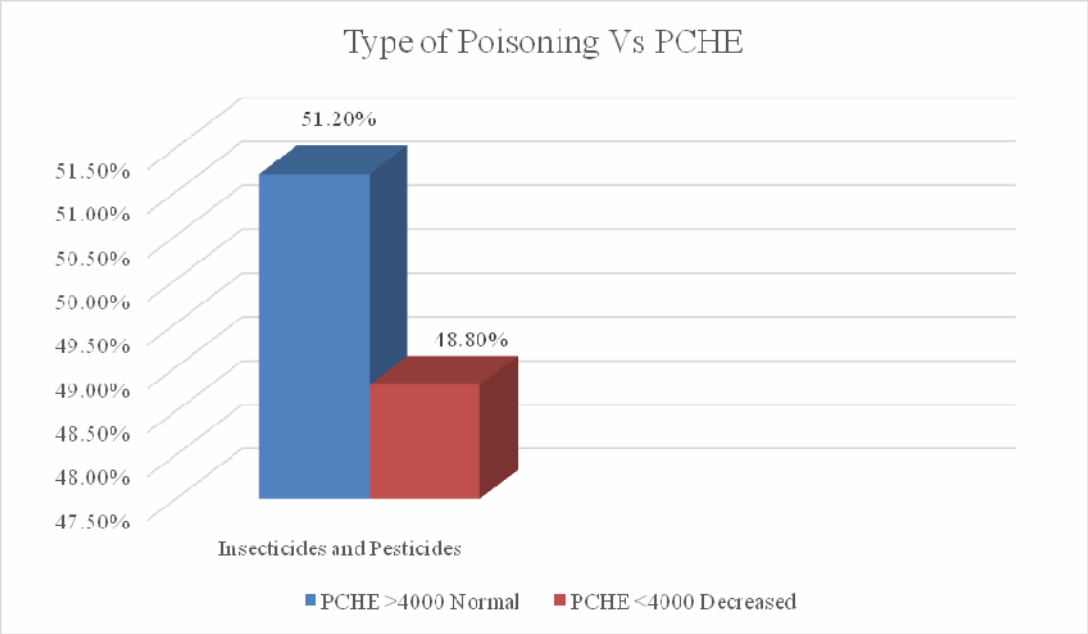



***Figure 11: Bar diagram showing association between type of poisoning and PCHE***

**Table 12: Association between Type of poisoning and PSS**

| | | PSS | | | | | Total |
|---|---|---|---|---|---|---|---|
| | | Death | Minor | Moderate | None | Severe | |
| Type of poison | Acids | 1 | 9 | 6 | 7 | 2 | 25 |
| | Alkalis | 0 | 5 | 3 | 6 | 1 | 15 |
| | Insecticides and Pesticides | 30 | 32 | 24 | 31 | 11 | 128 |
| | Plant poison | 0 | 3 | 0 | 16 | 1 | 20 |
| | Tablets | 1 | 24 | 13 | 40 | 4 | 82 |
| | Miscellaneous | 6 | 17 | 2 | 12 | 3 | 40 |
| Total | | 38 | 90 | 48 | 112 | 22 | 310 |

**$\chi$ 2 = 61.796, df = 20, p = <0.001****

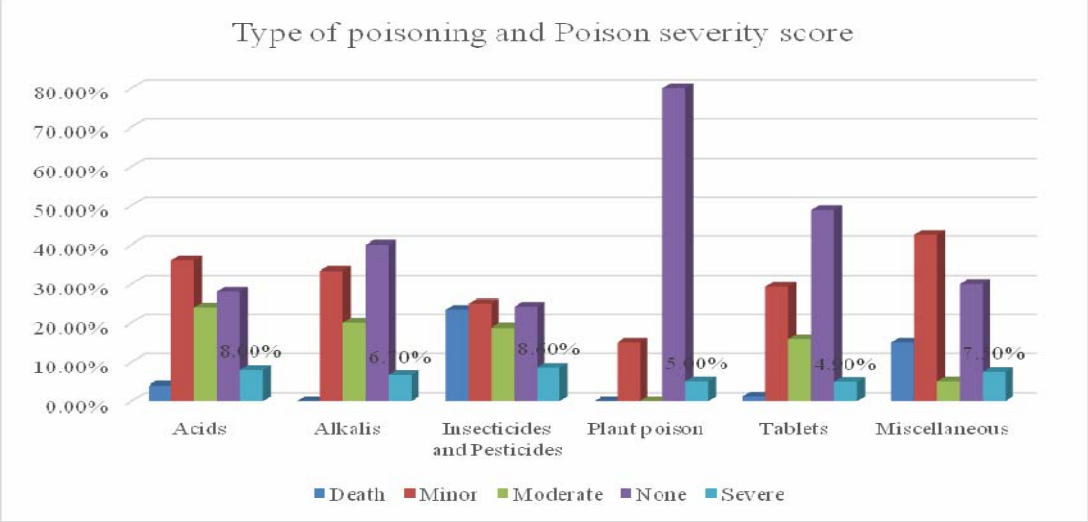



***Figure 12: Bar diagram showing association between type of poisoning and Poison severity score***

Poison severity score showed Majority i.e. 36% had normal score, 29% had minor score, 15.5% had moderate score, 12.3% had mortality, and 7.1% had severe score. There was statistically significant association between different types of poisoning and PSS. Severity (8.6%) and death (23.4%) was high among insecticide poisoning.

**Table 13: Association between Type of poisoning and Intubation**

| | | Intubation | | Total |
|---|---|---|---|---|
| | | No | Yes | |
| Type of poison | Acids | 25 | 0 | 25 |
| | Alkalis | 15 | 0 | 15 |
| | Insecticides and Pesticides | 58 | 70 | 128 |
| | Plant poison | 20 | 0 | 20 |
| | Tablets | 71 | 11 | 82 |
| | Miscellaneous | 40 | 0 | 40 |
| Total | | 229 | 81 | 310 |

**$\chi$ 2 = 96.32, df = 5, p = <0.001****

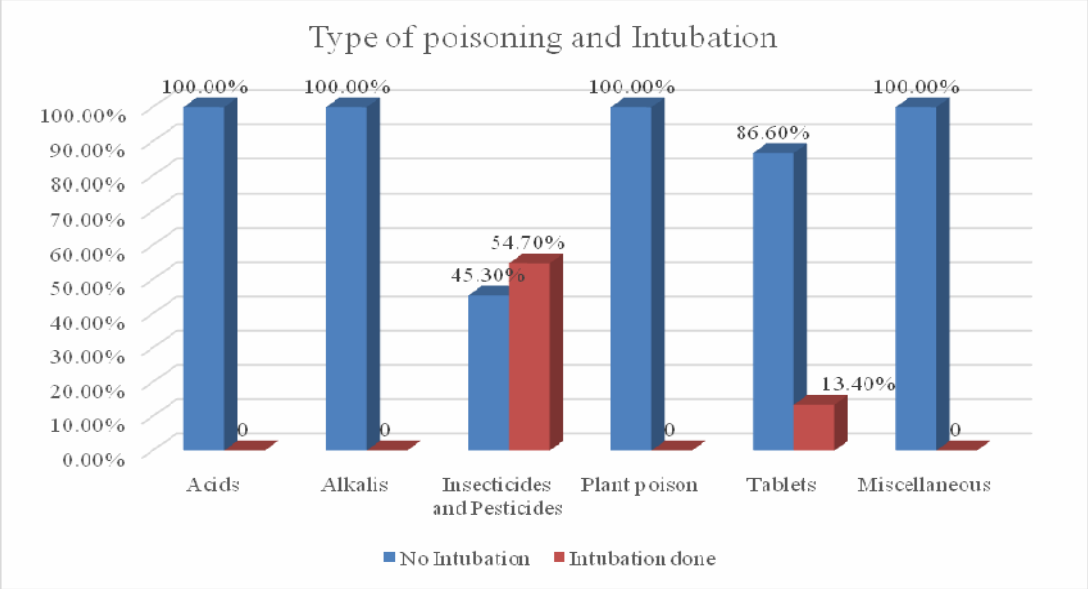



***Figure 13: Bar diagram showing association between type of poisoning and Intubation***

In the study intubation was done in 55% of insecticide poisoning and 13% of tablet poisoning. No intubation was done for other poisoning. This observation was statistically significant.

**Table 14: Association between Type of poisoning and Outcome**

| | | Outcome | | | Total |
|---|---|---|---|---|---|
| | | Complications | Death | Recovery | |
| Type of poison | Acids | 1 | 1 | 23 | 25 |
| | Alkalis | 0 | 0 | 15 | 15 |
| | Insecticides and Pesticides | 4 | 29 | 95 | 128 |
| | Plant poison | 1 | 0 | 19 | 20 |
| | Tablets | 6 | 1 | 75 | 82 |
| | Miscellaneous | 3 | 6 | 31 | 40 |
| Total | | 15 | 37 | 258 | 310 |

**$\chi$ 2 = 32.3, df = 10, p = 0.001****

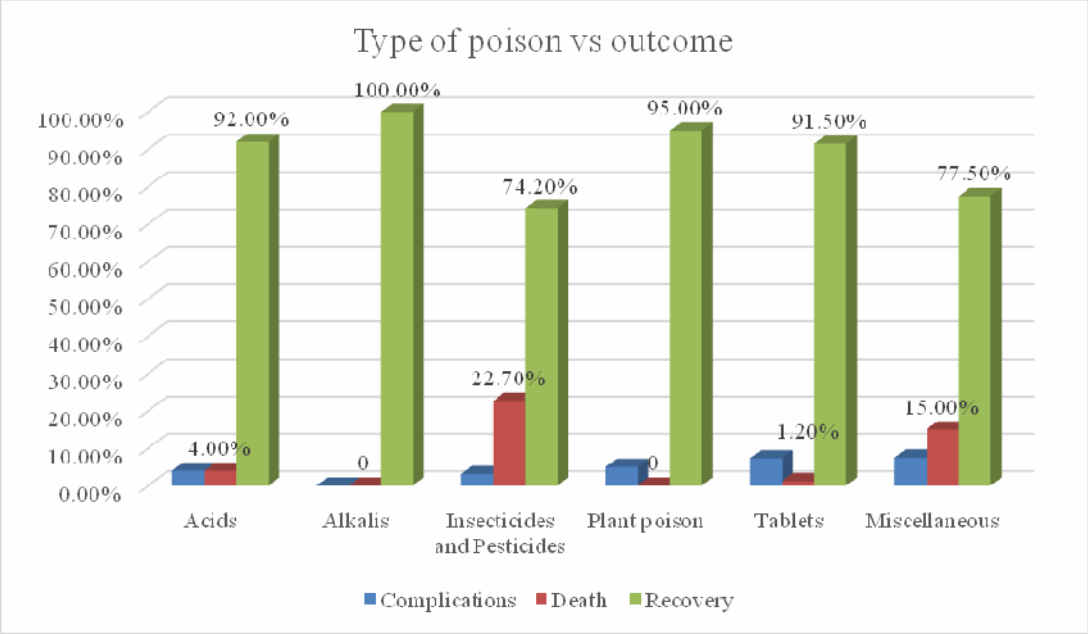



***Figure 14: Bar diagram showing association between type of poisoning and outcome***

In the study death occurred in 22.7% of insecticide poisoning, 15% of miscellaneous poisoning, 4% in acid poisoning and 1.2% in tablet poisoning. Majority of poisoning had high recovery rate. This was statistically significant.

**Table 15: Association between PSS and Outcome**

| | | Outcome | | | Total |
|---|---|---|---|---|---|
| | | Complications | Death | Recovery | |
| PSS | Death | 0 | 36 | 2 | 38 |
| | Minor | 0 | 1 | 89 | 90 |
| | Moderate | 6 | 0 | 42 | 48 |
| | None | 0 | 0 | 112 | 112 |
| | Severe | 9 | 0 | 13 | 22 |
| Total | | 15 | 37 | 258 | 310 |

**$\chi$ 2 = 361.268, df = 8, p <0.0001****

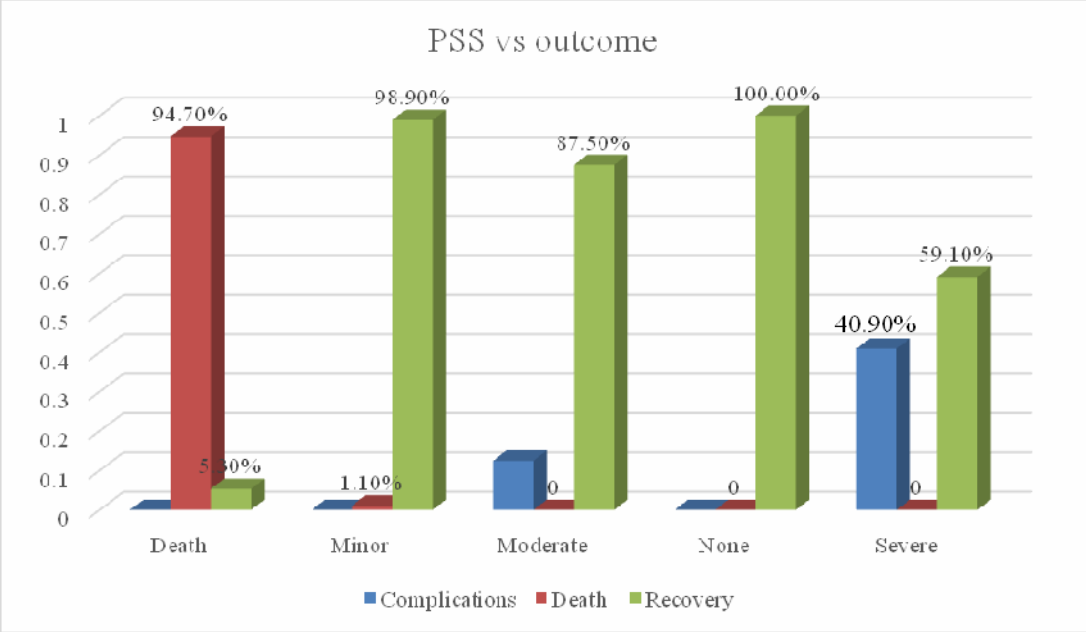



***Figure 15: Bar diagram showing association between PSS and outcome***

There was significant association between PSS score and Outcome in Poisoning. I.e. among patients with PSS score of death 94.7% died in the study. 40.9% of Severe PSS score had complications. Hence PSS can be a useful tool for predicting the outcome in Poisoning subjects.

**Table 16: Association between ABG and Outcome**

| | | Outcome | | | Total |
|---|---|---|---|---|---|
| | | Complications | Death | Recovery | |
| ABG | Acute respiratory acidosis | 3 | 4 | 19 | 26 |
| | Metabolic acidosis | 1 | 9 | 40 | 50 |
| | Mixed | 0 | 3 | 22 | 25 |
| | Normal ABG | 11 | 21 | 177 | 209 |
| Total | | 15 | 37 | 258 | 310 |

**Mixed = Respiratory acidosis plus metabolic acidosis**

**$\chi$ 2 = 7.46, df = 6, p = 0.280**

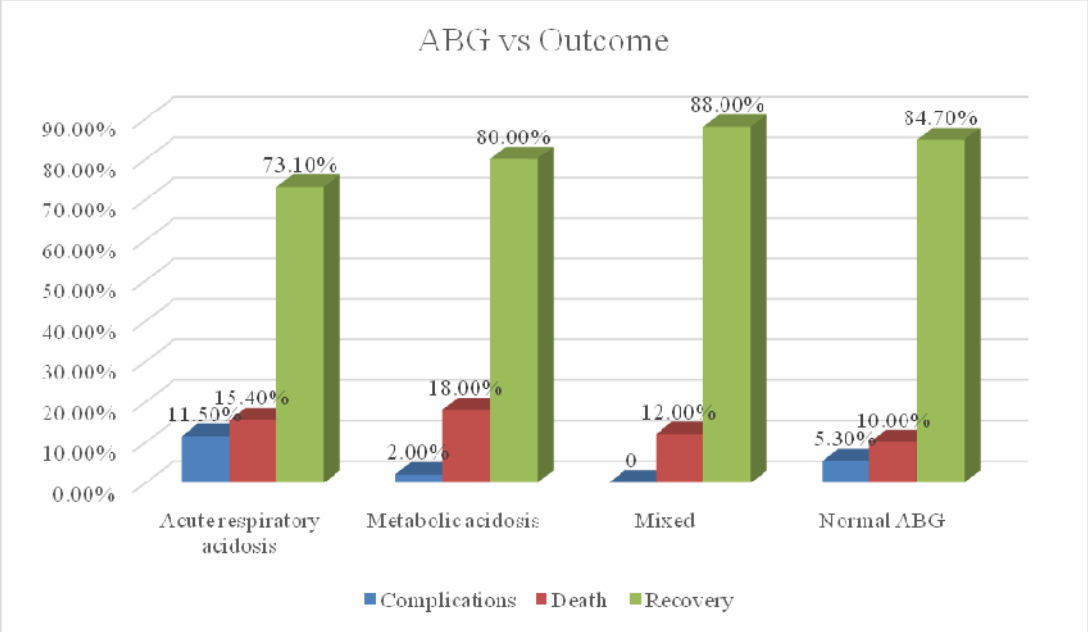



***Figure 16: Bar diagram showing association between ABG and Outcome***

In the study it was observed that among the subjects who died 18% had metabolic acidosis, 15.4% had acute respiratory acidosis, 12% had mixed ABG and 10% had Normal ABG**.** But ABG was not significantly associated with outcome.

**Table 17: Association between Trop T and Outcome**

| | | Outcome | | | Total |
|---|---|---|---|---|---|
| | | Complications | Death | Recovery | |
| Troponin T | Negative | 15 | 33 | 210 | 258 |
| | Positive | 0 | 4 | 48 | 52 |
| Total | | 15 | 37 | 258 | 310 |

**$\chi$ 2 =** 4.58**, df = 2, p = 0.101**

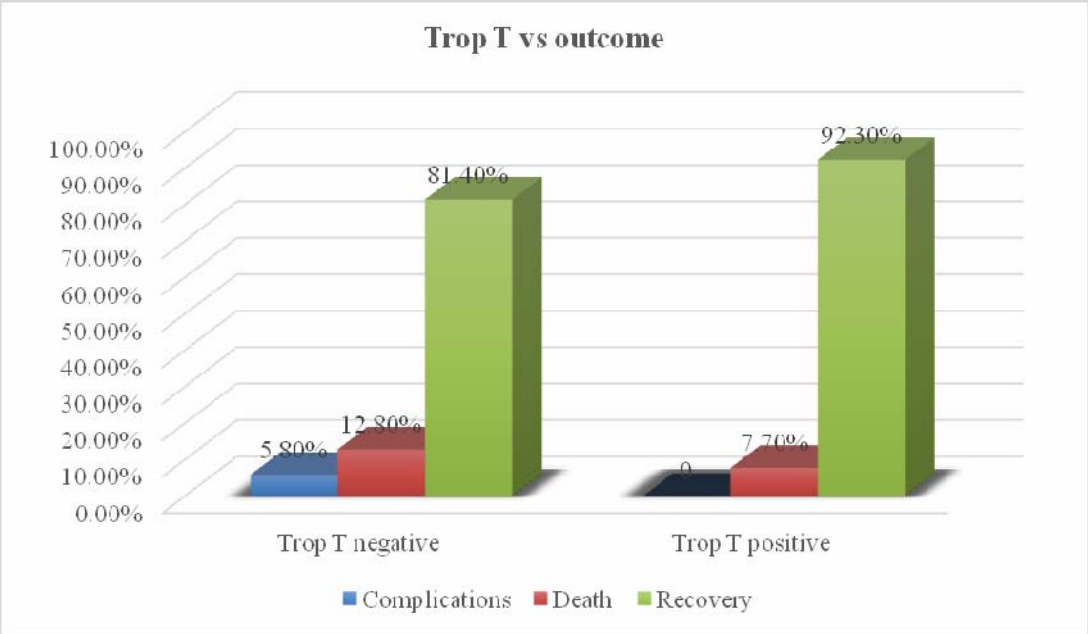



***Figure 17: Bar diagram showing association between Trop T and Outcome in poisoning subjects***

It was observed that among Trop T positive subjects 7.7% died and among Trop T negative subjects 12.8% was the mortality rate. But significant association was not observed between these two variables.

**Table 18: Association between ECG and Outcome**

| | | Outcome | | | Total |
|---|---|---|---|---|---|
| | | Complications | Death | Recovery | |
| ECG | Bundle branch blocks | 4 | 3 | 33 | 40 |
| | Extra systole | 0 | 2 | 13 | 15 |
| | NSR | 3 | 14 | 74 | 91 |
| | Sinus Tachycardia | 3 | 12 | 99 | 114 |
| | Sinus Bradycardia | 5 | 6 | 39 | 50 |
| Total | | 15 | 37 | 258 | 310 |

$\chi^2 = 9.46$, **df = 8, p = 0.305**

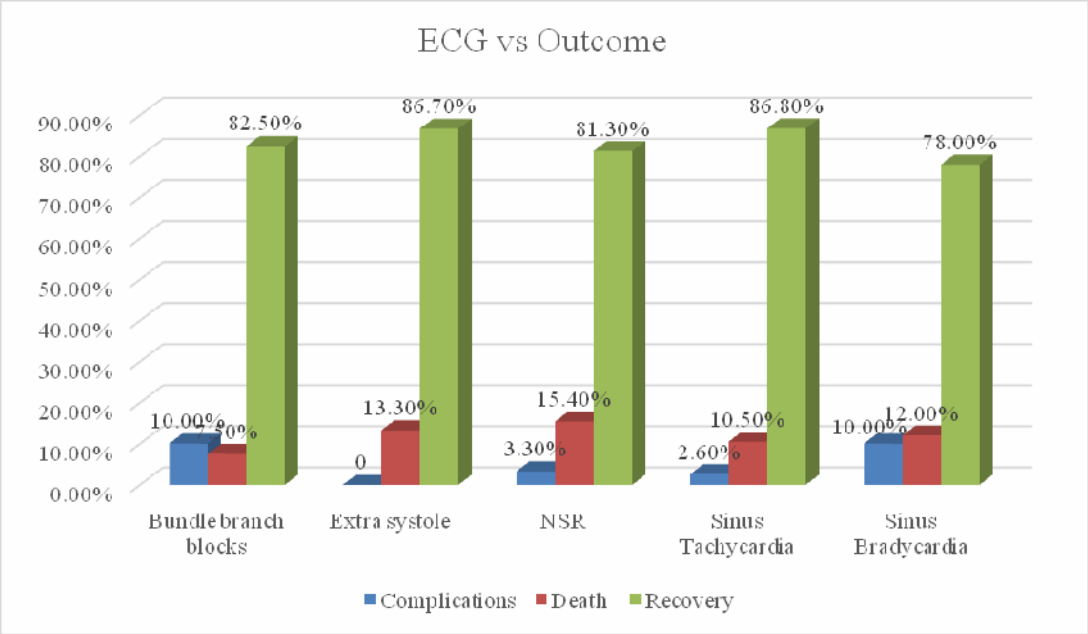



***Figure 18: Bar diagram showing association between ECG and outcome***

Among 37 deaths 15.4% had no ECG changes, 13.3% had extra systole, 12% had sinus bradycardia, 10.5% had Sinus tachycardia and 7.5% had Bundle branch blocks. But there was no significant association.

**Table 19: Association between Intubation and Outcome**

| | | Outcome | | | Total |
|---|---|---|---|---|---|
| | | Complications | Death | Recovery | |
| Intubation | No | 8 | 17 | 204 | 229 |
| | Yes | 7 | 20 | 54 | 81 |
| Total | | 15 | 37 | 258 | 310 |

**$\chi$ 2 =** 21.83**, df = 2, p = 0.0001****

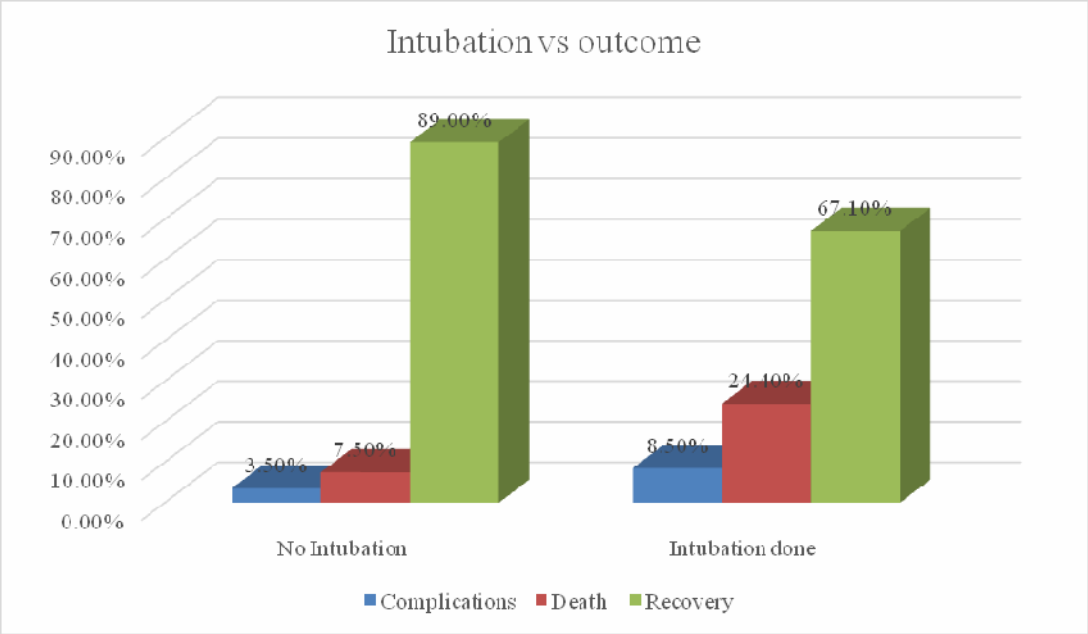



***Figure 19: Bar diagram showing association between Intubation and Outcome in poisoning patients***

It was observed that among the patients who were intubated the mortality was high i.e. 24.4% and among non-intubated patients mortality was 7.5%. Similarly complication rate was also high in intubated patients. This observation was statistically significant.

**Table 20: Association between RBS and Outcome among poisoning subjects**

| | | RBS | | Total |
|---|---|---|---|---|
| | | <140 Normal | >140 Increased | |
| Outcome | Complications | 3 | 12 | 15 |
| | Death | 18 | 19 | 37 |
| | Recovery | 86 | 172 | 258 |
| Total | | 107 | 203 | 310 |

**$\chi$ 2 = 4.82, df = 2, p = 0.089**

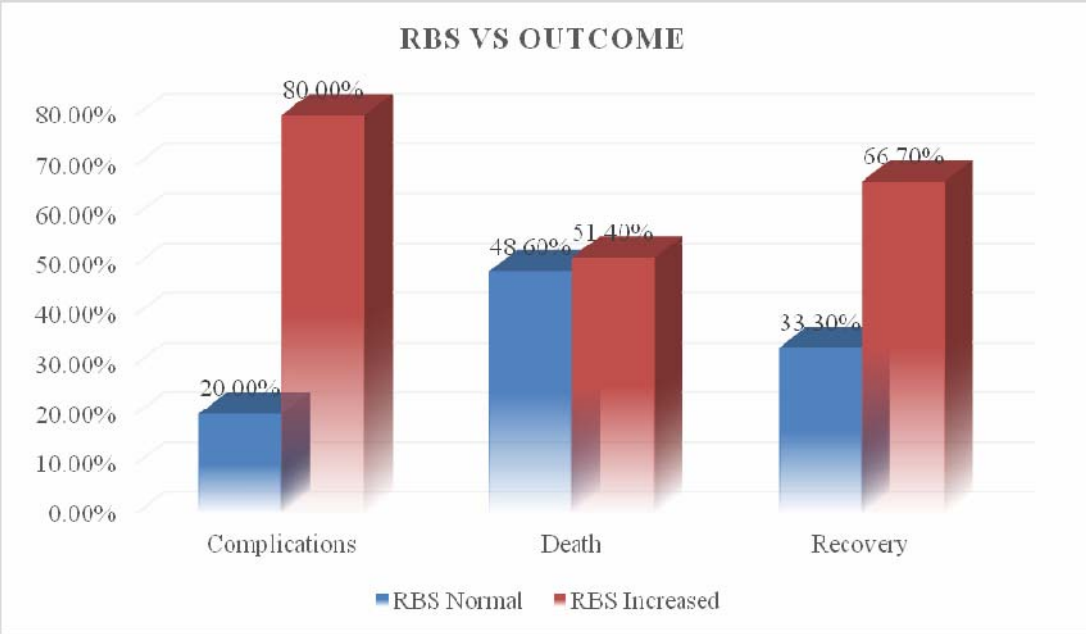



***Figure 20: Bar diagram showing association between RBS and outcome***

Among 37 patients who died 51.4% had raised RBS levels and 80% of subjects with complication had increased RBS level. This observation was not statistically significant

**Table 21: Association between CPK and Outcome among poisoning subjects**

| | | CPK | | Total |
|---|---|---|---|---|
| | | <195 Normal | >195 Increased | |
| Outcome | Complications | 8 | 7 | 15 |
| | Death | 23 | 14 | 37 |
| | Recovery | 178 | 80 | 258 |
| Total | | 209 | 101 | 310 |

**$\chi$ 2 = 2.11, df = 2, p = 0.348**

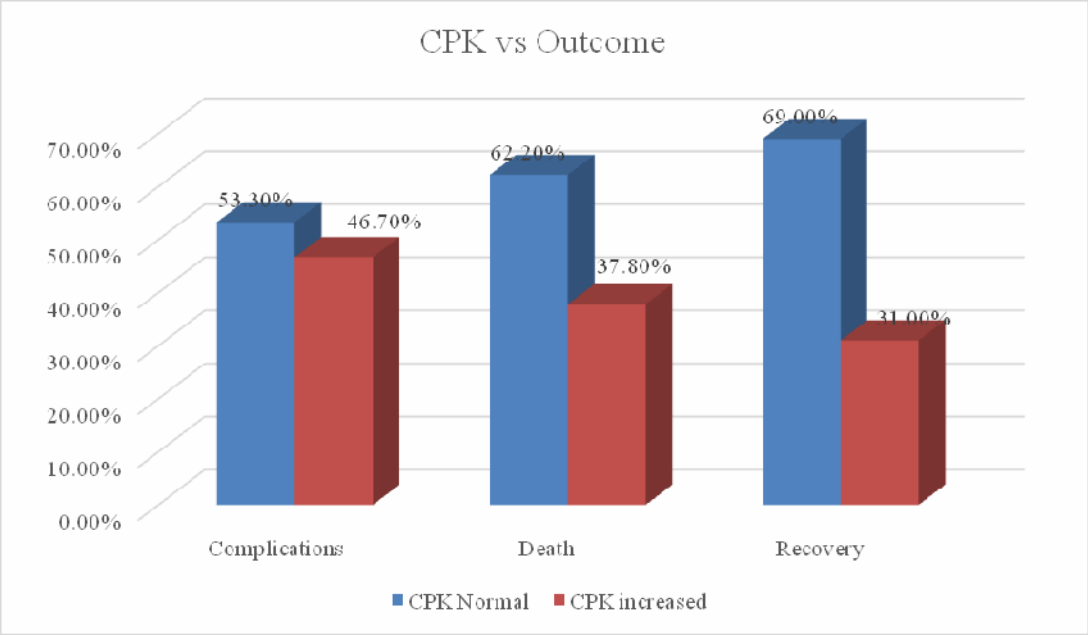



***Figure 21: Bar diagram showing association between CPK and Outcome***

It was observed that 46.7% of subjects with complications, 37.8% of subjects who died and 31% who recovered had increased CPK levels. This observation was not statistically significant.

**Table 22: Association between CKMB and Outcome among poisoning subjects**

| | | CKMB | | Total |
|---|---|---|---|---|
| | | <24 Normal | >24 Increased | |
| Outcome | Complications | 9 | 6 | 15 |
| | Death | 23 | 14 | 37 |
| | Recovery | 184 | 74 | 258 |
| Total | | 216 | 94 | 310 |

**$\chi$ 2 = 1.98, df = 2, p = 0.371**

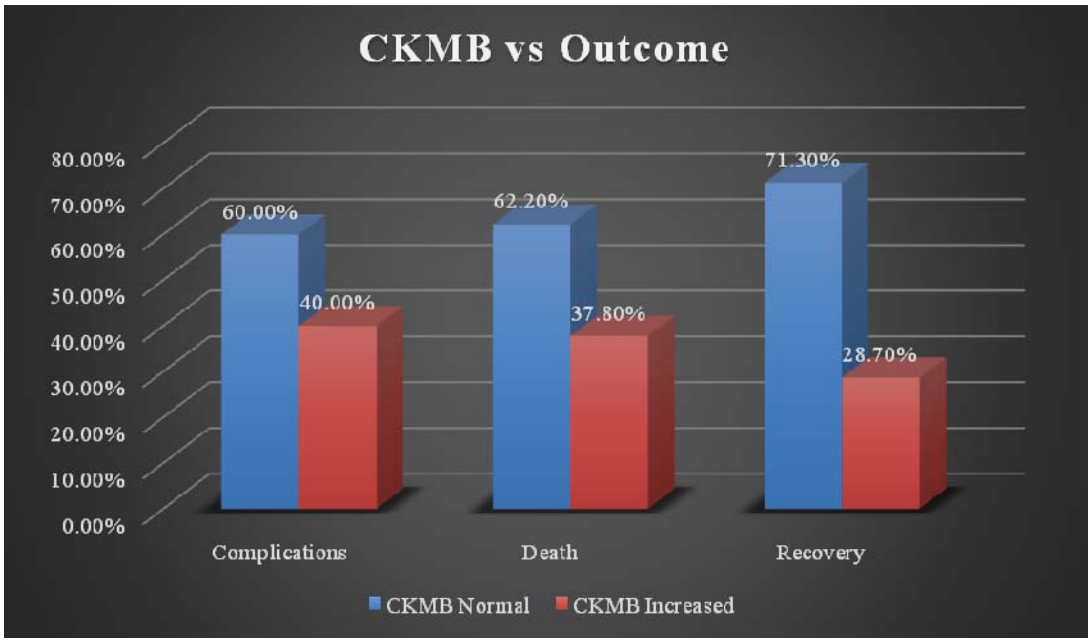



***Figure 22: Bar diagram showing association between CKMB and Outcome***

It was observed that 40% of subjects with complications, 37.8% of subjects with mortality and 28.7% who recovered had increased CKMB levels. This observation was not significantly associated.

**Table 23: Association between PCHE and Outcome among poisoning subjects**

| | | PCHE | | Total |
|---|---|---|---|---|
| | | >4000 Normal | <4000 Decreased | |
| Outcome | Complications | 4 | 0 | 4 |
| | Death | 16 | 12 | 28 |
| | Recovery | 45 | 51 | 96 |
| Total | | 65 | 63 | 128 |

**$\chi$ 2 = 4.916, df = 2, p = 0.086**

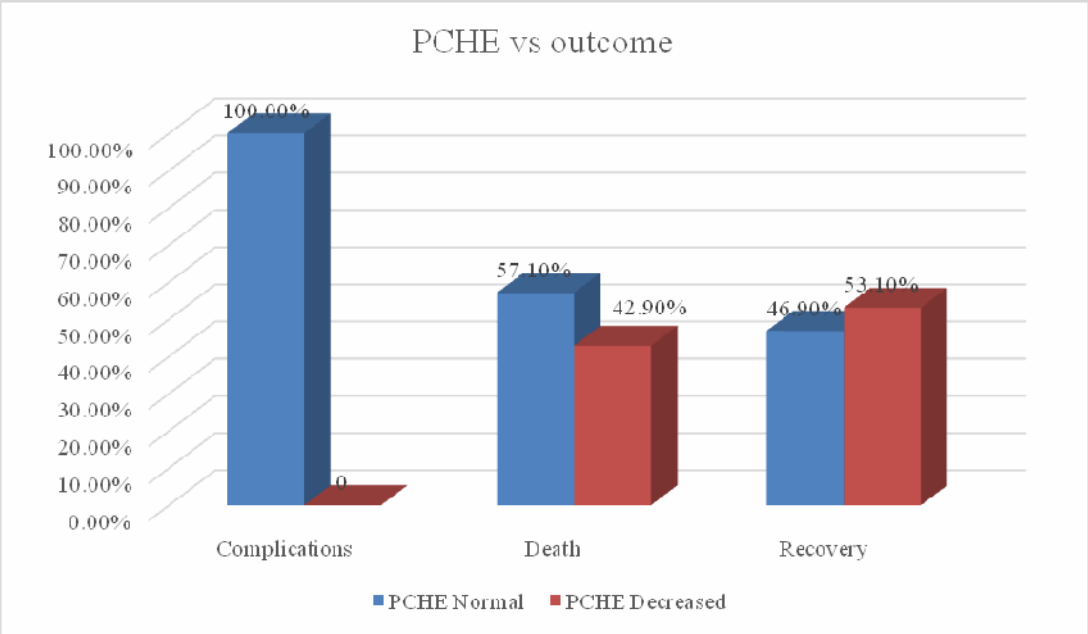



***Figure 23: Bar diagram showing association between PCHE and Outcome***

It was observed that 42.9% of subjects with mortality and 53.1% who recovered had decreased PCHE levels. This observation was not significantly associated.

**Table 24: Post mortem Stomach Mucosal appearances in various fatal poisoning cases**

| | Insecticides and Pesticides (n=29) | Acids (n=1) | Drugs (n=1) | Miscellaneous (n=6) |
|---|---|---|---|---|
| **Congestion** | 15 | 0 | 1 | 3 |
| **Hemorrhage** | 4 | 0 | 0 | 0 |
| **Erosive** | 5 | 1 | 0 | 2 |
| **Edematous** | 5 | 0 | 0 | 1 |

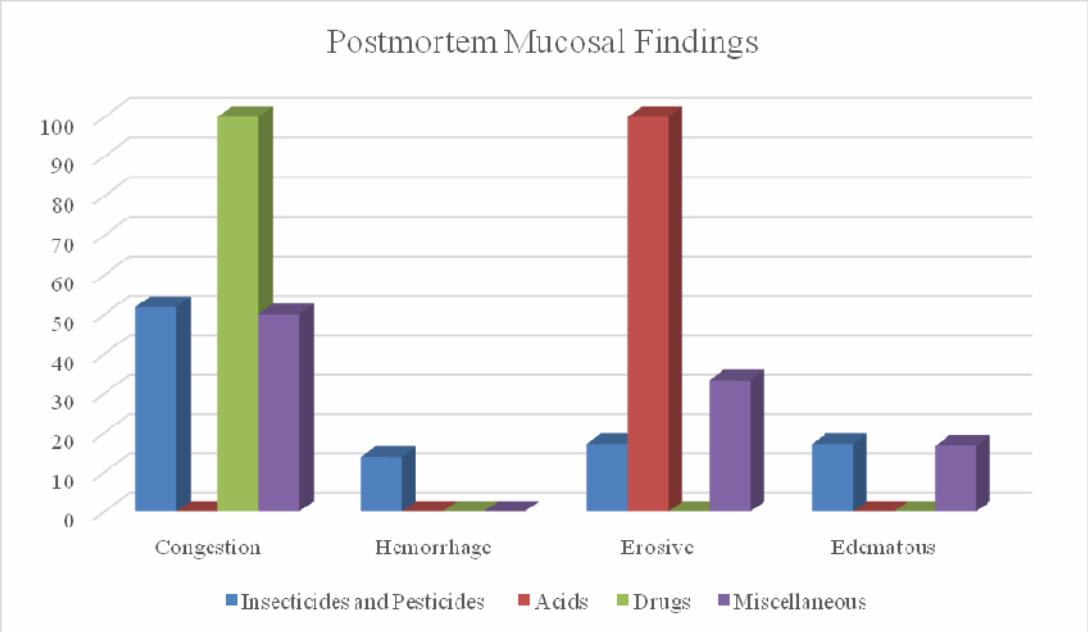



***Figure 24: Bar diagram showing postmortem findings of mucosal changes in various poisoning***

**Table 25: Poison detected by Chemical analysis among subjects who had mortality**

| Type of Poisoning | No | Percentage |
|---|---|---|
| **Organophosphorus Poison** | 26 | 70.3 |
| **Phosphide** | 4 | 10.8 |
| **Phenol** | 1 | 2.7 |
| **Oleander** | 1 | 2.7 |
| **No Result** | 5 | 13.5 |
| **Total** | 37 | 100 |

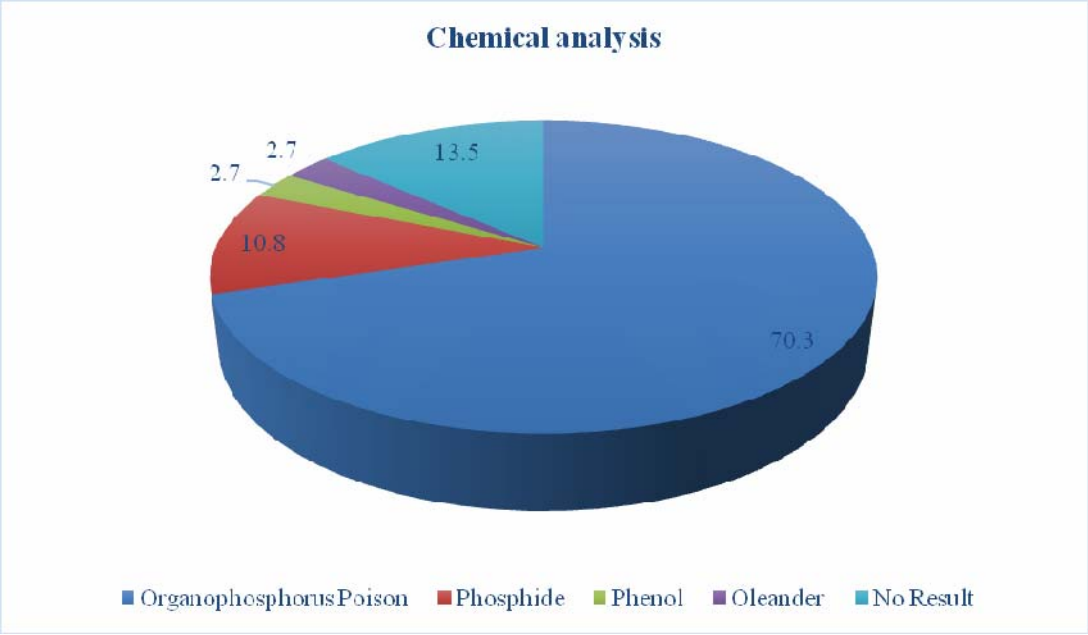
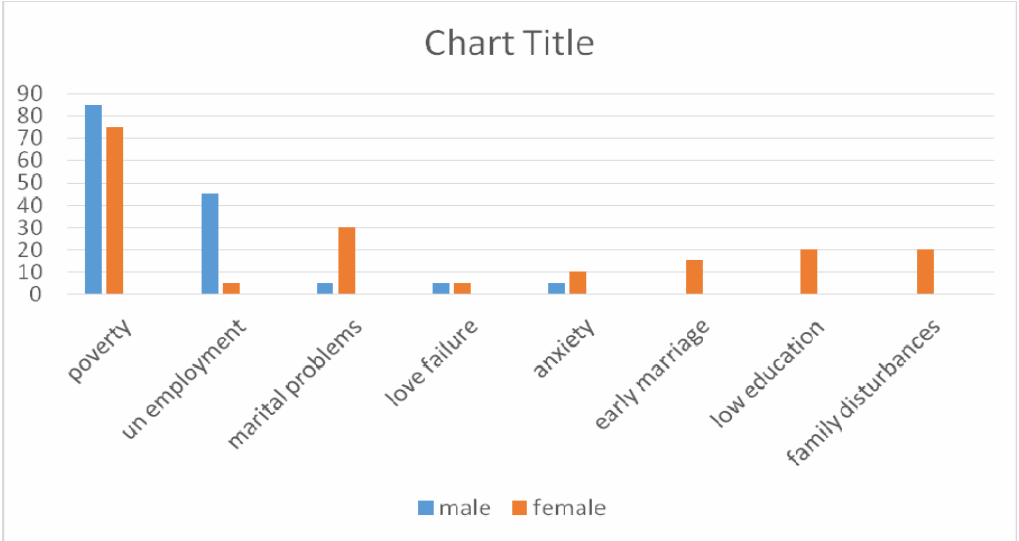



***Figure 25: Pie diagram showing chemical analysis findings***

**Table 26. Psychiatric evaluation for consumption of poison**

| Cause | Male | percentage | Female | percentage |
|---|---|---|---|---|
| **poverty** | 85 | 58.7% | 65 | 36.5% |
| **unemployment** | 45 | 31% | 5 | 3% |
| **Marital problems** | 5 | 3.5% | 30 | 18.2% |
| **Love failure** | 5 | 3.5% | 5 | 3% |
| **anxiety** | 5 | 3.5% | 10 | 6% |
| **Early marriage** | 0 | 0 | 15 | 9.1% |
| **Low education** | 0 | 0 | 20 | 12.1% |
| **Family disturbances** | 0 | 0 | 20 | 12.1% |
| **Total** | 145 | 100% | 165 | 100% |



# DISCUSSION

Insecticides and pesticides are most common group of poisons encountered in centre followed by tablet consumption, acids and alkalis, miscellaneous and plant poisons.

**COMPARITIVE DISCUSSION**:

**Age distribution of Subjects (Table 1)**

Majority 37.7% were in the age group 21 to 30 years. Hence it can be said that poisoning was common in younger age group. This correlates with other studies Aravind etal.[42],Patil. A et al. [13],Bansal M et al.[61] and Jaiprakash H et.al[62].Retrospective review, in a rural scenario it was found that the average age for marriage was between 21 and 30 years, this further could indicate this group are prone to have stress due to increased interpersonal marital conflicts. The second most common reason cited was increased prevalence of unemployment and lastly by student population who have to face a constant peer pressure. All these can lead to increase in the stress level.

**Sex distribution of subjects: (Table 2)**

Majority of subjects were females 53.2% and Males were 46.8%. this correlates with one of the studies in nepal done byPaudyal BP[63] andPandey K R et.al [64] But most of other studies such as Aravind et al[42], Patil. A et al [13], Bansal M et al[61] and Jaiprakash H et.al[62] show males are more effected than females. Our study we analyzed the fact such as early marriage, stoppage of education, dowry problem and husband is alcoholic were the major issues.

**Type of Poisoning among subjects (Table 3)**

It was observed that most common poison used among the subjects was Insecticides and Pesticides in 41.3%, followed by tablets 26.5%. in our study most common insecticide used as mode of poison are organoposphorous compounds followed by drugs. This is similar to studies done by Aravind et al.[42]Sheetu MK et.al[16]. organoposphorous is commonly consumed poison due to its easy availability and most of the people who consume are farmers. But study done by Patil. A et al [13] household products are major componds used for poison followed by pesticides. In M. Shoaib Zaheer et al.[65]Aluminium phosphide was the commonest poison consumed followed by zinc phosphide and organophosphorus compounds.

**Association between Type of Poisoning and symptoms among subjects (Table 4)**

It was observed that 52% of acid poisoning, 60% of Alkali poisoning, 40% of Insecticide and pesticide poisoning, 50% of plant poisoning, 37% of Tablet poisoning and 43% of miscellaneous poisoning had no symptoms. GI system was the most common system involved in all types of poisoning, CVS was involved in Insecticide and pesticide poisoning and miscellaneous poisoning. CNS, RS and Multisystem are involved mainly in Insecticide and pesticide poisoning.

**Association between Type of Poisoning and symptoms among subjects ( Table 5)**

Metabolic acidosis, Mixed ABG and Acute respiratory acidosis was commonly seen in Insecticide and pesticide poisoning. This association was statistically significant. This is in correlation with op compound study done by Asari M[65]

**Association between RBS among subjects: (Table 8)**

In the study RBS was increased in all most all the poisoning. There was significant association between RBS levels and type of poisoning. SHAHIN S et.al[66] noted hyperglycemia at the time of presentation in his study. Hyperglycemia in acute OPC poisoning has been reported many times in literature.[67]

**Association between PSS and Outcome (Table 15)**

There was significant association between PSS score and Outcome in Poisoning. I.e. among patients with PSS score of death 94.7% died in the study. 40.9% of Severe PSS score had complications. Hence PSS can be a useful tool for predicting the outcome in Poisoning subjects.

**Association between Type of poisoning and Intubation (Table 13)**

In the study intubation was done in 55% of insecticide poisoning and 13% of tablet poisoning. No intubation was done for other poisoning. This observation was statistically significant. Study done by Aravind et.al [42]showed OP compounds (23%) and Drug over dosage were the most common poisonings that required critical care management.

**Association between Type of poisoning and Outcome (Table 14)**

In the study death occurred in 22.7% of insecticide poisoning, 15% of miscellaneous poisoning, 4% in acid poisoning and 1.2% in tablet poisoning. Majority of poisoning had high recovery rate. This was statistically significant. This is significant to study done by Aravind et al[42] where 78.72% recovered and 10% of them died.

**Correlation between Elevated CKMB, Positive Troponin, ECG Changes, CPK with Outcome of the Patient (Table 17, 18, 21, 22).**

It was observed that 40% of subjects with complications, 37.8% of subjects with mortality and 28.7% who recovered had increased CKMB levels. This observation was not significantly associated. CK-MB in the study done by Pore NE et.al[69] in OPC poisoning were also found to be elevated

It was observed that among Trop T positive subjects 7.7% died and among Trop T negative subjects 12.8% was the mortality rate. But significant association was not observed between these two variables

Among 37 deaths 15.4% had no ECG changes, 13.3% had extra systole, 12% had sinus bradycardia, 10.5% had Sinus tachycardia and 7.5% had Bundle branch blocks. But there was no significant association.

It was observed that 46.7% of subjects with complications, 37.8% of subjects who died and 31% who recovered had increased CPK levels. This observation was not statistically significant. CPK in the study done by Pore NE et.al[69] in OPC poisoning were also found to be elevated

**Association between RBS and Outcome among poisoning subjects (Table 20)**

Among 37 patients who died 51.4% had raised RBS levels and 80% of subjects with complication had increased RBS level. This observation was not statistically significant. In the study done by Pore NE et.al[69] in OPC poisoning elevation of blood glucose concentration is noted due to accumulation of acetylcholine in the adrenals following inactivation of cholinesterase by the insecticides which stimulate the release of adrenaline into the blood, adrenaline

increases cell metabolism; it causes glycogenolysis in the liver and a consequent hyperglycemia.

**Association between Intubation and Outcome (Table 19)**

It was observed that among the patients who were intubated the mortality was high i.e. 24.4% and among non-intubated patients mortality was 7.5%. Similarly complication rate was also high in intubated patients. This observation was statistically significant. Most of the patients intubated are insecticide and pesticide group (OPC poison consumption) and mortality was also high in this group in our study. This is similar to study done by Singh B et.al[70]

**Poison detected by Chemical analysis among subjects who had mortality (Table 25)**

In our study out of 37 deaths, 32 (86.5%) of cases detected by chemical analysis, (70.3%) were positive for organophosphorus, (70.3%), phosphide (10.8%), lastly phenol and oleander (2.7%) each. These findings were correlating with history of poisoning.

The reasons for negative chemical analysis report in (13.5%) are due to the contributing predisposing disease of the victim or small quantity of poison intake causing difficulties to detect or due to faulty preservation and technique, poison vomited out, treatment undergone or the poison could have been detoxified, neutralized, conjugated or eliminated from the body causing difficulties in analytical procedures.

**Post mortem Stomach Mucosal appearances in various fatal poisoning cases (Table 24)**

Out of 29 cases (100%) of insecticides and pesticide poisoning stomach mucosa was congested for 15 (51.8%) patients, hemorrhagic for 4 (13.8%), erosive for 5(17.2%) and edematous for 5 (17.2%).

Organoposphorous poisoning mucosa of most of the patients were congested followed by hemorrhagic and erosive. Edematous mucosa was seen in aluminum phosphide cases. Similar autopsy

Features seen in Kishan RS et.al[15]

**Psychiatric evaluation:**

Depression is one of the major causes for suicidal deaths. Farmers and unemployed persons were more prone to death by poisoning in the present study. This is so because larger segment of our population comes from these groups

Suicide attempts among adults especially in age group of 21–30 years could be due to lack of employment, break-up in the family support system, failure of love affaire, inadequacy to cope with some immediate situation, impulsive behaviors, stress due to job and family, etc Early marriages, low education, poverty, marital problems, were the major factors which led females commit suicide.

## CONCLUSION

- Acute poisonings is one of the most common medical emergencies encountered in our hospital.
- The mean age at presentation was around 34 years.
- The incidence among women and men are 1.13:1 the reason being suicidal.
- The most common poison group is insecticide and pesticide, but there is also increase in consumptions of other group of poisons such as drugs, miscellaneous products.
- In the study death occurred in 22.7% of insecticide poisoning, 15% of miscellaneous poisoning (paraquat most common) 4% in acid poisoning and 1.2% in tablet poisoning. Majority of poisoning had high recovery rate.
- Equal importance should be given newer group of poisons as there is increase in mortality
- There was significant association between PSS score and Outcome in Poisoning. I.e. among patients with PSS score of death 94.7% died in the study. 40.9% of Severe PSS score had complications. Hence PSS can be a useful tool for predicting the outcome in Poisoning subjects
- Psychiatric evaluation done for poisoned patients and poverty is most common followed by unemployment in males, in females also poverty is primary cause followed by marital problems and family issues.

# SUMMARY

A total of 310 patients were studied over a period of one year. Majority of the patients were in the age group of 21-30 years (37.7%) followed by 31-40years (26%). 53.2% of the patients were females and the female to male ratio was 1.13:1.All were suicidal poisonings.

Insecticides and pesticides (41.3%) were the most common group of poison consumed in our study. Followed by tablet consumption (26.5%) and miscellaneous group (12.5%). GI system was the most common system involved in all types of poisoning, CVS was involved in Insecticide and pesticide poisoning and miscellaneous poisoning. CNS, RS and Multisystem are involved mainly in Insecticide and pesticide poisoning.

Poison severity score showed Majority i.e. 36% had normal score, 29% had minor score, 15.5% had moderate score, 12.3% had mortality, and 7.1% had severe score. There was statistically significant association between different types of poisoning and PSS. Severity (8.6%) and death (23.4%) was high among insecticide poisoning followed by miscellaneous group.

In our study intubation was done in 55% of insecticide poisoning and 13% of tablet poisoning. On admission and this was found to be statistically significant. Among the patients who were intubated the mortality was high i.e. 24.4% and among non-intubated patients mortality was 7.5%. Similarly complication rate was also high in intubated patients. This observation was statistically significant.

In our study death occurred in 22.7% of insecticide poisoning, 15% of miscellaneous poisoning, 4% in acid poisoning and 1.2% in tablet poisoning. Majority of poisoning had high recovery rate. This was statistically significant..

There was significant association between PSS score and Outcome in Poisoning. I.e. among patients with PSS score of death 94.7% died in the study. 40.9% of Severe PSS score had complications. Hence PSS can be a useful tool for predicting the outcome in Poisoning subjects

In our study out of 37 deaths, 32 (86.5%) of cases detected by chemical analysis, (70.3%) were positive for organophosphorus, (70.3%), phosphide (10.8%), lastly phenol and oleander (2.7%) each. These findings were correlating with history of poisoning.

Psychiatric evaluation done for poisoned patients and poverty is most common followed by unemployment in males, in females also poverty is primary cause followed by marital problems and family issues.

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# ANNEUXRES

# PROFORMA

## (A) IDENTIFICATION DETAILS

**SERIAL NO:**

NAME:

AGE/SEX: HOSP NO. :

ADDRESS: OCCUPATION:

DATE & TIME OF ADMISSION:

## (B) PRESENTING COMPLAINTS:

- NAME OF COMPOUND :
- MODE OF EXPOSURE:
- DATE & TIME OF EXPOSURE:
- INTERVAL BETWEEN EXPOSURE AND INITIATION OF THERAPY (hrs):
- IF INGESTED, RAW INGESTION OR WITH: ALCOHOL / WATER / KEROSENE / AERATED DRINK / Others:
- H/O : VOMITTING / SEIZURES / TREMORS / ALTERED SENSORIUM / DYSPNOEA Others (specify) -

## (C) PAST H/O

- DM / HTN / IHD / PTB / COPD / BA / CLD / Others.....
- H/O previous suicidal attempts – YES / NO; If YES, details -

## (D) PERSONAL H/O

- SMOKING / ALCOHOL / Pan chewing / IVDA /_____
- BOWEL / BLADDER HABITS-
- Other habits:

## (E) PHYSICAL FINDINGS

- PSS Score :
- VITAL SIGNS:
  - Pulse rate & rhythm:
  - BP: _____mm Hg in Right upper limb, supine
  - Respiratory rate & type:
  - Temperature:
- Pupils:
- Secretions : YES / NO
- Bowel incontinence – YES / NO
- Bladder incontinence – YES / NO

## (F)SYSTEMIC EXAMINATION*:*

| ORGAN | NONE | MINOR | MODERATE | SEVERE |
|---|---|---|---|---|
| | 0 | 1 | 2 | 3 |
| | No symptoms or signs | Mild, transient, and spontaneously resolving symptoms or signs | Pronounced or prolonged symptoms or signs | Severe or life-threatening symptoms or signs |
| GI-tract | | • Vomiting, diarrhea, pain | • Pronounced or prolonged vomiting, diarrhea, pain ileus | • Massive hemorrhage, perforation |
| | | • Irritation, 1st degree burns, minimal ulcerations in the mouth | • 1st degree burns of critical localization or 2nd and 3rd degree burns in restricted areas<br>• Dysphagia | • More widespread 2nd and 3rd degree burns<br>• Severe dysphagia |
| | | • Endoscopy: Erythema, edema | • Endoscopy: Ulcerative transmucosal lesions | • Endoscopy: Ulcerative transmural lesions, circumferential lesions, perforation |
| Respiratory system | | • Irritation, coughing, breathlessness, mild dyspnea, mild bronchospasm | • Prolonged coughing, bronchospasm, dyspnea, stridor, hypoxemia requiring extra oxygen | • Manifest respiratory insufficiency (e.g., severe bronchospasm, airway obstruction, glottal edema, pulmonary edema, ARDS, pneumonitis, pneumonia, pneumothorax) |
| | | • Chest X ray: Abnormal with minor or no symptoms | • Chest X ray: Abnormal with moderate symptoms | • Chest X ray: Abnormal with severe symptoms |
| Nervous system | | • Drowsiness, vertigo, tinnitus, ataxia | • Unconsciousness with appropriate response to pain | • Deep coma with inappropriate response to pain or unresponsive to pain |
| | | | • Brief apnea, bradypnea | • Respiratory depression with insufficiency |
| | | • Restlessness | • Confusion, agitation, hallucinations, delirium | • Extreme agitation |
| | | | • Infrequent, generalized, or local seizures | • Frequent, generalized seizures, status epilepticus, opisthotonos |
| | | • Mild extrapyramidal symptoms | • Pronounced extrapyramidal symptoms | |
| | | • Mild cholinergic/anticholinergic symptoms | • Pronounced cholinergic/anticholinergic symptoms | |
| | | • Paresthesia | • Localized paralysis not affecting vital functions | • Generalized paralysis or paralysis affecting vital functions |
| | | • Mild visual or auditory disturbances | • Visual and auditory disturbances | • Blindness, deafness |

| | | | | |
|---|---|---|---|---|
| Cardio-vascular system | | | • Sinus bradycardia (HR~40-50 in adults, 60-80 in infants and children, 80-90 in neonates) | • Severe sinus bradycardia (HR~<40 in adults, <60 in infants, <80 in neonates) |
| | | | • Sinus tachycardia (HR~140-180 in adults, 160-190 in infants and children, 160-200 in neonates) | • Severe sinus tachycardia (HR~>180 in adults, > 190 in infants and children, >200 in neonates) |
| | | • Isolated extrasystoles | • Frequent extrasystoles, atrial fibrillation/flutter, AV-block Ⅰ-Ⅱ, prolonged QRS and QTc-time, repolarization abnormalities | • Life-threatening ventricular dysrythmias, AV-block Ⅲ, asystole |
| | | | • Myocardial ischemia | • Myocardial infarction |
| | | • Mild and transient hypo/hypertension | • More pronounced hypo/hypertension | • Shock, hypertensive crisis |

| | | | | |
|---|---|---|---|---|
| Metabolic balance | | • Mild acid–base disturbances ($HCO_3^-$ ～15–20 or 30–40 mmol/L, pH～7.25–7.32 or 7.50–7.59)<br>• Mild electrolyte and fluid disturbances ($K^+$ 3.0–3.4 or 5.2–5.9 mmol/L)<br>• Mild hypoglycemia (～50–70 mg/dL or 2.8–3.9 mmol/L in adults)<br>• Hyperthermia of short duration | • More pronounced acid–base disturbances ($HCO_3^-$ ～10–14 or >40 mmol/L, pH～7.15–7.24 or 7.60–7.69)<br>• More pronounced electrolyte and fluid disturbances ($K^+$ 2.5–2.9 or 6.0–6.9 mmol/L)<br>• More pronounced hypoglycemia (～30–50 mg/dL or 1.7–2.8 mmol/L in adults)<br>• Hyperthermia of longer duration | • Severe acid–base disturbances ($HCO_3^-$ ～<10 mmol/L, pH ～<7.15 or >7.7)<br>• Severe electrolyte and fluid disturbances ($K^+$ <2.5 or >7.0 mmol/L)<br>• Severe hypoglycemia (～<30 mg/dL or 1.7 mmol/L in adults)<br>• Dangerous hypo– or hyperthermia |
| Liver | | • Minimal rise in serum enzymes (AST, ALT ～2–5 × normal) | • Rise in serum enzymes (AST, ALT ～5–50 × normal) but no diagnostic biochemical (e.g., ammonia, clotting factors) or clinical evidence of liver dysfunction | • Rise in serum enzymes (～>50 × normal) or biochemical (e.g., ammonia, clotting factors) or clinical evidence of liver failure |
| Kidney | | • Minimal proteinuria/hematuria | • Massive proteinuria/hematuria<br>• Renal dysfunction (e.g., oliguria, polyuria, serum creatinine of ～200–500 μmol/L) | • Renal failure (e.g., anuria, serum creatinine of >500 μmol/L) |

| | | | | |
|---|---|---|---|---|
| Blood | | • Mild hemolysis<br>• Mild methemoglobinemia (metHb ～10–30%) | • Hemolysis<br>• More pronounced methemoglobinemia (metHb ～30–50%)<br>• Coagulation disturbances without bleeding<br>• Anemia, leucopenia, thrombocytopenia | • Massive hemolysis<br>• Severe methemoglobinemia (metHb > 50%)<br>• Coagulation disturbances with bleeding<br>• Severe anemia, leucopenia, thrombocytopenia |
| Muscular system | | • Mild pain, tenderness<br>• CPK ～250–1500 IU/L | • Pain, rigidity, cramping, and fasciculations<br>• Rhabdomyolysis, CPK ～1500–10,000 IU/L | • Intense pain, extreme rigidity, extensive cramping, and fasciculations<br>• Rhabdomyolysis with complications, CPK ～> 10,000 IU/L<br>• Compartment syndrome |
| Local effects on skin | | • Irritation, 1st degree burns (reddening) or 2nd degree burns in <10% body surface | • 2nd degree burns in 10–50% of body surface (children: 10–30%) or 3rd degree burns in <2% of body surface | • 2nd degree burns in >50% of body surface (children: >30%) or 3rd degree burns in >2% of body surface |
| Local effects on eye | | • Irritation, redness, lacrimation, mild palpebral edema | • Intense irritation, corneal abrasion<br>• Minor (punctate) corneal ulcers | • Corneal ulcers (other than punctate), perforation<br>• Permanent damage |

## (G) INVESTIGATIONS

| **DATE** | | | |
|---|---|---|---|
| HB (gm %) | | T. PROTEIN | |
| TC | | ALBUMIN | |
| DC(N,L,E) | | BILIRUBIN | |
| ESR | | SGOT | |
| PLATELET COUNT | | SGPT | |
| HCT | | PSEUDO CHOLINESTERASE | |
| BT | | **ABG** | |
| CT | | | |
| PT | | PH | |
| INR | | PCO2 | |

| | | | |
|---|---|---|---|
| APTT | | PO2 | |
| | | HCO3 | |
| RBS | | SO2 | |
| SODIUM | | | |
| POTASIUM | | CPK | |
| S. AMYLASE | | CKMB | |
| PCHE | | TROP T | |
| HBsAG | | | |
| HIV | | Urine R/E | |
| | | | |
| CHEST X RAY | | | |
| ECG | | | |

Relevant PM findings (IN CASE OF DEATH)

Sent to FSL:

Chemical analysis report:

Cause of death:

SRI DEVARAJ URS MEDICAL COLLEGE & RESEARCH CENTRE,

TAMAKA, KOLAR

**PATIENT CONSENT FORM**

Case no

Title: "**STUDY OF PATTERN OF POISONINGS AND THEIR OUTCOME AMONG IN-PATIENTS ADMITTED IN A RURAL TERTIARY CARE CENTRE"**

Name of the investigator:

Name of the participant: _____

I _____d/o,w/o_____ give my full, free and voluntary consent to participate in the study entitled "**STUDY OF PATTERN OF POISONINGS AND THEIR OUTCOME AMONG IN-PATIENTS ADMITTED IN A RURAL TERTIARY CARE CENTRE**." I have read (or it has been read to me) and understood this consent form. I have understood that I have the right to refuse consent or withdraw it at any time during the study and this will not affect my treatment in any way. I was free to ask questions and undergo examination and they have been answered to my satisfaction. I have been explained about the intent of the study.

Signature / Thumb impression of the

Participant Name: _____ Date: _____ Time: _____

Researcher to Complete I …… certify that I have explained the nature and procedures of the research project to …… and consider that she/he understands what is involved.

Signed …….. Date ……..

Name and Address of Principal Investigator:

# KEY TO MASTER CHART

| | |
|---|---|
| P | patient |
| INSC and PEST | insecticides and pesticides |
| MISC | miscellaneous |
| RS SYMP | respiratory system |
| GI SYM | gastrointestinal symptoms. |
| MULTI SYSM | multisystem |
| CVS SYM | cardiovascular system |
| CNS SYM | central nervous system |
| RBS | random blood sugar |
| ABG | arterial blood gas, |
| RESP ALK | respiratory alkalosis |
| MET ACID | metabolic acidosis |
| CPK | creatine phosphokinase |
| TROP T | troponin T |
| CKMB | creatine kinase MB |
| ECG | electrocardiogram |
| S.BRANDY | sinus bradycardia |
| NSR | normal sinus rhythm |
| S.TACHY | sinus tachycardia |
| PCHE | pseudocholinesterase |
| PSS | poisoning severity score, |
| Hrs | hours |

# MASTER CHART

| SL NO | AGE | SEX | TYPE OF POISON | SYMPTOMS | DEATH | RBS | ABG | CPK | TROP T | CKMB | ECG | PCHE | PSS | INTUBATION | INTERVAL Hrs | OUTCOME |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| P1 | 40 | M | insc and pest | RS SYMP | | 70 | MIXED RESP ALK + MET ACID | 1045 | N | 33 | S.BRADY | 2129 | NONE | NO | <1 | RECOVERY |
| P2 | 30 | F | insc and pest | RS SYMP | | 65 | MIXED RESP ALK + MET ACID | 102 | N | 21 | BUNDLE BRANCH BLOCKS | 5650 | MODERATE | YES | 4 - 5 | RECOVERY |
| P3 | 19 | M | insc and pest | NO | | 74 | MIXED RESP ALK + MET ACID | 201 | N | 29 | NSR | 2434 | MINOR | YES | 2 -3 | RECOVERY |
| P4 | 32 | M | insc and pest | GI SYM | | 112 | METABOLIC ACIDOSIS | 288 | P | 58 | EXTRA SYSTOLE | 2358 | MINOR | NO | <1 | RECOVERY |
| P5 | 30 | M | insc and pest | MULTI SYSM | YES | 49 | MIXED RESP ALK + MET ACID | 206 | N | 30 | S.TACHY | 2973 | DEATH | NO | 4 - 5 | DEATH |
| P6 | 22 | M | insc and pest | GI SYM | | 69 | ACUTE RESPIRATORY ACIDOSIS | 146 | N | 16 | NSR | 5183 | NONE | NO | 1 -2 | RECOVERY |
| P7 | 21 | M | insc and pest | RS SYMPH | | 125 | METABOLIC ACIDOSIS | 199 | N | 33 | NSR | 200 | MODERATE | YES | > 5 | RECOVERY |
| P8 | 28 | M | insc and pest | MULTI SYSM | YES | 64 | METABOLIC ACIDOSIS | 150 | N | 40 | S. TACHY | 360 | DEATH | YES | > 5 | RECOVERY |
| P9 | 35 | M | insc and pest | MULTI SYSM | | 43 | ACUTE RESPIRATORY ACIDOSIS | 146 | P | 200 | NSR | 480 | MINOR | YES | 2 -3 | DEATH |
| P10 | 26 | F | insc and pest | NO | | 100 | MIXED RESP ALK + MET ACID | 143 | N | 29 | NSR | 200 | MINOR | NO | <1 | RECOVERY |
| P11 | 28 | M | insc and pest | GI SYM | | 65 | NORMAL ABG | 767 | P | 35 | S. TACHY | 823 | MODERATE | YES | 4 - 5 | RECOVERY |
| P12 | 21 | M | insc and pest | RS SYMPH | | 69 | MIXED RESP ALK + MET ACID | 423 | N | 16 | BUNDLE BRANCH BLOCKS | 2620 | NONE | NO | 4 - 5 | RECOVERY |
| P13 | 18 | M | insc and pest | MULTI SYSM | | 106 | MIXED RESP ALK + MET ACID | 135 | N | 20 | NSR | 370 | NONE | NO | 3 - 4 | RECOVERY |
| P14 | 30 | F | insc and pest | MULTI SYSM | YES | 61 | METABOLIC ACIDOSIS | 159 | N | 16 | NSR | 200 | DEATH | YES | > 5 | RECOVERY |
| P15 | 25 | F | insc and pest | GI SYMPH | | 95 | MIXED RESP ALK + MET ACID | 54 | N | 60 | NSR | 1775 | NONE | NO | > 5 | RECOVERY |
| P16 | 20 | F | insc and pest | NO | | 86 | ACUTE RESPIRATORY ACIDOSIS | 80 | N | 31 | NSR | 347 | NONE | NO | 2 -3 | RECOVERY |
| P17 | 25 | M | insc and pest | NO | | 89 | MIXED RESP ALK + MET ACID | 141 | N | 28 | NSR | 1425 | MODERATE | YES | 4 - 5 | RECOVERY |
| P18 | 18 | M | insc and pest | GI SYM | | 100 | NORMAL ABG | 240 | N | 45 | NSR | 456 | MINOR | NO | 4 - 5 | RECOVERY |
| P19 | 48 | M | insc and pest | GI SYMPH | YES | 96 | METABOLIC ACIDOSIS | 400 | N | 46 | S. TACHY | 261 | DEATH | NO | 1 -2 | DEATH |
| P20 | 23 | M | insc and pest | NO | | 128 | MIXED RESP ALK + MET ACID | 891 | N | 46 | S. TACHY | 128 | MINOR | NO | 2 -3 | RECOVERY |
| P21 | 25 | M | insc and pest | MULTI SYSM | | 158 | ACUTE RESPIRATORY ACIDOSIS | 133 | N | 35 | NSR | 1276 | MODERATE | YES | <1 | RECOVERY |
| P22 | 22 | M | insc and pest | GI SYM | | 96 | METABOLIC ACIDOSIS | 122 | N | 24 | NSR | 648 | MINOR | NO | 4 - 5 | RECOVERY |
| P23 | 60 | M | insc and pest | CVS SYM | YES | 89 | ACUTE RESPIRATORY ACIDOSIS | 210 | P | 92 | NSR | 2495 | DEATH | YES | 3 - 4 | DEATH |
| P24 | 50 | M | insc and pest | NO | | 155 | RESP ACIDOSIS | 207 | P | 147 | S. TACHY | 200 | MINOR | YES | <1 | RECOVERY |
| P25 | 18 | F | insc and pest | NO | | 117 | METABOLIC ACIDOSIS | 97 | N | 10 | S. TACHY | 200 | SEVERE | YES | > 5 | RECOVERY |
| P26 | 19 | F | insc and pest | CVS SYM | | 95 | METABOLIC ACIDOSIS | 159 | N | 30 | S. TACHY | 503 | NONE | NO | > 5 | RECOVERY |
| P27 | 20 | F | insc and pest | GI SYM | | 154 | METABOLIC ACIDOSIS | 137 | P | 136 | S. TACHY | 326 | NONE | NO | > 5 | RECOVERY |
| P28 | 20 | F | insc and pest | RS SYMPH | | 179 | ACUTE RESPIRATORY ACIDOSIS | 162 | N | 26 | S. TACHY | 200 | NONE | NO | 1 -2 | RECOVERY |
| P29 | 20 | M | insc and pest | GI SYMPH | | 98 | MIXED RESP ALK + MET ACID | 1470 | N | 92 | NSR | 1581 | MODERATE | YES | > 5 | RECOVERY |
| P30 | 22 | M | insc and pest | no | | 244 | METABOLIC ACIDOSIS | 124 | N | 23 | NSR | 4216 | NONE | YES | > 5 | RECOVERY |
| P31 | 25 | F | insc and pest | NO | | 80 | ACUTE RESPIRATORY ACIDOSIS | 445 | N | 14 | NSR | 6576 | MINOR | YES | > 5 | RECOVERY |
| P32 | 55 | M | insc and pest | GI SYMPH | YES | 112 | METABOLIC ACIDOSIS | 303 | N | 40 | NSR | 4450 | DEATH | YES | <1 | DEATH |
| P33 | 32 | F | insc and pest | CNS SYM | YES | 132 | METABOLIC ACIDOSIS | 561 | N | 34 | NSR | 200 | DEATH | YES | 4 - 5 | DEATH |
| P34 | 22 | M | insc and pest | NO | | 93 | METABOLIC ACIDOSIS | 57 | N | 73 | NSR | 200 | MINOR | YES | 4 - 5 | RECOVERY |

# MASTER CHART

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|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| P35 | 25 | F | insc and pest | NO | | 247 | METABOLIC ACIDOSIS | 502 | N | 31 | S. TACHY | 200 | SEVERE | YES | 2 -3 | RECOVERY |
| P36 | 40 | M | insc and pest | CNS SYM | | 82 | NORMAL ABG | 194 | N | 34 | S. TACHY | 200 | NONE | NO | > 5 | RECOVERY |
| P37 | 26 | F | insc and pest | NO | | 101 | ACUTE RESPIRATORY ACIDOSIS | 1149 | N | 47 | NSR | 5799 | MODERATE | YES | 1 -2 | COMPLICATIONS |
| P38 | 30 | M | insc and pest | NO | | 106 | METABOLIC ACIDOSIS | 107 | N | 26 | NSR | 4048 | NONE | YES | > 5 | RECOVERY |
| P39 | 35 | M | insc and pest | NO | | 111 | MIXED RESP ALK + MET ACID | 1367 | P | 62 | NSR | 1145 | MINOR | YES | 2 -3 | RECOVERY |
| P40 | 23 | M | insc and pest | NO | | 92 | METABOLIC ACIDOSIS | 365 | N | 27 | NSR | 1021 | MODERATE | YES | > 5 | RECOVERY |
| P41 | 25 | M | insc and pest | CNS SYMP | YES | 159 | ACUTE RESPIRATORY ACIDOSIS | 46 | N | 34 | NSR | 200 | DEATH | YES | 3 - 4 | DEATH |
| P42 | 30 | M | insc and pest | no | | 94 | ACUTE RESPIRATORY ACIDOSIS | 277 | N | 19 | NSR | 477 | NONE | YES | 4 - 5 | RECOVERY |
| P43 | 24 | M | insc and pest | CVS SYM | | 126 | MIXED RESP ALK + MET ACID | 96 | N | 28 | NSR | 3496 | MODERATE | YES | <1 | RECOVERY |
| P44 | 28 | M | insc and pest | NO | | 82 | METABOLIC ACIDOSIS | 122 | N | 22 | S. TACHY | 13000 | NONE | NO | > 5 | RECOVERY |
| P45 | 18 | M | insc and pest | CNS SYM | | 99 | ACUTE RESPIRATORY ACIDOSIS | 856 | N | 28 | NSR | 1103 | NONE | NO | 3 - 4 | RECOVERY |
| P46 | 50 | F | insc and pest | rs symp | YES | 86 | MIXED RESP ALK + MET ACID | 242 | N | 19 | S. TACHY | 381 | DEATH | NO | 4 - 5 | DEATH |
| P47 | 18 | F | insc and pest | CVS SYM | | 88 | MIXED RESP ALK + MET ACID | 202 | N | 15 | NSR | 4052 | NONE | NO | 4 - 5 | RECOVERY |
| P48 | 65 | F | insc and pest | GI SYM | | 345 | MIXED RESP ALK + MET ACID | 1600 | P | 126 | S. TACHY | 7387 | SEVERE | YES | <1 | RECOVERY |
| P49 | 27 | M | insc and pest | CNS SYMP | | 196 | ACUTE RESPIRATORY ACIDOSIS | 123 | N | 44 | S. TACHY | 219 | MINOR | NO | 1 -2 | RECOVERY |
| P50 | 21 | F | insc and pest | CVS SYM | YES | 118 | METABOLIC ACIDOSIS | 284 | N | 12 | NSR | 200 | DEATH | NO | 2 -3 | DEATH |
| P51 | 20 | M | insc and pest | NO | | 159 | METABOLIC ACIDOSIS | 190 | N | 16 | NSR | 200 | MINOR | NO | 1 -2 | RECOVERY |
| P52 | 33 | M | insc and pest | CNS SYMP | | 187 | MIXED RESP ALK + MET ACID | 136 | P | 126 | S. TACHY | 200 | MODERATE | YES | > 5 | RECOVERY |
| P53 | 20 | M | insc and pest | GI SYM | | 88 | ACUTE RESPIRATORY ACIDOSIS | 165 | N | 18 | NSR | 691 | DEATH | NO | > 5 | DEATH |
| P54 | 54 | F | insc and pest | CVS SYM | YES | 88 | MIXED RESP ALK + MET ACID | 54 | N | 37 | NSR | 985 | MINOR | NO | 3 - 4 | RECOVERY |
| P55 | 18 | M | insc and pest | GI SYMPH | | 299 | MIXED RESP ALK + MET ACID | 837 | P | 83 | S. TACHY | 299 | MINOR | NO | <1 | RECOVERY |
| P56 | 45 | M | insc and pest | RS SYMPH | | 131 | METABOLIC ACIDOSIS | 148 | N | 27 | NSR | 6645 | MINOR | NO | > 5 | RECOVERY |
| P57 | 26 | M | insc and pest | CNS SYMP | YES | 136 | METABOLIC ACIDOSIS | 1439 | N | 27 | NSR | 447 | DEATH | NO | > 5 | DEATH |
| P58 | 27 | M | insc and pest | CVS SYM | | 96 | METABOLIC ACIDOSIS | 600 | P | 164 | S. TACHY | 864 | MODERATE | YES | > 5 | RECOVERY |
| P59 | 35 | M | insc and pest | GI SYMPH | YES | 120 | METABOLIC ACIDOSIS | 265 | N | 15 | NSR | 476 | DEATH | YES | 3 - 4 | DEATH |
| P60 | 27 | F | insc and pest | NO | | 100 | METABOLIC ACIDOSIS | 463 | N | 22 | NSR | 2600 | NONE | NO | 4 - 5 | RECOVERY |
| P61 | 64 | M | insc and pest | MULTI SYSM | YES | 136 | NORMAL ABG | 152 | N | 14 | S. TACHY | | DEATH | NO | 4 - 5 | DEATH |
| P62 | 72 | M | TABLETS | NO | | 246 | NORMAL ABG | 251 | P | 15 | S. TACHY | | SEVERE | YES | <1 | RECOVERY |
| P63 | 18 | M | TABLETS | GI SYM | | 245 | ACUTE RESPIRATORY ACIDOSIS | 256 | P | 17 | S. TACHY | | MINOR | NO | 2 -3 | RECOVERY |
| P64 | 45 | M | TABLETS | CNS SYMP | | 128 | METABOLIC ACIDOSIS | 285 | P | 16 | S. TACHY | | NONE | NO | 1 -2 | RECOVERY |
| P65 | 51 | F | TABLETS | RS SYMPH | | 156 | NORMAL ABG | 245 | N | 16 | S.BRADY | | NONE | NO | 4 - 5 | RECOVERY |
| P66 | 52 | M | TABLETS | GI SYM | | 157 | METABOLIC ACIDOSIS | 236 | N | 15 | NSR | | NONE | NO | 4 - 5 | RECOVERY |
| P67 | 59 | M | TABLETS | GI SYM | | 189 | NORMAL ABG | 145 | N | 15 | EXTRA SYSTOLE | | DEATH | YES | 4 - 5 | DEATH |
| P68 | 42 | M | TABLETS | GI SYM | | 112 | NORMAL ABG | 152 | N | 14 | NSR | | NONE | NO | 4 - 5 | RECOVERY |
| P69 | 46 | F | TABLETS | GI SYM | | 128 | NORMAL ABG | 152 | N | 24 | NSR | | MINOR | NO | 3 - 4 | RECOVERY |
| P70 | 65 | M | TABLETS | CNS SYMP | | 119 | NORMAL ABG | 145 | N | 18 | BUNDLE BRANCH BLOCKS | | MODERATE | NO | 4 - 5 | RECOVERY |

# MASTER CHART

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|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| P71 | 41 | F | TABLETS | NO | | 136 | METABOLIC ACIDOSIS | 152 | P | 56 | S.BRADY | 1125 | NONE | NO | 2 -3 | RECOVERY |
| P72 | 42 | M | insc and pest | NO | | 193 | NORMAL ABG | 116 | N | 15 | S.BRADY | 5214 | SEVERE | YES | > 5 | RECOVERY |
| P73 | 74 | F | insc and pest | GI SYMPH | YES | 197 | NORMAL ABG | 169 | N | 15 | S.BRADY | 4859 | DEATH | YES | <1 | DEATH |
| P74 | 64 | F | insc and pest | NO | | 353 | NORMAL ABG | 365 | P | 14 | EXTRA SYSTOLE | 1485 | MODERATE | YES | 4 - 5 | RECOVERY |
| P75 | 62 | M | insc and pest | CNS SYMP | YES | 88 | NORMAL ABG | 95 | N | 13 | NSR | 5475 | DEATH | YES | 4 - 5 | DEATH |
| P76 | 69 | F | insc and pest | GI SYMPH | | 146 | NORMAL ABG | 965 | N | 45 | BUNDLE BRANCH BLOCKS | 8547 | MODERATE | YES | 1 -2 | RECOVERY |
| P77 | 31 | F | insc and pest | GI SYMPH | YES | 96 | METABOLIC ACIDOSIS | 100 | N | 9 | BUNDLE BRANCH BLOCKS | 6548 | DEATH | YES | > 5 | DEATH |
| P78 | 19 | M | insc and pest | NO | | 183 | NORMAL ABG | 123 | P | 78 | BUNDLE BRANCH BLOCKS | 1587 | MODERATE | YES | > 5 | RECOVERY |
| P79 | 21 | F | insc and pest | NO | | 173 | NORMAL ABG | 896 | N | 10 | S.BRADY | 1475 | MINOR | NO | 3 - 4 | RECOVERY |
| P80 | 28 | F | ACIDS | NO | | 79 | METABOLIC ACIDOSIS | 158 | N | 9 | S.BRADY | | MODERATE | NO | > 5 | RECOVERY |
| P81 | 24 | M | ACIDS | GI SYM | | 199 | NORMAL ABG | 189 | N | 11 | EXTRA SYSTOLE | | DEATH | NO | 2 -3 | DEATH |
| P82 | 60 | F | ACIDS | GI SYM | | 245 | NORMAL ABG | 190 | N | 16 | S. TACHY | | NONE | NO | 1 -2 | RECOVERY |
| P83 | 23 | F | ACIDS | GI SYM | | 135 | NORMAL ABG | 145 | N | 14 | S. TACHY | | SEVERE | NO | > 5 | COMPLICATIONS |
| P84 | 64 | M | ACIDS | NO | | 186 | NORMAL ABG | 165 | N | 18 | S. TACHY | | NONE | NO | <1 | RECOVERY |
| P85 | 31 | F | ALKALIS | NO | | 197 | ACUTE RESPIRATORY ACIDOSIS | 526 | N | 15 | S. TACHY | | NONE | NO | 4 - 5 | RECOVERY |
| P86 | 22 | M | ALKALIS | GI SYM | | 128 | METABOLIC ACIDOSIS | 185 | N | 12 | S. TACHY | | NONE | NO | 4 - 5 | RECOVERY |
| P87 | 19 | F | ALKALIS | GI SYM | | 193 | NORMAL ABG | 175 | N | 14 | S. TACHY | | NONE | NO | 3 - 4 | RECOVERY |
| P88 | 30 | F | ALKALIS | NO | | 191 | NORMAL ABG | 154 | N | 14 | S. TACHY | | MINOR | NO | > 5 | RECOVERY |
| P89 | 41 | M | ALKALIS | NO | | 158 | NORMAL ABG | 456 | N | 65 | S. TACHY | | MINOR | NO | 2 -3 | RECOVERY |
| P90 | 58 | F | ALKALIS | GI SYM | | 165 | NORMAL ABG | 165 | N | 7 | EXTRA SYSTOLE | | MODERATE | NO | > 5 | RECOVERY |
| P91 | 30 | F | ALKALIS | GI SYM | | 175 | MIXED RESP ALK + MET ACID | 145 | P | 58 | NSR | | MINOR | NO | > 5 | RECOVERY |
| P92 | 31 | M | ALKALIS | NO | | 185 | NORMAL ABG | 124 | N | 21 | NSR | | MINOR | NO | > 5 | RECOVERY |
| P93 | 25 | F | ALKALIS | NO | | 195 | NORMAL ABG | 185 | N | 4 | NSR | | NONE | NO | > 5 | RECOVERY |
| P94 | 47 | F | ALKALIS | NO | | 189 | METABOLIC ACIDOSIS | 165 | P | 25 | S.BRADY | | MINOR | NO | 1 -2 | RECOVERY |
| P95 | 22 | M | ACIDS | GI SYM | | 168 | NORMAL ABG | 589 | N | 2 | NSR | | MINOR | NO | 4 - 5 | RECOVERY |
| P96 | 23 | F | ACIDS | GI SYM | | 164 | NORMAL ABG | 125 | N | 13 | NSR | | MODERATE | NO | 4 - 5 | RECOVERY |
| P97 | 56 | M | ACIDS | NO | | 183 | NORMAL ABG | 465 | P | 5 | NSR | | NONE | NO | 2 -3 | RECOVERY |
| P98 | 31 | F | ACIDS | GI SYM | | 126 | NORMAL ABG | 145 | N | 10 | BUNDLE BRANCH BLOCKS | | NONE | NO | <1 | RECOVERY |
| P99 | 22 | M | ACIDS | NO | | 187 | NORMAL ABG | 258 | N | 6 | EXTRA SYSTOLE | | NONE | NO | 4 - 5 | RECOVERY |
| P100 | 50 | F | ACIDS | NO | | 198 | NORMAL ABG | 165 | N | 8 | NSR | | NONE | NO | 3 - 4 | RECOVERY |
| P101 | 29 | M | ACIDS | GI SYM | | 154 | NORMAL ABG | 587 | P | 19 | NSR | | MODERATE | NO | 4 - 5 | RECOVERY |
| P102 | 21 | F | ACIDS | GI SYM | | 124 | NORMAL ABG | 185 | N | 6 | NSR | | MINOR | NO | 2 -3 | RECOVERY |
| P103 | 23 | M | ACIDS | NO | | 165 | METABOLIC ACIDOSIS | 165 | N | 15 | BUNDLE BRANCH BLOCKS | | MODERATE | NO | <1 | RECOVERY |
| P104 | 57 | M | ACIDS | NO | | 112 | NORMAL ABG | 859 | N | 12 | S. TACHY | | MINOR | NO | > 5 | RECOVERY |
| P105 | 21 | M | ACIDS | GI SYM | | 171 | NORMAL ABG | 185 | N | 18 | S. TACHY | | MINOR | NO | > 5 | RECOVERY |
| P106 | 50 | F | ACIDS | NO | | 154 | MIXED RESP ALK + MET ACID | 168 | N | 18 | S. TACHY | | MINOR | NO | 2 -3 | RECOVERY |

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|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| P107 | 31 | M | ACIDS | NO | | 165 | NORMAL ABG | 195 | N | 17 | S. TACHY | | MINOR | NO | 1 -2 | RECOVERY |
| P108 | 29 | M | ACIDS | GI SYM | | 118 | NORMAL ABG | 165 | N | 14 | S. TACHY | | MODERATE | NO | > 5 | RECOVERY |
| P109 | 21 | M | ACIDS | GI SYM | | 165 | METABOLIC ACIDOSIS | 185 | P | 14 | S. TACHY | | MINOR | NO | > 5 | RECOVERY |
| P110 | 29 | M | ACIDS | GI SYM | | 158 | NORMAL ABG | 145 | N | 18 | EXTRA SYSTOLE | | MODERATE | NO | 2 -3 | RECOVERY |
| P111 | 50 | F | ACIDS | NO | | 168 | NORMAL ABG | 175 | N | 16 | S.BRADY | | MINOR | NO | 2 -3 | RECOVERY |
| P112 | 60 | F | ACIDS | NO | | 123 | NORMAL ABG | 135 | P | 15 | BUNDLE BRANCH BLOCKS | | SEVERE | NO | > 5 | RECOVERY |
| P113 | 18 | M | ACIDS | NO | | 165 | METABOLIC ACIDOSIS | 165 | N | 13 | NSR | | MINOR | NO | 3 - 4 | RECOVERY |
| P114 | 30 | M | ACIDS | NO | | 199 | NORMAL ABG | 145 | N | 15 | NSR | | NONE | NO | 4 - 5 | RECOVERY |
| P115 | 32 | M | ALKALIS | GI SYM | | 255 | NORMAL ABG | 185 | N | 14 | NSR | | MODERATE | NO | <1 | RECOVERY |
| P116 | 24 | M | ALKALIS | NO | | 235 | NORMAL ABG | 165 | N | 15 | NSR | | NONE | NO | > 5 | RECOVERY |
| P117 | 49 | M | ALKALIS | NO | | 86 | NORMAL ABG | 125 | P | 16 | BUNDLE BRANCH BLOCKS | | NONE | NO | 3 - 4 | RECOVERY |
| P118 | 32 | F | ALKALIS | NO | | 248 | METABOLIC ACIDOSIS | 569 | N | 15 | S.BRADY | | SEVERE | NO | > 5 | RECOVERY |
| P119 | 60 | F | ALKALIS | GI SYM | | 187 | NORMAL ABG | 258 | N | 15 | EXTRA SYSTOLE | | MODERATE | NO | > 5 | RECOVERY |
| P120 | 24 | M | TABLETS | NO | | 88 | NORMAL ABG | 154 | P | 14 | S. TACHY | | MINOR | NO | <1 | RECOVERY |
| P121 | 23 | F | TABLETS | NO | | 158 | NORMAL ABG | 123 | N | 14 | S. TACHY | | NONE | NO | 4 - 5 | RECOVERY |
| P122 | 49 | M | TABLETS | RS SYMPH | | 98 | NORMAL ABG | 165 | N | 15 | S. TACHY | | NONE | NO | 4 - 5 | RECOVERY |
| P123 | 23 | F | TABLETS | NO | | 165 | NORMAL ABG | 154 | N | 12 | S. TACHY | | MINOR | NO | 1 -2 | RECOVERY |
| P124 | 32 | M | TABLETS | CNS SYMP | | 178 | NORMAL ABG | 654 | N | 14 | S. TACHY | | NONE | NO | 2 -3 | RECOVERY |
| P125 | 22 | F | TABLETS | RS SYMPH | | 179 | METABOLIC ACIDOSIS | 987 | N | 16 | S. TACHY | | NONE | NO | 4 - 5 | RECOVERY |
| P126 | 21 | M | TABLETS | GI SYM | | 114 | NORMAL ABG | 164 | P | 45 | BUNDLE BRANCH BLOCKS | | MINOR | NO | 4 - 5 | RECOVERY |
| P127 | 32 | F | TABLETS | GI SYM | | 168 | NORMAL ABG | 158 | N | 18 | S. TACHY | | MINOR | NO | 3 - 4 | RECOVERY |
| P128 | 49 | M | TABLETS | NO | | 96 | NORMAL ABG | 145 | N | 15 | S. TACHY | | MINOR | NO | <1 | RECOVERY |
| P129 | 24 | F | TABLETS | NO | | 125 | NORMAL ABG | 458 | N | 14 | S. TACHY | | MODERATE | NO | 4 - 5 | RECOVERY |
| P130 | 32 | F | TABLETS | CNS SYMP | | 245 | NORMAL ABG | 165 | N | 14 | S. TACHY | | NONE | NO | 4 - 5 | RECOVERY |
| P131 | 23 | M | TABLETS | CNS SYMP | | 112 | NORMAL ABG | 145 | P | 56 | NSR | | NONE | NO | 4 - 5 | RECOVERY |
| P132 | 22 | F | TABLETS | NO | | 301 | NORMAL ABG | 175 | N | 15 | NSR | | SEVERE | NO | 4 - 5 | RECOVERY |
| P133 | 21 | M | TABLETS | GI SYM | | 89 | NORMAL ABG | 758 | N | 16 | S. TACHY | | MINOR | NO | 4 - 5 | RECOVERY |
| P134 | 48 | F | TABLETS | GI SYM | | 302 | NORMAL ABG | 154 | N | 12 | S. TACHY | | NONE | NO | 1 -2 | RECOVERY |
| P135 | 23 | M | TABLETS | GI SYM | | 301 | ACUTE RESPIRATORY ACIDOSIS | 169 | N | 48 | S. TACHY | | NONE | YES | 4 - 5 | RECOVERY |
| P136 | 33 | M | TABLETS | NO | | 115 | NORMAL ABG | 987 | P | 18 | EXTRA SYSTOLE | | NONE | NO | 4 - 5 | RECOVERY |
| P137 | 19 | M | TABLETS | GI SYM | | 169 | NORMAL ABG | 154 | N | 14 | S.BRADY | | MINOR | NO | 4 - 5 | RECOVERY |
| P138 | 26 | M | TABLETS | GI SYM | | 172 | NORMAL ABG | 185 | N | 12 | S.BRADY | | MODERATE | NO | 2 -3 | RECOVERY |
| P139 | 33 | M | TABLETS | GI SYM | | 136 | NORMAL ABG | 156 | P | 15 | BUNDLE BRANCH BLOCKS | | NONE | NO | <1 | RECOVERY |
| P140 | 48 | F | TABLETS | GI SYM | | 181 | NORMAL ABG | 658 | N | 85 | S. TACHY | | NONE | NO | > 5 | RECOVERY |
| P141 | 21 | F | TABLETS | NO | | 95 | METABOLIC ACIDOSIS | 168 | N | 16 | S. TACHY | | NONE | NO | > 5 | RECOVERY |
| P142 | 33 | F | TABLETS | GI SYM | | 244 | NORMAL ABG | 145 | N | 14 | S. TACHY | | MODERATE | YES | 3 - 4 | RECOVERY |

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|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| P143 | 29 | F | TABLETS | GI SYM | | 246 | NORMAL ABG | 485 | N | 15 | S. TACHY | | NONE | NO | > 5 | RECOVERY |
| P144 | 21 | F | TABLETS | GI SYM | | 185 | MIXED RESP ALK + MET ACID | 169 | N | 15 | S. TACHY | | NONE | NO | > 5 | RECOVERY |
| P145 | 34 | F | TABLETS | GI SYM | | 79 | NORMAL ABG | 174 | N | 15 | S. TACHY | | MINOR | NO | > 5 | RECOVERY |
| P146 | 30 | M | TABLETS | NO | | 158 | NORMAL ABG | 798 | N | 13 | BUNDLE BRANCH BLOCKS | | MINOR | NO | > 5 | RECOVERY |
| P147 | 23 | M | TABLETS | CNS SYMP | | 126 | NORMAL ABG | 145 | N | 15 | BUNDLE BRANCH BLOCKS | | MODERATE | NO | > 5 | COMPLICATIONS |
| P148 | 34 | M | TABLETS | NO | | 165 | NORMAL ABG | 146 | N | 98 | BUNDLE BRANCH BLOCKS | | NONE | NO | 3 - 4 | RECOVERY |
| P149 | 48 | M | TABLETS | NO | | 149 | NORMAL ABG | 185 | N | 14 | S.BRADY | | MINOR | NO | 3 - 4 | RECOVERY |
| P150 | 23 | M | TABLETS | GI SYM | | 159 | NORMAL ABG | 192 | P | 15 | S.BRADY | | MINOR | NO | 2 -3 | RECOVERY |
| P151 | 23 | F | TABLETS | GI SYM | | 112 | NORMAL ABG | 587 | N | 18 | EXTRA SYSTOLE | | MINOR | NO | 4 - 5 | RECOVERY |
| P152 | 24 | F | TABLETS | GI SYM | | 84 | NORMAL ABG | 120 | N | 19 | BUNDLE BRANCH BLOCKS | | MINOR | NO | 4 - 5 | RECOVERY |
| P153 | 34 | M | TABLETS | GI SYM | | 153 | METABOLIC ACIDOSIS | 160 | N | 12 | NSR | | MODERATE | NO | 4 - 5 | RECOVERY |
| P154 | 48 | M | TABLETS | NO | | 156 | NORMAL ABG | 165 | P | 15 | NSR | | NONE | NO | 3 - 4 | RECOVERY |
| P155 | 18 | M | TABLETS | GI SYM | | 154 | ACUTE RESPIRATORY ACIDOSIS | 123 | N | 17 | NSR | | NONE | NO | > 5 | RECOVERY |
| P156 | 23 | F | TABLETS | GI SYM | | 96 | NORMAL ABG | 112 | N | 85 | NSR | | NONE | NO | > 5 | RECOVERY |
| P157 | 22 | F | TABLETS | CNS SYMP | | 152 | NORMAL ABG | 584 | N | 14 | NSR | | MODERATE | NO | > 5 | COMPLICATIONS |
| P158 | 34 | M | TABLETS | CNS SYMP | | 175 | NORMAL ABG | 163 | N | 45 | S. TACHY | | NONE | NO | > 5 | RECOVERY |
| P159 | 30 | M | TABLETS | CNS SYMP | | 182 | NORMAL ABG | 965 | N | 18 | S. TACHY | | NONE | NO | 2 -3 | RECOVERY |
| P160 | 36 | M | TABLETS | NO | | 183 | MIXED RESP ALK + MET ACID | 145 | P | 89 | BUNDLE BRANCH BLOCKS | | NONE | NO | 4 - 5 | RECOVERY |
| P161 | 47 | F | TABLETS | CNS SYMP | | 86 | NORMAL ABG | 123 | N | 17 | S.BRADY | | NONE | NO | 4 - 5 | RECOVERY |
| P162 | 29 | F | TABLETS | GI SYM | | 145 | NORMAL ABG | 356 | N | 17 | EXTRA SYSTOLE | | NONE | NO | 4 - 5 | RECOVERY |
| P163 | 34 | F | TABLETS | GI SYM | | 118 | NORMAL ABG | 102 | P | 65 | S. TACHY | | NONE | NO | 3 - 4 | RECOVERY |
| P164 | 40 | F | TABLETS | GI SYM | | 146 | NORMAL ABG | 96 | N | 15 | S. TACHY | | NONE | NO | 4 - 5 | RECOVERY |
| P165 | 28 | M | TABLETS | GI SYM | | 153 | NORMAL ABG | 185 | N | 89 | S. TACHY | | NONE | NO | 4 - 5 | RECOVERY |
| P166 | 47 | M | TABLETS | NO | | 128 | NORMAL ABG | 165 | N | 21 | S. TACHY | | MINOR | NO | <1 | RECOVERY |
| P167 | 26 | M | TABLETS | NO | | 186 | NORMAL ABG | 485 | N | 68 | S. TACHY | | MINOR | NO | > 5 | RECOVERY |
| P168 | 34 | F | TABLETS | NO | | 189 | NORMAL ABG | 156 | N | 25 | S. TACHY | | MINOR | NO | > 5 | RECOVERY |
| P169 | 23 | F | TABLETS | NO | | 210 | NORMAL ABG | 789 | N | 98 | BUNDLE BRANCH BLOCKS | | MINOR | NO | 3 - 4 | RECOVERY |
| P170 | 47 | F | MISC | RS SYMPH | | 113 | NORMAL ABG | 185 | N | 22 | S. TACHY | | MODERATE | NO | 4 - 5 | RECOVERY |
| P171 | 25 | F | MISC | NO | | 246 | NORMAL ABG | 685 | N | 21 | S. TACHY | | DEATH | NO | 4 - 5 | DEATH |
| P172 | 35 | F | MISC | NO | | 256 | NORMAL ABG | 125 | N | 78 | S. TACHY | | NONE | NO | 2 -3 | RECOVERY |
| P173 | 26 | M | MISC | GI SYM | | 276 | NORMAL ABG | 145 | N | 24 | S. TACHY | | NONE | NO | 4 - 5 | RECOVERY |
| P174 | 40 | F | MISC | NO | | 117 | NORMAL ABG | 165 | N | 15 | S.BRADY | | NONE | NO | 3 - 4 | RECOVERY |
| P175 | 23 | F | MISC | CVS SYM | | 258 | METABOLIC ACIDOSIS | 125 | N | 14 | NSR | | NONE | NO | 1 -2 | RECOVERY |
| P176 | 19 | F | MISC | RS SYMPH | | 139 | NORMAL ABG | 163 | P | 65 | BUNDLE BRANCH BLOCKS | | DEATH | NO | > 5 | DEATH |
| P177 | 35 | F | MISC | RS SYMPH | | 246 | NORMAL ABG | 144 | N | 16 | S.BRADY | | MODERATE | NO | > 5 | COMPLICATIONS |
| P178 | 24 | F | MISC | GI SYM | | 215 | NORMAL ABG | 155 | N | 23 | EXTRA SYSTOLE | | MINOR | NO | > 5 | RECOVERY |

# MASTER CHART

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|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| P179 | 47 | F | MISC | GI SYM | | 243 | NORMAL ABG | 125 | N | 22 | S. TACHY | | MINOR | NO | > 5 | RECOVERY |
| P180 | 21 | F | MISC | GI SYM | | 118 | MIXED RESP ALK + MET ACID | 169 | N | 15 | S. TACHY | | DEATH | NO | > 5 | DEATH |
| P181 | 35 | F | MISC | NO | | 148 | NORMAL ABG | 125 | N | 18 | S. TACHY | | MINOR | NO | 3 - 4 | RECOVERY |
| P182 | 30 | M | MISC | GI SYM | | 159 | NORMAL ABG | 156 | N | 21 | S. TACHY | | MINOR | NO | 2 -3 | RECOVERY |
| P183 | 29 | M | MISC | NO | | 156 | NORMAL ABG | 125 | P | 32 | S. TACHY | | NONE | NO | 4 - 5 | RECOVERY |
| P184 | 56 | M | MISC | NO | | 158 | NORMAL ABG | 136 | N | 12 | S. TACHY | | NONE | NO | 1 -2 | RECOVERY |
| P185 | 58 | F | MISC | RS SYMPH | | 147 | NORMAL ABG | 458 | N | 65 | S. TACHY | | DEATH | NO | > 5 | DEATH |
| P186 | 27 | F | MISC | RS SYMPH | | 112 | NORMAL ABG | 587 | N | 25 | S. TACHY | | MINOR | NO | > 5 | RECOVERY |
| P187 | 26 | F | MISC | NO | | 157 | NORMAL ABG | 154 | N | 14 | S. TACHY | | MINOR | NO | 3 - 4 | RECOVERY |
| P188 | 46 | F | MISC | NO | | 153 | NORMAL ABG | 169 | P | 98 | EXTRA SYSTOLE | | MINOR | NO | > 5 | RECOVERY |
| 1P89 | 35 | F | MISC | GI SYM | | 165 | NORMAL ABG | 658 | N | 15 | S.BRADY | | SEVERE | NO | 2 -3 | RECOVERY |
| 1P90 | 24 | M | PLANT POISON | NO | | 145 | ACUTE RESPIRATORY ACIDOSIS | 125 | N | 16 | BUNDLE BRANCH BLOCKS | | SEVERE | NO | > 5 | COMPLICATIONS |
| P191 | 23 | M | PLANT POISON | GI SYM | | 116 | NORMAL ABG | 136 | N | 65 | S.BRADY | | NONE | NO | > 5 | RECOVERY |
| P192 | 60 | M | PLANT POISON | NO | | 149 | NORMAL ABG | 1269 | N | 20 | NSR | | NONE | NO | > 5 | RECOVERY |
| P193 | 36 | F | PLANT POISON | GI SYM | | 158 | NORMAL ABG | 154 | P | 65 | NSR | | NONE | NO | 1 -2 | RECOVERY |
| P194 | 36 | F | PLANT POISON | GI SYM | | 165 | ACUTE RESPIRATORY ACIDOSIS | 154 | N | 10 | EXTRA SYSTOLE | | MINOR | NO | 4 - 5 | RECOVERY |
| P195 | 46 | F | PLANT POISON | NO | | 115 | NORMAL ABG | 193 | N | 15 | S.BRADY | | NONE | NO | 2 -3 | RECOVERY |
| P196 | 33 | F | PLANT POISON | GI SYM | | 152 | NORMAL ABG | 987 | N | 14 | BUNDLE BRANCH BLOCKS | | NONE | NO | <1 | RECOVERY |
| P197 | 60 | M | PLANT POISON | NO | | 163 | NORMAL ABG | 185 | N | 15 | BUNDLE BRANCH BLOCKS | | NONE | NO | > 5 | RECOVERY |
| P198 | 22 | M | PLANT POISON | GI SYM | | 126 | NORMAL ABG | 165 | N | 12 | BUNDLE BRANCH BLOCKS | | NONE | NO | > 5 | RECOVERY |
| P199 | 37 | M | PLANT POISON | NO | | 172 | NORMAL ABG | 548 | N | 16 | BUNDLE BRANCH BLOCKS | | MINOR | NO | > 5 | RECOVERY |
| P200 | 39 | F | PLANT POISON | GI SYM | | 185 | NORMAL ABG | 785 | N | 15 | BUNDLE BRANCH BLOCKS | | NONE | NO | 1 -2 | RECOVERY |
| P201 | 46 | F | PLANT POISON | NO | | 129 | NORMAL ABG | 165 | N | 69 | NSR | | NONE | NO | > 5 | RECOVERY |
| P202 | 75 | F | PLANT POISON | NO | | 196 | METABOLIC ACIDOSIS | 856 | N | 12 | NSR | | NONE | NO | > 5 | RECOVERY |
| P203 | 18 | F | PLANT POISON | NO | | 148 | NORMAL ABG | 185 | N | 15 | NSR | | NONE | NO | > 5 | RECOVERY |
| P204 | 34 | M | PLANT POISON | NO | | 150 | NORMAL ABG | 145 | N | 65 | NSR | | NONE | NO | 3 - 4 | RECOVERY |
| P205 | 37 | M | PLANT POISON | GI SYM | | 151 | NORMAL ABG | 169 | P | 14 | S.BRADY | | NONE | NO | 4 - 5 | RECOVERY |
| P206 | 36 | M | PLANT POISON | NO | | 175 | NORMAL ABG | 987 | N | 78 | S. TACHY | | NONE | NO | 2 -3 | RECOVERY |
| P207 | 46 | F | PLANT POISON | GI SYM | | 173 | NORMAL ABG | 154 | N | 15 | S. TACHY | | NONE | NO | 4 - 5 | RECOVERY |
| P208 | 57 | F | PLANT POISON | GI SYM | | 172 | NORMAL ABG | 586 | N | 96 | S. TACHY | | NONE | NO | 1 -2 | RECOVERY |
| P209 | 21 | F | PLANT POISON | CNS SYMP | | 171 | NORMAL ABG | 136 | N | 15 | S. TACHY | | MINOR | NO | > 5 | RECOVERY |
| P210 | 35 | F | TABLETS | CNS SYMP | | 128 | NORMAL ABG | 156 | N | 14 | EXTRA SYSTOLE | | MODERATE | YES | > 5 | RECOVERY |
| P211 | 37 | F | TABLETS | NO | | 181 | NORMAL ABG | 145 | P | 14 | S.BRADY | | NONE | NO | > 5 | RECOVERY |
| P212 | 35 | M | TABLETS | NO | | 151 | NORMAL ABG | 658 | N | 15 | S.BRADY | | MINOR | NO | 1 -2 | RECOVERY |
| P213 | 56 | M | TABLETS | NO | | 152 | NORMAL ABG | 154 | N | 16 | BUNDLE BRANCH BLOCKS | | NONE | NO | 4 - 5 | RECOVERY |
| P214 | 34 | M | TABLETS | GI SYM | | 152 | NORMAL ABG | 789 | N | 13 | NSR | | NONE | NO | 3 - 4 | RECOVERY |

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|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| P215 | 39 | F | TABLETS | GI SYM | | 162 | NORMAL ABG | 169 | P | 18 | NSR | | NONE | NO | 4 - 5 | RECOVERY |
| P216 | 46 | F | TABLETS | CNS SYMP | | 161 | ACUTE RESPIRATORY ACIDOSIS | 258 | N | 12 | NSR | | MODERATE | YES | 2 -3 | COMPLICATIONS |
| P217 | 38 | F | TABLETS | CNS SYMP | | 192 | NORMAL ABG | 152 | N | 18 | S.BRADY | | NONE | NO | > 5 | RECOVERY |
| P218 | 36 | F | TABLETS | NO | | 185 | NORMAL ABG | 654 | N | 15 | S.BRADY | | MINOR | NO | > 5 | RECOVERY |
| P219 | 45 | F | TABLETS | CNS SYMP | | 254 | NORMAL ABG | 689 | N | 87 | BUNDLE BRANCH BLOCKS | | SEVERE | NO | > 5 | COMPLICATIONS |
| P220 | 19 | F | TABLETS | RS SYMPH | | 266 | METABOLIC ACIDOSIS | 896 | N | 15 | S. TACHY | | MINOR | NO | 3 - 4 | RECOVERY |
| P221 | 30 | M | TABLETS | NO | | 258 | NORMAL ABG | 169 | N | 95 | S. TACHY | | MODERATE | YES | > 5 | COMPLICATIONS |
| P222 | 65 | M | MISC | CNS SYMP | | 245 | NORMAL ABG | 789 | P | 13 | S. TACHY | | NONE | NO | > 5 | RECOVERY |
| P223 | 37 | M | MISC | NO | | 125 | NORMAL ABG | 125 | N | 14 | S. TACHY | | MINOR | NO | > 5 | RECOVERY |
| P224 | 54 | F | MISC | GI SYM | | 154 | NORMAL ABG | 487 | N | 68 | S. TACHY | | DEATH | NO | 3 - 4 | DEATH |
| P225 | 45 | F | MISC | NO | | 162 | NORMAL ABG | 1125 | N | 16 | S. TACHY | | MINOR | NO | 4 - 5 | RECOVERY |
| P226 | 30 | F | MISC | NO | | 131 | NORMAL ABG | 963 | P | 15 | S. TACHY | | MINOR | NO | 3 - 4 | RECOVERY |
| P227 | 38 | F | MISC | NO | | 158 | NORMAL ABG | 856 | N | 85 | S. TACHY | | MINOR | NO | 4 - 5 | RECOVERY |
| P228 | 43 | F | MISC | GI SYM | | 169 | NORMAL ABG | 156 | N | 16 | BUNDLE BRANCH BLOCKS | | MINOR | NO | 3 - 4 | RECOVERY |
| P229 | 53 | F | MISC | RS SYMPH | | 157 | NORMAL ABG | 154 | N | 15 | BUNDLE BRANCH BLOCKS | | DEATH | NO | > 5 | DEATH |
| P230 | 30 | F | MISC | GI SYM | | 154 | NORMAL ABG | 156 | N | 98 | BUNDLE BRANCH BLOCKS | | NONE | NO | > 5 | RECOVERY |
| P231 | 45 | F | MISC | NO | | 154 | NORMAL ABG | 145 | N | 15 | BUNDLE BRANCH BLOCKS | | NONE | NO | 2 -3 | RECOVERY |
| P232 | 38 | F | TABLETS | NO | | 165 | NORMAL ABG | 125 | N | 65 | BUNDLE BRANCH BLOCKS | | SEVERE | YES | 1 -2 | COMPLICATIONS |
| P233 | 29 | M | TABLETS | CNS SYMP | | 169 | ACUTE RESPIRATORY ACIDOSIS | 168 | P | 21 | S.BRADY | | MINOR | NO | 4 - 5 | RECOVERY |
| P234 | 62 | M | TABLETS | RS SYMPH | | 125 | NORMAL ABG | 195 | N | 21 | S.BRADY | | MINOR | NO | 4 - 5 | RECOVERY |
| P235 | 18 | M | TABLETS | NO | | 154 | NORMAL ABG | 185 | N | 19 | S.BRADY | | MODERATE | YES | 4 - 5 | RECOVERY |
| P236 | 34 | F | TABLETS | NO | | 186 | NORMAL ABG | 165 | N | 18 | S.BRADY | | NONE | NO | > 5 | RECOVERY |
| P237 | 52 | F | TABLETS | NO | | 173 | NORMAL ABG | 152 | N | 25 | S.BRADY | | NONE | NO | 3 - 4 | RECOVERY |
| P238 | 38 | F | TABLETS | NO | | 149 | NORMAL ABG | 169 | N | 15 | S.BRADY | | MODERATE | YES | 4 - 5 | RECOVERY |
| P239 | 34 | F | TABLETS | NO | | 158 | NORMAL ABG | 125 | P | 12 | NSR | | NONE | NO | 1 -2 | RECOVERY |
| P240 | 42 | F | TABLETS | GI SYM | | 141 | METABOLIC ACIDOSIS | 145 | N | 16 | BUNDLE BRANCH BLOCKS | | NONE | NO | 4 - 5 | RECOVERY |
| P241 | 28 | F | TABLETS | GI SYM | | 165 | NORMAL ABG | 136 | N | 13 | S. TACHY | | MODERATE | YES | 2 -3 | RECOVERY |
| P242 | 33 | F | MISC | NO | | 145 | NORMAL ABG | 159 | N | 18 | S. TACHY | | SEVERE | NO | 4 - 5 | COMPLICATIONS |
| P243 | 42 | M | MISC | NO | | 147 | NORMAL ABG | 125 | P | 14 | S. TACHY | | MINOR | NO | 4 - 5 | RECOVERY |
| P244 | 31 | M | MISC | GI SYM | | 165 | NORMAL ABG | 158 | N | 15 | S. TACHY | | MINOR | NO | 3 - 4 | RECOVERY |
| P245 | 26 | M | MISC | CNS SYMP | | 135 | NORMAL ABG | 168 | N | 15 | S. TACHY | | MINOR | NO | > 5 | RECOVERY |
| P246 | 33 | F | MISC | GI SYM | | 154 | MIXED RESP ALK + MET ACID | 162 | P | 12 | S. TACHY | | MINOR | NO | > 5 | RECOVERY |
| P247 | 31 | F | MISC | NO | | 185 | NORMAL ABG | 169 | N | 15 | S.BRADY | | MINOR | NO | > 5 | RECOVERY |
| P248 | 40 | F | MISC | GI SYM | | 125 | NORMAL ABG | 148 | N | 10 | BUNDLE BRANCH BLOCKS | | NONE | NO | > 5 | RECOVERY |
| P249 | 20 | F | MISC | CVS SYM | | 124 | NORMAL ABG | 154 | N | 9 | NSR | | NONE | NO | 1 -2 | RECOVERY |
| P250 | 21 | M | MISC | NO | | 185 | NORMAL ABG | 125 | N | 18 | NSR | | NONE | NO | 3 - 4 | RECOVERY |

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|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| P251 | 42 | M | MISC | GI SYM | | 146 | NORMAL ABG | 112 | N | 16 | S.BRADY | | SEVERE | NO | 2 -3 | COMPLICATIONS |
| P252 | 31 | M | insc and pest | GI SYMPH | | 1182 | NORMAL ABG | 125 | N | 65 | S.BRADY | 2154 | MINOR | NO | 3 - 4 | RECOVERY |
| P253 | 68 | F | insc and pest | CNS SYM | YES | 189 | METABOLIC ACIDOSIS | 126 | P | 16 | S. TACHY | 4582 | DEATH | YES | 1 -2 | DEATH |
| P254 | 40 | F | insc and pest | NO | | 131 | NORMAL ABG | 129 | N | 18 | S. TACHY | 6548 | MODERATE | YES | 4 - 5 | RECOVERY |
| P255 | 28 | F | insc and pest | RS SYMP | | 178 | NORMAL ABG | 154 | P | 35 | S. TACHY | 1254 | NONE | NO | 4 - 5 | RECOVERY |
| P256 | 31 | F | insc and pest | NO | | 165 | ACUTE RESPIRATORY ACIDOSIS | 145 | N | 18 | S. TACHY | 9587 | NONE | NO | 4 - 5 | RECOVERY |
| P257 | 42 | F | insc and pest | CNS SYM | YES | 176 | NORMAL ABG | 158 | N | 14 | S. TACHY | 6584 | DEATH | NO | 4 - 5 | DEATH |
| P258 | 27 | M | insc and pest | CVS SYM | | 114 | NORMAL ABG | 158 | N | 15 | S. TACHY | 6584 | MODERATE | YES | 4 - 5 | RECOVERY |
| P259 | 21 | M | insc and pest | NO | | 185 | NORMAL ABG | 165 | N | 15 | S. TACHY | 4587 | DEATH | YES | 4 - 5 | DEATH |
| P260 | 56 | F | insc and pest | CVS SYM | | 189 | NORMAL ABG | 148 | N | 22 | S.BRADY | 3562 | MINOR | NO | 4 - 5 | RECOVERY |
| P261 | 43 | F | insc and pest | NO | | 145 | NORMAL ABG | 951 | N | 55 | BUNDLE BRANCH BLOCKS | 3568 | MODERATE | YES | 2 -3 | RECOVERY |
| P262 | 39 | F | insc and pest | CNS SYM | | 178 | NORMAL ABG | 165 | N | 12 | S.BRADY | 4587 | SEVERE | YES | 3 - 4 | RECOVERY |
| P263 | 20 | M | insc and pest | NO | YES | 168 | METABOLIC ACIDOSIS | 196 | N | 33 | S.BRADY | 5486 | DEATH | YES | 3 - 4 | DEATH |
| P264 | 40 | M | insc and pest | RS SYMP | | 175 | NORMAL ABG | 158 | N | 24 | S.BRADY | 6548 | MINOR | YES | 1 -2 | RECOVERY |
| P265 | 43 | F | insc and pest | NO | | 165 | NORMAL ABG | 168 | N | 66 | BUNDLE BRANCH BLOCKS | 1458 | SEVERE | YES | > 5 | RECOVERY |
| P266 | 40 | F | insc and pest | CVS SYM | YES | 185 | NORMAL ABG | 125 | N | 25 | NSR | 6584 | DEATH | YES | > 5 | DEATH |
| P267 | 31 | F | insc and pest | NO | | 154 | NORMAL ABG | 111 | N | 98 | NSR | 4587 | MINOR | NO | 2 -3 | RECOVERY |
| P268 | 44 | M | insc and pest | RS SYMP | | 168 | NORMAL ABG | 153 | N | 20 | NSR | 9587 | MINOR | NO | 3 - 4 | RECOVERY |
| P269 | 28 | M | insc and pest | CVS SYM | | 158 | NORMAL ABG | 126 | N | 21 | NSR | 1458 | MODERATE | YES | 3 - 4 | RECOVERY |
| P270 | 57 | F | insc and pest | NO | | 117 | METABOLIC ACIDOSIS | 145 | P | 65 | NSR | 4585 | NONE | NO | 1 -2 | RECOVERY |
| P271 | 27 | F | insc and pest | CNS SYM | YES | 185 | NORMAL ABG | 152 | N | 23 | NSR | 6965 | DEATH | NO | > 5 | DEATH |
| P272 | 44 | F | insc and pest | NO | | 145 | NORMAL ABG | 165 | N | 54 | BUNDLE BRANCH BLOCKS | 1254 | NONE | NO | > 5 | RECOVERY |
| P273 | 23 | M | insc and pest | GI SYMPH | | 163 | NORMAL ABG | 125 | N | 21 | S.BRADY | 5876 | NONE | NO | 2 -3 | RECOVERY |
| P274 | 40 | M | insc and pest | CVS SYM | YES | 158 | NORMAL ABG | 112 | N | 98 | S.BRADY | 4586 | DEATH | YES | 4 - 5 | DEATH |
| P275 | 25 | F | insc and pest | NO | | 169 | NORMAL ABG | 115 | N | 25 | S.BRADY | 6584 | NONE | YES | 4 - 5 | RECOVERY |
| P276 | 20 | F | insc and pest | RS SY | | 158 | NORMAL ABG | 658 | N | 65 | S.BRADY | 9658 | SEVERE | YES | 2 -3 | COMPLICATIONS |
| P277 | 44 | F | insc and pest | CNS SYM | YES | 148 | NORMAL ABG | 356 | N | 12 | NSR | 3215 | DEATH | YES | 4 - 5 | DEATH |
| P278 | 32 | M | insc and pest | NO | | 158 | METABOLIC ACIDOSIS | 458 | N | 12 | S.BRADY | 4587 | MODERATE | YES | 4 - 5 | RECOVERY |
| P279 | 23 | M | insc and pest | GI SYMPH | | 169 | NORMAL ABG | 659 | P | 15 | NSR | 6584 | MINOR | NO | 4 - 5 | RECOVERY |
| P280 | 33 | F | insc and pest | NO | | 157 | NORMAL ABG | 154 | N | 12 | NSR | 7856 | MINOR | NO | 1 -2 | RECOVERY |
| P281 | 60 | F | insc and pest | RS SYMP | | 159 | METABOLIC ACIDOSIS | 146 | N | 15 | BUNDLE BRANCH BLOCKS | 4587 | MINOR | NO | 4 - 5 | RECOVERY |
| P282 | 44 | M | insc and pest | NO | | 158 | NORMAL ABG | 125 | N | 25 | NSR | 1254 | NONE | YES | 4 - 5 | RECOVERY |
| P283 | 24 | M | insc and pest | NO | | 165 | NORMAL ABG | 654 | N | 9 | NSR | 9587 | SEVERE | YES | 2 -3 | RECOVERY |
| P284 | 21 | F | insc and pest | GI SYMPH | YES | 116 | NORMAL ABG | 145 | N | 6 | S.BRADY | 5486 | DEATH | YES | 3 - 4 | DEATH |
| P285 | 56 | F | insc and pest | NO | | 254 | NORMAL ABG | 789 | N | 21 | NSR | 4587 | NONE | NO | 3 - 4 | RECOVERY |
| P286 | 44 | M | insc and pest | CNS SYM | | 255 | NORMAL ABG | 158 | N | 14 | NSR | 6974 | NONE | NO | 1 -2 | RECOVERY |

# MASTER CHART

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|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| P287 | 50 | M | insc and pest | NO | | 266 | NORMAL ABG | 152 | N | 22 | S.BRADY | 4587 | NONE | NO | > 5 | RECOVERY |
| P288 | 30 | F | insc and pest | RS SYMP | | 244 | NORMAL ABG | 654 | P | 15 | S. TACHY | 4587 | NONE | NO | > 5 | RECOVERY |
| P289 | 34 | F | insc and pest | NO | | 248 | NORMAL ABG | 154 | N | 15 | S. TACHY | 9856 | MINOR | NO | > 5 | RECOVERY |
| P290 | 29 | F | insc and pest | GI SYMPH | | 278 | NORMAL ABG | 156 | N | 18 | S. TACHY | 6584 | MODERATE | YES | > 5 | RECOVERY |
| P291 | 28 | F | insc and pest | NO | | 152 | METABOLIC ACIDOSIS | 128 | N | 11 | S. TACHY | 5287 | NONE | YES | 3 - 4 | RECOVERY |
| P292 | 34 | M | insc and pest | RSSYMP | | 118 | NORMAL ABG | 153 | N | 15 | BUNDLE BRANCH BLOCKS | 6587 | MODERATE | YES | 1 -2 | RECOVERY |
| P293 | 40 | M | insc and pest | NO | | 245 | NORMAL ABG | 987 | N | 25 | S.BRADY | 6598 | SEVERE | YES | 3 - 4 | COMPLICATIONS |
| P294 | 27 | F | insc and pest | GI SYMPH | YES | 154 | NORMAL ABG | 165 | N | 14 | S.BRADY | 4587 | DEATH | YES | 3 - 4 | DEATH |
| P295 | 54 | F | insc and pest | NO | | 245 | NORMAL ABG | 789 | N | 15 | S. TACHY | 6597 | MINOR | NO | 2 -3 | RECOVERY |
| P296 | 26 | F | insc and pest | RS SYMP | | 154 | NORMAL ABG | 158 | N | 12 | S. TACHY | 4587 | MINOR | NO | 4 - 5 | RECOVERY |
| P297 | 40 | F | insc and pest | NO | | 256 | NORMAL ABG | 654 | N | 18 | S. TACHY | 5698 | MINOR | NO | 4 - 5 | RECOVERY |
| P298 | 25 | F | insc and pest | RS SYMP | | 148 | ACUTE RESPIRATORY ACIDOSIS | 126 | P | 19 | S. TACHY | 4587 | MODERATE | YES | 4 - 5 | RECOVERY |
| P299 | 24 | F | insc and pest | NO | | 154 | NORMAL ABG | 456 | N | 20 | S. TACHY | 5879 | MINOR | NO | 4 - 5 | RECOVERY |
| P300 | 40 | F | insc and pest | NO | | 169 | ACUTE RESPIRATORY ACIDOSIS | 169 | N | 21 | S. TACHY | 5876 | MINOR | NO | 3 - 4 | RECOVERY |
| P301 | 60 | M | insc and pest | RS SYMPH | YES | 123 | NORMAL ABG | 135 | N | 15 | S. TACHY | 9875 | DEATH | NO | > 5 | DEATH |
| P302 | 23 | F | insc and pest | GI SYMPH | | 458 | METABOLIC ACIDOSIS | 125 | N | 17 | S. TACHY | 6587 | NONE | YES | > 5 | RECOVERY |
| P303 | 34 | F | insc and pest | NO | | 122 | NORMAL ABG | 165 | N | 16 | BUNDLE BRANCH BLOCKS | 6894 | MODERATE | YES | > 5 | RECOVERY |
| P304 | 35 | F | insc and pest | MULTI SYSM | YES | 154 | NORMAL ABG | 125 | N | 15 | S.BRADY | 4578 | DEATH | YES | > 5 | DEATH |
| P305 | 22 | F | insc and pest | NO | | 154 | METABOLIC ACIDOSIS | 654 | N | 12 | S.BRADY | 4987 | SEVERE | YES | > 5 | COMPLICATIONS |
| P306 | 36 | M | insc and pest | RS SYMP | | 152 | NORMAL ABG | 951 | N | 18 | S.BRADY | 4575 | MINOR | YES | > 5 | RECOVERY |
| P307 | 21 | F | insc and pest | NO | | 102 | ACUTE RESPIRATORY ACIDOSIS | 152 | N | 16 | NSR | 4658 | NONE | YES | > 5 | RECOVERY |
| P308 | 36 | F | insc and pest | RS SYMP | YES | 256 | NORMAL ABG | 654 | N | 21 | NSR | 5684 | MODERATE | NO | 1 -2 | RECOVERY |
| P309 | 34 | F | insc and pest | NO | | 145 | NORMAL ABG | 753 | N | 22 | NSR | 3449 | SEVERE | YES | 4 - 5 | RECOVERY |
| P310 | 28 | F | insc and pest | MULTI SYSM | YES | 175 | NORMAL ABG | 124 | N | 18 | NSR | 5876 | DEATH | YES | 3 - 4 | DEATH |