CHAPTER 21
FORENSIC PSYCHIATRY

Psychiatry deals with the study, diagnosis and treatment of mental illness. **Forensic psychiatry** deals with application of psychiatry in the administration of justice. Insanity or unsoundness of mind can be defined as a disease of the mind or the personality, in which there is derangement of the mental or emotional processes. The intelligence is weakened or perverted, but the insane person may not show physical weakness. The law has not defined insanity. The term is used for those persons who are unable to adopt themselves to the ordinary social requirements, due to mental disease. The law is most frequently concerned with "mental impairment" and not "mental illness." Different kinds and degrees of mental impairment are required for different legal issues.

An abnormal mental state may be due to a functional psychosis, to substance misuse, to an abnormal metabolic state, such as hypoglycaemia, or due to less common problems, such as learning disability, organic brain disease or head injury. In abnormal mental state, the effects of stress anxiety, fear or anger may coexist with intoxication (hallucinogenics and stimulants) of some kind and the behaviour, demeanour and appearance should be observed.

The following are some of the important terms in connection with insanity.

ABRECATION: Reviving and bringing into consciousness, forgotten and other traumatic experiences or repressed emotions from unconscious level by catharsis.

AFFECT : Emotion, feeling or mood, e.g., lability of mood, cyclothymia, flattening, incongruity and inappropriateness of affect.

AFFECTIVE DISORDER : Psychiatric disorder in which the chief feature is a relatively prolonged affective change of an abnormal degree, i.e., depression and mania.

APHASIA : The loss of ability to express meaning by the use of speech or writing (motor aphasia), or to understand spoken or written language (sensory or auditory aphasia).

DELIRIUM: It is a disturbance of consciousness in which orientation is impaired, the critical faculty is blunted or lost and thought content is irrelevant or inconsistent. In the early stage the patient is restless, uneasy and sleepless. He then completely loses self-control, becomes excited and talks furiously. Delusions and sometimes hallucinations are present. It usually occurs in physical diseases, in which there is continuous high temperature, and sometimes due to overwork, mental stress or drug intoxication. A person may become impulsive and violent and may commit suicide. Such person is not responsible for his criminal acts.

DELUSION : Delusion is a false belief in something which is not a fact, and which persists even after its falsity has been clearly demonstrated. A normal person can have a delusion, but is capable of correcting it by his reasoning power, by his past experience and by being convinced by others. A secondary delusion arises from some morbid experience. Delusion in insane person is a symptom of brain disease. It is under the control of emotional but not rational forces. They are found in epileptic, affective and schizophrenic psychoses. Delusions are not seen in anxiety neurosis and other neurotic illnesses.

Types: (1)Grandeur or exaltation : A man imagines himself to be very rich while in reality he is a pauper. They are seen in delirium tremens. **(2) Persecution (paranoid)** : The person imagines that attempts are being made to poison him by his nearest relatives like wife, sons or parents. They are seen in paranoid schizophrenia, dementia and depression. Delusions of grandeur and persecution are often present together in the same person. **(3) Reference:** The person believes that people, things, events, etc., refer to him in a special way. He believes that even strangers in the street are looking at him and are talking about him, or items in the radio or newspapers are referring to him. **(4) Influence** : They occur in schizophrenia. The person complains that his thoughts, feelings and actions are being influenced and controlled by some outside agency, like radio, hypnotism, telepathy, etc. **(5) Infidelity** : A man imagines his wife to be unfaithful while in fact she is chaste. **(6) Self-reproach** : The person blames himself for the past failures and misdeeds which are often of no importance. **(7) Nihilistic** : The person declares that he does not exist or that there is no world, etc. **(8) Hypochondriacal:** The person believes

that there is something wrong with his body, though he is healthy. **(9) Other types** are of jealousy, of religion, etc.

M.L.Importance : Delusion is never an isolated disorder, but is merely an indication of deep-seated, widespread disorder. For this reason, such person cannot be regarded as fully responsible for his antisocial acts. Suicide is a major risk. There may be a combination of murder and suicide.

EROTOMANIA: Erotomania is a delusion in which the person believes that someone is deeply in love with him/her. The eratomanic develops an obsession for a particular person and starts believing that the other person is reciprocating. The object is usually of a higher status, famous or superior at work, but can also be a complete stranger. The erotomanic tries to get close with the person through telephone calls, letters, gifts, visits, etc. The person is otherwise normal.

EMPATHY : The degree to which the observer is able to enter into the thoughts and feelings of the patient and establish good contact.

FUGUE: A state of altered awareness during which an individual forgets part or whole of his life, leaves home and wanders. It may occur in hysteria, depressive illness, schizophrenia and epilepsy.

HALLUCINATION: Hallucination is a false sense perception without any external object or stimulus to produce it. They are purely imaginary, and may affect any or all the special senses.

Types : (1) Visual : A person imagines of being attacked by a lion when no lion exists. **(2) Auditory:** A person hears voices and imagines that a person is speaking to him when no one is present. **(3) Olfactory:** A person smells pleasant or unpleasant odour when none is present. **(4) Gustatory:** A person feels sweet, sour, bitter, good or bad taste in the mouth, though no food is actually present. **(5) Tactile :** A man imagines rats and mice crawling into his bed, when there are none. **(6) Psychomotor:** A man will have feeling of movement of some part of the body in the absence of such movement.

Visual hallucinations are the commonest in organic mental disorders, while the auditory hallucinations are the commonest in functional disorders. Visual hallucinations are present in delirium tremens, focal CNS lesions, toxic disturbances, schizophrenia and drug withdrawal syndromes; auditory in schizophrenia, delirium, psychotic mood disorders, and toxic and metabolic encephalopathies; gustatory in organic brain diseases and temporal lobe epilepsy; olfactory in organic brain disease and major depressions and tactile in cocainism. Hallucinations occur in fevers, intoxications, and insanity. In command hallucinations, the patient is ordered by hallucinatory voices to do things/acts, which may be frightening or dangerous. They may be pleasant, but more often they are unpleasant. A person suffering from unpleasant hallucinations may be incited to commit suicide or homicide.

ILLUSION : Illusion is a false interpretation by the senses of an external object or stimulus which has a real existence, e.g., when a person sees a dog and mistakes it for lion, or hears the notes of birds and imagines them to be human voices, or imagines a string hanging in his room to be snake, or may mistake the stem of a tree for a ghost in the dark. A sane person may experience illusion, but is capable of correcting the false impressions. An insane person continues to believe in the illusions, even though the real facts are clearly pointed out. Illusions are a feature of psychoses, particularly of the organic type.

IMPULSE : This is a sudden and irresistible force compelling a person to the conscious performance of some action without motive or forethought. A sane person is capable of controlling an impulse. An insane person having no judgement and no reasoning power, and no capacity to understand the facts, may do things on impulse. These are usually seen in imbecility, dementia, acute mania, and epilepsy.

Types : (1) Kleptomania : An irresistible desire to steal articles of little value. **(2) Pyromania:** An irresistible desire to set fire to things. **(3) Mutilomania :** An irresistible desire to mutilate animals. **(4) Dipsomania :** An irresistible desire for alcoholic drinks at periodic intervals. **(5) Sexual impulses:** Compulsive urge to perform sexual intercourse which may often be in a perverted way. **(6) Suicidal and homicidal impulses.**

OBSESSION : In this , a single idea, thought, or emotion is constantly entertained by a person which he recognises as irrational, but persists inspite of all efforts to drive it from his mind. It is a disorder of content of thought. Any attempt to resist makes them appear more insistent, and yielding is the almost inevitable outcome. It is a borderline between sanity and insanity. They usually occur in

neurotic people, well able to discharge the ordinary responsibilities of life. These ideas are usually associated with some sort of dread or fear. A wife may continuously believe her husband to be unfaithful inspite of proof to the contrary. A person may go to bed at night after securely bolting the door of his room, but he soon gets up to see he has done so. A sane person may repeat it once or twice, but an insane person does not sleep, and spends the whole night in frequently seeing whether the door is bolted.

PHOBIA : It is an excessive or irrational fear of a particular object or situation. Phobias may develop to almost any object or situation. Acrophobia is morbid fear of high places. Agarophobia is fear of being in a large open space. Nyctophobia is morbid fear of darkness. Claustrophobia is fear of staying in a closed or confined space. Mysophobia is morbid fear of filth or contamination. Xenophobia is fear of strangers.

LUCID INTERVAL : This is a period occurring in insanity, during which all the symptoms of insanity disappear completely. The individual is able to judge his acts soundly, and he becomes legally liable for his acts. The period of lucid interval varies from person to person and from time to time in the same person, and as such one cannot be certain about the time when a person passes again in the state of insanity. Therefore if he commits an offence, he cannot be completely held responsible, because it is very difficult to know, whether he was suffering from some mental abnormality at the time of committing the offence. In mania and melancholia lucid intervals are common.

ONEIROID STATES (oneirophrenia): It is a dream-like state, which may last for days or weeks. The patient suffers from mental confusion, amnesia, illusions, hallucinations, disorientation, agitation and anxiety. It occurs in delirium and early schizophrenia.

TWILIGHT STATE (psychomotor automatism): It is a state of diminished awareness of acts of relatively short duration, of which he has no recollection. The patient may do some unaccustomed automatic acts sometimes in an aggressive way, and may suffer from visual hallucintions. It is usually seen in epilepsy.

PSYCHOPATH: Psychopath or sociopath is a person who is neither insane nor mentally defective, but fails to conform to normal standards of behaviour. Psychopaths have abnormal personality, persistently behave in an antisocial or disruptive manner, and are unable to appreciate the moral implications of their actions. It is not a ground for an insanity defence, but may provide a plea of diminished responsibility.

In this there is a failure of maturation of the personality, the individual retaining a child-like selfishness. They have no abnormality of thought, mood or intelligence, but their behaviour is unacceptable. Lack of emotional response, an unswerving desire for the gratification of their desires and complete lack of conscience are characteristic of psychopathic personality. Frustration of any whim is not tolerated and is met with

Table (21–1). Difference between lucid interval in insanity and head injury

	Trait	Insanity	Head Injury
(1)	History:	Of insanity present.	Of injury to the head.
(2)	Preceding symptoms:	Of insanity.	Of concussion.
(3)	Following symptoms:	Of insanity.	Of cerebral irritation and compression of the brain.
(4)	Occurrence:	Frequent.	Only once.

Table (21–2). Difference between psychosis and neurosis

	Trait	Psychosis	Neurosis
(1)	Nature:	A disease entity with a physical basis which is determined genetically.	A reaction to stressful circumstances due to adverse childhood experiences.
(2)	Severity:	Major.	Minor.
(3)	Empathy:	Absent.	Present.
(4)	Contact with reality:	Absent.	Present.
(5)	Insight:	Absent.	Present.

aggression, completely devoid of any regret or remorse. Immediate violence including murder can arise from any challenge to their egotism. They can plan and implement their antisocial acts in an efficient way. The basic defect appears to be moral, rather than psychological or neurological.

PSYCHOSES : They are characterised by a withdrawal from reality; a living in a world of fantasy. These mental illnesses supervene upon a normally developed mental faculty. There is deterioration in the personality and progressive loss of contact with reality. Such persons incorrectly evaluate the accuracy of their perceptions and thoughts and make incorrect inferences about external reality, even in the face of contrary evidence. Delusions and hallucinations are common. Certain drugs, such as alcohol, heroin, morphine, cannabis, cocaine and LSD used habitually may produce psychoses. Psychosis may occur in epilepsy and pregnancy.

(1) THE PSYCHOTIC KILLER : Such person is either: (a) incapable of knowing the nature of the act (as in cases of organic disorder), or (b) his judgement is faulty (due to delusions and hallucinations). Murders are common in depression. Schizophrenics commit murders due to delusions of persecution. Morbid jealousy associated with alcoholism may lead to murder of the spouse due to delusions of infidelity. Rarely maniacs and hypomaniacs commit murders due to delusions.

(2) THE PSYCHOPATHIC KILLER : The killing may be unintentional due to loss of control. Over-controlled murderer is one who has a high level of control over his aggression, but commits a murder due to an explosive response. After the aggressive act, he returns to his rigidly controlled behaviour. In some, normal emotional responses may be almost absent.

NEUROSES : The patient suffers from emotional or intellectual disorders, but he does not lose touch with reality. They occur mostly in the form of anxiety, depression or hysteria. The effects may be mild or may cause considerable distress to the patient, but they are not associated with severe affective change, nor with disturbances of thought.

NEURASTHENIA : It is a condition of nervous exhaustion due to physical or mental conditions. There is an abnormal fatigue and irritability of the nervous system.

MUTISM : There is complete loss of speech. It is seen is hysteria, catatonic schizophrenia, depression, organic brain lesion and malingering.

Confabulation is pathological loss of memory.

CAUSES OF INSANITY : (1) Hereditary, e.g. Huntington's chorea and amaurotic family idiocy. (2) Environmental factors, e.g., faulty parental attitude and lack of mental hygiene. (3) Psychogenic, e.g., unsuccessfully repressed mental conflicts. (4) Precipitating, e.g., financial and business worries, frustrations and disappointments in sexual affairs, death of close relative, etc. (5) Organic, e.g, head injury, atherosclerosis, senile degeneration, myxoedema, pernicious anaemia, etc.

CLASSIFICATION (W.H.O.1965)

(I) PSYCHOSES: (A) Organic psychoses: (1) Senile and presenile dementia. (2) Alcoholic psychosis. (3) Associated with intracranial infections, e.g., epidemic encephalitis, abscess, meningitis, tuberculosis, G.P.I., etc. (4) Associated with cerebral arteriosclerosis, epilepsy, intracranial tumours, degenerative disease, brain anomalies, etc. (5) Associated with other physical conditions, such as endocrine, metabolic and nutritional disorders, systemic infections, drug intoxication, childbirth,etc. **(B) Functional psychoses : (a) Schizophrenia:** (1) Simple (2) Hebephrenic type. (3) Catotonic type. (4) Paranoid and other atypical or unspecified forms. **(b) Affective type:** (1) Involutional melancholia. (2) Manic-depressive. (3) Paranoid states. (4) Other atypical forms.

(II) NEUROSES: (1) Anxiety neurosis. (2) Hysterical neurosis. (3) Phobic neurosis. (4) Obsessive-compulsive neurosis. (5) Depressive. (6) Depersonalisation syndrome. (7) Hypochondriacal. (8) Unspecified neurosis.

(III) Personality disorders (psychopathic).

(IV) Sexual deviation.

(V) Drug dependence.

MENTAL SUBNORMALITY (oligophrenia): Amentia is retarded, incomplete or abnormal mental development. The term amentia has been replaced by mental retardation. It includes mental subnormality and mental handicap. It signifies a condition of retarded, incomplete or abnormal mental development. I.Q. is below 70. It is characterised by incomplete maturation of attention, perception, cognition and social adaptability. It results from environmental, genetic, endocrinal, metabolic, infective, toxic causes and birth trauma.

(1) Idiocy : Idiots are defined as persons so defective in mind from birth or an early age that they are unable to guard themselves from ordinary physical dangers. Their mentality does not exceed

that of a normal child of three years. Intelligence quotient (I.Q.) (Binet-Simon test) 0 to 20. Physical abnormalities, like microcephaly and mongolism, and malformations of brain and of other organs of the body are seen.

(2) Imbecility : Imbeciles are persons who are so defective in mind from birth or an early age, that they are incapable of managing themselves or their affairs. An imbecile child is incapable of being taught. Their mentality ranges from that of normal child of three to seven years. I.Q. 20 to 50.

(3) Feeblemindedness (moron) : In these mental defectiveness, not amounting to imbecility exists from birth or an early age and they require care, supervision and control for their protection. Mental age 6 to 11; I.Q. 50 to 75.

Intelligence Quotient (I.Q): It is the intellectual capacity of a person in relation to his chronological age, which is expressed as a percentage. Normal adult I.Q. is 90 to 110%.

PSYCHOSIS ASSOCIATED WITH ORGANIC DISEASES

Mental illnesses which arise from some structural brain damage are called organic psychoses. The organic psychoses are characterised by lability of mood, failure of memory, deterioration of intellect, irritability, irrational anger, confusion, loss of social inhibitions, etc. which may lead such persons into unacceptable behaviour and even sudden violence.

Dementia is a condition in which there is degeneration of mental faculties after they have been fully developed. Memory, intellect and judgement are impaired. (1) PRESENILE DEMENTIA: It may be seen before 65 years. Pseudodementia is associated with schizophrenia. (a) Alzheimer's disease. (b) Pick's disease. (c) Creutzfeldt-Jacob disease. (d) Huntington's chorea.

(2) SENILE DEMENTIA : It is caused due to arteriosclerosis and old age. It usually starts after 65 years. A feeling of loneliness, of being unwanted, loss of prestige and death of a close relative cause the onset of the disease. The patient is confused, judgement and memory is impaired. Delusions, hallucinations and emotional outbursts are common.

(3) CEREBRAL TUMOURS : Mental symptoms may occur at any stage of growth of cerebral tumours involving prefrontal, frontal, temporal and parietal lobes.

(4) CEREBRAL TRAUMA : It can precipitate any type of mental illness.

(5) DRUG-INDUCED PSYCHOSIS: Dependence on barbiturates, amphetamines, cannabis, heroin, cocaine, etc., leads to psychosis. Cocaine, LSD, amphetamines and mescaline can produce clinical symptoms similar to schizophrenia.

(6) TOXIC PSYCHOSIS : Heavy metals, such as arsenic and mercury may produce mental degeneration.

(7) DEFICIENCY STATES: Deficiency of cyanocobalamin (pernicious anaemia), nicotinic acid (pellagra), 5-hydroxytryptamine (phenylketonuria), and hypoglycaemia produce mental degeneration.

(8) GENERAL PARALYSIS OF THE INSANE: This is a chronic progressive condition leading to paralysis and dementia. It is usually associated with menigovascular syphilis and tabes dorsalis. There is a chronic psycho-organic syndrome characterised by temperamental and personality changes. The memory is impaired and thought retarded.

(9) EPILEPTIC PSYCHOSIS : Short transitory fits of uncontrollable mania occur. There is general impairment of the mental faculties with loss of memory and self-control. Auditory and visual hallucinations are followed by delusions of persecution. Moral sensibility is lost and sometimes they are dangerous to themselves and to others. There may be progressive dementia.

(A) PRE-EPILEPTIC INSANITY : Instead of epileptic aura, the patient may occasionally develop violent fits of mania or extreme depression of mind. Hallucinations and delusions are common during this stage, and such persons may commit assault or other criminal acts.

(B) POST-EPILEPTIC INSANITY : The stupor following epileptic fit is replaced by automatic acts, of which the patient has no recollection. The patient is confused and terrified by visual and auditory hallucinations and delusions of persecution, and may commit crimes like thefts, incendiarism, sexual assaults and murders. These crimes are involuntary, automatic and unpremeditated. The patient never attempts to conceal them at the time of committing, but may try to conceal them on regaining consciousness. Automatic action tends to be of the same type in each attack. The action is usually habitual, e.g., a man walks into a shop, picks up something and walks out again, afterwards being arrested for theft, or one who exposes himself in a public place and is arrested for indecent conduct; or a person cutting something may inflict incised wounds or kill a child; or a person accustomed to firearms may shoot somebody.

TWILIGHT STATE: In this condition the field of consciousness is narrowed for a short time, followed by amnesia. The patient may do some automatic act and is aggressive and suffers from visual hallucinations. It is seen usually in epilepsy and rarely in hysteria,

punchdrunkenness and head injury.

(C) PSYCHOMOTOR EPILEPSY (temporal lobe, masked or psychic epilepsy): In this, instead of epileptic fit, the patient suffers from temporary and transient seizure of maniacal excitement with loss of consciousness. The patient may commit crimes without any motive.

(10) ALCOHOLISM: (A) Alcoholic blackouts (B) Delirium tremens. (C) Alcoholic hallucinosis. (D) Korsakov's psychosis. (E) Delusions of jealousy.

FUNCTIONAL PSYCHOSES : Mental illnesses which have no neurological basis are called functional psychoses. The functional psychoses are characterised by disorders of thought which have no physical basis. (a) In schizophrenic psychoses, disorders of the thought process are dominant. (b) In affective psychoses, mood abnormality is dominant. This is a disease of hereditary origin affecting young adults and forms a major group of all psychiatric illnesses.

SCHIZOPHRENIA : It is a condition of split personality, in which the patient loses his contact with his environment. It is primarily a disorder of thinking (cognition). This disorder can be in form, stream, possession or content of thought. It is characterised by splitting of different psychic functions. (1) Disorders of behaviour: withdrawal from reality, preoccupation with the self (narcissism), attribution of feeling of strangeness to outside influence (depersonalisation), and feelings that his mind and body are under control (passivity of feeling). (2) Disorders of thought: confused thoughts leading to thought block, devious thinking leading to incoherence of speech often with newly formed words (neologism). (3) Disorders of affect: depression, elation, inappropriate moods, lability of mood, anxiety and blunting of emotions. (4) Delusions: of grandeur, paranoid, hypochondriac and influence. (5) Hallucinations: commonly auditory, sometimes visual and tactile. (6) Personality deterioration: affecting his work, family and social relationships. It is the commonest type of insanity in homicidal crimes, especially where the victim is a stranger. The impulses are not sudden and the crime is usually preceded by much complaining and planning.

(1) SIMPLE SCHIZOPHRENIA: It begins in early adolescence. There is a gradual loss of interest in the outside world from which he withdraws. There is an all-round impairment of mental faculties. He becomes emotionally flat and apathetic and has difficulty in forming social relationships. Complete disintegration of the personality occurs later.

(2) HEBEPHRENIA: It begins in adolescents or young adults. Thinking process is disturbed. Wild excitement, illusions, hallucinations and bizarre delusions are present. Often conduct is impulsive and senseless. Ultimately the whole personality may disintegrate completely.

(3) CATATONIA: This is characterised by alternating stages of depression, excitement and stupor; impulsive suicidal or homicidal attacks and auditory hallucinations are common. This phase lasts for few hours to few days followed by a stage of stupor which begins with lack of interest, concentration and general indifference.

(4) PARANOID SCHIZOPHRENIA (paranoia, paraphrenia): Paranoia is the mild form, and is common in males. Paranoid schizophrenia develops insidiously in the fourth decade. It is characterised by suspiciousness, delusions of persecution and auditory hallucinations. At first delusions are indefinite, but later they become fixed on some person. The patient usually retains his memory and orientation. When delusions affect his behaviour, he is often a source of danger to himself and others. In paraphrenia, delusions and hallucinations are present, but the personality is relatively intact.

(5) SCHIZO-AFFECTIVE PSYCHOSIS: This is an atypical type of schizophrenia in which there are mood disturbances. Attacks of elation or depression, unmotivated rage, anxiety, panic, etc., occur.

(6) Pseudo-neurotic Schizophrenia: It may start with overwhelmingly permanent neurotic symptoms.

AFFECTIVE TYPES: These diseases are of hereditary origin affecting young adults, and form a major group of all psychiatric illnesses.

Manic-depressive psychosis shows wide swings of mood from euphoric elation to deepest depression, quite out of proportion or often totally unrelated to external circumstances. The two extremes of these moods are hyperactive, excitant at one and depressive stupor at the other. The primary disturbance is of affect. It occurs periodically. Isolated attacks of mania and depression may occur in the same patient and some show attacks of one type only.

(1) MANIC PHASE: This is a condition of exaltation of the emotions and the intellect. (A) Acute mania: It is characterised by euphoria or irritable mood, excitement, loss of self-control, flight of ideas and great muscular activity. Mood is elated, attention is fleeting and there is high degree of distraction. (B) Hypomania: It is the mildest form in which there is an exaggerated sense of self-importance. Offences, such as petty theft, deception, indecent assault, and fraud may be committed.

(2) DEPRESSIVE PHASE (MELANCHOLIA): It is an intense feeling of depression and misery without any cause. The sadness of mood is reflected in posture,

movements and facial expression. He retires from his usual social activities, avoids friends. Suicide is well planned and is of great danger to the patient. He may kill relatives, especially dependent young children. Homicidal and suicidal tendencies co-exist. They have feelings of self-reproach and guilt, and marked psychomotor disturbances.

TYPES: (1) Endogenous. (2) Reactive and neurotic. (3) Involutional. (4) Puerperal.

NEUROSES: This forms a group of personality disturbances resulting from reactions to life situations. They may occur singly or in combination.

(1) ANXIETY NEUROSIS: It results from autonomic stimuli. The patient has spells of dyspnoea, choking, palpitation, insomnia, faintness, trembling, headache, chest pain, etc. The person is apprehensive and depressed and worries about his health. Intolerance of noise, children and spouse may be seen.

(2) HYSTERICAL NEUROSIS: It is common in young female, but may occur in old ladies whose nervous system starts degenerating. The symptoms commence when faced with unpleasant life situations. The patient shows deafness, blindness, loss of smell, anaesthesia, paraesthesia, paralysis, aphonia, etc. Convulsions may occur. In the dissociative type, change in the patient's level of consciousness and identity are observed.

(3) PHOBIC NEUROSIS: Phobias are specific fear which the patient recognises as irrational. They often act as defence mechanisms that protect the patient from unpleasant life situations. The different types are : acrophobia (high places), agoraphobia (open spaces), claustrophobia (closed spaces), necrophobia (dead bodies), nyctophobia (darkness), phonophobia (sound), pathophobia (disease), thanatophobia (death), toxiphobia (being poisoned), etc. It interferes with daily life.

(4) OBSESSIVE-COMPULSIVE NEUROSIS: Anxiety, depression and fear are present in varying degrees. Obsessional thought may be associated with a whole range of objects. Doubt may exist whether doors are locked or lights switched off.

(5) DEPRESSIVE NEUROSIS: It resembles manic-depressive psychosis but the degree of depression is less, and mental retardation, delusions and hallucinations are absent. There is risk of suicide.

PERSONALITY DISORDERS: The person is said to suffer from a personality disorder, when the behaviour is inappropriate to the situation. Psychopathic personality refers to a special derangement of the mind. The important features are: (1) Lack of normal conscience. (2) Absence of normal feelings for other people, such as love, affection, sympathy, etc. (3) A tendency to antisocial impulsive acts. (4) Failure to learn from experience and to be prevented from crime by punishment. (5) Freedom from any other form of mental disorder. The symptoms usually appear in early childhood, but become pronounced in adulthood, and gradually diminish in later life. Sexual perversions and crimes are common.

SEXUAL DEVIATION: It may be a symptom of an underlying psychiatric disorder, such as schizophrenia, senile dementia, personality disorder. The person indulges in sexual perversions and unnatural sex practices.

DRUG DEPENDENCE: Drug addicts have a wide range of personalities. Most of them are emotionally unstable, immature, impulsive, angry with society, and unable to achieve their goals or face difficult situations in life. About 10% suffer from psychopathic personality disorders.

DIAGNOSIS OF INSANITY

In typical case of insanity the diagnosis is easy, but in early stages, especially when he has no permanent delusions, and in borderline cases, the correct diagnosis becomes difficult. The object of the clinical examination is to form an opinion about the patient's mind and the degree of responsibility.

(1) Preliminaries: Note the name, age, sex, and address of person. Time of beginning and end of each examination should be noted.

(2) Family History: Enquire into the mental condition of the patient's parents and siblings and whether any of them suffered from chorea, epilepsy, or frank mental illness, etc.

(3) Personal History: (1) History of previous mental disease in the parents. (2) Factors connected with environment, such as parents and homes, over-protection as a child, rejection, strictness, inferiority complex, discrimination by parents, emotional maladjustments during childhood, emotional fixation during adolescence to parents. (3) Psychogenic factors, such as repression, emotional conflict and anxiety states. (4) Organic diseases, like cerebral vascular accidents, head injury, acute fevers, advanced renal and cardiac disease, senile degenerative condition, toxaemias, etc. (5) Drug dependence to opium, pethidine, barbiturates, alcohol, cannabis, etc. (6) Domestic difficulties. (7) Emotional shock. (8) Frustration in life, love, sex, etc.

(4) Physical Examination: (1) Observe the patient's manner of dressing and walking, bearing and gestures. (2) Examine for the presence of deformities and malformations in the head or body.

(3) Note the pulse rate and body temperature, both of which may be increased. (4) The tongue may be furred. (5) The skin is dry and wrinkled and the hands and feet moist. A complete and detailed physical examination should be carried out to exclude any disease.

(5) Mental Condition: The following observations should be recorded. **(1) General appearance:** Naked, dressed properly, improperly, dirty or clean habits, and facial expression, whether vacant, grimacing, mask-like or makes faces. **(2) Talk:** Mutism, aphonia, distraction, irrelevant, neologism (coining his own vocabulary), echolalia (repeating identical words uttered by another person), perseveration (repetition of an act monotonously), wandering speech and talkative. **(3) Speech:** Coherent, incoherent, aphasia, lalling, lisping, drawling, slurring, stammering. **(4) Writing:** Agraphia, flight of ideas, obscene or insulting language, unintelligible. **(5) Behaviour:** Stereotypy, prese-veration, mannerism, impulsive, lazy, stupor, automatic obedience, negativism, seclusive, echopraxia (copying all actions of another), etc. **(6) Mood:** Emotion, euphoria, joy, anger, elation, exaltation, apathy, irritable, touchy, etc. **(7) Memory:** Good, bad, concentration, appreciation, grasp, etc. **(8) Sleep:** Insomnia, hyposomnia, somnambulism, somnolentia. **(9) Walking and gait:** Stealthy, hurried, etc. **(10) Attitude and posture:** Proud, peculiar, over-erect, aggressive, worried, etc. **(11) Sex behaviour:** Towards same sex and opposite sex. **(12) Attention:** Attentive, inattentive, fluctuating, etc. **(13) Thought process:** Retardation, preoccupied, ambivalence, double orientation, power of orientation, etc. **(14) Thought content:** Delusions, hallucinations, illusions, obsession, self-consciousness, etc.

(5) Other Investigations: Additional evidence can be collected from pathological examinations of the blood, urine and CSF, and X-ray and electroencephalographic recordings.

Observation: The person should be kept under observation in a general hospital or general nursing home, or psychiatric hospital or nursing home, or in any other suitable place, which should not exceed ten days, but with the permission of the Magistrate, he may be detained for further periods of ten days, up to a maximum of 30 days.

Violent and criminal persons should be kept in a prison. The person should be watched during different times of the day, when he is alone, in company and while he is working, eating, reading

Table (21–3) Difference between real insanity and feigned insanity.

Trait	Real insanity	Feigned insanity
(1) Onset:	Gradual.	Sudden.
(2) Motive:	Absent, e.g., no history of commission of crime.	Present, e.g., commission of crime.
(3) Predisposing factors:	Usually present, e.g., history of insanity in parents, or of sudden monetary loss, grief, etc.	Absent.
(4) Signs and symptoms:	Uniform and present whether the patient is being observed or not.	Present only when conscious of being observed; variable and always exaggerated, and do not resemble any particular mental disease.
(5) Facial expression:	Peculiar, e.g., vacant look or fixed look of excitement.	No peculiarity; frequently changing, exaggerated and voluntary.
(6) Insomnia:	Present.	Cannot persist; patient sleeps soundly after a day or two.
(7) Exertion:	Patient can stand exertion of fatigue, hunger and sleep for several days without breaking down.	Cannot stand exertion for more than a few days and breaks down.
(8) Habits:	Dirty and filthy.	Not dirty and filthy.
(9) Skin and lips:	Dry, harsh.	Normal.
(10) Frequent examination:	Does not mind.	Resents for fear of detection.

or writing, and when he is unaware of the fact of being observed.

Certification: A certificate should not be issued after single examination. Three examinations on different days and different hours are usually recommended, with the possibility of more, because a person may behave peculiarly at a single examination , either due to the effect of drugs, or due to delirium caused by fever. The certificate should contain a clinical description of the patient, and indicate the reasons for the diagnosis of the specified disorder.

Feigned Insanity: Insanity may be feigned by criminals to evade sentence of death or long terms of imprisonment, by soldiers and policemen to leave the service, and by businessmen to avoid contracts.

THE MENTAL HEALTH ACT, 1987: It was enacted to consolidate and amend the law relating to the treatment and care of mentally ill persons, to make better provision with respect to their property and affairs, and for matters connected therewith. This Act repeals the Indian Lunacy Act, 1912. The Act has changed the following terms : (a) Psychiatric hospital is used instead of mental hospital, (b) Mentally ill person instead of lunatic. (c) Mentally ill prisoner instead of criminal lunatic.

Some of the main provisions are: (1) "Mentally ill person" is defined as" a person who is in need of treatment by reason of any mental disorder other than mental retardation." (2) The Central Government and State Governments have to establish an Authority for Mental Health for regulation, development, direction and coordination with respect to Mental Health Services. (3) Establishment and maintenance of psychiatric hospitals or psychiatric nursing homes can be done only with licence, which has to be renewed every five years. The licence can be revoked if the hospital is not being maintained according to the provisions of the Act. If a mentally ill person is received or detained against the provisions of the Act, the punishment is imprisonment up to two years and also fine. (5) For every psychiatric hospital or nursing home, the Government should appoint not less than five visitors, of whom at least one should be a medical officer. Not less than three visitors shall make a joint inspection of hospital or nursing home at least once in every month, and examine every minor patient, and as far as possible all other mentally ill persons and the order for the admission of, and the medical certificates, and shall enter their remarks in a book.

RESTRAINT OF THE INSANE

Restraint may be: (1) Immediate, and (2) Admission to an asylum.

(I) Immediate Restraint: This can be done in case of (1) an insane person who is dangerous to himself or to others, or who is likely to injure or wastefully spend his property or that of others, (2) person suffering from delirium due to disease, and (3) delirium tremens.

Immediate restraint is done under the personal care of attendants, e.g., by safely locking-up in a room. The consent of the lawful guardian of the insane person has to be taken, but if there is no time to take the consent and the insane person is dangerous to himself or to others, he can be immediately restrained. Such restaint is lawful only as long as the danger exists.

(II) Admission in Psychiatric Hospital : The following procedures are adopted.

(1) Admission on Voluntary Basis : Any major person, who considers himself to be a mentally ill person, may request the medical officer-in-charge of psychiatric hospital (or psychiatric nursing home) for admission and treatment. In case of a minor, the guardian may make such request. The officer-in-charge should make such inquiry as he may deem fit within 24 hours, and if he is satisfied that the person requires treatment as an in-patient, he may admit such person. This is the most common method.

(2) Admission Under Special Circumstances: A mentally ill person may not be able to express his willingness for admission as a voluntary patient. Such person can be admitted in a psychiatric hospital (psychiatric nursing home) for a period of ninety days, if an application is made by a relative or friend. The application should be in the prescribed form and should be accompanied by two medical certificates, one of which shall be by a medical officer in the service of Government. If the application is not accompanied by medical certificates, the officer-in-charge of the psychiatric hospital, can get the mentally ill person examined by two doctors working in the hospital.

(3) Reception Order on Application : The officer-in-charge of a psychiatric hospital can make an application to the Magistrate in case of a mentally ill person who is undergoing treatment under a temporary treatment order, if he is satisfied that

(a) the treatment is required to be continued for more than six months, or (b) it is necessary in the interest of the health and personal safety of the mentally ill person, or for the protection of others.

The husband or wife of the mentally ill person, or any other relative, can make an application in the prescribed form to the Magistrate. The applicant must be a major and must have personally seen the patient within fourteen days of the date of the application. The exact manner of relationship, and the circumstances under which the application is being made, and whether any previous application had been made for inquiry should be stated. The application should be signed and verified by the applicant. Two medical certificates should be submitted, issued by two medical practitioners, who must have separately examined the patient within ten days of the presentation of the application. One certificate should be from a medical practitioner in the service of Government. Each doctor should certify "that the alleged mentally ill person is suffering from mental disorder of such a nature and degree, that such person should be admitted in a psychiatric hospital, and that such admission is necessary in the interest of the health and personal safety of that person, or for the protection of others".

On receipt of application, the Magistrate must consider the allegations in the petition, and the evidence of mental illness as disclosed by the medical certificates. He can personally examine the alleged mentally ill person. If he is satisfied, he may pass a Reception Order immediately, or he may fix a date for consideration of the petition. If he fixes a date, he must give notice to the petitioner and to any person to whom in his opinion, notice should be given. On the date fixed, the petition must be considered in private in the presence only of the petitioner, the alleged mentally ill person, a representative of the alleged mentally ill person, and such other persons as the Magistrate thinks should be present. If the Magistrate is satisfied that it is necessary to detain the alleged mentally ill person in a psychiatric hospital, he passes a Reception Order (order for admission and detention) which is valid for 30 days. If he is not satisfied, he may refuse the application, giving his reasons in writing, a copy of which is supplied to the applicant. A certified copy of the Reception Order is sent to the officer-in-charge of the psychiatric hospital.

(4) Reception Order on Production of Mentally Ill Person Before Magistrate : (a) An officer-in-charge of a police station is authorised to arrest a wandering or dangerous mentally ill person, and produce him before a Magistrate. A wandering mentally ill person is one who wanders aimlessly, and a dangerous mentally ill person is one who because of his violent behaviour is dangerous to himself or others. The Magistrate must examine such person, send him to be examined by a medical officer, and make such inquiries as he thinks necessary, and if he is satisfied that such person is a mentally ill person, he may pass a Reception Order. If a relative or friend executes a bond for the proper care of the mentally ill person, the Magistrate may hand him over to the friend or relative.

(b) An officer-in-charge of a police station, or any private person can report to a Magistrate, if he believes that any person is mentally ill, and is not kept under proper care and control, or that he is **cruelly treated or neglected** by any relative or guardian. The Magistrate may order to produce the mentally ill person before him and summon such relative or guardian. The Magistrate may make an order for the proper care and treatment of the mentally ill person. If there is no one who is legally bound to maintain the mentally ill person and even otherwise, the Magistrate may pass an order for the admission of the mentally ill person.

In any area where a Commissioner of Police has been appointed, he can exercise or discharge, all the powers and functions of the Magistrate in cases of dangerous, wandering, cruelly treated or neglected mentally ill persons.

(5) Admission after Judicial Inquisition : If a person possessing property is alleged to be mentally ill, the District Court may order an inquisition upon application made by any relative. The Court, if it is satisfied, may order to admit such person in a psychiatric hospital.

(6) Admission of Mentally Ill Prisoner : A mentally ill prisoner can be admitted into any psychiatric hospital, by an order passed by an appropriate authority under Prisoners Act, (1900), Air Force Act (1950), Army Act (1950), Navy Act, (1957), or under Section 330 or 335 of Cr.P.C. (1973).

(7) Admission of an Escaped Mentally Ill Person : A mentally ill person escaping from a psychiatric hospital, can be retaken by any police

officer, or any officer or servant of the psychiatric hospital and readmitted into such hospital.

Discharge of a Mentally Ill Person : (1) Voluntary patients should be discharged within twenty-four hours of the receipt of request for discharge made by the patient or the guardian. (2) A mentally ill person who is admitted on an application by a relative or friend can himself, or a relative or friend can apply to the Magistrate for his discharge. The Magistrate after making an enquiry can either allow or dismiss the application. (3) The officer-in-charge of a psychiatric hospital can order in writing discharge of any inpatient (other than voluntary patient), on the recommendation of two medical practitioners, one of whom shall preferably be a psychiatrist. (4) A mentally ill person detained under Reception Order made on application, shall be discharged, if the person on whose application the admission order was made, applies in writing to the officer-in-charge. Such person cannot be discharged if the officer-in-charge certifies in writing that the person is dangerous. (5) If a person detained on Reception Order, is subsequently found by any judicial inquisition to be of sound mind, he is to be discharged.

MENTAL DISORDER AND RESPONSIBILITY

Responsibility, in the legal sense means the liability of a person for his acts or omissions, and if these are against the law, the liability to be punished for them. The law presumes that every person is mentally sound until the opposite is proved. The type and degree of mental disorder which shall free a person from civil or criminal responsibilities is greatly controversial. Section 328 to 339 of Cr.P.C. 1973, relate to provisions as to accused persons of unsound mind, including fitness to stand trial and the subsequent procedures.

CIVIL RESPONSIBILITY

The question of civil responsibility arises in the following conditions.

(1) Management of Property and Affairs of Insane : If any relative or friend of an alleged mentally ill person possessing property gives an application, the Court may direct inquiry whether the person is of unsound mind and incapable of managing his property and affairs. The medical evidence is given in the form of a certificate, which should state, "that insanity is of such a degree as to make him incapable of managing his property and affairs". In case of doubt, it is safer to give an opinion in favour of sanity. If on inquiry, a person is found incapable of managing his property and affairs, but is not dangerous to himself or to others, the Court appoints a manager to look after his property, granting him necessary power. The Court may order the sale or disposal of the lunatic's property or the payment of his debts and expenses. The Court may order a second inquiry, if it is reported that unsoundness of mind had ceased and will order all proceedings in the lunacy to cease, if it is satisfied that the lunacy has ceased.

(2) Insanity and Contracts : A contract is invalid if one of the parties at the time of making it was incapable of understanding what he was doing due to insanity. Contract entered into with a mentally ill person may be valid, if the other party can show that he did not know that the other party was mentally ill, and that the contract is a fair contract. Insanity which develops subsequent to the contract does not make it invalid, unless performance of services becomes impossible. A lunatic is responsible for the payment of the simple necessities of life, such as food, shelter, clothing, medical care, etc., but he is not responsible if the order is grossly excessive or unreasonable, or if the seller has taken undue advantage of his insanity. The mental disorder of partner does not itself dissolve the partnership, unless steps be taken for dissolution. A person who is usually of unsound mind, but occasionally of sound mind, may make a contract when he is of sound mind. A person who is usually of sound mind, but occasionally of unsound mind, may not make a contract when he is of unsound mind.

(3) Insanity and Marriage Contract : A marriage is considered invalid, if at the time of marriage, either party (1) is incapable of giving valid consent due to insanity, or (2) though capable of giving valid consent, has been suffering from such a kind or degree of mental disorder as to be unfit for marriage and procreation, or (3) has been suffering from recurrent attacks of insanity or epilepsy.

(4) The Competence of Insane to be a Witness: An insane person is not competent to give evidence if he cannot understand the necessity of telling the truth due to insanity. A person of unsound mind who suffers from delusions, but is able to tell what he has seen, and who understands the obligation of an oath, is competent to give evidence. An insane

person is competent to give evidence during the period of lucid interval.

(5) Consent and Insanity : Consent to certain acts like sexual intercourse or hurt is not valid, if such consent is given by a person who from unsoundness of mind, is unable to understand the nature and consequences of that act.

(6) Insanity and Testamentary Capacity : Testamentary capacity (testament=will) is the mental ability of a person to make a valid will. "Will" denotes any testamentary document (S.31, I.P.C.). The requirements for a valid will are as follows. A written and properly signed and witnessed document must exist. The testator must be major, and of sound disposing mind, at the time of making the will. Force, undue influence, or dishonest representation of facts, should not have been applied by others. A sound disposing mind is a mind which has capacity of recollecting, judging and feeling the relations, connections and obligations of his family and blood relations. **Holograph will** is one which is written by a testator in his own handwriting.

Doctors are sometimes called upon to witness the execution of the will of a sick person. The doctor should proceed in the usual way : physical examination, mental state including intelligence testing, and laboratory investigations. The testator is said to be of sound mind if he is capable of disposing of his property with understanding and reason. The following tests are recommended to find out whether the testator is of sound and disposing mind. (1) Ask the testator preliminary questions, e.g., about his relatives, their number and the degree of social contact with them; his opinions on family, friends and business partners; his age, politics and hobbies. (2) Ask general questions for testing awareness as regards time, place, etc. (3) Ask him about the nature, extent and value of his properties, and the manner of distribution desired by him. If any unusual or unjust distribution is to be made, find out whether it is intentional, and if so the reasons for it, and whether he is able to repeat the main provisions of will. (4) Test the patient's powers of concentration by simple sums of arithmetic, etc. (5) Ask all other persons to leave the room, and then ask the patient whether there was any pressure or influence on him by any one. The doctor should exclude any disease, infirmity, pain, strain, influence of drug or drink or any insane delusion, which are bound to affect a normally sound and disposing state of mind. The most common symptom of absence of legal capacity is impairment of memory. Prejudices, dislikes and hatred, however ill-found, or however strongly entertained cannot be classed as insane delusions. Dislike of ones relatives without reason is not necessarily proof of want of capacity. The most important thing to determine is whether at the time of making the will, the testator understood the business in which he was engaged, and knew how he wanted to dispose of his property.

(1) A person affected by an insane delusion can make a valid will, if the delusion is not related in any way to disposal of the property, or the persons affected by the will. (2) Persons can make valid wills during lucid interval. (3) A will is considered valid even though the testator committed suicide shortly after making a will, if there is no other evidence of mental disorder. (4) Persons of extreme age and feeble health with defective memory can make a valid will, unless their mind has become so impaired, that they are unable to understand its nature and consequences. (5) A person suffering from motor or sensory aphasia, agraphia (failure to communicate by writing), and alexia (failure to understand by reading), or who is blind, can make a valid will, if he knows what he does by it, if he can make clear by gestures that he wishes to make a will, and is able to understand the meaning of questions put to him in this connection. (6) Wills made by persons in extremis (at the point of death) may be regarded with suspicion, because at that time a clear mind is unusual. (7) A will executed by a dying person during delirium would be invalid. (8) Partial drunkenness does not invalidate a contract or will, but when drunkenness has caused a temporary loss of reasoning powers, the person cannot make a valid will.

CRIMINAL RESPONSIBILITY

A person may plead insanity to avoid : (1) conviction, if the accused was insane when the alleged crime was committed, (2) trial, when the accused is insane and cannot plead, and (3) capital punishment, when a condemned prisoner is insane. A person is "unfit to plead" or "insane on arraignment", or "under disability" to a charge of crime, when he suffers from such disease of the mind which prevents him from understanding the nature of proceedings in Court, from distinguishing between a plea of guilty and not guilty, from examining witnesses or instructing the lawyer on his

behalf, or otherwise making a proper defence, or from following the evidence intelligently. If insanity is proved, the accused person is found "not guilty", and is ordered to be kept in a psychiatric hospital or other suitable place of custody.

The law presumes that every person is sane and responsible for his actions. The defence has to prove that the accused is insane. The law also presumes that for every criminal act, there must be criminal intent or mind, **mens rea,** (mens=mind; rea=criminal) motivating it. **Actus reus** means the actual physical act causing death.

The following are the tests for determining criminal responsibility.

(1) Mc Naughten Rule (the right or wrong test; the legal test) : English Courts, in dealing with the responsibility of the insane in criminal cases, are guided by the rules laid down after the Mc Naughten trial in 1843. Daniel Mc Naughten a 29 year old Scotsman, was probably suffering from paranoid schizophrenia. For many years he had a delusion that spies sent by Catholic priests, with the help of Tories (the party then in power in England), were constantly following him, harassing him and hatching a conspiracy against him. He also, probably, had auditory hallucinations with Tories accusing him of crimes of which he said he was not guilty. Therefore, he decided to kill the Tories Prime Minister, Sir Robert Peel, making elaborate plans for the criminal act. On 20th January 1843, he shot Sir Peel's Private Secretary, Edward Drummond, in the back, mistaking him for the Prime Minister. During the trial, he admitted that he 'was driven to desperation by persecution'. Ten physicians (nine for defence and one for prosecution) found him insane. He was found 'not guilty on grounds of insanity', and was sent to Bethlem Mental Hospital for life. The verdict lead to unprecedented public outcry. Queen Victoria, summoned the House of Lords to a special session. The Lord Chancellor, Lord Lyndhurst put to a panel of 14 judges, 5 hypothetical questions designed to clarify the legal position. The answers, given on 19th June 1843, came to be known as the 'Mc Naughten Rules'.

The most important of these rules is as follows. "An accused person is not legally responsible, if it is clearly proved, that at the time of committing the crime, he was suffering from such a defect of reason from abnormality of mind, that he did not know the nature and quality of the act he was doing, or that what he was doing was wrong".

This legal test has also been accepted in India as the law of criminal responsibility and is included in Sec. 84, I.P.C. which is as follows : "Nothing is an offence which is done by a person, who at the time of doing it, by reason of unsoundness of mind, is incapable of knowing the nature of the act, or that he is doing what is either wrong or contrary to law"

COMMENT : Clearly Proved : The insanity must be directly related to the offence in such a way as to satisfy the Court that the mental abnormality had a direct causative relationship to the offence, and that the offence would not have occurred if there was no mental abnormality.

DEFECT OF REASON : It is necessary to show that the intellectual or cognitive faculties of the accused were so disordered, that his reasoning powers as to facts and actions were not functioning normally.

ABNORMALITY OF THE MIND: When an insanity defence is used it must be clearly established that a defect of reason resulted from the 'abnormality of the mind'. The 'abnormality of the mind' is a legal, and not a psychiatric concept. The English Law recognizes as abnormality of the mind any disease which is capable of producing mental dysfunction. The law is not concerned with the brain but with the mind, as the term is used in lay terminology meaning reason, memory and understanding. However, when mental dysfunction is attributable to external factors (e.g. alcohol and drugs consumed voluntarily), this is not called as the abnormality of the mind. It is usually assumed to mean one of the major functional or organic psychoses.

WRONG : A crime is an act declared by the law of the land to be an offence at a particular time. It is a behaviour which is in violation of the law and is punishable. A crime consists of two main elements. (1) The actus reus (i.e. the guilty act which is against the criminal law). (2) The mens rea (i.e. the intent). Both these elements must be present before the accused can be said to have committed certain crimes.

The rule concerns itself with the ability of the accused to distinguish between 'right' and 'wrong' with reference to the particular crime. If at the time of the commission of the crime, the accused had the capacity to know that his act was wrong, he will be fully responsible, even if he was mentally ill, and unable to refrain from doing the act at that time. If a person commits a crime under the influence of an insane delusion, he is judged as though the delusionary facts were real.

Examples : (1) If due to an insane delusion, a person thinks that another man is attempting to kill, and he kills that man in self-defence, he has no criminal responsibility. (2) If under the influence of an insane delusion, a person thinks another to be a wild animal and kills him, he has no criminal responsibility, because he does not know the physical nature of the act. (3) If under the influence of an insane delusion, a person thinks that he is the State Executioner, and that he has to execute the victim as a part of his job, he is exempt from punishment. (4) If under an insane delusion, a person thinks that another person has caused a serious injury to his character and fortune and kills him, he becomes responsible, because under the law no one can kill a person in revenge.

The defect of Mc Naughten rule is that, for deciding that a person is insane, only intellectual factors (reason) are taken into consideration, but not the emotional and volitional factors, delusional beliefs, hallucinations and the ability of the individual to control the impulses.

(2) DURHAM RULE (1954) : In 1954, Judge David Bazelon formulated the product rule or test in the Monte Durham case (Durham vs US) in the District of Columbia, USA. "An accused person is not criminally responsible, if his unlawful act is the product of mental disease or mental defect". The term mental disease referred to mental disorder while the term mental defect referred to mental retardation. In this the causal connection between the mental abnormality and the alleged crime should be established. This broad standard of causality, referred to as the 'but-for-cause' standard would free almost all defendants who could show any degree of mental disease or defect. A lot of confusion was caused by the terms 'product', 'mental disease' and 'mental defect'. The rule was discarded 18 years later in the Brawner case (US vs Brawner, 1972).

(3) CURREN'S RULE (1961) : "An accused person is not criminally responsible, if at the time of committing the act, he did not have the capacity to regulate his conduct to the requirements of the law, as a result of mental disease or defect".

(4) THE IRRESISTIBLE IMPULSE TEST (NEW HAMPSHIRE DOCTRINE): An accused person is not criminally responsible, even if he knows the nature and quality of his act and knows that it is wrong, if he is incapable of restraining himself from committing the act, because the free agency of his will has been destroyed by mental disease". The irresistible impulse has been criticized for several reasons. It has often been asked in such cases whether the impulse was strong (and irresistible) or the offender weak (not resisting the impulse voluntarily). This is a metaphysical question for which psychiatry as well as law have no answer. This test is never relied on as the sole test but is combined with the "right or wrong" test.

(5) THE AMERICAN LAW INSTITUTE (ALI) TEST (1972) : "A person is not responsible for criminal conduct, if at the time of such conduct, as a result of mental disease or defect, he lacks adequate capacity either to appreciate the criminality of his conduct, or to adjust his conduct to the requirements of the law.

As used here, the terms 'mental disease or defect' do not include any abnormality manifested only by repeated criminal or otherwise antisocial conduct. This accepts the theory of the combined Mc Naughten and irresistible impulse tests, which takes into consideration the impairment of volitional capacity and the impairment of the cognitions. However, it rejects both of these tests as too narrow. It points out that both of these tests demand that the impairment be complete. It is a giant step forward from Mc Naughten. Instead of knowing "the difference between right and wrong", the defendant is subject to the requirement of "appreciating" it. Instead of proving the act a "product" of disease, it has only to be shown that the disease resulted in a loss of "substantial capacity" to obey the law.

INSANITY AND MURDER : In criminal cases where insanity is pleaded as a defence, the defence has to prove it. The opinion of the medical witness must be based on his own personal observations. The doctor should obtain detailed history from the accused person and from other sources, and then carry out physical examination and investigations. The following factors which are helpful, should be taken note of : **(1) History :** History of the accused and his family with regard to mental disease **(2) Motive :** There is no motive in insanity. **(3) Preparation :** Prearrangement or preplanning are absent in insanity. **(4) Accomplices:** Not present. **(5) Nature of crime :** An insane person may kill several persons including his friends and relatives. **(6) Conduct of the criminal at the time of the crime :** An insane person does not try to destroy evidence. **(7) Conduct of the criminal after the crime :** An insane person may even notify police about the crime.

Sections 328 to 339 of Cr.P.C. deal with provisions as to accused persons of unsound mind including fitness to stand trial and the subsequent procedures. The law considers the state of mind only at the time of the alleged offence and not generally

The mental status of responsibility at the time of the alleged offence has to be determined. The capacity to stand trial is determined at the time of the actual trial. The interval between these two events may be days, months or years. The psychiatrist conducts the examination of the alleged insane person at some time after the alleged crime, and before the trial. The psychiatrist's task becomes difficult with the increase in the interval between the alleged crime and his examination. Psychiatric prediction has limited reliability. An opinion of the present mental state of an individual usually carries the lowest risk of error; an opinion about a recent past mental state has greater risk of error, and an opinion about the future carries the greatest error and least reliability. The greater the interval between commission of crime and psychiatric examination, the lower will be the reliability of the opinion for legal purposes. The opinion of the psychiatrist about the mental state of an accused at a previous period of time depends on his knowledge about the accused's present clinical condition, past history obtained from relatives and medical sources, his behaviour before, during and after the act in question, and his knowledge of, course and natural history of the disorder of mind of the accused. Therefore, at the very best the psychiatrist's views concerning the accused's degree of responsibility and self-control at the time of alleged offence will be matters of inference. If he finds evidence of frank psychosis, epilepsy or other illness accepted as disease or disorder of mind, he can justifiably infer that such illness played a part in the offence with which the accused is charged. There is probably greater difficulty in giving a defensible opinion with respect to a causal relationship between a retrospectively established mental illness and a particular criminal act. He will also be required to show that the accused either fulfils the requirements of the Mc Naughten Rules, or that his illness has caused a substantial impairment of his responsibility.

All the mental disorders do not free a person from criminal responsibility for his acts. If the disorders impair the cognitive faculties of the accused, i.e., the faculty of understanding the nature of his act and its consequences, he is not held responsible. Cognition includes all aspects of perceiving; thinking and remembering. If insanity affects only the emotions and the will, but not the cognitive faculties, the person is held responsible for his acts. The law recognises a guilty intent, the mens rea, as an essential part of the crime. Only those persons who are completely incompetent, demented or wild are considered to lack the ability to have a guilty intention. The most serious problem for Courtroom presentation of psychiatric and psychological evidence is the inherent uncertainty of the field itself. There is often a confusion between findings and opinions in the individual patient. Genuine disagreement between experts is common, even in areas of diagnosis and prognosis. When a medical witness deposes regarding the existence, characters, and extent of the mental disease, the Judge has to decide whether the disease justifies an acquittal on the ground of insanity. The question whether in given circumstances, a man was sane or insane is for the Court to decide.

Doctrine of Diminished Responsibility : Diminished responsibility is a term used for border line mental state. It recognises that there are different degrees of mental disorder, and in general a defence of diminished responsibility requires evidence of a state of mind bordering upon but not amounting to insanity. This includes certain organic states, depressions, obsessional states and some paranoid states.

The defence of diminished responsibility is usually applied for people with a lesser degree of mental abnormality (whether arising from a condition of arrested development or any inherent causes or induced by disease or injury) than could be brought within the Mc Naughten rules. On the other end, this defence is also not used for simulated insanity, mild character defects, bad temper, jealousy, hatred, drug use, racial characteristics, low intelligence, poor judgement, political fanatism and similar unfavourable personal feelings. Such person may be punished for culpable homicide not amounting to murder.

AUTOMATISM : Automatism is conduct that is performed by a person whose consciousness is impaired to such an extent, that he is not fully aware of his actions. There may be no consciousness at all of the actions in question, or there may be awareness that falls below the level of normal consciousness. It is an apparently purposeful, and complex behaviour which occurs without conscious control, and for which there is amnesia later. This alteration of consciousness may be produced by

organic factors or by non-organic factors, such as stress or shock. The main factors producing automatism recognised by the criminal Courts are: (1) Epilepsy (insane automatism). (2) Concussion or cerebral disease. (3) Hypoglycaemia, and (4) Somnambulism (non-insane automatism). However, the concept of automatism is very vague and unsatisfactory. The Indian law has no special provision for automatism.

SOMNAMBULISM : It means, walking during sleep. A person leaves his bed and walks in the house or out of the house without any awareness of his actions, but rarely injures himself. He is not asleep but in a state of dissociated consciousness, in a hallucinatory state, unrelated to his immediate environment. It is similar to an automatism. Such persons are usually well-adjusted in life, socially well-behaved, and not aggressive. A large percentage of adults have psychiatric problems attended with acute anxiety. There may have been immediate stress or concealed mental conflict preceding the walk. The crime is not wilful or premeditated. The mental faculties are partially active and are so concentrated on one particular idea, that he may solve a difficult problem or commit a crime, e.g., theft or murder. He may commit suicide, fall in a well or meet with an accident. There is no recollection of the event, but in some cases, the events of one fit are remembered in a subsequent fit and carried out similarly. Such person is not criminally responsible for his acts.

SOMNOLENTIA (semisomnolence) : It is often called sleep-drunkenness and is midway between sleep and waking. If such a person is suddenly aroused from a deep sleep, he may commit some crime due to confusion of the mind, especially when he is having a dream at that time. Such person is not criminally responsible for his acts.

Impulse : Some crimes are committed due to an impulse, in which the person loses self-control, such as sudden violent anger. Such persons are criminally responsible, unless insanity is present.

Hypnotism or Mesmerism : This is a sleep-like condition produced by artificial means or by suggestions. During a hypnotic trance, a person may perform acts suggested by the hypnotist, but does not remember them afterwards. A hypnotised person usually cannot be tricked into doing some immoral or dishonest act. Medical hypnosis is safe and is used in the treatment of many conditions of ill-health including states of depression and other mental disorders.

Delirium : A delirious person may commit criminal acts due to delusions and hallucinations. He is not legally responsible for the acts committed during delirium.

Drunkenness : An act done by a person, who is incapable of knowing the nature of the act due to intoxication is not an offence, if the thing which intoxicated him was administered to him without his knowledge or against his will (Sec. 85. I.P.C.). An intoxicated person (voluntary drunkenness) is criminally responsible, if he had the intention or knowledge of committing a crime (S.86, I.P.C.). Thus, if a person commits criminal abortion due to which the woman dies, the abortionist may be guilty of murder, for he intended to commit an unlawful act. Mental disorder brought about by drugs or delirium tremens due to drink, frees one of criminal responsibility.

If the evidence shows that a crime was committed due to violent passion due to the drunken condition, an inference can be drawn that the accused intended the natural consequences of his act. But, if the evidence shows that the accused was so much intoxicated as to be incapable of forming the specific intent, it may be a ground for altering the nature of the offence.

A person is not responsible for his criminal acts done during post-traumatic automatism, twilight states and oneroid states.

CHAPTER 22
ARTEFACTS

Artefact is any change caused or feature introduced in a body after death (accidental or physiologically unrelated finding to the natural state of the body), that is likely to lead to misinterpretation of medico-legally significant ante-mortem findings. The responsibility of medical jurist is very great. Often the doctor is the chief source of evidence upon which legal decisions are made, and the freedom or imprisonment, or the life or death of an accused person depends on his evidence. Therefore, the doctor should learn to draw conclusions logically and correctly, instead of forming hasty judgement. The autopsy pathologist should be able to distinguish them from the significant ante-mortem changes. Further, if the doctor misinterprets the artefacts, he will have a tough time in the Court during cross-examination, for a lawyer aware of these pitfalls, may attempt to discredit his evidence.

(A) ARTEFACTS INTRODUCED BETWEEN DEATH AND AUTOPSY

(I) Agonal Artefacts : (1) Regurgitation and aspiration of gastric contents is a common agonal artefact. It may be seen in natural deaths, as a terminal event, or due to handling of the body, or due to resuscitation. (2) One of the effects of asphyxia is to cause vomiting due to medullary suboxia. As a result, the air-passages may be filled at the end of asphyxial event by inhaled vomit. The findings, especially in infants should not be assumed to be the cause of asphyxia ; it is more likely to be the result. In case of choking, particles may be drawn into the bronchioles, which distinguish the condition from those cases in which food is forced up the oesophagus and falls in the larynx after death. (3) **Oesophagogastromalacia** is rarely seen in persons who die within hours or days after receiving severe head injury with cerebral damage. This occurs due to autodigestion; stomach contents are spilled into left chest cavity or left subphrenic area. The tissue affected is grayish-white to black and very friable. It may occur immediately before or shortly after death. It is an agonal or postmortem artefact.

(II) Resuscitation Artefacts : (1) The injection marks of resuscitation are usually found in the cardiac region or on the extremities. In intracardiac injection, heart may show contusion and blood may collect in the pericardium. Some of the injection marks may be associated with post-mortem bruises. (2) A defibrillator applied to the chest may produce a ring-like contusion. (3) External massage may cause bruising of the anterior chest wall, haemorrhage into subcutaneous tissues and pectoral muscles, fracture of several ribs and sometimes of sternum. The sternum fractures through the middle of the body and manubrium. There may be associated tears of the lungs with release of free air into the tissues. Haemorrhages in the lungs are usually associated with remote parenchymal and subpleural hexagonal foci of aspirated blood. There may be rupture of atria and ventricles. Lacerations of the liver occur due to direct manipulation of the liver area and also by a push and pull effect on the liver ligaments and the diaphragm. Less commonly, the spleen is lacerated due to the same mechanism. In most of these cases, fracture sites do not show haemorrhage and intercostal muscles do not show contusions. In such cases, the lungs show bone marrow or fat emboli in about 20% of cases. (4) Vigorous resuscitation with a thoracotomy and internal cardiac massage, produces air embolism. (5) When positive pressure breathing apparatus (respirator) is used for resuscitation, it produces acute emphysema, sometimes with subpleural blebs, air in the mediastinum or tension pneumothorax. The administration of oxygen by mask or tube may cause rupture of oesophagus and lung. (6) Contusions of soft tissues of the neck may be mistaken for homicidal strangulation. These resuscitation injuries may be mistaken for those due to assault or from streering-wheel impact injuries. In such cases, history is very important. (7) Damage to the mouth, palate, pharynx and larynx can occur from attempts to introduce a laryngoscope. Mouth-to-mouth breathing may cause contusions of the face, neck, and damage to the lips and inner gums, when the face and neck have been gripped by a hand.

(III) Artefacts due to the Handling of the Body: (1) Occasionally, fractures of the ribs or the bones of extremities, or of cervical spine may occur by rough handling of bodies, especially if there is severe osteoporosis. They are commonly produced

during attempts to straighten the limbs contracted due to rigor mortis. Bleeding is usually absent in such post-mortem fractures. (2) Contusion of the occipital region may be caused, if the head of corpse is allowed to fall on a hard surface during handling. (3) Fresh abrasions may be produced due to dragging of the body which were originally free from them, during the transfer of the body from the scene of crime. (4)Undertaker's fracture is a subluxation of the lower cervical spine due to tearing of the intervertebral disc at about C6–C7.

(IV) Artefacts Related to Rigor Mortis : (1) The handling of the body may cause breaking of the rigor at least partially, which may mislead the doctor in the estimation of the time of death. (2) The onset and duration of the rigor may be altered by atmospheric conditions like extreme heat or cold, or ante-mortem conditions like muscular state, exhaustion, wasting diseases, and hyperthermia due to infections. (3) Rigor affecting the heart may simulate concentric hypertrophy of the heart. (4) Rigor in stomach may accentuate the rugae or fix a point of contraction so as to give a pseudo-hourglass, which is readily removed by traction. (5) Rigor in pylorus causes it to be unduly firm and contracted.

(V) Artefacts Related to Post-mortem Lividity: (1) The colour of the post-mortem stains is usually bluish-purple. Certain poisons may change the colour of the hypostatic area, e.g., cherry-red colour in CO poisoning, bright-red colour in HCN poisoning, brown or chacolate colour in poisoning by nitrites, potassium chlorate and aniline, dark-brown colour in phosphorus poisoning. The post-mortem stains are of pink colour in bodies exposed to cold and in refrigerated bodies. (2) Patches of haemorrhage, sometimes quite large and confluent, can occur in the tissues behind the oesophagus at the level of the larynx. These lie on the anterior surface of the cervical vertebrae and are caused by distension and leakage from the venous plexuses that lie in this area, and can be mistaken due to strangulation (3) 'Banding' of the oesophagus may be seen especially when the tissues are congested. These bands are pale areas in the mucosa caused by post-mortem hypostasis being prevented from settling by the external pressure of adjacent anatomical structures, including parts of the larynx, trachea, and aortic arch. It is commonly seen in routine non-traumatic autopsies. (4) Large petechiae or ecchymoses, sometimes with raised blood blisters may be seen in the dependent skin of persons who have died a congestive death, or when upper part of the body hangs down after death. They are commonly seen over the upper part of front of chest and on the back of the shoulders. The face may show haemorrhages when the head is dependent.

(VI) Artefacts due to Burns : (1) Heat ruptures may resemble lacerated or incised wounds. (2) Heat haematoma may simulate extradural haemorrhage. (3) An unburnt groove around the neck due to tightness of the clothes, e.g. collar, may resemble a strangulation mark. In severely burnt bodies, fat droplets may be found in the pulmonary vessels, which should not be mistaken for ante-mortem pulmonary fat embolism. (4) Radiant heat reaching the body after death may cause loosening or drying and tanning of the skin.

(VII) Artefacts in Firearm Wounds : (1) Drainage wounds may be mistaken for firearm wounds. (2) In decomposition, there may be peeling of skin and loss of hair and gunpowder from the skin around an entrance wound. The margins of an entry wound may become ragged due to disintegration of the tissue at the margins, and it becomes difficult to distinguish entrance wound from exit. (3) Sometimes, unexpectedly a bullet may be found in the body, which may be densely encapsulated due to old injury, whereas the actual cause of death may be something else.

(VIII) Artefacts due to Animal and Insect Bites: (1) Rodents gnaw away tissue over localised areas. They produce shallow craters with irregular borders by nibbling and leave long grooves. (2) The bites by dogs are clear-cut, with deep impressions of teeth in small area. Individual punctures may resemble stab wounds. (3) Cat bites are usually very small and round. Face and neck are usually

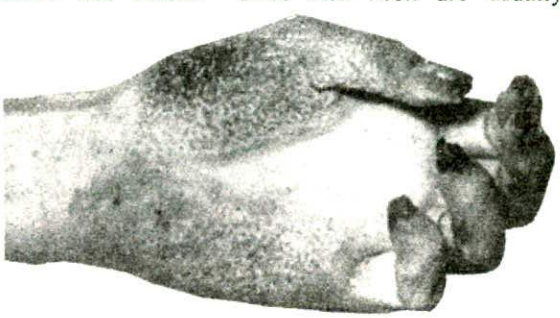

Fig. (22–1). Erosion of the skin by rodents.

involved in the recumbent body. They may sometimes resemble knife wounds, especially in bones. (4) Marks produced by insects (ants or roaches) are dry, brown with irregular margins, and are usually seen in moist parts of the body, e.g., ears, armpits, groin, scrotum, anus, etc. They resemble ante-mortem abrasions. As they become dry, they resemble brush burns. They do not show vital reaction. Extensive linear ant lesions around the neck resemble ligature abrasion. (5) Rarely, injuries, caused by crabs, may simulate stab wounds. (6) Post-mortem injuries produced from the bumping of the body into rocks, coral or marine structures should be distinguished from ante-mortem trauma. (7) Similarly, fractures caused by fall into the water from a height due to the body striking forcibly against some solid object, or mutilation from boat propellers, or loss of fingers, toes, eyelids, lips, genitals or rarely whole portions of the body occurring from the attacks of marine animals should also be distinguished. (8) Leeches, which become attached to the skin around the eyes, and detach when the body is removed from the water may produce haemorrhagic lesion simulating a black eye. (9) Crabs and other crustaceans, turtles and fish tend to gnaw the soft tissues around the eyes, ears, mouth, genitals, anus and edges of the surface wounds of the body. Any animal attacking the dead body usually selects those areas where the skin is broken. Ante-mortem wounds are thus greatly enlarged.

(IX) Artefacts in Brain : (1) Flattening of the convolutions of brain is seen in cases of oedema of the brain, which is generalised. Regional flattening of the cerebral convolutions is a post-mortem artefact, which is seen in those parts of the brain which are in contact with the cranium, especially the occipital lobes. (2) Grooving of the unci is seen in cases of raised intracranial pressure. Uncal grooving is also found in normal brains as an artefact.

(X) Artefacts in Liver : (1) The undersurface of the liver in contact with transverse colon shows greenish colouration due to putrefaction. (2) The liver surface is also stained due to bile.

(XI) Post-mortem Haemorrhage : (1) Before the blood clots, a post-mortem injury may damage a blood vessel and produce haemorrhage. (2) After death, blood may collect in the pleural cavities due to wounds produced on the chest wall and the lung tissue. Intercostal veins bleed much more than arteries. (3) After death, blunt impact may lacerate blood vessels and displace red cells into the tissue spaces. Patches of haemorrhage, sometimes large and confluent can occur in the tissues behind the oesophagus on the anterior surface of the cervical vertebrae, due to distension and leakage from the venous plexuses that lie in this areas.

(XII) Artefacts Related to Hair : The beard may appear to grow after death in some cases, whereas the growth of hair stops immediately after death. The cause of this post-mortem apparent growth of beard is the shrinkage of the skin, due to which greater part of the hair shaft is exposed above the epidermis.

(XIII) Artefacts due to Decomposition : (1) Intense localised lividity of skin due to hypostasis, or displacement of internal pools of blood by pressure of gases of decomposition produces pseudo-bruising which may simulate ante-mortem bruises. (2) Internal hypostasis with haemolysis of red cells may resemble haemorrhage, especially in the meninges, kidneys and retroperitoneal tissues. (3) In a dead body lying on its back, blood accumulates in the posterior part of the scalp due to gravity. In advanced decomposition, due to lysis of red cells and breakdown of the vessels, blood seeps into the soft tissues of the scalp. This appears as a confluent bruising and cannot always be differentiated from true ante-mortem bruising. (4) Bloody fluid may be found in the mouth and nose in decomposed bodies, which is marked in conditions which produce pulmonary oedema. In such cases, the cause of death should not be mistaken for haemorrhage. (5) Accumulation of blood in the tissues of the neck in drowning may simulate ante-mortem haemorrhage due to strangulation. (6) The blood becomes darker in decomposition, due to which the brain, lungs, heart, etc. appear congested, which may be mistaken for signs of asphyxia. (7) Due to decomposition, gases collect in the tissues, cavities, and hollow viscera under considerable pressure, and subcutaneous tissues become emphysematous. These changes may cause a false impression of ante-mortem obesity. (8) A deep groove simulating ligature mark of strangulation may be seen around the neck in decomposed bodies and also in mummification, if the deceased has been wearing buttoned shirt at the time of death. (9) Air in the right side of the heart due to decomposition may be mistaken for air embolism. Oxygen in right heart will indicate air

embolism, because it is not present in appreciable quantity, if the gases were those of decomposition. In cases of air embolism, the volume of air is much larger than that due to decomposition. (10) Blebs formed due to putrefaction may be mistaken for blebs from burns. (11) Regurgitated gastric juices may cause tanning of the skin of the face and neck, which may simulate ante-mortem burning. (12) Separation of sutures of the skull in a child due to gases of decomposition within the brain, and bursting of the abdomen with protrusion of the abdominal viscera due to advanced decomposition should not be mistaken for trauma. (13) In advanced decomposition, small miliary granules or plaques one to 3 mm. in diameter may be seen on serous or endothelial surfaces of the body, such as pleura, peritoneum, pericardium and endocardium. They resemble grayish-white colonies of bacteria growing on surface of an agar. They consist of calcium, fat, endothelial cells and bacteria, and should not be mistaken for inflammatory lesions or the effect of a poison. (14) Lungs may show post-mortem bacterial colonies. (15) The pancreas is one of the most vulnerable organs to post-mortem autolysis, because of the action of its own enzymes. At first it is softened and haemorrhagic. Later, it may resemble haemorrhagic pancreatitis. Histological changes of necrosis are usually seen within a few hours after death. Inflammatory reaction and fat necrosis are absent in such cases. (16) Fissures or splits in the skin formed due to putrefaction may simulate ante-mortem lacerations or incised wounds. (17) Small round holes produced by maggots may simulate bullet holes. (18) Excessive flaccidity of vaginal orifice with effusion of bloody fluid and spontaneous detachment of portions of mucous membrane of vagina may simulate ante-mortem sexual assault.

(XIV) Artefacts due to Chemicals : In automobile accidents or air-plane crashes, exposure to gasoline causes post-mortem detachment of epidermis. On exposure to air, the underlying dermis has a yellow to brown colour as drying occurs. They resemble thermal burns or abrasions. They occur particularly when the fire is put out with water.

(XV) Artefacts due to Refrigeration : Pink hypostasis is seen in bodies kept in cold storage. Post-mortem refrigeration of infants usually solidify the subcutaneous fat which produces a prominent crease where there was a normal skin fold of the neck, which resembles strangulation mark.

(XVI) Embalming Artefacts : (1) The trocar wound may simulate a stab wound. Some blood may be forced out of injured blood vessels due to pressure and collect in the tissues and may be mistaken for ante-mortem haemorrhage. (2) The skin tends to become transparent due to embalming and minor haemorrhages become visible, which may sometimes create problems. (3) A homicidal stab wound may be enlarged by the embalmer to approach an artery or he may pass a trocar through a gunshot wound. This will modify the dimensions of the wounds. (4) The trocar may also disturb the track of the weapon or bullet and produce false tracks.

(XVII) Interment and exhumation Artefacts: (1) In bodies which have been buried, fungus growth is usually seen at body orifices, eyes and at the sites of open injuries. After the removal of the fungus, the colour of the underlying skin resembles bruising. (2) Grave-diggers can produce post-mortem fractures, abrasions, and lacerations.

(XVIII) Toxicological Artefacts : (1) Faulty technique in collecting the sample, especially blood sample, can give false results. (2) When blood is collected from the heart with a long needle, it may be contaminated with stomach contents or regurgitated oesophageal contents. (3) If blood is contaminated with pericardial or pleural fluids, false results are obtained as regards alcohol, because significant diffusion of alcohol occurs after death, from the stomach to the pleural and pericardial fluid. (4) Certain anticoagulants used for blood, e.g., formalin, heparin, methenamine, and EDTA give a positive test for methanol. (5) Decomposition of the tissues after death produces ethyl alcohol and other higher alcohols. Many of the bacteria can produce alcohol, the values of which are less than 200 mg%. (6) Decomposition also causes an increase of concentration of CO in the blood up to 19%. (7) Significant amounts of cyanide are also produced due to decomposition. (8) In cases of death due to burns, significant amounts of cyanide may be found in the blood, possibly due to inhalation of hydrogen cyanide. (9) Many substituted phenols are found in decomposing tissues, especially P-hydroxyphenyl derivatives. (10) In buried bodies, arsenic may be imbibed from the surrounding earth. Keratin tissues absorb arsenic by external contamination due to which, the concentration in hair and nail may be

much greater than the concentration of arsenic in the contaminating fluid.

(B) ARTEFACTS INTRODUCED DURING AUTOPSY : (I) Air in Blood Vessels: Pulling of the dura in the sagittal line will cause the air to enter the blood vessels at the top of the brain. Similarly air may enter the veins of the neck during the reflection of the skin. This may lead to erroneous diagnosis of air embolism.

(II) Skull Fractures : Fractures of skull usually in the middle fossae may be produced due to partial sawing and forceful pull of the skull cap or due to partial sawing and then using chisel and hammer to loosen the skull cap. This may produce additional fractures or may cause extension of already present ante-mortem fractures. In such cases, differentiation between ante-mortem and post-mortem fractures may become very difficult.

(III) Visceral Damage : (1) Rough handling of the brain during removal may produce tears of the midbrain. (2) Rough handling of the liver during removal may produce tears of the diaphragmatic surface, which simulate ante-mortem lacerations. (3) If the neck structures are pulled too hard during autopsy to drag out the thoracic viscera, they may be torn, and also transverse intimal tears may be produced in the descending aorta.

(IV) Extravasation of Blood : (1) In case of suspected cranial injury, the body should be opened, and the cardiovascular system decompressed by opening heart before the head is opened. If blood has not been drained from vessels of the head, damage to the dura and the dural venous sinuses on removal of skull cap, may lead to an escape of blood into the subdural space, simulating an ante-mortem subdural haemorrhage. (2) Large blood vessels may be cut while opening the thoracic and abdominal cavities, and considerable amount of blood escape into the pleural and peritoneal cavities. (3) Air may be drawn back into the circulation and enter coronary vessels and give false impression of air embolism. (4) During autopsy, the handling of organs and the incision of the vessels may result in extravasation of blood into the tissues. (5) The removal of the neck structures *en block* as in routine autopsies, may produce artefacts in the neck tissues which resemble bruises seen in the case of throttling. Therefore, the neck structures should be dissected *in situ* and in a bloodless field.

(V) Fracture of Hyoid Bone : (1) When the tongue and neck structures are firmly grasped and pulled upon while removing the neck organs, the hyoid bone and thyroid cartilage may be fractured, especially in old persons. Surrounding the fracture regions, haemorrhages are not seen. (2) Osseous union between the segments of hyoid may be unilateral. Such unilateral mobility or artefact by dissection may lead to the erroneous impression of ante-mortem fracture.

(VI) Injury to Blood Vessels : While dissecting the neck structures, if toothed dissecting forceps is used, it may damage the intima of the carotid artery which resembles a tear, as is seen in case of strangulation.

(VII) Toxicological Artefacts : They may be introduced due to : (1) Contamination of viscera with stomach contents during autopsy, or by putting all the organs in one container, or by using contaminated instruments or containers. (2) Faulty technique in collecting the sample. (3) Faulty storage or use of preservatives.

CHAPTER 23

FORENSIC SCIENCE LABORATORY

Forensic science is the study and application of scientific examination and evaluation of evidence, for legal purposes. Forensic sciences include : (1) Forensic medicine. (a) Forensic pathology. (b) Forensic psychiatry. (2) Forensic toxicology. (3) Forensic immunology. (4) Forensic odontology. (5) Forensic anthropology. (6) Forensic police sciences. (a) Criminalistics, wherein evidence, such as blood stains, glass, soil, clothing and firearms is compared, identified, individualised and interpreted. (b) Questioned documents examination involves the scientific examination of handwriting, typewriting, printing ink, paper, or other aspects of a document for the purpose of determining various legal questions asked about the document. (c) Trace evidence. (d) Ballistics. (7) Other forensic science specialities, which include voice print examination, polygraph technology, fingerprinting, etc.

ORGANISATION : Such institutes should provide three major categories of service : clinical, pathological and laboratory. In addition, it should have stores, exhibit room, workshop and library. Usually only laboratory services are provided by the forensic science laboratory.

CLINICAL SERVICES : They include examination of victims of assault, sexual crime, drunkenness, etc.

PATHOLOGY SERVICES : They include chemical analysis, toxicology, serology, biology, photography, fingerprints, ballistics, etc.

MUSEUM : Every laboratory should establish a museum containing fingerprints, bullets and cartridge cases, tyre tread patterns, animal hair, soils, typewritten specimens, inks, rope and cordage, cloth, photographs of various crystal poisons, etc.

STAFFING : (1) Director, medical or scientific. (2) Clinical services. Physician and obstetrician. (3) Pathology services : Pathologist. (4) Laboratory services : Biologist, physicist, serologist, microanalyst, photographer, fingerprint expert, ballistic expert, etc. (5) Others : Librarian, liason officer.

Functions : (1) To examine, compare and evaluate physical evidence, so as to link a suspect to the victim, or to the scene of a crime. In most cases, the laboratory supplements the work of police investigator in order to convert suspicion into a reasonable certainty of either guilt or innocence. (2) Protection of the innocent, e.g., a person arrested for selling narcotics, is set free if the chemical analysis of the material shows it to be harmless. It determines facts, which are not subject to the bias and prejudice and other human failings of the eyewitness. (3) Training of the police investigators as to what constitutes physical evidence, how it is to be found, collected, preserved and delivered to the laboratory.

It is not a solution for all the difficulties that confront the police in searching out crime; it is merely an aid in crime detection. Its results are more often rather negative than positive.

Material : The items which are most commonly handled by the laboratory and which frequently serve as evidence are knives, blunt instruments, blood and seminal stains, chemical substances, poisons, fingerprints and footprints, hair, fibres, firearms, bullets, cartridge cases and wad, tools and tool marks, broken glass, paint chips, oil, grease, petroleum products, soils, clothing, pieces of papers, cigars, cigarette stumps, matches, documents and fragments of various materials. Sometimes, laboratory technicians are called to the scene of a crime to collect specimens with which investigating officers are not qualified to deal.

Criminal Investigation : All criminal investigation is concerned either with people or with material objects. Only people commit crimes but they invariably do so through the medium of objects. It is these objects that together constitute physical evidence. The term **physical evidence** (trace evidence) includes any and all objects, living or dead, solid, liquid or gas, and the relationship between all objects as they relate to the problem in question, e.g., a crime. A knife, burglar tool, firearms, bullets, blood and seminal stains, saliva, pus, milk, poisons, fingerprints, hair, fibres, glass, paint, oil, dust, signature, microscopic fragments of all types, bacteria and even an odour are all physical evidence. The microscopic evidence persists at the scene of a crime long after all the visible and obvious evidence has been removed, and may solve the problem. When there is even a reasonable chance of finding significant evidence, it should never be neglected, even when the crime was committed long back. Physical evidence is useful in two ways :

(1) It is often the decisive factor in determining guilt or innocence. It can do this by supplying the demonstrable facts, thus resolving discrepancies in ordinary testimony. (2) It can be a material aid to link a suspect, a weapon or a scene to a crime. Evidence should be marked or labelled so that it can be positively identified. Date, time, place, from whom and by whom it was taken or found should be recorded. The chain of evidence must be intact and complete. Evidence should be preserved in the same condition in which it was found. The forensic scientist must routinely work with forensic scientists of various other disciplines in the investigation of a criminal matter or civil dispute.

Locard's Exchange Principle : When any two objects come into contact, there is always a transfer of material from each object on the other. Traces from the scene may be carried away on the person or tools of the criminal, and at the same time, traces from all or any of these may be left at the scene. Wherever a criminal goes, whatever he touches, and whatever he leaves will serve as silent evidence against him, e.g., fingerprints, footprints, hair, fibres from clothes, broken glass, tool marks, paints, scratches, blood or seminal stains, etc. It is actual evidence, and its presence is absolute proof of the crime. The evidence of eyewitnesses may be wrong as a result of their partisanship, faulty memory, or defective observation. Physical evidence cannot be wrong and completely absent. Only its interpretation can be wrong. Only human failure to find it, study and understand it, can diminish its value. The laboratory must be devoted to this study and understanding. Large numbers of criminals escape because the physical evidence is not fully understood and utilised. More laboratory failures are due to inadequate collection of existing evidence, than are caused by the failure of the laboratory to examine it properly. All laboratory findings are related to a probability, and a single piece of evidence is rarely sufficient in itself to establish proof of guilt or innocence.

The laboratory functions in three main categories: (1) RECONSTRUCTION : The police investigator can reconstruct the events leading up to, during and sometimes preceding a crime in most cases. The laboratory helps to make these events more clear. Usually, an investigation cannot be effective without such reconstruction of events from physical evidence. The arrest and conviction of a criminal would be difficult if the investigator fails to know what happened at the scene of crime. (2) Corpus Delicti: When a substance is confiscated and the holder is charged with possession of narcotics, the detection of the substance by laboratory analysis will establish the body of crime. (3) CONNECTIVE-DISCONNECTIVE MODE : If certain material found on the suspect are also found at the scene of crime and vice versa, and of common or similar origin, it will connect the suspect to the crime. If there is no similarity in physical evidence collected from the suspect and scene of crime, it will aid in disconnecting the suspect from the crime.

The following are some of the illustrations of the usefulness of forensic science laboratory in criminal investigations. The following paragraphs should be correlated with the appropriate chapters.

(1) PERSONAL IDENTITY : The main problem of the criminal investigator is the establishment of personal identity of the criminal. Fingerprints, foot prints, hair, blood, semen, etc., are unique to the individual.

The criminal may be identified indirectly through the tool he used, the gun he fired, the clothes he wore, writing he made, the soil, glass, paint, etc., he removed from the scene of crime. No two objects are ever completely identical. In physical evidence the term identity must be understood to signify practical and determinable identity only.

(2) BLOOD : In murder, assault, rape, etc., blood from the victim may be present at the scene of the crime, and on the person and clothing of accused and weapons. The distribution and appearance of bloodstained areas on the victim and his clothing may be used to interpret and reconstruct details of the crime. The criminal may be injured in the course of struggle or accidentally either by fall while moving in the dark, or by protruding nail, or broken window glass, in the act of breaking into a house. Blood groups are very useful in cases of disputed paternity.

(3) SEMEN : Stains may be found on the clothes of the accused and victim, pubic hair and person, on the bedding, mattress, floor or ground on which the offence was committed or on the piece of cloth used by the culprit or the victim for wiping after the offence. Ultraviolet light is useful in fluorescence tests, such as examining stains on garments.

(4) FIREARMS : A bullet recovered from a dead body can be examined to determine the type of gun which fired it , and the type of ammunition fired. By careful study of the markings on the bullet, the gun which fired it can be determined.

(5) FINGERPRINTS : A criminal can be identified especially by means of latent prints left at the scene of a crime, on a weapon, or in another incriminating location. Fingerprints are also useful in identification

of dead bodies, persons suspected of operating under aliases, amnesia victims, etc.

(6) HAIR : Traces of certain elements are deposited in our hair because of diet, drug intake and atmospheric conditions. The proportions of these differ considerably in different persons and these can be measured through neutron activation analysis. Hair from a criminal may be pulled out by objects at the scene. Similarly, hair from the victim may adhere to the criminal's person, clothing or weapon. In a sexual offence, hair from the victim may be found on the genitals of the accused and vice versa. Hair found sticking to a motor vehicle involved in an accident are useful in the identity of vehicle. Animal hair are very important in case of bestiality, and cattle thefts.

(7) FIBRES : The fibres may be of animal, vegetable, mineral, and synthetic origin. A crime against person often involves contact between the criminal's clothes or weapon on one side and the victim's clothes on the other. Even in burglary, or theft, the criminal often handles or touches several objects in the premises, due to which clothing fibres are transferred from the criminal to the scene of crime and vice versa, because the clothes constantly carry loose fibres. If it is found that a fibre from one source exactly matches one from another source, there is definite probability that the two sources have come into contact with each other.

(8) POISONS : In a suspected case of poisoning, the identification of the poison is necessary. It must be remembered that the presence of injuries or a disease sufficient to account for death does not rule out the possibility of poisoning.

(9) WEAPONS AND TOOLS : A wide variety of tools are used in the commission of crimes, e.g., knives, screwdrivers, bars, saws, pliers, cutters, hammers, drill, etc. Some of these leave marks which are very characteristic, and by which the tool may be quite accurately identified. The tool may also carry traces from the scene in the form of paint, oil, particles of wood or scratches from nails or other hard objects, and these may lead to the detection of crime. The examination of the wound will indicate the type of the weapon used, and the microscopic evidence can connect the weapon with the perpetrator or perhaps even with wound.

(10) CLOTHES : Fibres, paint, grease or dust may be found on the suspect's clothes in a burglary, and stains of semen or blood on the clothes of both the victim and the assailant in sexual offences.

(11) GLASS : In hit-and-run traffic accident, cyclist's rear lamp may be broken, the glass fragments of which may be found on bumper or other parts of the vehicle. Also, traces of glass from the headlamp of the vehicle may be found at the scene. The burglar in trying to enter the house may break window glass, the fragments of which may be carried in his clothes, etc. The refractive index, specific gravity and exposure to ultraviolet light of the glass fragment help to identify their probable source. The composition of glass can be checked chemically or by spectroscopic examination.

(12) WOOD : If a piece of wood from the handle of the tool used by the criminal is found at a scene, it can be identified by matching it with the handle of the tool seized from the suspect. Particles or splinters of the wood found on the suspect's clothes or tool should be compared with the wood of the door or window broken for the forced entry. Faults, marks, bruises and other individual pointers assist in matching a piece of wood with another piece from which it has been separated. Paint and external factors also indicate a relation between broken pieces. Microscopical examination of the cell structures is useful in identification of sawdust.

(13) METALS : Most evidence which is metallic in nature is in the form of tools and weapons. Like wood, metal pieces from the tool used by the criminal may be found at the scene of the crime, and metal fragments from the door and window fittings, and from the boxes and safes, may be recovered from the criminal's clothing or tool. Metallic fragments can be examined chemically or with the spectroscope and can be identified wih a specimen sample of the metal.

(14) TOOL MARKS : The two main types of tool marks are compression and scrap marks with a combination of both and also cutting marks. Every tool has its own peculiarities and the wear causes an individuality which are transmitted to the object on which the tool has been used. The density, pigment distribution, spectrographic analysis to determine the chemical composition of the mineral constituents of paint samples, and microscopic examination give positive proof.

(15) PAINT : In a road accident, flakes of paint from the vehicle may be found on the ground or on the person, animal or object hit by it, and traces of paint from the object may be seen on the vehicle. A burglar may carry on his body, tool or clothing, paint from the wall or doors of the house which he has burgled or from the safe which he has broke open.

(16) DUST AND DIRT : A criminal invariably carries soil in varying quantities on his feet or footwear, fom the earth on which he has walked, and on his body or clothing, from the ground on which he has lied down during the commission of the crime, or fallen in the course of a struggle. Soil usually consists of

mineral constituents, decomposed organic matter, broken leaves, pollen, grains, etc., capable of direct identification.

(17) VEGETABLE MATERIAL : Anyone committing a crime out of doors is likely to get plant material on his clothing, the identification of which will connect him with the scene. Algae and fungi are usually found on damp walls, in buildings, the soils, on vegetation and on domestic articles. The burglar climbing the damp wall or fallpipe may get smears of green algae. Seeds, portions of leaf, bark and other vegetable fragments are useful and can be identified to belong to particular areas of country. Grass fragments, particularly uncommon types, pollen, weeds and seeds are identifiable with their particular source of origin.

(18) STRINGS AND ROPES : The criminal may have brought his tools to the scene tied up in a bundle with string or cord, and may have left it behind. If it is identical in structure, size, shape, and appearance with another found in the suspect's possession, it is of great value.

(19) TYRE MARKS : It may be possible to trace a car by means of its tyre marks. A tyre mark should be compared with a test mark and not directly with the tyre.

(20) DOCUMENTS : They are physical evidence like blood, hair, glass, etc. Questioned documents may necessiate : (1) Physical and chemical examination including a study and identification of: (a) writing materials, e.g., paper, pen, ink, pencil, typewriter, (b) erasures, obliterations, and alterations, (c) order and age of writing, typing or other markings, (2) identification of the authorship of the writing. The individual should be asked to write something for the purposes of comparison with a questioned document. Infrared light will assist in the examination of closed letters, questioned documents, etc.

(21) PHOTOGRAPHY : Photography provides life-like reporductions which serve to refresh memory, and are a useful evidence. In the forensic sciences photographs are used (1) as a means to record a phenomenon observed, and (2) to reveal that cannot normally be seen. The first category includes the recording of simple matching techniques, photomicrography, and photomacrography. The second category includes the effect of infrared and ultraviolet radiations which helps in seeing things which are not seen in ordinary light, such as faint letter marks, and the production of radiographs using X-rays.

(22) LIE DETECTION: (A) **Polygraph** : It is an instrument used to detect lies. Keeler polygraph, and Stoelling deceptograph are in common use. Polygraph makes a continuous record of blood pressure, pulse, respiration and electrodermal reaction changes in response to stimuli in the form of questions. It is based on the theory, that when the person tells a lie in answer to a question, and there is fear that lie will be detected, the emotion of fear results in stimulation of sympathetic nervous system which results in certain physiological changes, some of which may be easily recorded. There is relative rise in blood pressure and recovery, slowing down of the breathing, and many times suppression and a relative change in the skin resistance of the individual.

In pre-test interview, the test questions are framed with the mutual consent of the subjects and to the satisfaction of examiner, that they are adequate to serve the purpose of the particular examination. A basic explanation of the attachments in the polygraph is given to the subject. An attempt is made to answer the subject's questions regarding the procedure. The questions are framed in such a way that they are clearly understood by the subject and they call for only 'Yes' or 'No' as answer. The questions usually number ten. Relevant and irrelevant questions are mixed up. The control questions are put to reduce the natural nervousness, the natural stigma of the issue at stake, and the natural slight resentment of the accusatory nature of the matter involved in the investigation. A question is asked every 20 to 25 seconds, and polygraph chart recorded in 3 to 4 minutes. Usually, the same test is repeated twice or thrice as a check on any possible error. An experienced and competent polygraph examiner can correctly detect truth or lie in about 80 to 90% cases. The few errors that do occur favour the innocent, since the known mistakes in diagnosis almost always involve a failure to detect lies of deceptive subjects. Offenders, suspects, complainants, witnesses and informants are examined by this method to test truth of their statements. It is also useful in civil cases, e.g., paternity cases, insurance claims, pre-employment screening by banks and other institutions.

(B) Narcoanalysis : ("truth serum" drugs): This is based on the principle, that at a point very close to unconsciousness, the subject will be mentally incapable of resistance to questioning, and incapable of inventing the falsehoods that he has used to conceal his guilt. The methods used are : (1) Half mg. of scopolamine hydrobromide, s.c., followed by one-fourth mg. every twenty minutes, for an average

of 3 to 6 injections, until the subject reaches the proper stage for questioning. (2) Sodium amytal or sodium pentothal (truth serum) 2.5 to 5% solution i.v. at a rate not to exceed one ml., until the proper stage is induced. (3) 0.1g. sodium seconal, one and half hours before induction; 45 minutes later 15 mg. morphine sulphate and half mg. scopolamine hydrobromide are given s.c To save time all three drugs may be given intravenously. Person loses inhibitions and becomes talkative. It depresses CNS, lowers B.P. and slows heart rate. In a state of relaxation, the suspect is susceptible to suggestion and reveals repressed feelings or memories. Large number of false negatives are common.

(C) Hypnosis : It may have a definite value in reviving the memory of the cooperative subject. Some people under the influence of hypnosis are able to recall incidents and details that they had long forgotten.

(D) Word Association : Changes in reaction time of the subject's reply to word stimuli, either visual or auditory, or by stereotype of answers, or by exhibition of uncoordinated physical movements, have been employed in attempts to detect deception.

BRAIN MAPPING: (Brain fingerprinting) In brain mapping the architectural details including the structure and function of the normal and abnormal human brain are generated. It uses a multidisciplinary approach involving brain imaging, neuropsychology, clinical neuroscience, computer science and bioinstrumentation, to integrate information on a scale ranging from whole brain structure to the microscopic level. Modern brain scanning technique consists of electroencephalography (EEG), magneto encephalography (MEG), positron emission tomography (PET), magnetic resonance imaging (MRI and functional MRI) and computed tomography (CT). It is developed by Dr. Lawrence A. Farwel of U.S.A. The equipment called **"electro-cap"** is fixed on the suspect's head. The suspected person is questioned about the crime and also shown the visuals of the crime scene (victim, weapon, time, place and how he committed the crime, etc. along with irrelevant words, photographs, etc.) to stimulate his brain and encourage a reaction on a computer monitor. Apart from his verbal replies another computer keeps track of the neuro impulses (brain waves, chemical responses) emitted whenever the visual is seen. The intensity of the brain waves shoots up whenever a quesion or the visual stimuli matches the information stored in the brain, if the suspect is really the perpetrator of the crime. This can be tracked and printed to provide the best scientific evidence. It is said to be more accurate than polygraph test.

SECTION II

TOXICOLOGY

CHAPTER 24

GENERAL CONSIDERATIONS

Toxicology is the science dealing with properties, actions, toxicity, fatal dose, detection estimation of, interpretation of the result of toxicological analysis and treatment of poisons. **Forensic toxicology** deals with the medical and legal aspects of the harmful effects of chemicals on human beings. **Poison** is a substance (solid, liquid or gaseous), which if introduced in the living body, or brought into contact with any part thereof, will produce ill-health or death, by its constitutional or local effects or both. The definition of poison is vague and unsatisfactory for (1) a substance which is harmless in small quantities may act as poison, and cause death when taken in large amount, and (2) bacterial toxins are not regarded as poisons in ordinary sense of the term. **Clinical toxicology** deals with human diseases caused by, or associated with abnormal exposure to chemical substances. **Toxinology** refers to toxins produced by living organisms which are dangerous to man, e.g. poisonous plants, the venom of snakes, spiders, bees, etc. and bacterial and fungal toxins.

HISTORY: The early history of poisons is described in the ancient Indian Shastras, Egyptian Papyri, Sumerian, Babylonian, Hebrew and Greek records. Menes Pharohs I (3000 B.C) is said to have studied many poisons. Atharva Veda (1500 B.C.) describes poisons. In Kalpasthana, Chikitasasthana, and Uttarasthana of the Shastras, symptoms and antidotes of poisons are given in detail. Susruta (350 B.C.) described how the poisons were mixed with food and drink, unguintment oils, perfumes, medicines, bathing water, snuff, or sprinkled over clothes, shoes, beds, jewellery or put in the ears, eyes, etc. Kautily in his Arthashastra (about second century B.C) states that the art and science of poisoning was extensively studied as a separate branch, and used both as an offensive and defensive measure against the enemy. Gradually, there arose a class of 'professional poisoners', who could ingeniously mask the bitter taste or strange odours of the poisons with sweet-tasting and pleasant substances. The Ebers Papyrus (1500 B.C) contains information extending back many centuries. Of the more than eight hundred recipes given, many contain recognised poisons, such as hemlock, aconite, opium, lead, copper and antimony. Hippocrates added a number of poisons in fourth centurey B.C. in Greek medicine. In the mythology and literature of Greek history many references to poisons and their use are found. Theophrastus (fourth century B.C) included numerous references to poisonous plants in De Historia plantarum. Dioscorides, a Greek physician, in the Court of Emperor Nero, attempted at a classification of poisons which remained a standard for sixteen centuries. The Greeks and later the Romans made considerable use of poisons, often political. In 82 B.C. Sulla issued the Lex Cornelia in Rome, which appears to be the first law against poisoning and it later became a regulatory statute directed at careless dispensers of drugs. In the seventh, eighth and ninth centuries Avicenna. Rhazes and Jaber, the Arab pharmacologist-physicians developed the art and science of pharmacology and therapeutics. With the spread of Muslim rule over Eurpoe and Asia, the use of this science also spread. Bhoja-prabhanda (980 A.D.), has a reference to the inhalation of medicaments before surgical operations, and an anaesthetic called "sammohini" is said to have been used in the time of Buddha.

The Italians brought the art of poisoning to its zenith prior to the Renaissance period and extended into that period. Paracelsus created the basic scientific discipline of toxicology in the late Middle Ages. Orfila (Spanish chemist, 1787 to 1853), was the first to attempt a systematic correlation between the chemical and biologic information of the poisons known then. He improved on the work of others and carried on his own experimentation on animals. He pointed to the necessity of chemical analysis for legal proof of lethal intoxication and he devised methods for detecting poisons. He published several books on poisoning. At about the same time Marsh, Magendie, Tardieu, Ambrose Pare, Stas, Scheelle and Reinsch made valuable contributions. Robert Christison (1779 to 1882) was markedly influenced by Orfila and produced a major work on poisons (1845). Rudolf Kobert, also wrote a text book on toxicology in the style of Orfila (1893).

THE LAW ON POISONS:

(1) THE DRUGS AND COSMETICS ACT, 1940: It regulates the import, manufacture, distribution and sale of all kinds of drugs. One of its main features is the control of the quality, purity and strength of drugs. Any patent or proprietary medicine should display on the label or container, either the true fromula or a list of ingredients contained in it. This Act empowered the Central Government to form a Drugs Technical Advisory Board, and to establish a Central Drugs Laboratory, to help and advice both the Central and State Governments. This Act was amended in 1964.

(2) THE DRUGS AND COSMETIC RULES 1945: They were framed under the Drugs Act, 1940, to regulate the importation of drugs, the functions and procedures of the Central Drugs Laboratory, the appointment of licensing authorities, and the manufacture, distribution and sale of drugs. These rules have classified drugs into Schedules as follows. Schedule C: Biological and special products; E: List of poisons; F: Vaccines and sera; G: Hormone preparations; H: Drugs (poisons to be sold only on the prescription of a registered medical practitioner); J: List of diseases for the cure of which no drug should be advertised; L: Antibiotics, antihistaminics and other recent chemotherapeutic agents.

The supply of any drug on a prescription must be recorded at the time of supply in a prescription register specially maintained for the purpose. The following particulars must be entered. (1) Serial number of the entry, (2) the date of supply, (3) the name and address of the prescriber, (4) the name and address of the patient, (5) the name of the drug or preparation and the quantity, (6) the name of the manufacturer, the batch number and the date of expiry of potency in the case of Schedule C and L drugs, and (7) the signature of the qualified person supplying the medicine. Both Schedule H and L drugs must not be sold by retail, except on a prescription by a registered medical practitioner.

(3) THE PHARMACY ACT, 1948: It was passed in order to make better provision for the regulation of the profession of pharmacy and to constitute Central Council of Pharmacy and State Councils of Pharmacy. The object of this Act is to allow only the registered pharmacists to compound, prepare, mix or dispense any medicine on the prescription of a medical practitioner. This does not apply to the dispensing by a doctor for his own patients.

(4) THE DRUGS CONTROL ACT, 1950: It provides for the control of sale, supply and distribution of drugs, the issue of cash memo for sale, marking of prices, and exhibiting list of prices and stocks. It gives power to fix the maximum price of any drug, which may be charged by a dealer or producer.

(5) THE DRUGS AND MAGIC REMEDIES (Objectionable Advertisement) Act, 1954: The object of the Act is to ban advertisements which offend decency or morality, and to prevent self-medication and treatment which cause harmful effects. Advertisements of magic remedies for procuring abortion or prevention of conception, increase of sexual potency, correction of menstrual disorders, and treatment of venereal diseases is completely prohibited.

(6) NARCOTIC DRUGS AND PSYCHOTROPIC SUBSTANCES (NDPS) ACT, 1985: It was amended in 1989 and 2001. It repeals three acts: (1) The Opium Act, 1857. (2) The Opium Act, 1878. (3) The Dangerous Drugs Act, 1930. The Act consolidates and amends the existing laws relating to narcotic drugs, strengthens the existing laws relating to narcotic drugs, strengthens the existing controls over drugs of abuse, enhances the penalties particularly for illegal trading offences, makes provision for exercising effective control over psychotropic substances, and makes provision for the implementation of international conventions relating to narcotic drugs and psychotropic substances. A psychotropic drug is one that alters mental function by its action. A narcotic drug means Cocoa leaf, Cannabis, Opium, Poppy straw and includes all manufactured drugs. "Psychotropic substance", means any substance, natural or synthetic, or any natural material or any salt or preparation of such substance or material, included in the list of psychotropic substances specified in a Schedule to the Act. This schedule lists 77 psychotropic substances, e.g., LSD, amphetamine, tranquilisers, barbiturates, LSD, benzodiazipines, methaqualone, psilocybine, phencyclidine, mescaline, etc.

Cultivation of poppy, cannabis and coca plnts require licence.

(7) PREVENTION OF ILLICIT TRAFFIC IN NARCOTIC DRUGS AND PSYCHOTROPIC SUBSTANCES ACT, 1988.

Medico-legal Aspects of Poisons : Sections, 176, 193, 201, 202, 284, 299, 300, 304A, 309, 320, 324, 326 and 328 I.P.C. and S.39, 40 and 175, Cr.P.C. deal with offences relating to administration of poisonous substances. Sections 272 to 276, I.P.C. deal with adulterated foods and drugs.

The intention with which any act is committed is an important element in law. Accurate definition of poison is not absolutely necessary in law, for administration of any substance with the intention of causing injury or death, and which causes injury or death as a result, is legally sufficient for awarding

Table (24–1)
Common household poisons

Preparation	Toxic substance
(A) Domestic Poisons :	
(I) Cosmetics :	
(1) Cuticle remover :	Potassium hydroxide; trisodium phosphate.
(2) Depilatories :	Barium sulphide; thallium.
(3) Hair wave lotions :	Thioglycollate salts; perborates; bromates.
(4) Nail polish removers :	Acetone.
(5) Suntan lotions :	Denatured alcohol; methyl salicylate.
(6) Baby powder :	Boric acid.
(II) Kitchen :	
(1) Baking powder :	Tartaric acid 50%.
(2) Baking soda :	Sodium bicarbonate.
(3) Dish washing compounds :	Sodium polyphosphates; sodium carbonate; sodium silicates.
(4) Fire extinguishing fluids :	Carbon tetrachloride; sodium carbonate; methyl bromide.
(5) Matches :	Antimony; phosphorus; sesqui-sulphide, potassium chlorate.
(III) Rat Poisons :	
(1) Rat paste :	Aluminium phosphide; zinc sulphide; zinc phosphide; arsenious oxide; red squill; thallium sulphate, phosphorus, barium carbonate, strychnine, norbromide, warfarin, sodium fluoroacetate.
(2) Rodine :	Yellow phosphorus.
(IV) Roach powder :	Sodium fluoride.
(V) Sanitary :	
(1) Deodorant tablets :	Formaldehyde; naphthalene.
(2) Drain cleaners :	Sodium hydroxide.
(3) Disinfectants :	Phenol; bleaching powder (Calcium hydrochlorite).
(VI) Miscellaneous :	
(1) Insecticide spray :	D.D.T., Gammexane, etc.
(2) Moth balls :	Naphthalene.
(3) Marking ink :	Aniline.
(4) Ink remover :	Sodium hypochlorite 5%
(5) Anti-rust products :	Ammonium sulphide; hydrofluoric acid; naphtha; oxalic acid.
(6) Cleaning solvents :	Petroleum hydrocarbons; carbon tetrachloride; trichloroethylene.
(7) Flourescent lamps :	Beryllium
(8) Furniture polish :	Turpentine; petroleum hydrocarbons.
(9) Paint remover :	Sodium hydroxide; lead acetate.
(10) Shoe polish :	Aniline; nitrobenzene.
(11) Hair bleach :	Potassium permanganate; hydrogen peroxide.
(12) Toys (paints) :	Lead.
(13) Fireworks :	Arsenic; mercury; antimony; lead; phosphorus; thiocyanate.
(14) Crayons (chalk) :	Salts of arsenic, copper, lead.
(15) Crayons (wax) :	Para-nitroaniline.
(B) Garden poisons :	
(1) Insecticides :	Organophosphorus compounds; chlorinated hydrocarbons; nicotine; tar oils.
(2) Fungicides :	Lead arsenate; copper compounds; organic mercurials; lime; sulphur.
(3) Weed-killers :	Sodium chlorate; arsenious oxide, and arsenites; dinitrocresol; paraquat.
(C) Therapeutic Poisons :	
(1) Antiseptics :	Iodine, benzoin.
(2) Tonic tablets :	Iron.
(3) Tonic syrup :	Strychnine.
(4) Sleeping tablets :	Barbiturates.
(5) Headache tablets :	Aspirin.
(6) Cough remedies :	Codeine.
(7) Throat tablets :	Potassium chlorate.
(8) Pep tablets :	Benzedrine.
(9) Others :	Antidepressants; tranquilisers.

punishment, whether the substance is one which can be called poison or not. The law does not make any difference between murder by means of poisons and murder by other means. Deliberate administration of poison proves the intention of the accused to cause death, or other such bodily injury which is sufficient to cause death, and will bring culpable homicide within the scope of murder. Sec. 284, I.P.C. deals with negligent conduct with respect to poisonous substances. Whoever does, with any poisonous substance, any act in a manner so rash or negligent as to endanger human life, or to be likely to cause hurt or injury to any person, or knowingly or negligently omits to take sufficient care to guard against probable danger to human life, shall be punished with imprisonment up to six months, or with fine. In cases of hurt, the law provides for greater punishments when any corrosive substance or any substance which is harmful to human body to swallow or to take into the blood is used (Sec. 324 and 326 I.P.C.). Whoever administers to or cause to be taken by any person, any poison or any stupefying, intoxicating or unwholesome drug, or other thing with intent to cause hurt to such person, or with intent to commit or to facilitate the commission of an offence, shall be punished with imprisonment up to ten years and also fine (Sec.328, I.P.C.). Adulteration of drugs is punishable with imprisonment up to 6 months (S. 274, I.P.C.). Administering poison to a person with criminal intent is by itself a criminal offence, whether actual hurt is caused or not.

EPIDEMIOLOGY: Poisoning both accidental and intentional are a significant contributor to mortality and morbidity throughout the world. According to WHO, three million acute poisoning cases with 2,20,000 deaths occur annually. Of these 90% of fatal poisoning occur in developing countries particularly among agricultural workers. Acute poisoning forms one of the commonest causes of emergency hospital admissions. Pattern of poisoning in a region depends on variety of factors, such as availability of the poisons, socioeconomic status of the population, religious and cultural influences and availability of drugs.

The exact incidence of poisoning in India is uncertain due to lack of data at central level as most cases are not reported, and as mortality data are a poor indicator of incidence of poisoning. It has been estimated that about 5 to 6 persons per lakh of population die due to poisoning every year.There are more than four thousand species of medicinal plants growing as herbs, shrubs and trees in India, many of which are poisonous when administered in large doses. The toxic principles belong to alkaloids, glycosides, toxalbumins, resins, cannabinoides and polypeptides. Suicidal and homicidal cases of poisoning are common in India, as poisons can be easily obtained and many poisonous plants grow wild, e.g. datura, oleanders, aconite, nux vomica, etc. Many Indians consider the taking of life by bloodshed a greater crime than poisoning, strangling etc. Accidental poisoning occurs from the use of philters or love potions, and quack remedies containing poisonous drugs, and snake bites. A love philter is a drug which is supposed to increase the love between the giver and taker. All aphrodisiacs such as cantharides, arsenic, alcohol, opium, cocaine and cannabis, are supposed to act as love philters. In India, the common poisons are insecticides and pesticides, such as organophosphates, chlorinated hydrocarbons, aluminium phosphide, carbamates and pyrethroids. Other poisons are corrosives, sedatives, alcohol, datura, oleanders, calotropis, croton and cleaning agents. In children kerosene, pesticides, drugs and household chemicals are commonly involved.

The commonest cause of poisoning in India and other developing countries is pesticides, the reasons being agriculture based economics, poverty and easy availability of highly toxic pesticides. Occupational poisoning due to pesticides are also common in developing countries, due to unsafe practices, illiteracy, ignorance and lack of protective clothing. In India, organophosphates form the largest bulk of pesticide poisoning. Since 1985, aluminium phosphide poisoning has been reported as the commonest cause of intentional poisoning in northern parts of India, viz., Haryana, Punjab and Rajasthan.

Among the adults, females predominate in all age groups, with an evident preponderance in the second and third decades of life. Acute poisoning in children is almost entirely accidental, while in adults it is mainly suicidal.

Mortality varies from country to country depending on the nature of the poison and availability of facilities and treatment by qualified persons.

OCCUPATIONAL AND ENVIRONMENTAL TOXICOLOGY: Occupational toxicology deals with the chemicals found in the place of work. Persons working in various industries may be exposed to various agents during the synthesis, manufacture or packaging of these substances or through their use during the occupation. Under the Workmen's Compensation Act, 1923, if workman contracts any disease specified therein as an occupational disease peculiar to that employment, such as anthrax, primary

cancer of the skin, pathological manifestations due to X-rays, radium, etc., poisoning by lead, arsenic, mercury, phosphorus, etc., it is deemed to be an injury by accident for purpose of compensation.

HAZARDOUS MATERIALS are substances that can potentially cause adverse health effects in individuals through contact with skin or mucous membrane or absorption through skin, respiratory or gastrointestinal absorption. They include chemicals, bilogical agents and radioactive substances, which may be in the solid, liquid or gaseous state.

Potential toxic exposures on the farm include a wide assortment of pesticides, noxious fumes, solvents, corrosive agents, fertilisers, envenomations (bites by snakes, scorpions, bees and wasps, centipedes, spiders, ticks, marine animals, caterpillars, etc.), and natural toxic aerosols.

Anthracosis, asbestosis (and its complications, such as pulmonary adenocarcinoma and mesothelioma), silicosis, brucellosis occur due to occupational exposure. Gas and wood stoves, chemically treated furniture and fabrics and domestic pest control are some of the examples of non-occupational exposures.

ENVIRONMENTAL TOXICOLOGY: It deals with potentially harmful impact of chemicals present as pollutants of the environment, to living organisms. Environment includes all the surroundings of living organisms, especially the air, soil and water. A pollutant is a substance present in the environment due to human activity, and which has harmful effect on living organisms. More than 60,000 chemicals are said to be in common. use. With advances in technology, pollution is increasing. The main causes of pollution are the production and use of industrial chemicals, increased use of insecticides, etc., in agriculture and production and use of energy. Threshold limit values (TLV) for about 600 chemicals commonly used have been prepared in USA.

All substances causing methaemoglobinaemia, and smoke inhalation can cause upper airway obstruction, lower airway obstruction, bronchospasm, pulmonary oedema and tissue hypoxia. Smoke may contain acrolein (aldehyde), ammonia, CO, cyanide, oxides of nitrogen, hydrogen sulphide, sulphur dioxide, hydrogen chloride, chlorine, phosgene, isocyanates, etc., depending on the material burnt. Acute respiratory failure may occur due to atelectasis, airway obstruction, or pulmonary oedema. Toxin-induced effects may contribute to adult respiratory distress syndrome (ARDS), sepsis, pneumonia, leading to prolonged illness or delayed death. Toxic combustion products depress CNS by acting as anaesthetic agents.

ECOTOXICOLOGY: It is concerned with the toxic effects of chemical and physical agents on living organisms, especially in populations and communities within defined ecosystems. It includes the transfer pathways of those agents and their interactions with the environment.

Poison Information Centres: National Poisons Information Centre has been established in AIIMS, New Delhi. It uses a computer software on poisons (INTOX) compiled by WHO. National Institute of Occupational Health at Ahmedabad has also a centre. Regional centres are located in Chennai and Cochin (POISINDEX). These centres provide toxicity assessment and treatment recommendations over the telephone throughout the day for all kinds of poisons.

Prevention: (1) Education is a major component of any poison prevention programme. (2) All drugs and toxic substances should be kept in locked cabinets. (3) All household poisons must be kept separate from food. (4) All products, pesticides and medicines should be kept in their original containers. (5) Keep caps and tops on bottles properly closed. (6) Do not keep household cleaners on the floor, under the kitchen sink, or in low cupboards that a child can easily open. (7) The label should be read before using the drug. (8) No drug should be given or taken in the dark. (9) All drugs whether expired or otherwise should be disposed in a safe manner. (10) Drugs in child-proof packages only should be purchased. (11) Children should be taught not to eat plants or berries. (12) Wherever cooking gas is used, adequate ventilation should be provided. (13) In persons showing suicidal tendencies, special care should be taken. (14) In chemical factories air-pollution should be prevented. (15) The workers in all factories should be properly educated, and safety equipment provided.

Nature of Poisoning : (1) Ideal Homicidal Poison : The characters of an ideal homicidal poison should be: (1) cheap, (2) easily available, (3) colourless, odourless and tasteless, (4) capable of being administered, either in food, drink or medicine, without producing any obvious change to prevent suspicion, (5) highly toxic, (6) signs and symptoms should resemble a natural disease, or the serious ill-effects should be delayed sufficiently long for the accused to escape suspicion, (7) there should not be any antidote, (8) there should be no post-mortem changes, (9) should not be detected by chemical tests, or other methods, and (10) must be rapidly

destroyed or made undetectable in the body.

Organic compounds of fluorine (used as rodenticides), and thallium satisfy several of the above criteria. Arsenic and aconite are commonly used.

(2) Ideal Suicidal Poison : The characters of an ideal suicidal poison should be: (1) cheap, (2) easily available, (3) highly toxic, (4) tasteless or of pleasant taste, (5) capable of being easily taken in food or drink, and (6) capable of producing painless death. Opium and barbiturates satisfy several of the above criteria. Organophosphorus compounds and endrin are commonly used as suicidal poisons.

(3) Stupefying : Datura, cannabis indica, chloral hydrate.

(4) Abortion : Calotropis, oleanders, aconite, croton, semecarpus, cantharides, ergot, lead, arsenic, mercury, potassium permanganate, etc.

(5) Accidental: Household poisons. **Non-accidental poisoning** may occur as an extension of the syndrome of child abuse, usually in children below 30 months.

(6) Rare: Bacteria, insulin.

(7) Cattle Poisoning : The usual motive is destruction of cattle of an enemy, or to obtain the hides. The usual poisons are abrus precatorius, oleanders, calotropis, organophosphorus, arsenic, aconite, strychnine, zinc phosphide, nitrate, etc.

(8) Arrow Poisons : Abrus precatorius, croton oil, calotropis, aconite, strychnine, curare and snake venom are used as arrow poisons.

(9) Aphrodisiacs: Cantharides, cocaine, cannabis, opium, strychnine, arsenic.

Classification : Poisons may be classified according to the chief symptoms which they produce. **(I) Corrosives:** (1) **Strong acids:** (a) **Mineral or inorganic acids:** Sulphuric, nitric, hydrochloric. (b) Organic acids: Carbolic, oxalic, acetic, salicylic. (2) **Strong alkalis:** Hydrates and carbonates of sodium, potassium and ammonia. (3) **Metallic salts:** Zinc chloride, ferric chloride, copper sulphate, silver nitrate, potassium cyanide, chromates and bichromates.

(II) Irritants: (1) **Agricultural.** (2) **Inorganic:** (a) **Non-metallic:** Phosphorus, iodine, chlorine, bromine, carbontetrachloride. (b) **Metallic:** Arsenic, antimony, copper, lead, mercury, silver, zinc. (c) **Mechanical :** Powdered glass, diamond dust, hair, etc. (3) **Organic:** (a) **Vegetable:** Abrus precatorius, castor, croton, calotropis, aloes. (b) **Animal:** Snake and insect venom, cantharides, ptomaine.

(III) Systemic: (1) Cerebral: (a) **CNS depressants:** Alcohols, general anaesthetics, opioid analgesics, hypnotics, sedatives. (b) **CNS stimulants:** Cyclic antidepressants, amphetamine, caffeine, methylphenidate. (c) **Deliriant:** Datura, belladonna, hyocyamus, cannabis, cocaine, etc. **(2) Spinal:** Nux vomica, gelsemium. **(3) Peripheral:** Conium, curare. **(4) Cardiovascular:** Aconite, quinine, oleander, tobacco, cyanide. **(5) Asphyxiants:** CO, CO_2 ,hydrogen sulphide.

(IV) Miscellaneous: Food poisoning, botulism.

Poisons may also be classified according to their **morbid anatomic** manifestations. (1) No morphologic changes are present which can be attributed to direct chemical action by the toxic agent, e.g. acute CNS depressants (alcohols, sedatives, hypnotics, tranquillisers, salicylates); chemical asphyxiants (carbon monoxide, hydrogen cyanide); organophosphates and most alkaloids. The abnormalities seen at autopsy result from shock and terminal anoxia. (2) Systemic lesions are produced without injury at the portal of entry, e.g. acute haemolytic poisons (arsine, nitrobenzene). (3) Injury is present at portal of entry without systemic injuries, e.g., corrosives, chlorine, sulphur dioxide. Death may be caused by local tissue changes (pulmonary oedema) or acute vasomotor collapse. (4) Local and systemic injuries are present, e.g., heavy metals.

Criminal Offences: The administration of a poison is a criminal offence whenever: (1) it is with intent to kill, (2) with intent to cause serious injury, (3) used recklessly even though there is no intent to kill, (4) for stupefying to facilitate a crime, e.g., robbery or rape, (5) to procure an abortion, (6) to annoy the victim , (7) to throw poison on another person with intention to injure him.

Poisoning may result from: (1) The administration of a poison for criminal purposes. (2) The swallowing of poison in mistake for harmless substance. (3) The inhalation through ignorance or accident, of the vapours of a poison. (4) The incorrect preparation of medicines containing a poison. (5) The accidental taking of a large dose of medicine containing a poison. (6) Excessive self-medication. (7) Addiction to drugs. (8) Bite by a poisonous animal. (9) Food infected with bacteria or their toxins.

Routes of Administration: In order of rapidity

of action: (1) Inhaled in gaseous or vapourous form. It usually involves a volatile substance, gas, dust, smoke or aerosol. Volatile solvents, such as benzene, toluene, xylene, acetone, methylene chloride, methyl chloroform, and carbon tetrachloride poisoning in industrial exposures; solvent sniffing among adolescents, or accidents in the home; gases such as CO, hydrogen sulphide and methane in industries; smokes and dusts of industrial origin may involve lead, mercury, silicon, asbestos and beryllium. (2) Injection into blood vessels. (3) Intramuscular, subcutaneous and intradermal injection. (4) Application to a wound. (5) Application to a serous surface. (6) Application to a bronchotracheal mucous membrane. (7) Introduction into stomach. (8) Introduction into the natural orifices, e.g. rectum, vagina, urethra, etc. Some drugs can be given by rectal route to produce a systemic effect, e.g., aspirin, barbiturates, chloral hydrate, chlorpromazine, etc. (9) Application to unbroken skin. Organic phosphates, nicotine, some organic solvents and lewisite gas can penetrate the skin and produce intoxication and death Other substances which are absorbed through the skin are: phenol and its derivatives, endrin, methyl salicylate, mercury, tetraethyl lead and alkylated compounds, cantharidin, hydrocyanic acid, hormones, such as oestrogen, progesterone, testosterone and desoxycorticosterone, vitamin D and K.

FATE OF POISONS IN THE BODY: The greater part of a poison is thrown out of the body as a result of vomiting and purging. The portion absorbed is mainly deposited in a less soluble form in the liver, which either partially metabolises or completely destroys it. The unaltered portion enters into the general circulation and acts on the body as a whole, or on the particular organs with which it has special affinity, provided the poison is not destroyed or made harmless by the kidneys and muscles. Some inorganic poisons like arsenic and antimony are retained in certain tissues, such as nails, hair, bones, etc., for a considerable time. Certain poisons like chloroform, phosphorus, nitrates and acetic acid disappear by evaporation or oxidised or destroyed in the body and no trace of them can be detected in the viscera or tissues if post-mortem is delayed.

Drugs Secreted into the Stomach: (1) **Acids:** Salicylic acid, barbital, probenecid, p-Hydroxypropiophenone, phenylbutazone, thiopental. (2) **Bases:** Theophylline, antipyrine, aminopyrine, quinine, levorphanol, acetanilid, aniline, phencyclidine, dextromorphan, tolazoline.

Routes of Elimination: The absorbed portion of poison is mainly excreted by the kidneys and to some extent by the skin. Other routes are bile, milk, saliva, mucous and serous secretions. The unabsorbed portion is excreted in the vomit and faeces.

Levels of consciousness:

Grade 0 : Fully conscious.
Grade 1 : Drowsy but responding to verbal command.
Grade 2 : Maximum response to minimal painful stimuli.
Grade 3 : Minimal response to maximum painful stimuli.
Grade 4 : No response to painful stimuli, loss of all reflexes including the pharyngeal, laryngeal and corneal.

The most painful stimulus is probably rubbing one's knuckles over the patient's sternum. This hurts more than pressing the eyeball and is potentially less dangerous.

ACTION OF POISONS: (1) LOCAL: The local action by coming in direct contact with the part. (1) Chemical destruction by corrosives. (2) Congestion and inflammation by irritants. (3) Effects on motor and sensory nerves, e.g., tingling of skin and tongue by aconite, dilation of pupils by belladonna or datura.

(2) REMOTE: Remote action produced either by shock acting reflexly through severe pain caused by corrosives, or by poison being first absorbed into the system through the blood, and then exerting a specific action on certain organs and tissues, e.g., cantharides acting on kidneys produces nephritis, nux vomica acting on the spinal cord causes tetanic convulsions.

(3) COMBINED: Drugs like carbolic acid, oxalic acid, phosphorus, etc., have local and remote actions.

CAUSES MODIFYING ACTION OF POISONS

(1) QUANTITY : More the quantity, more severe are the toxic effects. A large quantity of poison taken orally may cause excessive vomiting, causing its rapid elimination and decreased toxicity, e.g., alcohol, copper sulphate, etc. The action of a poison varies with the dose, e.g., a very large dose of arsenic may produce death by shock without causing irritant symptoms, while moderate doses produce irritant symptoms and small doses produce therapeutic action.

(2) FORM : (A) PHYSICAL STATE : Poisons act most rapidly when gaseous and less when liquid. In case of solids, the action depends on their solubility.

(B) CHEMICAL COMBINATION : The action of a poison depends upon the solubility or insolubility resulting from a chemical combination, e.g., silver nitrate and hydrochloric acid are both strong poisons,

but when combined, form an insoluble salt of silver chloride which is harmless. A substance may be harmless in metallic state but its salt may be toxic, e.g., arsenic is not poisonous but its salts are poisonous. Certain poisons which are not soluble in water may become dissolved in the acid secretion of the stomach and absorbed into the blood, e.g., lead carbonate and copper arsenite.

(C) MECHANICAL COMBINATION : The action of a poison may be altered if combined mechanically with inert substances, e.g., small dose of concentrated mineral acid produces corrosive action, but the same dose largely diluted with water is harmless.

(3) MODE OF ADMINISTRATION : The rapidity of the action is in the order described under routes of administration. As a rough guide if the active dose by the mouth is considered as unit, the rectal dose is about one-and-half to two, and the hypodermic dose about one-fourth. A lethal dose is usually ten or more times the maximum medicinal dose.

The rate of absorption from the alimentary canal is variable. Absorption by the stomach occurs more rapidly when the stomach is empty than when it is full. Absorption may be hastened if nature of stomach contents is such as will dissolve the poison, e.g., action of phosphorus will be hastened if oil is taken immediately after it is swallowed. Gastroenterostomy hastens the entry of poisons into the small bowel. Sleep, narcosis and trauma causing gastrointestinal stasis will retard it. Retardation during gastrointestinal absorption, dilution and alteration during digestion, or metabolism by the action of the liver render some piosons almost inactive and greatly reduce the potency of others. The skin is on the whole a bad absorptive organ.

(4) CONDITION OF THE BODY (A) AGE : Age has a considerable effect upon the dosage of drugs. Poisons have greater effect at the two extremes of age. A child under two years of age has not yet fully developed the drug-metabolising enzymes of the liver, and does not have an effective blood-brain barrier, and as such is much more susceptible to the effect of most drugs. There are some drugs of which children can take more than their proportionate dose, e.g., mercury and belladonna. There are some of which they cannot take even a proportionate dose e.g., morphine.

(b) IDIOSYNCRACY : It may be defined as the inherent personal hypersensitivity to the agent in question. Certain people are sensitive for certain drugs and even articles of diet, e.g., shellfish, eggs and fruit. The symptoms usually occur in the skin as an urticaria, but may be of a more general nature with dyspnoea, rigors, fever, diarrhoea, haemorrhage from the bowel and albuminuria. Fatal cases are comparatively rare, but symptoms may be alarming or dangerous. Iodine, bromine, opium, belladonna, cocaine, aspirin, penicillin, and mercury are common examples of drugs to which many people are allergic.

(c) HABIT : The effect of certain poisons decreases with habituation. Tolerance is the ability of an organism to show less response to a specific dose of a chemical than it showed on a previous occasion from the same dose. It results from a decreased reaction between the chemical and the biologic effector substance. Opium preparations frequently taken, lose much of their effect after a time, and require to be administered in increased doses. Addicts can tolerate quantities of the drug which would endanger life if they had been initial doses. Tolerance is seldom a natural phenomenon. The same effect of habit occurs from the use of tobacco, alcohol, cocaine, morphine and other alkaloids. It is more usually a feature of natural substances, less of syntheitc drugs, such as sulphonal, barbiturates, chloral, etc. Tolerance for mineral substances is limited, but it occurs in connection with arsenic to a certain extent.

(d) STATE OF HEALTH : A healthy person tolerates better than a diseased. General debility, senility, chronic or disabling disease may cause death of a person to a dose that is ordinarily safe, e.g. ,CO may kill at a blood saturation of only 25 to 30%. In some diseases, larger doses of certain drugs may be given without harmful effects, e.g., opium in tetanus, delirium tremens and mania, and strychnine in paralysis, while in other diseases certain drugs cannot be given even in small doses, e.g., opium in granular kidney and bronchial asthma and mercury in chronic nephritis.

(e) SLEEP AND INTOXICATION : The action of a poison is delayed if a person goes to sleep soon after taking it. The action is also delayed if one takes a poison in an intoxicated condition.

(f) CUMULATIVE ACTION : Poisons which are eliminated slowly may accumulate in the body when given in repeated doses for a long time and may ultimately produce symptoms of poisoning.

TYPES OF POISONING: (1) **Acute poisoning** is caused by an excessive single dose, or several smaller doses of a poison taken over a short interval of time. (2) **Chronic poisoning** is caused by smaller doses over a period of time, resulting in gradual worsening. The poisons which are commonly used for the purpose of chronic poisoning are arsenic, phosphorus, antimony and opium. (3) **Subacute poisoning** shows features of both acute and chronic poisoning. (4) **Fulminant poisoning** is produced by a massive dose. In this death occurs

rapidly, sometimes without preceding symptoms.

Parasuicide (attempted suicide, or pseudocide) is a conscious, often impulsive, manipulative act, undertaken to get rid of an intolerable situation. Drug ingestion is the commonest form. Most persons are psychologically disturbed.

Diagnosis of Poisoning: (1) In the Living : There is no single symptom, and no definite group of symptoms, which are absolutely characteristic of poisoning. The closest resemblance to disease, may be produced by thallium poisoning. The symptoms of a disease may simulate acute poisoning, e.g., the sudden onset of intestinal obstruction may be mistaken for irritant metal poisoning. A detailed clinical history is of great importance.

The following conditions should arouse suspicion of poisoning. (1) The symptoms appear suddenly in a healthy person. (2) The symptoms appear immediately or within a short period after food or drink. (3) The symptoms are uniform in character, and rapidly increase in severity. (4) When several persons eat or drink from the same source of poison in the food or drink at the same time, all suffer from similar symptoms at or about the same time. (5) The discovery of poison in food taken, in the vomit or in the excreta is strong proof of poisoning.

The following groups of symptoms are suggestive of poisoning. (1) The sudden onset of abdominal pain, nausea, vomiting, diarrhoea and collapse. (2) The sudden onset of coma with constriction of pupils. (3) The sudden onset of convulsions. (4) Delirium with dilated pupils. (5) Paralysis, especially of lower motor neurone type. (6) Jaundice and hepatocellular failure. (7) Oliguria with proteinuria and haematuria. (8) Persistent cyanosis. (9) Rapid onset of the neurological or gastrointestinal illness in persons known to be occupationally exposed to chemicals.

Collect : (1) Stomach wash (entire quantity). (2) Ten ml. blood. (3) Urine, as much as possible. 100 mg. of sodium fluoride for 10 ml. blood acts both as a preservative and as an anticoagulant.

Symptoms of Chronic Poisoning : (1) The symptoms are exaggerated after the administration of suspected food, fluid or medicine. (2) Malaise, cachexia, depression and gradual deterioration of general condition of the patient is seen. (3) Repeated attacks of diarrhoea, vomiting, etc., are seen. (4)When the patient is removed from his usual surroundings, the symptoms disappear. (5) Traces of poison may be found in the urine, stool or vomit.

A detailed history of the quality and quantity of the poison administered, the character of the symptoms with reference to their onset, and the time that passed between the taking of the poison and the development of symptoms, the duration of illness, the treatment given, and the time of death, should be obtained from the relatives of the deceased.

In the Dead : Collect all information from the inquest report and from the relatives of the deceased.

(I) Post-mortem Appearances : External : (1) The surface of the body and the clothes may show **stains** or marks of vomit, faeces or the poison itself. The colour changes in the corroded skin and mucous membrane are: (a) sulphuric and hydrochloric acid: grey, becoming black from blood; (b) nitric acid: brown; (c) hydrofluoric acid: reddish-brown; (d) carbolic acid: greyish-white; (e) oxalic acid: grey, blackened by blood; (f) cresols: brown, leathery; (g) casustic alkalis: greyish-white; (h) mercuric chloride: bluish-white; (i) zinc chloride: whitish; (j) chromic acid and potassium chromate: organge, leathery. (2) Colour of **post-mortem staining :** The skin may be dark-brown or yellow in phosphorus and acute copper poisoning; cherry-red in poisoning by carbon monoxide; chacolate-coloured in cases of death from poisoning by nitrites, aniline, nitrobenzene, acetanilide, bromates, chlorates, et . due to the formation of methaemoglobin. (3) **Smell about the mouth and nose :** Substances which may be recognised by their odour are (a) **Garlick-like:** Phosphorus, arsine gas, arsenic (breath and perspiration), thallium, selenium, dimethylsulphoxide, tellurium, parathion, malathion. (b) **Sweet or fruity:** Ethanol, chloroform, nitrites. (c) **Acrid:** Paraldehyde, chloral hydrate. (d) **Rotten eggs:** Hydrogen sulphide, mercaptans, disulfiram. (e) **Fishy or musty:** Zinc phosphide. Other substances are: cyanides, phenol, opium, ether, camphor, etc. (4) **The natural orifices,** e.g., mouth, nostrils, rectum and vagina may show the presence of poisonous material or the signs of its having been used. (5) **Injection marks** should be looked for with care. (6) **Skin** should be examined for lesions, e.g. hyperkeratosis and pigmentation may be found in chronic arsenical poisoning. Jaundice may occur in poisoning from phosphorus, senecio, and in susceptible persons by potassium chlorate. (7) Any evidence of **marks of violence,** such as bruises, or wounds of any nature, may suggest some form of

death other than poison.

The bodies of persons poisoned are not more rapidly decomposed than those of others. Some poisons may delay the action of the putrefactive bacteria to some extent.

Internal : There is no special routine peculiar to poisoning cases. All organs must be examined and all contents preserved.

(1) Smell : On opening the body, note any peculiar smell. The skull should be opened first to detect unusual odours in the brain tissues, because such odours are masked by the opening of the body cavities. This is useful in cyanide, alcohol, phenol, cresol, ether, chloroform and camphor poisoning.

(2) Mouth and Throat : Examine the tongue, mouth and throat for any evidence of inflammation, erosion or staining. Areas of necrosis of the pharynx may be seen in death associated with agranulocytosis caused by amidopyrine, thiouracil, dinitrophenol, sulphonamides and barbiturates.

(3) Oesophagus : Corrosive alkalis produce marked softening and desquamation of the mucous membrane. In acute cantharidin poisoning, the mucous membrane is often swollen and engorged and may show patches of ulceration. Perforation of oesophagus may occur in poisoning from paraquat and fluorides.

(4) Upper Respiratory Tract : Examine the larynx, trachea and bronchi for evidence of volatile irritants or inhaled poisonous matter. Oedema of glottis, and congestion and desquamation of mucous membrane of the trachea and bronchi may be seen in corrosive acid or alkali poisoning when it enters the respiratory tract.

(5) Stomach : Toxic substances may be held in high concentrations in the rugae and crypts of the mucosa, or even in the blood in the actual stomach wall. The colour and appearance may be normal, though poison is present.

(a) Hyperaemia : Hyperaemia of the mucous membrane caused by an irritant poison is generally marked at the cardiac end and greater curvature of stomach, but rarely at the pyloric end. It is usually patchy and of a deep crimson colour. The ridges are more involved in poisoning than in disease. The mucous membrane is often covered with a sticky secretion and shows small haemorrhagic foci. Redness of mucous membrane of the stomach is found after death, but is usually limited to the posterior wall. In this case, there is no thickening of mucous membrane, nor any thick mucus over its surface. Redness of the mucosa is also found during digestion, in asphyxial deaths due to general venous congestion, and when it is exposed to atmosphere. Hyperaemia caused by disease is uniformly spread over the whole surface and not in patches. Putrefactive changes will alter the colour of a healthy stomach, but the destructive changes of poisoning are usually present . Histological examination helps in cases of doubt.

Colours other than red may be present due to various causes. The green colour of ferrous suplhate tablets; the blue of amytal capsule; the pink of soneryl are characteristic . Mercury usually causes a slate-coloured stain; arsenic may show white particles adherent; strong sulphuric, acetic or hydrochloric acids, and concentrated oxalic acid are likely to blacken or char the wall; nitric acid may cause yellow colour, carbolic acid may produce buff or white colour and shrivelling; cresols produce brown colour; copper produces a blue or green colour. The colour may also be due to bile when there will be no signs of inflammation, or to fruit juice or food, when it is uniform and without signs of inflammation.

(b) Softening : Corrosives and irritants produce immediate contraction of the muscularis, due to which the superficial epithelium is damaged, while the depths of the glands are protected by compression of their necks by the spasm. Excess of mucus is secreted by the glands due to the neighbouring irritation. If life is prolonged, the poison passes deeper and deeper. Spasm of the pylorus holds a poison at this point, which is the site most often involved. Softening of mucous membrane of stomach, especially at its cardiac end and greater curvature is usually caused by corrosive poisons, chiefly alkaline corrosives. It is also seen in mouth, throat and oesophagus. In disease, it is limited to stomach and is usually found at its cardiac end. Softening due to putrefaction begins in most dependent parts and affects all the coats of the stomach without detachment of its mucosa, and softened patch is not surrounded by an inflamed area.

(c) Ulcers : Ulceration due to corrosive or irritant poisons is usually found at the greater curvature of the stomach. It appears as an erosion with thin, friable margins. The surrounding mucosa is softened due to inflammation, and there is diffuse hyperaemia. An ulcer from disease is usually seen on the lesser curvature and the margins are well-

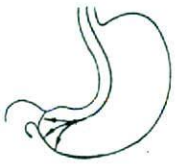

Fig. (24–1). Magenstrasse is the term applied to the pathway acidic agents follow.

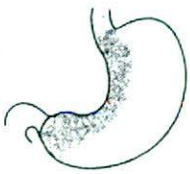

Fig. (24–2). Shaded rigion indicates the area of greatest injury caused by acid ingestion in a food-filled stomach.

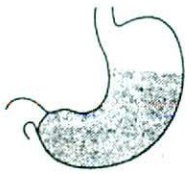

Fig. (24–3). Shaded region indicates the area of greatest injury caused by acid ingestion in an empty stomach.

defined, thickened and indurated.

(d) Perforation : Perforation is occasionally observed, when the strong mineral acids have been taken, especially sulphuric acid; it is much less common with other acids. Ammonia can cause perforation of stomach. The stomach, in such cases is blackened and extensively destroyed, the aperture is irregular, the edges sloughing, and the adjacent tissues easily torn. The acid escapes into the abdomen and causes peritonitis. Perforation by irritant poisons is rare. In chronic gastric ulcer, it is oval or rounded and has a punched-out appearance and may show chronic adhesion to neighbouring organs.

In autolysis from post-mortem digestion, the change is confined to the stomach alone, and it is commonly found only at the cardiac end. The opening is large and irregular, with rough and pulpy edges. The surrounding mucous membrane is softened and gelatinous. Peritonitis is not seen.

The Contents of the Stomach : The ligatured stomach should never be sent for analysis without being opened, as putrefaction may obscure changes in the mucous membrane, and the gases produced may result in the lid of the jar being forced off in transit. The stomach is opened along its greater curvature in a clean porcelain dish. The wall is examined for fragments of poison adhered to it, such as powdered poisons, fragments of capsules, starch from tablets, fragments of leaves or fruit, cantharides, etc. The contents must be carefully observed and written notes made, regarding the volume, colour and contents, including food. The presence of seeds, leaves, capsules and foreign bodies, such as nails, pins, glass, etc., must be noted.

The cells of plants in the alimentary canal, retain their characteristic shape, dimensions, surface ornamentation and other characters, which can be identified in vomit or material from gastrointestinal tract, by microscopic examination.

(6) The Duodenum and Intestines : A strongly acid reaction is of greater significance here than in the stomach contents. Sodium hydroxide can rarely cause perforation of duodenum. Ulceration beyond the pylorus is usually due to natural disease The only characteristic change which occurs in the intestine is seen in mercury poisoning. This change which usually involves the ascending and transverse colons, is a diphtheretic colitis, which may resemble the enteritis of acute bacillary dysentery.

A normal gastrointestinal tract rules out poisoning by corrosive acids and alkalis, phenols, mercury and arsenic.

(7) Liver : Substances, such as arsphenamine, phosphorus, chloroform, trinitrotoluene, carbon tetrachloride and senecio, may produce liver necrosis. Arsenic, carbon tetrachloride, amanita phalloides, yellow phosphorus and rarely ferrous sulphate produce a fatty liver. Jaundice may be produced by phosphorus, senecio and potassium chlorate, due to acute haemolytic anaemia.

(8) Respiratory System: Oedema of the glottis and congestion and desquamation of the mucous membrane of the trachea and bronchi may be seen in corrosive poisoning when the acid or alkali has entered the respiratory tract. The lungs show non-specific signs of congestion and oedema.

(9) Kidneys : Parenchymatous degenerative changes are commonly found in irritant metal poisoning, and in cantharidin poisoning. Extensive necrosis of proximal convoluted tubules may be found in deaths from poisoning by mercuric chloride, phenol, lysol and carbon tetrachloride.

(10) Heart : Subendocardial haemorrhages in the left ventricle occur in most cases of acute arsenic poisoning.

(11) The **bladder,** and in females the **vagina and uterus** should be particularly examined, for poison is occasionally introduced into the body by these routes. In criminal abortion, it may be necessary to send the vagina and uterus for analysis.

Many poisons, such as alkaloids do not produce characteristic tissue changes. The presence of wounds or of a disease sufficient to cause death, does not rule out the use of poison. A poison can cause death without leaving any naked eye changes, and proof of poisoning must be obtained from other sources, or from chemical examination. **No poison kills without producing some symptoms of illness, if no signs after death.** Therefore, enquiry as to symptoms in life is very important. The conclusion that death was caused by poison depends on evaluation of clinical, toxicologic and anatomic

evidence.

Chemical Analysis: If during autopsy any organ is removed from the body, it should never be placed on any surface, or in any container which is not clean. If this is not done, a doubt may arise, whether the poison found might have been accidentally introduced in the vessel used. If a refrigerator is available, all organic substances, should be kept in it as soon as possible after removal from the body. Chemical compounds should not be added, as they may confuse the issue. Decomposition may produce substances not in the original stomach, but allowances can almost always be made for these without confusion.

The specimens of blood, urine, bile and vitreous should be placed in glass containers, but not in plastic containers, as these fluids can leach out plastic polymers from the wall of a plastic container, which may mask some compounds and interfere with analysis.

Blood is the specimen of choice for detection of poisons, as it gives the best indication of the quantity of drug exerting an effect on the person at the time of death. The urine concentrates the drug or poison in many cases. It is suitable for single direct spot test, because there is no protein-binding to prevent extraction. The concentration of poisons found in urine is not important in evaluating the quanitity ingested or the toxicity. In delayed deaths, the poison may be found in urine, when none is found in viscera.

The muscle, especially of thigh is well preserved in advanced decomposition. Levels of drugs in the muscle more accurately reflect blood levels than the liver or kidney.

Postmortem diffusion of the drugs occurs from the stomach into the liver, mainly the left lobe. Diffusion also occurs in the base of left lung, spleen and pericardial fluid and to a lesser degree into heart, aorta and inferior vena cava.

In a living person, the concentration of a poison is lower in the venous blood as compared to arterial, because tissues may take up the compound from the arterial supply. Portal blood has higher concentration of a poison that is being absorbed from the intestine. After death, variation in concentration is caused by uneven destruction by enzymatic and microbiological activity and from diffusion from sites of higher concentration. Postmortem levels of many poisons are unreliable because the barriers formed by living cell membranes breakdown after death, and molecules can easily move through the tissues into blood vessels.

It is essential to prevent contamination of the solid viscera with the contents of the gastrointestinal tract, because an idea of the length of time since ingestion may be had from the relative amounts of poison in the stomach, intestines and the solid organs. If the poison is only found in the contents of the stomach, and none in the solid viscera and is not an irritant, doubts may occur about the actual cause of death. Poison found in liver or kidney is proof of absorption. Therefore, it is important to keep the contents of the alimentary canal in separate bottles. Poison found in urine, unless added with evil intention is a proof of absorption and excretion. If the poison is also found in the food or medicine preserved, this would be very strong additional evidence. The stomach contents are of primary value for estimating the quantity ingested in acute overdoses, and qualitatively in identifying substances which have been recently ingested.

INFORMATION SUPPLIED TO THE LABORATORY: The pathologist should provide all available information to the toxicologist, i.e. (1) brief details of symptoms if any, and length of illness, (2) if poison taken is not known, drugs or poisons to which the deceased was known to have access, including medication being taken, (3) history obtained from family members and friends, (4) empty containers or medications found at the scene, (5) autopsy findings, and (6) any special risk with the samples, e.g. hepatitis B virus, AIDS, etc. It is very important to recover and send the container in which the toxic substance had been kept, which narrows the toxicologist's search to one or more specific compounds. The pathologist should not ask the toxiocologist to look for a "general unknown poison" in the viscera preserved.

THE ANALYTIC PROCEDURE

For toxicologic analysis poisons can be divided into five groups.

ANALYSIS: Toxicological analysis of biological tissues involves: (1) Separation of the drug from the biological tissue. For this, the contents of stomach are diluted in water, and the solid viscera are cut into small pieces and macerated in water. Then a solvent is used to extract the poison. (2) Purification of the drug. This is done by additional extraction procedures using alkaline and acid solutions. (3) Analytical detection and quantitiation. This is done by thin-layer chromotography (TLC), gas chromatography (GC), gas chromatography-mass sepectrometry (GC-MS), and rarely UV spectrophotometry. Except for gas chromatography-mass spectrometry, none of the methods is totally specific. If a method of analysis other than GC-MS is used for initial identification, then

often it is easier to make positive identification and even quantitation using the GC-MS.

GROUP I. GASES: Gases are separated from blood or lungs by simple aeration procedures and specific tests applied. Air samples collected at scene of exposure give better results.

GROUP II. STEAM VOLATILE POISONS: They include both organic and inorganic substances, which are separated from biological materials by steam distillation from an acidic or basic medium, e.g. ethyl and methyl alcohol, phenol, chlorinated hydrocarbons, benzene, amphetamines, nicotine, yellow phosphorus, etc.

Steam distillation of a sample of finely minced tissue containing tartaric acid separates volatile acidic and neutral substances. The residue is made alkaline and redistilled, which separates volatile basic substances. Individual qualitative tests are carried out on suitable portions of the distillate. If some volatile compound is identified in distillate, a fresh weighed sample of tissue is used for quantitative analysis.

GROUP III. METALLIC POISONS: (1) IN DRY ASHING PROCEDURE, the weighed and minced tissue is dried in an oven and then placed in muffled furnace at 450°C until all the organic matter is destroyed. The remaining ash is leached with mineral acids and resulting solutions subjected to qualitative and quantitative analysis for individual metals. Arsenic, antimony and mercury are volatile at 450°C and would be lost in such procedure.

(2) The WET ASHING PROCEDURE employs a mixture of nitric, sulphuric and perchloric acids to oxidise the organic matter. The remaining solution is the ash which is used for analysis of various metals.

GROUP IV. NONVOLATILE ORGANIC POISONS: This group includes all compounds that are alcohol and water soluble. (1) Compounds which may be extracted from an acidic aqueous medium by chloroform or ether include organic acids and organic netural compounds, such as barbiturates, acetanilid, phenacetin, etc. (2) Compounds which may be extracted from a basic aqueous medium by chloroform or ether include organic bases, such as cocaine, quinine, strychnine, phenothiazines, imipramine, nicotine, demerol, etc. (3) Compounds which may be extracted from aqueous solution, which is faintly alkaline with ammonia or sodium bicarbonate, by chloroform with 10% ethanol include morphine, dionin, dilaudid, etc.

For the above substances 200 to 500 g. of tissue is finely ground, and treated with alcohol, filtered and alcohol evaporated, and process repeated and a final residue is obtained and tests, such as TLC, GC, GC-MS carried out to find out specific poison.

GROUP V. MISCELLANEOUS: This includes all substances which are not classified in any of the above four groups, such as non-metallic inorganic substances and water and alcohol insoluble organic compounds. For identification, special individual procedures for each substance must be employed

FALSE POSITIVES : Many poisons enter the body regularly in small amounts with food, water or air. Arsenic, lead and mercury compounds are sprayed and dusted on fruits and vegetables and are ingested regularly. Nicotine is present in the blood of smokers. Many poisons are used therapeutically, e.g. arsenic and strychnine, sedatives, tranquilisers, etc. These conditions produce false positive results.

CAUSE OF DEATH: The blood level of the drug or chemical is useful to determine the cause of death, in correlation with clinical and anatomic findings. A lethal level does not by itself establish the cause of death. The blood level of a drug need not always be in the lethal range for it to reflect the cause of death, especially in a treated case. When the presence of a highly toxic material is established even in trace amounts, the inference that the poisoning is cause of death is justified.

Toxic and Lethal Drug Levels : Fatal concentrations of poisons vary depending upon: (1) analytical techniques which vary widely both in method and accuracy, (2) site of sampling, (3) fatal level being attributed to one substance without considering the levels of other toxic substances that the deceased may have taken, and of which the pathologist or analyst may not be aware. Many victims who die due to poisoning, have lower blood concentrations of the responsible agent than those usually regarded as fatal. The causes for this may be : (1) Unusual susceptibility to the drug; (2) combinations of drugs can interact in an additive fashion; (3) some pre-existing natural disease may have contributed to death; (4) rapid but not complete absorption of drug; (5) metabolic degradation of the drug during a prolonged survival in which respiratory complications and hypoxic encephalopathy maintain coma and act as the immediate causes of death.

Toxicity : The "**therapeutic index**", or the ratio of the toxic to the effective dose of a drug, indicates the relative toxicity of drugs. Toxicity of the chemicals have been devised depending on the amounts which poduce harm.

The **lethal dose** is the dose that kills. "Minimal lethal dose" is the smallest dose that has been recorded as fatal to a healthy person. The usual lethal dose of a poison is ten times the therapeutic dose.

Young's rule: The dose of a drug for a child is obtained by multiplying the adult dose by the age in years and dividing the result by the sum of the child's age plus 12.

Interpretation of Toxicological Results : The following factors should be considered in the interpretation of the result of toxicological analysis. (1) Age and weight of the deceased. (2) Presence of a natural disease condition. (3) Presence of traumatic lesions. (4) Degree of tolerance of the individual. (5) Hypersensitivity reaction.

Putrefaction and Toxicologic Analysis : In post-mortem decomposition, many poisons present in the tissues undergo chemical changes, and cannot be detected. Putrefaction of normal tissue may produce substances which give chemical reaction similar to those obtained from toxic compounds. Most volatile compounds are lost due to putrefaction, but ethyl alcohol and cyanide may be produced from normal tissue. Neurin, muscarin, mydalein, etc., are produced due to putrefaction, the toxicity of which is equal to the well-known alkaloids. In an embalmed body, it is very difficult to detect and identify most volatile poisons.

Failure to Detect Poison : In some cases, no trace of poison is found on analysis, although from other circumstances, it is almost or quite certain that poison was the cause of illness or death. The possible explanations of negative findings are : (1) The poison may have been eliminated by vomiting and diarrhoea, e.g., in irritant poisons. (2) The whole of the poison has disappeared from the lungs by evaporation or oxidation. (3) The poison after absorption may be detoxified, conjugated and eliminated from the system. (4) Some vegetable alkaloidal poisons cannot be definitely detected by chemical methods. (5) Some drugs are rapidly metabolised, making extraction difficult. (6) Some organic poisons especially alkaloids and glucosides may by oxidation during life, or due to faulty preservation, or a long interval of time, or from decomposition of the body, may deteriorate and cannot be detected chemically. (7) Biological toxins and venoms which may be protein in nature cannot be separated from body tissues. Immunoassay procedures can detect these compounds. (8) If the poison acts slowly and death is delayed following production of irreversible organic changes (e.g. hydrogen sulphide or cyanide), the poison may be completely excreted. (9) Many drugs may be present in very small amount and these may require considerable amount of viscera for their identification. (10) The wrong or insufficient material may have been sent for analysis.

Table (24-2) Toxicity Rating (Gosselin, et. al).

< 5 mg/kg	6 (super toxic)
5 to 50 mg/kg	5 (extremely toxic)
50 to 500 mg/kg	4 (very toxic)
0.5 to 5g/kg	3 (moderately toxic)
5 to 15 g /kg	2 (slightly toxic)
>15g / kg	1 (non-toxic)

(III) Experiments on Animals : This is not an ideal test, for signs and symptoms may be due to other causes. Absence of signs and symptoms may be due to insusceptibility of the animal to the particular poison, e.g., rabbits are insusceptible to belladonna, hyocyamus and stramonium; pigeons are not affected by opium. Cat and dog are affected by poisons almost in the same way as man. They may be fed with the suspected food, or with the poison after it is separated from the viscera and the symptoms noted.

(IV) Moral and Circumstantial Evidence : Such evidence may consist of motive, the evidence of witness about the recent purchase of the poison, his behaviour before and after the commission of the offence, and the recovery of the poison from the possession of the accused.

Types of Drug Fatalities : Drug-related deaths can be : (1) Primary drug fatalities are those in which death is due to the toxic or adverse effect of the chemical agent, with or without the contributory influence of pre-existing, unrelated natural disease. (2) Secondary drug fatalities are those arising from medical complications of drug abuse, such as viral hepatitis and bacterial endocarditis. (3) Drug associated fatalities are those caused by homicidal, accidental and suicidal violence arising directly or indirectly from activities related to the obtaining and use of illicit drugs.

Drug - Automatism : According to this hypothesis, the patient develops a state of toxic delirium after ingesting one or several doses of a drug (usually depressant drugs, alcohol, or a combination of these), and in the delirious or automatism state, takes additional doses of the drug without realising it. It is difficult to prove or disprove this hypothesis.

Duties of a Medical Practitioner in a case of Suspected Poisoning : The duties are: (I) **Medical**: Care and treatment of the patient. (II) **Legal**: Assist the police to determine the manner of death. (1)

Note preliminary particulars of the patient, i.e., age, sex, address, date and time, identification marks, etc. (2) In case of suspected homicidal poisoning, the doctor must confirm his suspicion before expressing an opinion. For this he must : (a) Obtain history of route of exposure, quantity consumed, time elapsed since ingestion, etc. Collect vomit and urine, and submit for analysis. (b) Carefully observe and record the symptoms in relation to food; any change in the colour, taste or smell of the food or drink; other persons affected at the same time; the condition of the patient; explanation offered by the patient for the symptoms, and statements of other persons present which appear to be relevant. (c) Consult in strict confidence senior practitioner and keep him informed about the case. (d) Remove the patient to hospital. If the patient refuses to be moved, the doctor should engage nurses of his confidence, who should administer themselves the medicine and food, and allow no one to be with the patient alone, and maintain detailed records of the condition of the patient and of the treatment. (e) If a particular person is suspected, attempts should be made to outwit him by changes of diet and the alteration of meal times. Such person should be allowed to visit the patient only in the presence of a nurse or doctor. (f) Suspicion may arise when a person insists on preparing all the food and serving the patient personally, and also if the person insists on throwing away all the food which the patient leaves. (g) The doctor should keep detailed records of the number of his visits, the symptoms and signs observed, and treatment given from time to time. (3) Once the suspicions are confirmed, he should request the removal of the patient to hospital. If the victim is an adult who retains his mental faculties, it might be desirable to speak to him about the steps to be taken. (4) Any suspected articles of food, excreta, and stomach wash samples should be preserved. Full or empty bottles, capsules, paper packets, or liquids lying about should be collected and preserved. Any recent stains on bedclothes, furniture, etc., should be preserved if possible. Non-compliance is punishable under S. 201, I.P.C. if it is proved that the doctor did it with the intention of protecting the accused. (5) If a private practitioner is convinced that the patient is suffering from homicidal poisoning, he is bound under S. 39, Cr. P.C. to inform the police officer or Magistrate. Non-compliance is punishable under S. 176, I.P.C. (6) If he is sure that patient is suffering from suicidal poisoning, he is not bound to inform the police, since S. 309 of the I.P.C. is not included in the section of the I.P.C for which information has to be given under S.39, Cr.P.C. (7) If the practitioner is summoned by the investigating police officer, he is bound to give all information regarding the case that has come to his notice under S. 175, Cr. P.C. If he conceals the information, he is liable to be prosecuted under S. 202, I.P.C. If he gives false information, he is liable to be charged under S. 193, I.P.C. (8) A Government medical officer is required to report to police all cases of suspected poisoning, whether accidental, suicidal or homicidal attended in the hospital. (9) If the condition of the patient is serious, he must arrange to record the dying declaration. (10) If the patient dies, he should not issue a death certificate, but he should inform the police. (11) In case of food poisoning, public health authorities must be notified.

TREATMENT OF POISONING

(I) Immediate resuscitative measures in comatose patients should be adopted to stabilise respiration, circulation and to correct CNS depression. (ABCD of resuscitation.) (A) **Airway:** Opening up and cleaning up the airway (oral cavity, nostrils) of secretions, vomit or any other foreign body might be life saving. Protecting and securing the airway by means of endotracheal intubation may be necessary. (B) **Breathing:** If the arterial blood gas cannot be maintained inspite of establishing an effective airway, then graduated supplemental oxygen therapy either by a ventimask or through endotracheal tube should be administered. (C) **Circulation:** I.V. fluid administration may be life sustaining line. (D) **Depression of CNS** should be corrected. An unconscious patient should be turned to lie on one side to stop the tongue blocking the throat and to allow fluid to come out of the mouth (recovery position). Most of the poisoning cases, whether they are conscious or unconscious recover with supportive care alone.

(II) Removal of Unabsorbed Poison from the Body. (1) Inhaled Poisons : If the poison is inhaled as a gas, the patient must be removed into fresh air, artificial respiration and oxygen (six to eight litres per minute) should be given. The air-passages should be kept free from mucus by postural drainage or by aspiration. Nikethamide 2 ml. i.v. should be given if necessary. Give aminophyline 250 to 500 mg. if there is severe bronchospasm and diuretics

Fig. (24–4). Position of patient for gastric lavage.

if there is pulmonary oedema.

(2) Injected Poisons : If the poison has been injected subcutaneously from a bite or an injection, a tight ligature should be applied immediately above the wound, which must be loosened for one minute after every ten minutes, to prevent gangrene. The wound should be excised, the poison sucked out, and the poison neutralised by suitable chemical substance. Local vasoconstriction can be produced by injection of adrenaline. Immersion of the extremity in water at 10°C. slows capillary blood flow and limits absorption.

(3) Contact Poisons : Patient's contaminated clothes, contact lenses and jewellery should be removed immediately. If poison is applied to the skin or wound, or is inserted into the vagina, rectum or urinary bladder, it should be removed by washing with water for 30 minutes or should be neutralised by specific chemical. Eyes should be irrigated with normal saline for at least 15 minutes.

(4) Ingested Poisons : **GASTRIC LAVAGE** It is useful any time wihin 3 hours after ingestion of a poison. It is done using a stomach tube (Ewalds or Boas, tube) or ordinary soft, non-collapsible rubber tube of one cm. diameter and one-and-half metre length, with a glass funnel attached at one end, and a mark about 50 cm. from the other end, which should be rounded with lateral openings. At about the midpart of the tube there is a suction bulb, used to pump out the stomach contents. A wooden mouth gag has a hole at its mid-part to allow the passage of the tube through it. One end of the gag is pointed so that it can be forcefully inserted by the side of the mouth in non-cooperative patients. Dentures must be removed and a mouth gag is placed in right position in between the teeth of two jaws, so that the teeth do not bite the tube. Care should be taken in unconscious persons, who are likely to regurgitate and then aspirate stomach contents into respiratory tract and die from asphyxia. Patient should be lying on his left side or prone with head hanging over the edge of the bed, and face down supported by an assistant, so that the mouth is at a lower level than larynx, so that any fluid which may leak out through the sides of the tube will not trickle down inside the larynx and trachea. The end is lubricated with olive or sweet oil, liquid paraffin or glycerine, and is passed into the stomach by depressing the tongue with two fingers or tongue depressor, and slowly passing it downwards through the pharynx and oesophagus into the stomach, till the 50 cm. mark is reached. If there are no marks on the tube, the tube should be passed for a distance equal to that measured between the bridge of the nose and the tip of the xiphoid process. Force must not be used to insert the tube. Absence of coughing and of breath sounds in the funnel will confirm that the tube has not entered into the trachea. Whenever in doubt, test by keeping the free end of the tube just below a water surface. Air from the stomach is usually expelled completely in 2 to 3 expirations, whereas air from the lungs causes bubbling at each expiration. About one-fourth litre of warm water (35°C) should be passed through the funnel held high up above the patient's head. When funnel is empty, compress the tube below it between the finger and thumb and lower it below the level of the stomach, and its contents will be emptied by syphon action on releasing the pressure on the rubber tubing. If stomach pump is used, applying suction on the bulb will siphon the stomach contents. Preserve this for chemical analysis. If there is any bleeding, abandon the procedure. Gastric lavage may be done with water ; 1:5000 potassium permanganate; five per cent sodium bicarbonate ; four percent tannic acid; one percent sodium or potassium iodide; one to three percent calcium lactate; saturated lime water or starch solution, or 0.9% saline. Next, use about half litre of suitable solution and repeat till clear and odourless fluid comes out. This indicates that there is no further interaction between the antidote and poison. At this stage, the stomach is not completely emptied but a small quantity of the fluid containing the antidote or activated charcoal suspension (one

gm/kg body weight, or/and a cathartic) is left behind in the stomach, so that it may neutralise whatever small quantity of the poison is left behind. Ryle's tube or a number 10 to 12 French catheter can be used for infants and children, and about 25 cm. is necessary to reach the stomach. After a recent heavy meal, the bulky contents are first removed by emetics. Stomach wash is better than emesis because of the discomfort caused to the patient in vomiting.

In poisoning with salicylates, phenothiazines, tricyclic antidepressants, antihistamines, lavage can be done up to 12 to 18 hours after ingestion of the poison.

Contraindications: The only absolute contraindication is corrosive poisoning (except carbolic acid), owing to the danger of perforation. In the following conditions stomach wash can be done by taking proper precautions. (1) Convulsant poisons, as it may lead to convulsions. Lavage should be done after controlling the convulsions. (2) Comatose patients, because of the risk of aspiration of fluid into the air-passages. The airway should be sealed by cuffed endotracheal tube (8 to 9 mm) and lavage done. (3) Volatile poisons, which may be inhaled. (4) Upper alimentary disease, e.g., oesophageal varices. (5) In patients with marked hypothermia, and haemorrhagic diathesis.

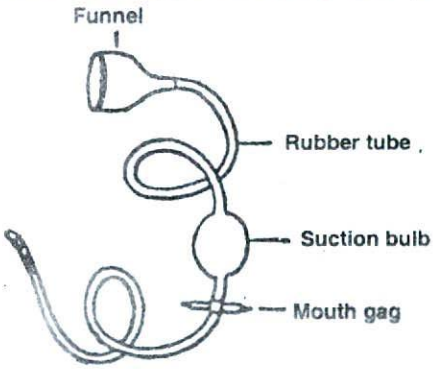

Fig. (24–5). Stomach pump.

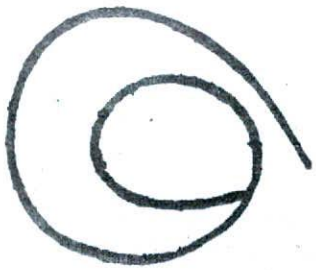

Fig. (24–6). Ryle's tube.

Emetics : Emetics should be used only if there is difficulty in obtaining or using a stomach tube. Vomiting can be produced only if the medullary centres are still responsive. Due to the danger of inhaling gastric contents, vomiting should only be induced when a conscious patient is lying on his side with the head dependent. Ipecacuanha powder one to 2 g. or 30 ml. of ipecac syrup for adults, 15 ml (1 to 12 years), 10 ml (9 to 12 months), 5 ml (6 to 9 months) followed by several glasses of water induces vomiting in 90 to 95% of patients within 20 to 30 minutes. Syrup of ipecac contains cephaeline and emetine. It induces vomiting by local activation of peripheral sensory receptors in the GIT, and stimulation of vomiting centre. The dose is repeated if vomiting does not occur in half hour. This is the only and best method of producing vomiting. Ingestion of excessive amount of salt water may cause fatal hypernatraemia. **Household emetics,** i.e. mustard powder (one teaspoon) and common salt are not effective and can lead to complications. Apomorphine, copper sulphate, tartar emetic and zinc sulphate are absolete

Contraindications : Same as for stomach wash and (1) severe heart and lung diseases, (2) advanced pregnancy, and (3) after ingestion of a CNS stimulant, because further stimulation associated with vomiting may produce convulsions.

(III) Administration of Antidotes : Antidotes are substances which counteract or neutralise the effects of poisons. Common modes of action of antidotes are : (1) Inert complex formation, e.g. chelating agents for heavy metals, dicobalt edetate for cyanide. (2) Accelerted detoxification, e.g. thiosulphate for cyanide. (3) Reduced toxic conversion, e.g. ethanol for methanol. (4) Receptor site blockade, e.g. naloxone for opiates. (5) Receptor site blockade, e.g. atropine for organophosphates at muscarinic receptor sites. (6) Toxic effect bypass, e.g. 100% oxygen in cyanide poisoning.

Tickling throat : Make patient lie face down or sit well forward with the head lower than the chest, and ask the patient to touch the back of the throat with is fingers or with yourown finger or a blunt object, such as a spoon handle or a wooden tongue depressor. This is usually ineffective.

(I) Mechanical or Physical Antidotes : They neutralise poisons by mechanical action or prevent

their absorption. (1) **Activated charcoal** is a fine, black, odourless powder. It is produced by the destructive distillation of various organic materials, usually from wood pulp, and then treating at high temperatures with a variety of activating agents, such as steam, CO_2, etc., to increase its adsorptive capacity. The particles are small, but the surface area is very large. It can be used by mixing with water to form a soup-like slurry. It acts mechanically by adsorbing and retaining within its pores organic, and also to a less degree mineral poisons, and thus delays the absorption from the stomach. Barbiturates, atropine, benzodiazipines, opiates, quinine, strychnine, phenothiazines, digitalis, amphetamines, antidepressants, antiepileptics, antihistamines, chloroquine, cimetidine, tetracycline, theophylline pyrethrins, aluminium phosphide are well adsorbed. In multipe doses it significantly increases the total body clearance of opium, cyanide and phenobarbital. Phenol, salicylates, kerosene, paracetamol are moderately adsorbed. It is not useful in poisoning with corrosives, heavy metals cyanide, hydrocarbons and alcohol. The initial dose is 60 to 100 g. in adults and 15 to 30 g. in children. Adsorption may lead to release of the offending chemical as the pH of the environment changes during the passage of the material through the gastrointestinal tract. Repeat doses of 50 g. every four hours can be given in poisoning by aspirin, phenobarbital, theophylline and carbamazepine, up to 2 days. (2) **Demulcents** are substances which form a protective coating on the gastric mucous membrane and thus do not permit the poisons to cause any damage, e.g. milk, starch, egg-white, mineral oil, milk of magnesia, aluminium hydroxide gel, etc. Fats and oils should not be used for oil-soluble poisons, such as kerosene, phosphorus, organophosphorus compounds, DDT, phenol, turpentine, aniline, acetone, carbontetrachloride, etc. (3) **Bulky food** acts as a mechanical antidote to glass powder by imprisoning its particles within its meshes, and thus prevent damage being effected by the sharp glass particles.

Multidose activated charcoal: It facilitates the passage of substances from plasma into the intestinal lumen (by creating a concentration gradient between the blood and bowel fluid) where the concentration of toxin has been significantly lowered by intraluminal charcoal adsorption, and significantly decreases the half-life of several drugs. Initial loading dose is 1 to 2 g/kg. Repeat doses of 0.5 to 1 g/kg are given at 4 to 6 hours intervals. It can also be administered by continuous infusion of 0.25 to 0.5 g/kg/hour through a nasogastric tube.

(2) Chemical Antidotes: They counteract the action of poison by forming harmless or insoluble compounds or by oxidising poison when brought into contact with them. (1) Common salt decomposes silver nitrate by direct chemical action, forming the insoluble silver chloride. (2) Albumen precipitates mercuric chloride. (3) Dialysed iron is used to neutralise arsenic. (4) Copper sulphate is used to precipitate phosphorus. (5) Potassium permanganate has oxidising properties. 1:5000 solution is used in poisoning for opium and its derivatives, strychnine, phosphorus, hydrocyanic acid, cyanides, barbituric acid and its derivatives, atropine and other alkalis. When it reacts with the poison in the stomach, it loses its pink colour. The wash must be continued till the solution coming out of the stomach is of the same pink colour as the solution put in. (6) A solution of tincture iodine or Lugol's iodine 15 drops to half a glass of warm water precipitates most alkaloids, lead, mercury, silver, quinine and strychnine. (7) Tannic acid 4%, or tannin in the form of a strong tea or one teaspoonful of tannic acid in water tends to precipitate apomorphine, cinchona, strychnine, nicotine, cocaine, aconite, pilocarpine, lead, silver, aluminium, cobalt, copper, mercury, nickel and zinc. (8) Alkalis neutralise acids by direct chemical action. It is safer to give little weak solution of an alkaline hydroxide, magnesia or ammonia. Bicarbonates should not be given, because of the possible risk of rupturing the stomach due to liberated CO_2. (9) Acids neutralise alkalis by direct chemical action. Only those substances which are by themselves harmless should be given e.g. vinegar, lemon juice, canned fruit juice. Neutralisation of acids with alkali and vice versa should be avoided because exothermic reaction of neutralisation can cause additional injury.

Socalled **universal antidote** consisting of activated charcoal, magnesium oxide and tannic acid is not recommended.

(3) Physiological or Pharmacological Antidotes: They act on the tissues of the body and produce symptoms exactly opposite to those caused by the poison. They are used after some of the poison is absorbed into the circulation. Their use is somewhat limited and not without danger. These agents act on the principle of antagonism by

interfering with another's action upon the enzymes, tissue cells or opposing nerve systems. Most of the known antidotes are only partial in their action. Atropine and physostigmine are two real physiological antidotes, as both of them affect nerve endings and produce opposite effects on the heart rate, state of the pupils, and glandular secretory activity. Other examples are: cyanides and amyl nitrite; barbiturates and picrotoxin or amphetamine; strychnine and barbiturates.

Chelating Agents: Chelating agents (metal complexing agents) are used in the treatment of poisoning by heavy metals. They have a greater affinity for the metals as compared to the endogenous enzymes. The complex of the agent and metal is more water soluble than the metal itself, resulting in higher renal excretion of the complex.They can form stable, soluble complexes with calcium and certain heavy metals.

(A) B.A.L. (British anti-lewisite; dimercaprol; dimercaptopropanol): It is used as a physiological antidote in arsenic, lead, bismuth, copper, mercury, gold, thallium and antimony. Many heavy metals have great affinity for sulphydryl (SH) radicles and combine with them in tissues and deprive the body of the use of respiratory enzymes of tissue cells. Dimercaprol has two unsaturated sulphydryl groups which combine with the metal, and thus prevent union of arsenic with the SH group of the respiratory enzyme system. The compound formed by the heavy metal and dimercaprol is relatively stable, which is carried into the tissue fluids particularly plasma, and is excreted in the urine. In severe poisoning a dose of 3 to 4 mg/kg. is given. Each ml. contains 50 mg. Three ml of 10% BAL and 20% benzyl benzoate in arachis oil is injected deep i.m. fourth hourly for the first two days, and then twice daily for ten days or till recovery. It should not be used when liver is damaged. BAL may induce haemolysis in the G-6-PD deficient individuals.

(B) E.D.T.A. (ethylenediaminetetraacetic acid; calcium disodium versenate; edathemil; edetic acid; versene): It is a chelating agent and is effective in lead, mercury, copper, cobalt, cadmium, iron and nickle poisoning. The usual dose is 25 to 35 mg/kg. body weight in 250 to 500 ml. of 5% glucose or normal saline i.v. over a one to 2 hour period twice daily for five days, and may be repeated after two to three days. It forms chelates with lead which are water-soluble, non-toxic, non-ionised, non-metabolised and excreted intact in the urine. It is superior to B.A.L. for the treatment of poisoning by arsenic and mercury.

(C) Penicillamine (cuprimine; dimethyl cystine): It is a hydrolysis product of penicillin. It has a stable SH group. It is given in a dose of 30 mg/kg. body weight up to a total of 2 g. per day in 4 divided doses orally for about 7 days. One to 3 g. can be given in slow normal saline drip daily for 2 to 4 days. It is the chelating agent of maximum efficiency for the heavy metals.

(D) DMSA, succimer (Meso-2, 3-dimercaptosuccinic acid): It is used in lead, mercury and arsenic poisoning. It is superior to EDTA in the treatment of lead poisoning, as it does not lead to redistribution of lead to the brain. It is less toxic to the kidneys. It can be given in patients with G-6PD deficiency. It is given in a dose of 10 mg/kg orally every 8 hours for 5 days, followed by the same dose every 12 hours for 14 days. A combination of succimer and EDTA is said to be more effective.

DMSA and DMPS possess the same dithiol (sulphydryl) chelating grouping as dimercaprol and the molecules are more hydrophilic. They have a better therapeutic index.

(E) DMPS: (2,3-dimercaptopropane 1-sulfonate) is effective in the treatment of mercury, lead and arsenic poisoning. It is given in a dose of 5 mg/kg. i.v. in 6 divided doses, followed by 100 mg. orally twice a day for 24 days.

(F) Desferrioxamine: It contains trivalent iron as a chelate and is very useful in acute iron poisoning. 8 to 12 g. is given orally daily to absorb iron in the stomach. Two g. in five percent of laevulose solution is given i.v. to bind absorbed iron, repeated twelve hourly if necessary. It is also used to promote removal of radioactive heavy metals.

(IV) Elimination of Poison by Excretion: Indications: (1) Severe poisoning. (2) Progressive deterioration inspite of full supportive care. (3) When there is high risk of serious morbidity or mortality. (4) When normal route of excretion of the toxic compound is impaired. (5) When the poison produces delayed but serious toxic effects. (6) When the patient is having cardiovascular, respiratory or other diseases that increase the hazards.

(1) Renal Excretion : It may be improved by giving large amounts of fluid, tea or lemonade orally. Forced diuresis may cause pulmonary or

cerebral oedema. Urinary acidification is not recommended.

(2) Purging: Thirty g. sodium sulphate with large amounts of water, hastens the elimination of poison in the stool. Magnesium sulphate should be avoided, since sufficient may be absorbed to produce central nervous system depression in cases of renal failure. To remove unabsorbed material from the intestinal tract, poorly absorbable material, such as liquid petroleum which is a solvent for fat-soluble agents is effective. Sorbitol 50 ml of 70% solution is a better purgative, but in young children it may cause fluid and electrolyte imbalance.

(3) Whole-bowel irrigation: Whole bowel irrigation involves the use of a polythylene glycol with electrolyte lavage solution which is a non-absorbable, osmotically active compound. This is administered usually by nasogastric tube (0.5 litres/hour to children less than 5 years of age and 2 litres/hour to adults) continuously until the rectal effluent is clear. It takes about 2 to 4 hours. It is useful in patients who have ingested large quantitites of substances that are difficult to remove, eg, iron and lithium overdose, sustained release preparations, cocaine, heroin, etc.

(4) Diaphoretics: In most cases, it is doubtful whether this speeds up the excretion of toxic agents. In most cases application of heat (blankets, hot water bottles), and administration of hot beverages (hot tea, hot milk, hot lemonade) will cause increased perspiration. Profuse perspiration will be produced by five mg. of pilocarpine nitrate, s.c. and a less marked effect may be produced by cutaneous irritation and cutaneous vasodilation produced by alcohol, salicylates and antipyretics.

(5) Forced alkaline diuresis, achieving a urinary pH of 7.5 to 9 promotes, excretion of drugs that are weak acids, such as salicylates, phenobarbital, chlorpropamide, methotrexate, etc. A solution of sodium bicarbonate 50 to 100 meq. added to one litre of 0.45% saline may be administered at 250 to 500 ml/hr for the first 1 to 2 hours. Alkaline solution and diuretics should be administered to maintain a urinary output of 2 to 3 ml/kg/hr.

(6) Peritoneal Dialysis: Alcohols, long-acting barbiturates, chloral hydrate, lithium, salicylates, bromides, inorganic mercury, quinidine, theophylline, and sodium chlorate are effectively removed by peritoneal dialysis. For adults, the exchange is usually 2 litres; for children under 5 years, 200 ml. It is only 10 to 25% as effective as haemodialysis. Exchange transfusion especially in children is useful in barbiturate, CO and salicylate poisoning.

(7) Haemodialysis: It is very useful for removing ethanol, methanol, ethylene glycol, chloral hydrate, lithium, trivalent arsenic, acetaminophen, bromides, phenobarbital, bromides, salicylates, fluoride, sodium chlorate, digitalis, methaqualone, boric acid and thiocyanates.

(8) Charcoal Haemoperfusion: This is useful even with highly protein-bound substances that have a large volume of distribution and are lipid-soluble. They include barbiturates, salicylates, paraquat, phenytoin, theophylline, chloral hydrate, digitalis, glutethimide, methaqualone, methotrexate, pentobarbital, carbamozepine, theophylline and paracetamol. Blood is circulated extracorporeally from an arterial source through a filter filled with adsorptive materials, i.e. charcoal coated with various polymers (acrylic hydrogel is commonly used), or resins and then back to the patient's venous side. The circuit is heparinised and primed with saline.

(V) Symptomatic Treatment: It refers to the adoption of general measures to support the life of the patient and to lessen suffering. The symptoms should be treated on general lines.

(VI) Adequate follow-up is necessary to treat the complications if any. In suicidal cases, psychiatric treatment is necessary.

CHAPTER 25
AGRICULTURAL POISONS

More than thousand chemicals are currently used as insecticides and pesticides. They can be classified as follows depending on the toxicity.

(I) VIRTUALLY HARMLESS: (1) Phenoxyacetic acid plant hormones, e.g. M.C.P.A., D.C.P.A., T.C.P.A. They are used for dock and thistle control. (2) Copper oxides, oxychlorides, used as fungicides. (3) Lime-sulphur washes, used as orchard fungicides. (4) Petroleum washes, used as orchard insecticides. (5) Tar-oil emulsion, used as orchard ovicides.

(II) COMPARATIVELY HARMLESS: (1) Sulphuric acid (20%), used as weed-killer. (2) Sodium chlorate used as mass herbicide for roads and rail tracks.

(III) MILDLY TOXIC: (1) Chlorinated hydrocarbon insecticides: (a) DDT. (b) Gammexane. (c) Methoxachlor. (d) Chlordan, aldrin and dieldrin. They are used to control fly, louse, tick, as agricultural insecticides and cattle disinfestors.

(IV) HIGHLY TOXIC: (1) Arsenical compounds: (a) Sodium arsenite. (b) Lead and calcium arsenate. (c) Paris green. They are used as weed-killers and orchard insecticides. (2) Nicotine, sulphates, tannates. They are used as horticultural insecticides. (3) Hydrocyanic acid, KCN, NaCN. They are used as a disinfestor and raticide. (4) Dinitro compounds, e.g. D.N.P. (dinitrophenol), D.N.O.C. (dinitro-orthocresol). They are used as selective weed-killers, ovicides and insecticides. (5) Organic polyphosphates: (a) HETP (hexaethyltetraphosphate). (b) TEPP (tetraethyl-pyrophosphate). (c) OMPA (octamethylpyrophos-phoramide). (d) Parathion (diethyl nitrophenyl thiophosphate). They are used as insecticides and acaricides.

INSECTICIDES OF VEGETABLE ORIGIN: They are nicotine, pyrethrins and rotenone.

(I) CHEMICAL INSECTICIDES: (I) Inorganic: They are phosphorus and compounds of antimony, arsenic, barium, mercury, thallium, zinc and fluorides.

(II) SYNTHETIC ORGANIC CHEMICAL INSECTICIDES: They can be divided into: (1) Phosphate esters. (2) Carbamates. (3) Chlorinated hydrocarbons: (a) Indane derivatives (chlordane, heptachlor, aldrin, dieldrin, endrin, diendrin). (b) Chlorobenzene derivatives: (DDT, chlorophenothane). Fatal dose: 150 mg/kg. body weight. (c) Benzene hexachloride (lindane, gammexane). Fatal dose: 15 g. (d) Chlorinated camphenes. (toxaphene, strobane). Fatal dose: 2 g.

ORGANOPHOSPHORUS POISONS

They are esters of phosphoric acid and form two series of compounds.

(A) Alkyl phosphates: (1) HETP. (2) TEPP (Tetron). (3) OMPA. (4) Dimefox. (5) Isopestox. (6) Malathion (Kill bug; Bugsoline). (7) Sulfotepp. (8) Demeton (9) Trichlorfon.

(B) Aryl phosphates: (1) Parathion (nitrostigmine) (Follidol; Kill phos; Ekato). (2) Paraoxon. (3) Methyl- parathion (Metacide). (4) Chlorthion. (5) Diazinon (Diazion; Tik 20). They are available as dusts, granules and liquids.

Absorption: They are absorbed by inhalation, through the skin, mucous membranes and the gastrointestinal tract. When sprayed in air, absorption in the plants occurs through leaves and stems.

Metabolism occurs in the liver. Detoxification occurs via cytochrome P_{450} monooxygenase. The aryl organophosphates require liver activation to become toxic. Excretion of metabolites occurs in the urine.

They are mixed with a solvent, usually aromax, which is responsible for kerosine-like smell in the body cavities, stomach contents, vomit, froth, etc. Some of the solvents used are odourless.

Action: Traces of acetylcholine are produced at the myoneural junction, which is hydrolysed to choline and acetic acid spontaneously. Hydrolysis is greatly increased by cholinesterases, which are present in plasma and on the membranes, or within the cytoplasm of many cells. Organophosphorus compounds are powerful inhibitors of carboxylic esterase enzymes, including acetylcholinesterase (true cholinesterase found in red cells, nervous tissue and skeletal muscle) and pseudocholinesterase (found in plasma, liver, heart, pancreas and brain). Organophosphorus compounds bind firmly to the esterase enzyme, inactivating it by phosphorylation, at the myoneural junctions and synapses of the ganglions. A proportion of the enzyme inhibited by a single dose is restored to activity within a few hours but some remains permanently inactivated and can ony be replaced by the synthesis of new proteins. These enzymes and particularly acetylcholine esterase (AChE) are important in the maintenance of impulse transfer between nerve and

skeletal muscle cells. Organic phosphates inhibit AChE in all parts of the body, due to which acetylcholine accumulates at the parasympathetic, sympathetic and somatic sites and transfer of nerve impulses across synapses at the autonomic ganglia at the nerve-muscle junction is prevented. Phosphorylated acetylcholinesterase loses an alkyl group, due to which the enzyme cannot spontaneously hydrolyse and becomes permanently inactivated. They are called **cholinesterase inhibitors.** This produces a syndrome of over-activity due to unhdrolysed acetylcholine. The inactivation of cholinesterase enzymes becomes irreversible after 24 to 36 hours. Symptoms appear in both the sympathetic and parasympathetic nervous system. Mild poisoning usually occurs when cholinesterase activity is 20 to 50% of normal; moderate poisoning occurs when activity is 10 to 20% of normal, and severe poisoning when activity is less than 10% normal. Small repeated exposures may gradually depress the cholinesterase activity to very low levels, often resulting in minimal symptoms. Symptoms are similar to those resulting from overdosage of acetylcholine, pilocarpine, physostigmine or muscarine. They have three distinct toxic actions. (1) A **muscarine-like effect** which potentiates postganglionic parasympathetic activity and affects pupils, bronchial muscles, salivary and sweat glands (stimulated), urinary bladder (contracted), cardiac sinus node (blocked). (2) **Nicotine-like stimulation followed by paralysis** of preganglionic and somatic motor nerves, causing twitchings of the eyelids, tongue and facial muscles followed by neuromuscular block and paralysis. (3) **Central nervous system stimulation** followed by depression causing headache, giddiness, restlessness, apprehension, tremors, ataxia, insomnia, coma and death.

Signs and Symptoms: Ocular exposure causes persistent miosis. Onset of systemic symptoms is most rapid following inhalation, and least rapid following absorption from the skin. Involuntary muscles and secretory glands are affected first, then voluntary muscles and finally vital brain centres. Respiratory symptoms may resemble an attack of asthma. With massive ingestion or inhalation, symptoms may begin within five minutes, or may be delayed for half to one hour and are at a maximum in two to eight hours. Signs and symptoms appear when the cholinesterase level drops to thirty percent of its normal activity. The respiratory or gastrointestinal symptoms are more marked depending on the route of entry.

(I) Muscarinic manifestations: These symptoms can be easily remembered by the acronym SLUDGE: Salivation, lachrymation, urination, defaecation, gastrointestinal distress, and emesis. (1) **Bronchial tree:** Bronchoconstriction, increased bronchial secretions, dyspnoea, cyanosis, pulmonary oedema. (2) **G.I.T. :** Anorexia, nausea, vomiting, cramps, diarrhoea, faecal incontinence, tenesmus. Pancreatitis may develop. (3) **Sweat glands:** Increased sweating. (4) **Salivary glands:** Increased salivation. (5) **Lacrimal glands:** Increased lacrimation. (6) **C.V.S.:** Bradycardia, hypotension. (7) **Pupils:** Miosis, occasionally unequal or dilated. (8) **Ciliary body:** Blurred vision. (9) **Bladder:** Urinary incontinence.

(II) Nicotinic manifestations: (1) **Striated muscle:** Initial stimulus results in contraction. Later there is paralysis due to persistent depolarisation. Muscle weakness is due to accumulation of acetylcholine. Muscular fasciculations, cramps, weakness, areflexia. (2) **Sympathetic ganglia:** Hypertension, tachycardia, pallor, mydriasis.

(III) CNS manifestations: Restlessness, emotional lability, headache, tremor, drowsiness, confusion, slurred speech, ataxia, generalised weakness, coma, convulsions, depression of respiratory and cardiovascular centres.

In some cases muscarinic or nicotinic or CNS effects only are seen, but most cases show a combination of all three.

Mild poisoning: Signs and symptoms are: nausea, malaise, fatigue, minimal muscle weakness, cramping without diarrhoea.

Moderate poisoning: SLUDGE and/or tremors, weakness, fasciculations, confusion, lethargy, anxiety.

Severe poisoning: SLUDGE, and respiratory insufficiency, weakness, fasciculations, coma, paralysis, seizures, autonomic dysfunction.

Bradycardia and A-V block occur due to cholinergic agents (organophosphates, carbamates, endrin), antiarrhythmic agents, beta blockers, digitalis and lithium.

Porphyrinaemia, resulting in **chromo-lachryorrhoea** (shedding of red tears) due to accumulation of porphyrin in the lachrymal glands is seen very rarely.

Intermediate Syndrome: In some cases after

one to four days muscle weakness and paralysis characterised by motor cranial nerve plasies, weakness of neck flexor and proximal limb muscles, and acute respiratory paresis are seen due to prolonged cholinesterase inhibition and muscle necrosis. It does not respond to oximes or atropine.

Delayed Sequelae: Delayed peripheral neuropathy can occur one to 5 weeks after exposure to certain compounds, such as parathion, malathion, trichlorfon, etc. It begins with paraesthesias and pain or cramps in the calves followed by ataxia, weakness, and toe drop. It rapidly progresses to a flaccid paresis which can ascend similar to Guillain-Barre syndrome. Reflexes are diminished. The disease may progress for 2 to 3 months, and muscle wasting occurs.

Chronic Poisoning: It usually occurs as an occupational hazard in agriculturists. The main features are: muscle cramps, weakness, gait disorders, paraesthesias, drowsiness, confusion, irritability, anxiety, etc.

Fatal Dose:

TEPP 50 mg. i.m. or 100 mg. orally.
OMPA 80 mg. i.m. or 175 mg. orally.
Parathion 80 mg. i.m. or 175 mg. orally.
HETP 60 mg. i.m. or 350 mg. orally.
Malathion and diazinon one g. orally.

Fatal Period: Usually within 24 hours in untreated cases, and within ten days in those treated cases when treatment is not successful. In non-fatal cases, the acute effects last for six to thirty hours which disappear in 2 to 3 days, but may sometimes persist for two weeks. Complete recovery occurs in ten days in patients treated early, unless hypoxic encephalopathy intervenes.

Cause of Death: Death is caused by paralysis of respiratory muscles, respiratory arrest due to failure of respiratory centre, or intense broncho-constriction. Late death, as long as 15 days after acute ingestion may be caused by ventricular arrhythmias.

Diagnosis: (1) Five ml of heparinised blood should be collected for cholinesterase determination. Alternatively, samples can be frozen. The cholinesterase activity of blood and plasma fall by 22 to 88%. The average normal values are 77 to 142 in the red cells and 41 to 140 in the plasma. The plasma cholinesterase is more sensitive and will fall more rapidly and before that of red cells. Thus, if there is dissociation of the two, i.e., if the plasma is down and red cells relatively little changed, the amount of exposure is less, than if they are both down. The plasma value will approach normal in seven to ten days. In untreated cases, plasma cholinesterase levels may require 4 weeks to normalise. The cholinesterase at the motor end-plate can be demonstrated histochemically in muscles kept at room temperature for one to two days, and up to several months in the tissues stored at 4 to 6°C. Fixation of tissue with phosphate buffered formalin and cold acetone for 24 hours or the embalming of the body does not affect the cholinesterase activity at the myoneural junctions.

(2) Diagnosis may be confirmed by giving two mg. of atropine. In a normal person this causes marked atropinisation, but in a case of poisoning by organophosphorus, symptoms are relieved without atropinizing. Estimations of cholinesterase are confirmatory.

Treatment: (1) The patient is removed from the source of exposure, the contaminated clothing removed and the exposed areas are washed with soap and water, followed by ethanol and water, or some alkaline solution. If eye is contaminated irrigate the conjunctival recesses, cornea, bulbar conjunctiva, internal and external palpebral surfaces. (2) The airway should be kept patent. Tracheastomy may be required. (3) When the poison is ingested, the stomach should be washed with 1:5,000 potassium permanganate solution. (4) Activated charcoal one g/kg. Ipecac should not be used. A cathartic, e.g. sorbitol or magnesium citrate can be given once unless diarrhoea has occurred. (5) Avoid physostigmine, endorphonium chloride and succinylcholine. (6) Atropine sulphate arrests the muscarine effects of postganglionic parasympathetic (peripheral) activity (muscarinic receptor antagonist) and arrests CNS effects. It has no effect on nicotinic actions. It is ineffective on respiratory centre in the presence of severe asphyxia and also if B.P. falls, Heart rate exceeding 140 beats/min should be avoided. 2 to 4 mg. is given i.v. (paediatric dose 0.05 mg/kg.) as a test dose. If there is no effect this dose may be doubled every 5 to 10 min. until muscarinic symptoms are relieved. Atropine should be continued until the tracheobronchial tree is cleared of the secretions and most secretions are dried, but not pupillary status. The average patient requires 40 mg per day, but as much as 1000 mg/day has been used. It can be given in continuous

infusion. Tachycardia is not a contraindication. Once signs of adequate atropinisation occur, the dose should be adjusted to maintain this effect for at least 24 hours. When cyanosis is present, maximal oxygenation should be achieved before atropine is given for avoiding increased risk of ventricular tachyarrhythmias associated with hypoxia. (6) Specific cholinesterase reactivators like diacetyl monoxime (DAM), or 2-pyridine aldoxime methiodide (pralidoxime iodide, 2- PAM), or pralidoxime chloride (Protopan, 2-PAM chloride) act by competing for the phosphate moiety of the organophosphorus compound and release it from the cholinesterase enzyme. Its action is marked at nicotinic sites, often improving muscle strength within 10 to 40 min. It also decreases muscarinic and CNS symptoms. The adult dose is one to 2 g. i.v., either as a 5% solution given over 5 min., or in 150 ml. of saline and infused over half-an-hour. This can be repeated in one hour if muscle weakness and fasciculations are not relieved. This dose should be repeated at 6 to 12 hours intervals for 24 to 48 hours to ensure distribution to all affected sites. Maximum dose should not exceed 12 g. in a 24 hour period. Alternatively, two-and-half percent solution can be given as continuous infusion of half g./hour. If signs and symptoms of cholinergic excess persist, continued dose may be necessary until symptoms subside. Pralidoxime and atropine work synergistically, and should be used together. **Oximes** reactivate inhibited cholinesterase, remove the block at neuromuscular junction, prevent formation of phosphorylated enzyme, and directly detoxify organophosphates. They do not cross blood-brain barrier. They should be started as early as possible, although they may be beneficial when given after 24 to 36 hours of exposure. They are given until the patient is clinically well and not requiring atropine. Pralidoxime decreases the amount of atropine required and also potentiates the action of atropine. Oximes are ineffective or minimally effective for: dimefox, dimethoate, ciodrin, methyl diazinon, methyl phenocapton, phorate, schradan and wepsyn. (7) Obidoxime chloride is more potent, but its toxicity is slightly greater. Dose: 250 mg. i.v. or i.m. It can be given if oximes are not available. (8) HI-6 and H10-7 appear to have activity against all known organophosphates. (9) Convulsions can be controlled with i.v. diazepam 10 to 20 mg at a rate of 0.5 ml (5 mg. per minute), repeated if necessary after 30 to 60 minutes. This may be followed by i.v. infusion to a maximum of 3 mg/kg of body weight over 24 hours. If convulsions persist, use i.v. phenobarbital 10 to 20 mg/kg body wt. or i.v. phenytoin 18 mg/kg. For status epilepticus, general anaesthesia may be used. Diazepam decreases anxiety and counteracts some aspects of CNS toxicity which are not affected by atropine. (10) Pulmonary oedema and bronchospasm should be treated with oxygen, intubation, atropine and positive pressure ventilation. (11) Antibiotics to prevent pulmonary infections.

Prophylaxis: The precautions to be taken are: (1) Protective clothing consisting of overall of white cotton, a white cloth hood to cover the head and neck, rubber apron, gloves and boots, eye- shields and respirator. (2) The face and the hands should be thoroughly washed after spraying with soap water. (3) Not more than 2 hours spraying a day should be done by a worker, and he should not work for more than 6 successive days on spraying. A person suffering from cold, bronchitis, etc. should not be engaged in spraying operation. (4) The workers should be properly instructed and their work supervised. (5) The workers should not smoke, chew or drink in the spraying area. (6) Spraying machines, tanks, containers, hoses, etc. should be thoroughly washed at the end of the work and before repairs are carried out. (7) Stop spraying immediately if you get a rash or feel sick, if your eyesight troubles you or you begin to sweat more than usual or feel unusually thirsty or have a headache.

Post-mortem Appearances : Signs of asphyxia are found. The face is congested and there is cyanosis of the lips, fingers and nose. Blood- stained forth is seen at the mouth and nose. The stomach contents may smell of kerosene. The mucosa of the stomach is congested with submucous petechial haemorrhages. Respiratory passages are congested and contain frothy haemorrhagic exudate. The lungs show gross congestion, excessive oedema and subpleural petechiae. Heart is sometimes soft and flabby. The internal organs are congested. The brain is congested and oedematous; meninges are congested. Petechial haemorrhages are present. The cholinesterase in erythrocytes and at myoneural junctions is below normal. Organophosphorus can be detected in putrefied bodies.

Chronic Poisoning: It is seen in persons engaged in pesticide spraying of crops, due to

inhalation or skin contamination. Symptoms include, weakness, anxiety, gait disorders, muscle cramps, paraesthesias, drowsiness, confusion, irritability and psychiatric manifestations.

Poisoning : Certain groups of people are at risk. (1) Those who are engaged in the manufacture and packing of the compounds. (2) Those who use these compounds as sprays or dusts in the open as insecticides. (3) Children of users. (4) Research workers. (5) Suicide is very common. (6) Homicide is rare. Hazards to consumers of sprayed crops appear to be very slight as they rapidly breakdown into non-toxic substances. Poisoning usually occurs from cutaneous absorption, ingestion or inhalation.

CASE: (1) In one case, one drop of parathion fell on the skin of forearm, which was not washed for two minutes, caused death.

(2) A child of nine weeks died after having been given deliberately two drops of parathion (Seifert, 1954).

CHEMICAL TEST : In parathion poisoning, P-nitrophenol is excreted in the urine. Sodium hydroxide added to steam distillate of urine gives a strong yellow colour.

CARBAMATES : They are derivatives of carbonic acid. They are marketed in the form of dusts or solutions, such as aldicarb (Temik), aminocarb (Matacil), aprocarb (Baygon), carbaryl (Sevin), carbofuran (Furaxdan). Absorption occurs through all routes. Carbaryl, carbofuran, methomyl (Lannate) and propoxur are highly toxic. Aldicarb, carbendezim and triallate are moderately toxic.

Signs and Symptoms: Symptoms begin in 15 minutes to 2 hours. Carbamates differ toxicologically from organophosphates: (1) They will spontaneously hydrolyse from the cholinesterase enzymatic site within 24 to 48 hours, whereas organophosphates will not. (2) They do not effectively penetrate into the CNS, and as such CNS toxicity is limited. All other clinical manifestations are similar to organophosphates.

Treatment: Atropine is the specific antidote. Pralidoxime may diminish the severity of symptoms and help prevent some morbidity. It improves respiratory functions and patient's well-being.

ORGANOCHLORINES

The organochlorine (chlorinated hydrocarbons) can be divided into four categories: (1) DDT and analogues: DDT, methoxychlor. (2) Benzene hexachloride: gamma hexachlorobenzene (Lindane). (3) Cyclodienes and related compounds: aldrin, chlordane, chlordecone, dieldrin, endosulfan, endrin, hepatachlor, isobenan, mirex. (4) Toxaphene and related compounds. All of these pesticides are absorbed through skin, orally and via inhalation. DDT is the least well absorbed. These agents are highly lipid soluble. They are partially metabolised in the liver and directly excreted in the urine, faeces and milk. Endrin is rapidly metabolised and eliminated and does not persist in body tissues.

Action: They interfere with nerve impulse transmission. CNS is first stimulated and then depressed.

Fatal Dose: DDT; 30 g. ; gammexane 15 g.

ENDRIN: Endrin belongs to the group of cyclodiene insecticides and is a polycyclic, polychlorinated hydrocarbon. It is a stereoisomer of dieldrin. It is soluble in aromatic hydrocarbons and ketones, sparingly in alcohols, but is not soluble in water. Its taste is bitter. It melts at 245°C. It is also called **" plant penicillin"**, because of its broad spectrum of activity against various insect pests. It is sold in the market under the trade names of Endrin-We-16, Endox-DB 50, Endtox EC-20, Endrex, Tafdrin, etc. These products contain about 20 to 50% of endrin mixed with petroleum hydrocarbon, such as aromax, which smells like kerosene. It is commonly used as sprays or as dusts, diluted with inert clays.

Symptoms : These begin within one to 6 hours. They are salivation, nausea, vomiting, abdominal pain, rarely diarrhoea, hoarseness of voice, coughing, froth at the mouth and nose, dyspnoea, headache, giddiness, restlessness, hyperirritability, dilated pupils, incoordination, ataxia, mental confusion, tremors, tonic and clonic convulsions, coma and death due to respiratory failure. In non-fatal cases, most of the persons feel well after twenty-four hours.

Chronic Poisoning : Long term exposure to some of these compounds results in cumulative toxicity characterised by loss of weight, weakness, ataxia, tremors, mental changes, oligospermia and increased tendency to leukaemias, purpura, aplastic anaemia and liver cancer.

Fatal Dose : 5 to 6 g. By ingestion it is 3 times as toxic as aldrin, dieldrin and 10 times as toxic as DDT.

Fatal Period : One to several hours.

Post-mortem Appearances : The mouth and stomach contents smell of kerosene. Signs of

asphyxia are present. Endrin resists putrefaction and can be detected in the viscera quite some time after death.

Treatment : (1) Clothing should be removed and skin washed with soap and water. (2) Gastric lavage, or the stomach evacuated by emetics and cathartics. (3) Give activated charcoal. (4) Cholestyramine is a non-absorbable bile acid binding anion exchange resin, which increases the faecal excretion of organochlorines. It is given 16 g. per day in divided doses for several days. (5) There is no specific antidote. (6) Maintain and assure adequate airway, breathing and circulation. (7) If the mental state is altered give dextrose, naloxone and thiamine. (8) Control convulsions with diazepam followed by phenobarbital. If necessary general anaesthesia is given. (9) Calcium gluconate is useful.

Circumstances of Poisoning : Human poisoning occurs from occupational or accidental exposure to endrin. Suicide is very common. Homicide is rare, but it is sometimes given mixed with food or sweets, or alcohol is used to conceal the smell. Accidental deaths are very rare.

CHLOROPHENOXYACETATES: They are used as weed-killers. They include 2, 4, -D, MCPA, mecoprop, dichloroprop (DCPP), 2, 4, 5-T. They irritate skin, mouth, GIT and damage muscles, nerves and brain. They are absorbed through skin, lungs and GIT.

SYMPTOMS: They cause redness and irritation of skin and eyes. Symptoms include: burning in mouth, coughing and choking, pain in abdomen, vomiting, diarrhoea, confusion, muscle weakness, twitchings, hypotension, fast breathing, convulsions, coma and death in few hours. If patient survives for more than a few hours liver and kidney damage occurs.

TREATMENT: Symptomatic.

CHLORATE : It is used as a weedkiller, in match heads and in fireworks. Sodium salt (resembles table sugar) is more toxic than potassium salt. Fatal dose is about 20 to 30 g. It is a powerful oxidising agent, and it attacks all body cells. It reacts with thiol groups on red cells and may cause it to lyse. It oxidises haemoglobin to methaemoglobin. It is also a potent nephrotoxin. Haemolysis may liberate large quantities of potassium ion making the serum level sufficiently high to reach fatal levels.

SYMPTOMS : They develop one to 4 hours after ingestion. They include : nausea, vomiting, diarrhoea, abdominal pain, shallow breathing, blood, protein and haemoglobin in urine, oliguria or anuria and renal failure, haemolysis, jaundice and hepatic failure, methaemoglobinaemia which may reach 40% or more, agitation, generalised weakness, muscular twitchings or convulsions, and death due to renal failure. On skin and eyes, it causes irritation, redness, burns and ulcers.

TREATMENT : (1) Stomach wash. (2) Methylene blue 25 ml. 1% sol. i.v. (3) Sodium thiosulphate 2 to 5 g. in 200 ml. of 5% sodium bicarbonate as a drink. (4) Ascorbic acid one g. every 4 hours as a drink or by slow i.v. injection (5) Peritoneal dialysis or haemodialysis. (6) Symptomatic.

PARAQUAT: It is a bipyridylium compound and used as herbicide and weed-killer. It is produced commercially as a brownish concentrated liquid of the dichloride salt in 10 to 30% strength, under the trade name, 'Gramoxone' and for horticultural use, as brown granules called "Weedol" at about 5% strength. Deaths occur due to ingestion. Deaths by inhalation while spraying are very rare.

ACTION: Paraquat undergoes a NADPH (nicotinamide adenine dinucleotide phosphate) dependent reduction to form the free radical, which reacts with molecular oxygen to reform the cation and produce a superoxide free radicals and hydroxyl radical (OH), which disrupt cellular function, structure and cell death. Concentrated solutions corrode G.I. mucosa.

ABSORPTION AND EXCRETION: Absorption through inhalation, skin or eye contact is minimal. 5 to 10% of the dose is absorbed, and the rest is excreted in the faeces. It is distributed to all the organs, but the highest concentrations are found in the kidneys and the lungs, followed by muscles from which paraquat can redistribute back into the circulation as plasma concentration decreases. More than 90% of the absorbed paraquat is excreted unchanged in the urine within the first 24 hours but can be detected in urine up to three weeks after ingestion.

FATAL DOSE: Three to five gm.

FATAL PERIOD: 2 to 7 days.

SIGNS AND SYMPTOMS: Doses of less than 1.5 g produce transient vomiting and diarrhoea which resolve within days. Ingestion of more than 50 mg/kg kill within 72 hours. LOCAL: Irritation and inflammation of skin, nails, cornea, conjunctivae and nasal mucosa. G.I.T.: Oropharyngeal ulceration and corrosion, nausea, vomiting, haematemesis, diarrhoea; painful mucosal ulceration, dysphagia, aphonia, prominent pharyngeal membranes, perforation of oesophagus, mediastinitis and pneumothorax. KIDNEYS: Oliguria or non-oliguric renal failure due to acute tubular necrosis; proximal tubular dysfunction. LUNGS: Cough, haemoptysis, dyspnoea due to pulmonary oedema, haemorrhage or fibrosis. PANCREAS: Pancreatitis. LIVER: Centrilobular

hepatic necrosis and cholestasis. C.V.S.: Hypovolaemia, shock, arrhythmias. C.N.S.: Late coma, convulsions, cerebral oedema. ADRENAL: Insufficiency due to necrosis. BONE MARROW: Polymorphonuclear leucocytosis early; anaemia late.

CAUSE OF DEATH: Death occurs from multi-organ failure or corrosive effects in the G.I. tract. Death from oesophageal perforation and mediastinitis can occur within 2 to 3 days of ingestion. In ingestions of less than 3 g. death occurs from 5 days to several weeks.

TREATMENT : (1) Gastric lavage may be beneficial if done within one hour of exposure. Emetics and cathartics are contraindicated. (2) One litre of a 15 to 30% aqueous suspension of Fuller's earth or 7% bentonite are given to adsorb paraquat, followed by 200 ml. of 20% mannitol. If the adsorbent has not appeared in the stool within 6 hours of its administration, the dose of the cathartic should be repeated. (3) If the above adsorbents are not available, activated charcoal (1 to 2 g. /kg) can be given. (4) Haemodialysis and haemoperfusion is useful if done within 12 hours of ingestion. (5) Avoid oxygen therapy. (6) Remove all clothing and wash the patient thoroughly with soap and water. (7) Analgesics should be given for pain.

POST-MORTEM APPEARANCES : There may be ulceration around the lips and mouth. The kidneys may show cortical pallor and diffuse tubular damage. If rapid death from acute hepatorenal failure does not occur, then progressive lung damage may cause death within the next two weeks. In the first day or two, there is damage to the pneumocytes, with vacuolation, desquamation and necrosis. A hyaline is often seen. Diffuse pulmonary oedema and haemorrhages occur. Within a few days repair begins. The mesenchymal interstitial cells and the alveolar lining cells divide rapidly and fill the alveoli. Both granular and membranous pneumocytes are involved. Within the first week, the air spaces become occluded by mononuclear cells forming rounded-up fibroblasts. If patient continues to survive, the alveoli begin to fibrose, with reticulin and collagen being laid down to form a rigid, stiff lung. The lungs may be mistaken for a diffuse pneumonia. There may be a fibrinous pleurisy and sometimes slight bloody pleural effusion. The stomach may show erosions and patchy haemorrhages or may be normal. The liver may show pallor, mottled fatty change and centrilobular necrosis.

PYRETHRINS & PYRETHROIDS

They are extracted from crysanthemum plant. Pyrethroids are synthetic analogues. Toxicity is very low due to their rapid metabolism. They are used as insect repellents, insecticides and pesticides. They are available as sprays, dusts, powders, mats and coils. Pyrethrum, allethrin, D-allethrin, permethrin, deltamethrin, decamethrin, cypermethrin, fenvalerate, flavalinate are some of the examples.

FATAL DOSE: 1 gm/kg. body weight.

ACTION: They prolong the inactivation of the sodium channel by binding to it in the open state.

SIGNS AND SYMPTOMS: Skin contact causes dermatitis and blistering. Paraesthesias, nausea, vomiting, vertigo, fasciculations, hyperthermia, altered mental state, convulsions, pulmonary oedema and coma. Inhalation causes rhinorrhoea, sore throat, wheezing, and dyspnoea.

TREATMENT : (1) Stomach wash with activated charcoal. (2) Oils and fats should be avoided. (3) Atropine and oximes are contraindicated. (4) Skin should be washed with soap and water. (5) Give adrenaline 0.5 to 1 ml. of 1:1000, i.m. for allergic reactions. Antihistamines such as chlorphenamine or pyromethazine by slow i.v. injection after adrenaline injection.

DINITRO COMPOUNDS : Dinitro-orthocresol and dinitrophenol are commonly used weed-killers. They are also used to kill insects and fungi and to preserve wood. They act by greatly increasing the cellular metabolism of any cell with which they come in contact. It is absorbed orally, through the skin, and respiratory tract. Inhalation and ingestion may occur during spraying of crops. Fatal dose one to 2 g. and fatal period 4 to 15 days.

SYMPTOMS : They appear rapidly and resemble a thyrotoxic crisis. CONTACT : Yellow colour of the skin and hair and rashes. Burns of lips and buccal mucosa. CNS : Anxiety, restlessness, tiredness, insomnia, convulsions, coma. C.V.S : Tachycardia and arrhythmias. R.S. : Tachyapnoea and pulmonary congestion. METABOLIC : Hyperpyrexia and intense perspiration. RENAL : Acute renal failure. HEPATIC : Acute liver necrosis. EYES: severe irritation, redness, watering.

TREATMENT : Symptomatic.

FLUORIDES: Sodium fluoride is used in rat poison and cockroach powders. Sodium silicofluoride, fluoroacetamide and fluoroacetate are used as rodenticides. Fluoride compounds react with acid in the stomach and form hydrofluoric acid which is corrosive. After absorption, fluoride ions bind clacium ions and to some extent potassium and magnesium ions and cause hypocalcaemia, hypokalaemia and hypomagnesaemia. Fluoride ions also inactivate proteolytic and glycolytic enzymes and act as general protoplasmic poison.

SIGNS AND SYMPTOMS : Ingestion causes burning pain in the mouth and epigastrium, thirst, salivation, vomiting, diarrhoea, haematemesis and haematuria, coma and convulsions. Death may occur within minutes from respiratory and cardiac failure.

TREATMENT: (1) Calcium orally and intravenously. (2) Stomach wash with lime water or milk.

FATAL DOSE: 5 mg/kg. body weight.

ZINC PHOSPHIDE: It is a steel-grey crystalline powder with a garlicky odour. Symptoms are similar to those of aluminium phosphide, but are usually slower to start because of the slow release of phosphine.

It is used to preserve grain and as rat poison, which causes death due to asphyxia.

FATAL DOSE: Five g.

FATAL PERIOD: 24 hours.

TREATMENT: (1) Move patient to fresh air if there are poisonous gases or fumes. (2) Stomach wash with potassium permanganate. (3) Activated charcoal. (4) Purgatives.

POST-MORTEM APPEARANCES: Garlicky odour in stomach contents. Blood is cherry-red. Congestion and oedema of the lungs. Fatty degeneration and necrosis of the liver.

ALUMINIUM PHOSPHIDE

Aluminium phosphide (ALP) is a solid fumigant pesticide, insecticide and rodenticide. In India it is available as white tablets of Celphos, Alphos, Quickphos, Phostoxin, Phosphotex, etc., each weighing 3 g. and has the capacity to liberate one gram of phosphine (PH_3). On coming in contact with moisture ALP liberates phosphine. Phosphine is a systemic poison and affects all organs of the body. The chemical reaction is accelerated by the presence of HCL in the stomach. ALP has garlicky odour. It is widely used as grain preservative. Phosphite and hypophosphite of aluminium which are non-toxic residues are left in the grains.

Absorption and Excretion: Phosphine is rapidly absorbed from the GI tract by simple diffusion and causes damage to the internal organs. It is also rapidly absorbed from the lungs after inhalation. After ingestion, some ALP is also absorbed and is metabolised in the liver, where phosphine is slowly released accounting for the prolongation of symptoms. Phosphine is oxidised slowly to oxyacids and excreted in the urine as hypophosphite. It is also excreted in unchanged form through the lungs.

Action: Phosphine inhibits respiratory chain enzymes and has cytotoxic action. It acts by inhibiting the electron transport resulting from preferential inhibition of cytochrome oxidase.

Fatal Dose: 1 to 3 tablets.

Fatal Period: One hour to four days. Majority die within twenty-four hours.

Signs and Symptoms: They depend on the dose and severity of poisoning.

Inhalation: Mild inhalation exposure produces irritation of mucous membranes and acute respiratory distress. Other symptoms are dizziness, easy fatigue, tightness in the chest, nausea, vomiting, diarrhoea and headache. Moderate toxicity produces ataxia, numbness, paraesthesia, tremors, diplopia, jaundice, muscular weakness, incoordination and paralysis. Concentration of PH_3 in air higher than 0.3 ppm causes severe illness. Severe toxicity produces adult respiratory distress syndrome, cardiac arrhythmias, congestive heart failure, pulmonary oedema, convulsions, and coma.

Ingestion: Mild intoxication produces nausea, vomiting, headache and abdominal pain, and the patients usually recover.

In moderate and severe poisoning, systemic manifestations are early and progressive and mostly fatal. **G.I.T.:** Nausea, vomiting, diarrhoea, retrosternal pain. **C.V.S.:** Hypotension, shock, arrhythmias, myocarditis, pericarditis, acute congestive heart failure. **R.S.:** Cough, dyspnoea, cyanosis, pulmonary oedema, respiratory failure. **Hepatic:** Jaundice, hepatitis, hepatomegaly, **Renal:** Renal failure. **C.N.S.:** Headache, dizziness, altered mental state, restlessness, convulsions, acute hypoxic encephalopathy, coma. **Rare**: Muscle wasting and tenderness and bleeding diathesis, due to widespread capillary damage. Cardiogenic shock is the most common cause of death. Complications include pericarditis, acute congestive cardiac failure, acute massive gastrointestinal bleeding and ARDS Mortality is high, 35 to 100%.

Post-mortem Appearances: Garlic-like odour is present at the mouth and nostrils and in the gastric contents. Blood-stained froth is found in the mouth and nostrils. Mucous membrane of the lower part of oesophagus, stomach and duodenum are congested. Decreasing congestion of the G.I. tract is seen in the small intestine. The lungs, liver, spleen, kidneys and brain are congested. Centrizonal haemorrhagic necrosis of the liver may be seen.

Histopathology: (1) Stomach: Congestion, oedema, leucocytic infiltration, sloughing of gastric

mucosa. (2) **Lungs:** Congestion, oedema, desquamation of respiratory epithelium, thickened alveoli, lymphocytic infiltration. **Kidneys:** Congestion, necrosis, tubular degeneration and regeneration. **Adrenals:** Congestion, haemorrhage, necrosis, area of lipid depletion in cortex. **Heart:** Congestion, oedema, fragmentation of fibres, focal necrosis, leucocytic infiltration. **Brain:** Congestion, oedema.

CHEMICAL TEST: (1) Five ml. of gastric aspirate and 15 ml. of water are put in a flask and the mouth is covered with a filter paper impregnated with silver nitrate (0.1N). The flask is heated at 50°C for 15 to 20 minutes. If phosphine is present, the filter paper turns black. (2) A piece of filter paper impregnated with 0.1 N silver nitrate solution is used in the form of a mask through which the patient breathes for 5 to 10 minutes. The filter paper turns black, if phosphine is present. (3) The filter paper impregnated with $AgNO_3$ (0.1N) is used in the form of face mask and the patient is asked to breathe in and out of this filter paper for 15 to 20 minutes. Presence of PH_3 is indicated by the blackening of filter paper. In breath, the test is positive only in patients who have ingested more than six gm. of ALP.

Treatment: (1) Gastric lavage with potassium permanganate is done and repeated 2 or 3 times to oxidise the poison. (2) Give activated charcoal 100 g. orally to adsorb phosphine. (3) Antacids reduce symptoms pertaining to the stomach and reduce absorption of phosphine. (4) Liquid paraffin is given for excretion of ALP and phosphine from the gut. (5) There is no specific antidote. (6) Magnesium sulphate reduces organ toxicity, corrects hypomagnesaemia and arrhythmias. The usual dose is one g. repeated for the next two hours, and then 1 to 1.5 g. every 6 hours for 5 to 7 days in the form of continuous i.v. infusion. (7) To treat shock 4 to 6 litres of fluids are to be administered during the first 3 to 6 hours; of this 50% must be normal saline. (8) Low dose dopamine infusion, 4 to 6 microgram/kg/min is useful. (9) I.V. hydrocortisone 400 mg every 4 to 6 hours is highly effective. It reduces the dose of dopamine. (10) Hypoxia is treated with oxygen. (11) Shock should be treated with fluids and hydrocortisone. (12) Metabolic acidosis should be corrected with i.v. sodium carbonate. Peritoneal or haemodialysis is useful.

Poisoning is usually suicidal, occasionally accidental and rarely homicidal.