Q 1: Attempt all the following questions.

i. **Give the general treatment for Poison.**
   Ans. Emesis, Gastric lavage, administration of general/ universal antidote, increase urine output by administration of diuritis, dialysis

ii. **Which subjects were used in Clinical Trial Phase- I?**
    Ans. Healthy human volunteers (male) and patients like AIDS & Cancer. Women and children are not including.

iii. **What is Drug Interaction?**
    Ans. Drug Interaction can be defined as the modification of the effect of one drug by prior or concomitant administration of another drug. DI may either enhance or diminish the intended effect of one or both drugs. The interaction may modify the diagnostic or therapeutic activity of either drug.

iv. **Write the symptoms of Hepatitis.**
    Ans. Initial symptoms are fever, chills, headache, fatigue, general weakness, aches especially on right side of abdomen followed anorexia, Nausea, vomiting along with by passage of dark urine then develop jaundice.

v. **Write the diagnostic tool of CHF.**
    Ans. Weight gain due to salt intake and water retention, easy fatiguability, weakness, Nocturia, Oliguria, Impairment of memory, confusion, Insomnia, anxiety, Peripheral edema

vi. **Write the symptoms of Depression.**
    Ans. Depressive illness, phobic states, obsessive compulsive behavior and certain anxiety disorders, with serious distortion of thought, behavior, capacity to recognize reality and perception.

vii. **Write non pharmacological approaches of DM**
    Ans. Control body weight, exercise, Balance diet, avoid sugar reach food, avoid administration drug induce DM

viii. **Write the demography of Peptic Ulcer.**
    Ans. Duodenal ulcer is largely a disease of adult males. Women seem to be peculiarly immune to DU during child bearing age. Gastric ulcers occur most frequently in the
older age group and in lower socio economic status people. DU is common young adult.

ix. **Define the terms OTC. Give some examples.**

*Ans.* Over The Counter drugs. An OTC drug is safe when it has low incidence of adverse reactions and side effects under conditions of widespread availability.

x. **Classify UTI.**

*Ans.* Complicated (Acute and Chronic UTI), Uncomplicate (Acute Cystitis and Phylonephritis) and Urosepsis

xi. **Give some examples of Drug induce Hepatitis.**

*Ans.* Paracetamol, Tetracycline, CPZ, Estrogen, Asprine, Valporic acid, furosemide

xii. **Classify the type of UTI.**

*Ans.* LUTI (Urethritis, Cystitis) and UUTI (Ureteritis and Phylonephritis)

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**SECTION B**

Q2. Give etiology, pathophysiology, manifestations and treatment of T.B.

*Ans.* Tuberculosis (TB) is a common, and in many cases lethal, infectious disease caused by various strains of mycobacteria, usually *Mycobacterium tuberculosis*. Tuberculosis typically attacks the lungs, but can also affect other parts of the body. It is spread through the air when people who have an active TB infection cough, sneeze, or otherwise transmit respiratory fluids through the air. Most infections are asymptomatic and latent, but about one in ten latent infections eventually progresses to active disease which, if left untreated, kills more than 50% of those so infected.

One third of the world's population is thought to have been infected with *M. tuberculosis*, with new infections occurring at a rate of about one per second. The distribution of tuberculosis is not uniform across the globe; about 80% of the population in many Asian and African countries test positive in tuberculin tests, while only 5–10% of the United States population tests positive. More people in the developing world contract tuberculosis because of compromised immunity, largely due to high rates of HIV infection and the corresponding development of AIDS.

**Signs and symptoms**

General signs and symptoms include fever, chills, night sweats, loss of appetite, weight loss, and fatigue, and significant finger clubbing may also occur.
**Pulmonary**

If a tuberculosis infection does become active, it most commonly involves the lungs (in about 90% of cases). Symptoms may include chest pain and a prolonged cough producing sputum. About 25% of people may not have any symptoms (i.e. they remain "asymptomatic"). Occasionally, people may cough up blood in small amounts, and in very rare cases, the infection may erode into the pulmonary artery, resulting in massive bleeding (Rasmussen's aneurysm). Tuberculosis may become a chronic illness and cause extensive scarring in the upper lobes of the lungs. The upper lung lobes are more frequently affected by tuberculosis than the lower ones. The reason for this difference is not entirely clear. It may be due either to better air flow, or to poor lymph drainage within the upper lungs.

**Extrapulmonary**

In 15–20% of active cases, the infection spreads outside the respiratory organs, causing other kinds of TB. These are collectively denoted as "extrapulmonary tuberculosis". Extrapulmonary TB occurs more commonly in immunosuppressed persons and young children.

**Transmission**

When people with active pulmonary TB cough, sneeze, speak, sing, or spit, they expel infectious aerosol droplets 0.5 to 5.0 µm in diameter. A single sneeze can release up to 40,000 droplets. Each one of these droplets may transmit the disease, since the infectious dose of tuberculosis is very low (the inhalation of fewer than 10 bacteria may cause an infection).

**Pathogenesis**

About 90% of those infected with *M. tuberculosis* have asymptomatic, latent TB infections (sometimes called LTBI), with only a 10% lifetime chance that the latent infection will progress to overt, active tuberculous disease. In those with HIV, the risk of developing active TB increases to nearly 10% a year. If effective treatment is not given, the death rate for active TB cases is up to 66%. TB infection begins when the mycobacteria reach the pulmonary alveoli, where they invade and replicate within endosomes of alveolar macrophages. The primary site of infection in the lungs, known as the "Ghon focus", is generally located in either the upper part of the lower lobe, or the lower part of the upper lobe. Tuberculosis of the lungs may also occur via infection from the blood stream. This is known as a Simon focus and is typically found in the top of the lung. This hematogenous transmission can also spread infection to more distant sites, such as peripheral lymph nodes, the kidneys, the brain, and the
bones. All parts of the body can be affected by the disease, though for unknown reasons it rarely affects the heart, skeletal muscles, pancreas, or thyroid.

**Diagnosis**

**Active tuberculosis**

Diagnosing active tuberculosis based merely on signs and symptoms is difficult, as is diagnosing the disease in those who are immunosuppressed. A diagnosis of TB should, however, be considered in those with signs of lung disease or constitutional symptoms lasting longer than two weeks. A chest X-ray and multiple sputum cultures for acid-fast bacilli are typically part of the initial evaluation. Interferon-\(\gamma\) release assays and tuberculin skin tests are of little use in the developing world. IGRA have similar limitations in those with HIV. A definitive diagnosis of TB is made by identifying *M. tuberculosis* in a clinical sample (e.g. sputum, pus, or a tissue biopsy). However, the difficult culture process for this slow-growing organism can take two to six weeks for blood or sputum culture. Thus, treatment is often begun before cultures are confirmed.

**Vaccines:** - The only currently available vaccine as of 2011 is bacillus Calmette–Guérin (BCG) which, while it is effective against disseminated disease in childhood, confers inconsistent protection against contracting pulmonary TB. Nevertheless, it is the most widely used vaccine worldwide, with more than 90% of all children being vaccinated. However, the immunity it induces decreases after about ten years.

**Management**

Treatment of TB uses antibiotics to kill the bacteria. Effective TB treatment is difficult, due to the unusual structure and chemical composition of the mycobacterial cell wall, which hinders the entry of drugs and makes many antibiotics ineffective. The two antibiotics most commonly used are isoniazid and rifampicin, and treatments can be prolonged, taking several months. Latent TB treatment usually employs a single antibiotic, while active TB disease is best treated with combinations of several antibiotics to reduce the risk of the bacteria developing antibiotic resistance. People with latent infections are also treated to prevent them from progressing to active TB disease later in life. Directly observed therapy, i.e. having a health care provider watch the person take their medications, is recommended by the WHO in an effort to reduce the number of people not appropriately taking antibiotics. The evidence to support this practice over people simply taking their medications independently is poor. Methods to remind people of the importance of treatment do, however, appear effective.
Q3. Define Toxicology, write its branches. How can you treat Barbiturate and Opiod Poisoning Patients?

**Ans.** It is the study of poisonous effect of drugs and other chemicals (Household, environmental pollutant, industrial, agricultural, homicidal) with emphasis on detection, prevention and treatment of poisonings. It also includes the study of effects of drugs, since the same substance can be drug or a poison, depending on the dose.

**Barbiturate poisoning**

Barbiturate poisoning is mainly dealt like the cases of other CNS depressant poisoning. Manifestations are due to excessive CNS depression- patient is flabby and comatose with shallow and falling respiration, fall in BP and cardiovascular collapse, renal shut down, pulmonary complications.

Lethal dose depends on lipid solubility. It is 2-3 g for the more lipid soluble agents (short acting barbiturates) and 5-10 g for less lipid soluble phenobarbitone.

**Treatment:**

1. Gastric lavage: leave a suspension of activated charcoal in the stomach to prevent absorption of the drug from intestines.
2. Supportive measures: such as, patent airway assisted respiration, oxygen, maintenance of blood volume by fluid infusion and use of vasopressors- dopamine may be be preffered for its renal vasodilating action.
3. Alkaline dieresis: with sodim bicarbonate 1meq/kg i.v. with or without mannitol is helpful only in the case of long acting barbiturates which are eliminated primarily by renal excretion.
4. Haemodialysis and haemoperfusion (through a column of activated charcoal or other absorbents) is highly effective in removing long acting as well as short acting barbiturates.

There is no specific antidote for barbiturates. In the past, analeptics like metrazol, bemegride, etc have been used in the attempt to awaken the patient. This is dangerous, may precipitate convulsions while the patients is still comatose- mortality is increased. The emphasis now is on keeping the patient alive till the poison has been eliminated.

**OPIOID POISONING**

Acute opioid (morphine) poisoning consists of stupor or coma, flaccidity, shallow and occasional breathing, cyanosis, pinpoint pupil, fall in BP and shock; convulsions may be seen in few, pulmonary edema occurs at terminal stages, death is due to respiratory failure.
Treatment: consists of respiratory support (positive pressure respiration also decreases pulmonary edema formation) and maintenance of BP (i.v. fluids, vasoconstrictors).

Gastric lavage should be done with pot. Permanganate to remove the unabsorbed drug. Lavage is indicated even when morphine has been injected; being a basic drug it is partitioned to the acid gastric juice, ionizes there and does not diffuse back into the blood.

Specific antidote: Naloxone 0.4-0.8 mg i.v. repeated every 2-3 minute till respiration picks up, is the preferred specific antagonist because it does not have any agonistic action and does not per se depress respiration. It has a short duration of action. Injection should be repeated every 1-4 hour later on, according to the response. Nalorphine is no longer used.

Q 4. What do you mean Asthma? Write its sign, symptom and diagnostic tool. How can you treat the Asthma?

Ans. Asthma is a common chronic inflammatory disease of the airways characterized by variable and recurring symptoms, reversible airflow obstruction, and bronchospasm. Common symptoms include wheezing, coughing, chest tightness, and shortness of breath.

Asthma is thought to be caused by a combination of genetic and environmental factors. Its diagnosis is usually based on the pattern of symptoms, response to therapy over time, and spirometry. It is clinically classified according to the frequency of symptoms, forced expiratory volume in one second (FEV1), and peak expiratory flow rate. Asthma may also be classified as atopic (extrinsic) or non-atopic (intrinsic) where atopy refers to a predisposition toward developing type 1 hypersensitivity reactions. Treatment of acute symptoms is usually with an inhaled short-acting beta-2 agonist (such as salbutamol) and oral corticosteroids. In very severe cases intravenous corticosteroids, magnesium sulfate and hospitalization maybe required. Symptoms can be prevented by avoiding triggers, such as allergens and irritants, and by the use of inhaled corticosteroids. Long-acting beta agonists (LABA) or leukotriene antagonists may be used in addition to inhaled corticosteroids if asthma symptoms remain uncontrolled. The prevalence of asthma has increased significantly since the 1970s. As of 2011, 235–300 million people were affected globally, including about 250,000 deaths.

Asthma is characterized by recurrent episodes of wheezing, shortness of breath, chest tightness, and coughing. Sputum may be produced from the lung by coughing but is often hard to bring up. During recovery from an attack it may appear pus like due to high levels of white blood cells called eosinophils. Symptoms are usually worse at night and in the early morning or in response to exercise or cold air. Some people with asthma rarely experience symptoms, usually in response to triggers, whereas others may have marked and persistent symptoms.
**Pathophysiology**

Asthma is the result of chronic inflammation of the airways which subsequently results in increased contractability of the surrounding smooth muscles. This among other factors leads to bouts of narrowing of the airway and the classic symptoms of wheezing. The narrowing is typically reversible with or without treatment. Occasionally the airways themselves change. Typical changes in the airways include an increase in eosinophils and thickening of the lamina reticularis. Chronically the airways' smooth muscle may increase in size along with an increase in the numbers of mucous glands. Other cell types involved include: T lymphocytes, macrophages, and neutrophils. There may also be involvement of other components of the immune system including: cytokines, chemokines, histamine, and leukotrienes among others.

**Diagnosis**

While asthma is a well recognized condition, there is not one universal agreed upon definition. It is defined by the Global Initiative for Asthma as "a chronic inflammatory disorder of the airways in which many cells and cellular elements play a role. The chronic inflammation is associated with airway hyper-responsiveness that leads to recurrent episodes of wheezing, breathlessness, chest tightness and coughing particularly at night or in the early morning. These episodes are usually associated with widespread but variable airflow obstruction within the lung that is often reversible either spontaneously or with treatment". There is currently no precise test with the diagnosis typically based on the pattern of symptoms and response to therapy over time. A diagnosis of asthma should be suspected if there is a history of: recurrent wheezing, coughing or difficulty breathing and these symptoms occur or worsen due to exercise, viral infections, allergens or air pollution. Spirometry is then used to confirm the diagnosis. In children under the age of six the diagnosis is more difficult as they are too young for spirometry.

**Management**

While there is no cure for asthma, symptoms can typically be improved. A specific, customized plan for proactively monitoring and managing symptoms should be created. This plan should include the reduction of exposure to allergens, testing to assess the severity of symptoms, and the usage of medications. The treatment plan should be written down and advise adjustments to treatment according to changes in symptoms. The most effective treatment for asthma is identifying triggers, such as cigarette smoke, pets, or aspirin, and eliminating exposure to them. If trigger avoidance is insufficient, the use of medication is recommended. Pharmaceutical drugs are selected based on, among other things, the severity
of illness and the frequency of symptoms. Specific medications for asthma are broadly classified into fast-acting and long-acting categories. Bronchodilators are recommended for short-term relief of symptoms. In those with occasional attacks, no other medication is needed. If mild persistent disease is present (more than two attacks a week), low-dose inhaled corticosteroids or alternatively, an oral leukotriene antagonist or a mast cell stabilizer is recommended. For those who have daily attacks, a higher dose of inhaled corticosteroids is used. In a moderate or severe exacerbation, oral corticosteroids are added to these treatments.

Q 5. Define Drug Information Centre and outline its various activities/functions.

Ans. In hospital the drug information services are available to all health care professionals who include clinicians, pharmacist, nurse, biochemists, microbiologist etc. The clinical pharmacist is relied upon as an expert to provide the information about drugs to the health professionals and public. The clinicians are the largest user of DIS. The information is obtained about DIS through:

1. Medical representative
2. Attending Lecture and symposia
3. Medical literature
4. Product data sheet
5. Articles from medical press
6. Reports from the committee on safety of medicine and publication like drug and therapeutic bulletin
7. From text books

The DIC provides information about Pharmaceutical and Therapeutic applications of the drugs.


Therapeutic:- Pharmacodynamics, Pharmacokinetics, Contraindications, uses, Mode of action, Toxicology, comparative pharmacology.

**SOURCES OF INFORMATION**

A Drug information centre must have a good collections of the source materials in order to deal with the questions as they arise. This should be continuously updated and maintained so that approx 90% of the questions can be resolved.
Information source may be

1. **Primary source:**
   Periodicals are also referred as journals, serials, magazines, bulletins etc are published as issues e.g. Weekly, monthly or quarterly volumes. They contain scientific information, research articles, review articles, book reviews and even advertisements.
   Periodicals are again of two types
   - **Primary periodicals:** Contains report of original research.
   - **Secondary periodicals:** Portion of original research according to the needs in the condensed form.
   On the basis of scope they are again classified into
   - a. **Scientific periodicals** - Original research articles which are reviewed by experts like IJPS.
   - b. **Professional periodicals** - Contains articles which have practical aspect. They are less technically and scientifically as compared to above like Pharma time and AJPE.
   - c. **Commercial periodicals** - They contain information that is useful to trade people information regarding new product entry, price change, information, promotional etc. e.g. CIMS, Drug index etc.

2. **Secondary source**
   Pharmacopoeia and formularies. These contain standards for drugs and related articles with description, tests and formulas for preparing the same which is given by a recognized authority.

Drug Information Centre location and functions:-
DIC/DIS should be located in a separate section of pharmacy. It contains medical texts, journals, large number of reference texts, photocopying facility and audio visual arrangements.

**Objectives:-**

1. To uplift the profession of pharmacy by bringing better interaction between pharmacist and community.
2. To improve patient compliance with drug dosage regimens and to improve therapeutic outcome.
3. DIC by using suitable technique and methods such as verbal written or audio visual communication, educate and counsel the patient about
a. Advertisements and educating patients about pharmacology of drug, storage and cost.
b. Clarifying doubts regarding sexual problem, contraceptives and family planning.
c. Drug abuse, alcoholism, smoking hazards and other socio medical problems.
d. Drug toxicity, overdose and poisoning of drug.
e. Advice on health issue.
g. Maintenance of records:- Different files should be prepared to differentiate the technical data and easy search.

QUALIFICATION OF PHARMACIST TO RUN DIS: -
1. To critically evaluate the drug literature
2. To edit the information to facilitate decision making.
3. He should have good communication skills.
4. Computer knowledge
5. Member of PTC.
6. Should know the research methodology.
7. Aware of the source of information.

Q 6. Describe the factors that affect placental transfer of drugs. Name drugs which cross placental barrier Name some drugs which can be (a) safely administered during pregnancy; (b) unsafe drugs during pregnancy.

Ans. Placental membranes are lipoidal and allow free passage of lipophilic drugs, while restricting hydrophilic, while restricting hydrophilic drugs. Placental efflux P-gp also serves to limit foetal exposure to maternally administered drugs. However, restricted amounts of non-lipid soluble drugs, when present in high concentrations or for long periods in maternal circulation, gain access to foetus. Some influx transporters also operate at the placenta. Thus, it is an incomplete barrier and almost any drug taken by the mother can affect the foetus or the new born (Drug taken just before delivery eg morphine).

Factors affecting the placental transfer of drugs:
1. Plasma protein binding:-
   Many drugs possess physiochemical affinity of plasma proteins. Acidic drugs generally bind to plasma albumin and basic drugs to alpha-1 acid glycoprotein. A drug's efficiency may be affected by the degree to which it binds to the proteins within blood plasma. The less bound a drug is, the more efficiently it can traverse cell
membranes or diffuse. Common blood proteins that drugs bind to are human serum albumin, lipoprotein, glycoprotein, α, β, and γ globulins.

A drug in blood exists in two forms: bound and unbound. Depending on a specific drug's affinity for plasma protein, a proportion of the drug may become bound to plasma proteins, with the remainder being unbound. If the protein binding is reversible, then a chemical equilibrium will exist between the bound and unbound states, such that:

\[
\text{Protein + drug} \rightleftharpoons \text{Protein-drug complex}
\]

Notably, it is the unbound fraction which exhibits pharmacologic effects. It is also the fraction that may be metabolized and/or excreted. For example, the "fraction bound" of the anticoagulant warfarin is 97%. This means that of the amount of warfarin in the blood, 97% is bound to plasma proteins. The remaining 3% (the fraction unbound) is the fraction that is actually active and may be excreted.

Protein binding can influence the drug’s biological half-life in the body. The bound portion may act as a reservoir or depot from which the drug is slowly released as the unbound form. Since the unbound form is being metabolized and/or excreted from the body, the bound fraction will be released in order to maintain equilibrium.

Since albumin is alkalotic, acidic and neutral drugs will primarily bind to albumin. If albumin becomes saturated, then these drugs will bind to lipoprotein. Basic drugs will bind to the acidic alpha-1 acid glycoprotein. This is significant because various medical conditions may affect the levels of albumin, alpha-1 acid glycoprotein, and lipoproteins.

**Examples:**

- **To albumin**- Barbiturates, BZD, Nsaids, Valproic acid, Phenytoin, Penicillins, Sulphonamides, tetracyclines, tobutamide, warfarin.
- **To alpha-1 acid glycoprotein**- Beta blockers, Bupivacaine, Lidocaine, disopyramide, Imipramine, Methadone, Prazosin, Quinidine, Verapamil.

2. **Drug concentrated in tissues:**

Sometimes the drugs are concentrated in the tissues and leads to the sustained action of drugs and sometimes leads to toxicity.

Examples:

a. Skeletal muscle and heart- digoxin, emetine (bound to muscles protein).

b. Liver- Chloroquine, tetracycline

c. Kidney- Digoxin, Chloroquine

d. Thyroid- Iodine
e. Brain- Chlorpromazine, acetazolamide, isoniazid.
f. Retina- Chloroquine
g. Bone and teeth- Tetracycline, heavy metals.

**Drugs safe and unsafe during pregnancy**

<table>
<thead>
<tr>
<th>Drug class</th>
<th>Unsafe drug</th>
<th>Safer alternative</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anti emetics</td>
<td>Domperidone. Ondensatron</td>
<td>Promethazine, cyclizine, dicuclomine</td>
</tr>
<tr>
<td>Peptic ulcer and GERD</td>
<td>Cemetidine, omeprazole, cisapride, lansoprazole</td>
<td>Ranitidine, famotidine</td>
</tr>
<tr>
<td>Laxatives</td>
<td>Senna, biscacodyl</td>
<td>Isabghula, lactulose</td>
</tr>
<tr>
<td>Analgesics</td>
<td>Aspirin, COX-2 inhibitors, codeine, tramadol</td>
<td>Paracetamol, ibuprofen</td>
</tr>
<tr>
<td>Anti allergics</td>
<td>Cetrizie, loratidine, astemizole</td>
<td>Chlorpheniramine, promethazine</td>
</tr>
<tr>
<td>Anti tubercular</td>
<td>Pyrizinamid, ethambutol</td>
<td>Isoniazid, rifampsin</td>
</tr>
<tr>
<td>Anti amoebic</td>
<td>Metronidazole, tinidazole</td>
<td>Diloxanide furoate</td>
</tr>
<tr>
<td>Anti malarial</td>
<td>Quinine, mefloquine, primaquine</td>
<td>Chloroquine, proguanil</td>
</tr>
<tr>
<td>Anti helminthics</td>
<td>Albendazole, mebendazole</td>
<td>Piperazine, niclosamide</td>
</tr>
</tbody>
</table>

**Q7. Discuss in detail `diabetes' management.**

**Ans.** Diabetes mellitus, often simply referred to as diabetes, is a group of metabolic diseases in which a person has high blood sugar, either because the body does not produce enough insulin, or because cells do not respond to the insulin that is produced. Diabetes mellitus derived from two Greek words. I. Diabetes - a siphon or running through. II. Mellitus - sweet. The rare disease diabetes insipidus has similar symptoms as diabetes mellitus, but without disturbances in the sugar metabolism. Diabetes results in abnormal levels of glucose in the bloodstream. This can cause severe short-term and long term consequences ranging from brain damage to amputations and heart disease. Another major micro vascular complications i.e. (retinopathy, nephropathy and neuropathy) of is also well-established

**History**

Diabetes mellitus was first described in Egypt 3000 years ago. About 400 B.C., Charak and Shusrut in India noted not only the sweetness of the urine, but also the correlation between obesity and diabetes. Near the beginning of the Christian era the Romans Aretaeus and Celsus described the disease.
Classification
Generally speaking, if not specified, diabetes mellitus means primary or idiopathic diabetes mellitus. Secondary diabetes mellitus is occurrence of hyperglycemia associated with some identifiable causes such as due to chronic pancreatitis, post-pancreatectomy, hormone producing tumors, certain drugs, hemochromatosis and genetic endocrinologic disorders. Diabetes mellitus is classified into four broad categories: type 1, type 2, gestational diabetes and "other specific types"

Type 1 diabetes
The term "type 1 diabetes" has replaced several former terms, including childhood-onset diabetes, juvenile diabetes, and insulin-dependent diabetes mellitus (IDDM). Type 1 diabetes mellitus is characterized by loss of the insulin-producing beta cells of the islets of Langerhans in the pancreas, leading to insulin deficiency. This type can be further classified as immune-mediated or idiopathic. The majority of type 1 diabetes is of the immune-mediated nature, in which beta cell loss is a T-cell-mediated autoimmune attack.

Type 2 diabetes
Type 2 diabetes is the most common type. The term "type 2 diabetes" has replaced several former terms, including adult-onset diabetes, obesity-related diabetes, and noninsulin-dependent diabetes mellitus (NIDDM).
Type 2 diabetes mellitus is characterized by insulin resistance, which may be combined with relatively reduced insulin secretion. The defective responsiveness of body tissues to insulin is believed to involve the insulin receptor. However, the specific defects are not known.
In the early stage of type 2, the predominant abnormality is reduced insulin sensitivity. At this stage, hyperglycaemia can be reversed by a variety of measures and medications that improve insulin sensitivity or reduce glucose production by the liver. Eventually, 80% diabetic are obese 50 % obese are diabetic.

Gestational diabetes
Gestational diabetes mellitus (GDM) resembles type 2 diabetes in several respects, involving a combination of relatively inadequate insulin secretion and responsiveness. It occurs in about 2%–5% of all pregnancies and may improve or disappear after delivery. Gestational diabetes is fully treatable, but requires careful medical supervision throughout the pregnancy. About 20%–50% of affected women develop type 2 diabetes later in life.
Other specific types:

- Latent autoimmune diabetes of adults (LADA) is a condition in which type 1 DM develops in adults. Adults with LADA are frequently initially misdiagnosed as having type 2 DM, based on age rather than etiology.
- Genetic defects of beta-cell function and Genetic defects in insulin action.
- Diseases of the exocrine pancreas.
- Pancreatitis.
- Trauma/pancreatectomy.
- Endocrinopathies.
- Aldosteronoma.
- Drugs or chemicals induced.

Pathophysiology

Insulin is the principal hormone that regulates uptake of glucose from the blood into most cells (primarily muscle and fat cells, but not central nervous system cells). Therefore, deficiency of insulin or the insensitivity of its receptors plays a central role in all forms of diabetes mellitus.

Humans are capable of digesting some carbohydrates, in particular those most common in food; starch, and some disaccharides such as sucrose, are converted within a few hours to simpler forms, most notably the monosaccharide glucose, the principal carbohydrate energy source used by the body. The rest are passed on for processing by gut flora largely in the colon. Insulin is released into the blood by beta cells (β-cells), found in the islets of Langerhans in the pancreas, in response to rising levels of blood glucose, typically after eating. Insulin is used by about two-thirds of the body's cells to absorb glucose from the blood for use as fuel, for conversion to other needed molecules, or for storage. Insulin is also the principal control signal for conversion of glucose to glycogen for internal storage in liver and muscle cells. Lowered glucose levels result both in the reduced release of insulin from the β-cells and in the reverse conversion of glycogen to glucose when glucose levels fall. This is mainly controlled by the hormone glucagon, which acts in the opposite manner to insulin. Glucose thus forcibly produced from internal liver cell stores (as glycogen) re-enters the bloodstream; muscle cells lack the necessary export mechanism. Normally, liver cells do this when the level of insulin is low (which normally correlates with low levels of blood glucose). Higher insulin levels increase some anabolic ("building up") processes, such as cell growth and duplication, protein synthesis, and fat storage. Insulin (or its lack) is the
principal signal in converting many of the bidirectional processes of metabolism from a catabolic to an anabolic direction, and vice versa. In particular, a low insulin level is the trigger for entering or leaving ketosis (the fat-burning metabolic phase).

If the amount of insulin available is insufficient, if cells respond poorly to the effects of insulin (insulin insensitivity or resistance), or if the insulin itself is defective, then glucose will not have its usual effect, so it will not be absorbed properly by those body cells that require it, nor will it be stored appropriately in the liver and muscles. The net effect is persistent high levels of blood glucose, poor protein synthesis, and other metabolic derangements, such as acidosis. When the glucose concentration in the blood is raised to about 9-10 mmol/L (except certain conditions, such as pregnancy), beyond its renal threshold (i.e. when glucose level surpasses the transport maximum of glucose reabsorption), reabsorption of glucose in the proximal renal tubuli is incomplete, and part of the glucose remains in the urine (glycosuria). This increases the osmotic pressure of the urine and inhibits reabsorption of water by the kidney, resulting in increased urine production (polyuria) and increased fluid loss. Lost blood volume

**Signs and symptoms**
The classical symptoms of diabetes are polyuria (frequent urination), polydipsia (increased thirst) and polyphagia (increased hunger)

**Diagnosis**

<table>
<thead>
<tr>
<th>Conditions</th>
<th>2 hour glucose mmol/l (mg/dl)</th>
<th>Fasting glucose mmol/l (mg/dl)</th>
<th>HbA1c %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>&lt;7.8 (&lt;140)</td>
<td>&lt;6.1 (&lt;110)</td>
<td>&lt;6.0</td>
</tr>
<tr>
<td>Impaired fasting glycaemia</td>
<td>&lt;7.8 (&lt;140)</td>
<td>≥ 6.1(≥110) and &lt;7.0(&lt;126)</td>
<td>6.0-6.4</td>
</tr>
<tr>
<td>Impaired glucose tolerance</td>
<td>≥7.8 (≥140)</td>
<td>&lt;7.0 (&lt;126)</td>
<td>6.0-6.4</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>≥11.1 (≥200)</td>
<td>≥7.0 (≥126)</td>
<td>≥6.5</td>
</tr>
</tbody>
</table>

Diabetes mellitus is characterized by recurrent or persistent hyperglycaemia, and is diagnosed by demonstrating any one of the following

- Fasting plasma glucose level ≥ 7.0 mmol/l (126 mg/dl)
- Plasma glucose ≥ 11.1 mmol/l (200 mg/dL) two hours after a 75 g oral glucose load as in a glucose tolerance test
- Symptoms of hyperglycaemia and casual plasma glucose ≥ 11.1 mmol/l (200 mg/dl)
- Glycated haemoglobin (Hb A1C) ≥ 6.5%
Treatment of Diabetes Mellitus

Non-Pharmacological Management of Diabetes Mellitus

1. Achieve and maintain ideal body weight.
2. Derive 55 to 60% of total caloric intake from carbohydrates.
3. Consume foods containing unrefined carbohydrate with fiber, attempting to take in 40g of soluble fiber per day.
4. Consume only modest amounts of sucrose.
5. Exercise

Oral Hypoglycaemic Agents

<table>
<thead>
<tr>
<th>Drug class/dose</th>
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</thead>
<tbody>
<tr>
<td><strong>First generation</strong></td>
<td><strong>Second generation</strong></td>
</tr>
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4. BIGUANIDES (after food)

| 1. Phenoforphmin   |   |
| 2. Metformin       |   |

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