



7

CLINICAL CHEMISTRY

7.1 INTRODUCTION

The clinical chemistry measures chemical changes in the body for diagnosis, therapy and prognosis of disease. Primarily testing is performed using body fluids such as serum, plasma, and urine to determine the chemical components.



OBJECTIVES

After reading this lesson, you will be able to:

- describe the regulation of blood sugar
- explain various blood glucose laboratory tests
- describe laboratory tests of Urea, Calcium and Phosphates
- describe Lipid Profile, Urine Creatinine

7.2 BLOOD SUGAR

The blood sugar concentration or blood glucose level is the amount of glucose (sugar) present in the blood of a human or animal. The body naturally tightly regulates blood glucose levels as a part of metabolic homeostasis. Glucose is the primary source of energy for the body's cells. Glucose is transported from the intestines or liver to body cells via the bloodstream, and is made available for cell absorption via the hormone insulin, produced by the body primarily in the pancreas.

The mean normal blood glucose level in humans is about 100 mg/dL; however, this level fluctuates throughout the day. Glucose levels are usually lowest in the morning, before the first meal of the day (termed "the fasting level"), and rise after meals for an hour or two by a few milligram. The normal blood glucose level (tested while fasting) for non-diabetics, should be between 70 and 100



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milligrams per deciliter (mg/dL). Blood sugar levels for those without diabetes and who are not fasting should be below 125 mg/dL. The blood glucose target range for diabetics, according to the American Diabetes Association, should be 70–130 (mg/dL) before meal, and less than 180 mg/dL after meals (as measured by a blood glucose monitor).

Blood sugar levels outside the normal range may be an indicator of a medical condition. A persistently high level is referred to as hyperglycemia; low levels are referred to as hypoglycemia. Diabetes mellitus is characterized by persistent hyperglycemia from any of several causes, and is the most prominent disease related to failure of blood sugar regulation. Intake of alcohol causes an initial surge in blood sugar, and later tends to cause levels to fall. Also, certain drugs can increase or decrease glucose levels.

7.2.1 Regulation

The body's homeostatic mechanism keeps blood glucose levels within a narrow range. It is composed of several interacting systems, of which hormone regulation is the most important.

There are two types of mutually antagonistic metabolic hormones affecting blood glucose levels:

- catabolic hormones (such as glucagon, cortisol and catecholamines) which increase blood glucose
- anabolic hormone (insulin), which decreases blood glucose.

7.2.2 Abnormality in blood sugar levels

7.2.3 High blood sugar

If blood sugar levels remain too high the body suppresses appetite over the short term. Long-term hyperglycemia causes many of the long-term health problems including heart disease, eye, kidney, and nerve damage. The most common cause of hyperglycemia is diabetes. When diabetes is the cause, physicians typically recommend an anti-diabetic medication as treatment. From the perspective the majority of patients, treatment with an old, well-understood diabetes drug such as metformin will be the safest, most effective, least expensive, most comfortable route to managing the condition. Diet changes and exercise implementation may also be part of a treatment plan for diabetes.

7.2.4 Low blood sugar

If blood sugar levels drop too low, a potentially fatal condition called hypoglycemia develops. Symptoms may include lethargy, impaired mental functioning; irritability; shaking, twitching, weakness in arm and leg muscles; pale complexion; sweating; paranoid or aggressive mentality and loss of consciousness.

7.2.5 Glucose measurement

7.2.5.1 Sample type

Glucose is measured in whole blood, plasma or serum. Historically, blood glucose values were given in terms of whole blood, but most laboratories now measure and report plasma or serum glucose levels. Because red blood cells (erythrocytes) have a higher concentration of protein (e.g., hemoglobin) than serum, serum has a higher water content and consequently more dissolved glucose than does whole blood. Collection of blood in clot tubes for serum chemistry analysis permits the metabolism of glucose in the sample by blood cells until separated by centrifugation. Red blood cells, for instance, do not require insulin to intake glucose from the blood. Higher than normal amounts of white or red blood cell counts can lead to excessive glycolysis in the sample, with substantial reduction of glucose level if the sample is not processed quickly. Ambient temperature at which the blood sample is kept prior to centrifuging and separation of plasma/serum also affects glucose levels. At refrigerator temperatures, glucose remains relatively stable for several hours in a blood sample.

Loss of glucose can be prevented by using Fluoride tubes since fluoride inhibits glycolysis. However, these should only be used when blood will be transported from one hospital laboratory to another for glucose measurement. Red-top serum separator tubes also preserve glucose in samples after being centrifuged isolating the serum from cells. Arterial, capillary and venous blood has comparable glucose levels in a fasting individual. Following meals, venous levels are somewhat lower than those in capillary or arterial blood; a common estimate is about 10%.

7.2.6 Measurement techniques

Two major methods have been used to measure glucose. The first, still in use in some places, is a chemical method exploiting the nonspecific reducing property of glucose in a reaction with an indicator substance that changes color when reduced. The more recent technique, using enzymes specific to glucose, is less susceptible to this kind of error. The two most common employed enzymes are glucose oxidase and hexokinase. In either case, the chemical system is commonly contained on a test strip which is inserted into a meter, and then has a blood sample applied. Test-strip shapes and their exact chemical composition vary between meter systems and cannot be interchanged. More precise blood glucose measurements are performed in a medical laboratory, using hexokinase, glucose oxidase or glucose dehydrogenase enzymes.

7.2.7 Blood glucose laboratory tests

1. fasting blood sugar (i.e., glucose) test (FBS)
2. two-hr postprandial blood sugar test (2-h PPBS)
3. oral glucose tolerance test (OGTT)



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4. intravenous glucose tolerance test (IVGTT)
5. glycosylated hemoglobin (HbA_{1C})
6. self-monitoring of glucose level via patient testing
7. Random blood sugar (RBS)
8. Average blood glucose may be estimated by measuring glycated hemoglobin (HbA_{1c})

7.2.8 Clinical Correlation

The fasting blood glucose level, which is measured after a fast of 8 hours, is the most commonly used indication of overall glucose homeostasis, largely because disturbing events such as food intake are avoided. The metabolic response to a carbohydrate challenge is conveniently assessed by a postprandial glucose level drawn 2 hours after a meal or a glucose load. In addition, the glucose tolerance test, consisting of several timed measurements after a standardized amount of oral glucose intake, is used to aid in the diagnosis of diabetes.

Finally, there are several influences on blood glucose level aside from food intake. Infection, for instance, tends to change blood glucose levels, as does stress either physical or psychological. Exercise, especially if prolonged or long after the most recent meal, will have an effect as well.

7.3 UREA

7.3.1 Blood urea nitrogen (BUN)

The liver produces urea in the urea cycle as a waste product of the digestion of protein. Normal human adult blood should contain between 6 to 20 mg of urea nitrogen per 100 ml. Individual laboratories may have different reference ranges, and this is because the procedure may vary.

7.3.2 Interpretation

BUN is an indication of renal health. Normal ranges 8-20 mmol/L. If Glomerular Filtration Rate (GFR) and blood volume decrease (hypovolemia) then BUN will increase. Other factors responsible for its increment are fever, increased catabolism, high protein diet and gastrointestinal bleeding.

7.4 CALCIUM-PHOSPHATES

Calcium metabolism or calcium homeostasis is the mechanism by which the body maintains adequate calcium levels. Derangements of this mechanism lead to hypercalcemia or hypocalcemia, both of which can have important consequences for health.

7.4.1 Calcium location and quantity

Calcium is the most abundant mineral in the human body. The average adult body contains in total approximately 1 kg, 99% in the skeleton in the form of calcium phosphate salts. The extracellular fluid (ECF) contains approximately 22.5 mmol, of which about 9 mmol is in the serum. Approximately 500 mmol of calcium is exchanged between bone and the ECF over a period of twenty-four hours.



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7.4.2 Biological functions

- Structural function: Supporting material in bones. Present as calcium phosphate.
- Signalling function: Intracellular calcium functions as a second messenger for some hormones.
- Enzymatic function: Calcium acts as a coenzyme for clotting factors.

Calcium also causes the release of Acetylcholine from Pre-synaptic terminal in the transmission of nerve impulse. Calcium causes the contraction of muscles.

7.4.3 Normal ranges

The serum level of calcium is closely regulated with normal total calcium of 2.2-2.6 mmol/L (9-10.5 mg/dL) and normal ionized calcium of 1.1-1.4 mmol/L (4.5-5.6 mg/dL). The amount of total calcium varies with the level of serum albumin, a protein to which calcium is bound. The biologic effect of calcium is determined by the amount of ionized calcium, rather than the total calcium. Ionized calcium does not vary with the albumin level, and therefore it is useful to measure the ionized calcium level when the serum albumin is not within normal ranges, or when a calcium disorder is suspected despite a normal total calcium level.

7.4.4 Effector organs

7.4.4.1 Absorption

Calcium enters the body in a normal diet and is absorbed across the intestinal brush border membrane. Calbindin is a vitamin D-dependent calcium-binding protein inside intestinal epithelial cells actively transports calcium into the body.

7.4.4.2 Excretion

The kidney excretes 250 mmol a day in pro-urine, and resorbs 245 mmol, leading to a net loss in the urine of 5 mmol/d. In addition to this, the kidney processes Vitamin D into calcitriol, the active form that is most effective in assisting intestinal absorption. Both processes are stimulated by parathyroid hormone.



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7.4.4.3 The role of bone

Although calcium flow to and from the bone is neutral, about 5 mmol is turned over a day. Bone serves as an important storage point for calcium, as it contains 99% of the total body calcium. Calcium release from bone is regulated by parathyroid hormone. Calcitonin stimulates incorporation of calcium in bone, although this process is largely independent of calcitonin. Low calcium intake may also be a risk factor in the development of osteoporosis.

7.4.5 Interaction with other chemicals

7.4.5.1 Potential positive interactions

- Vitamin D is an important co-factor in the intestinal absorption of calcium, as it increases the number of calcium binding proteins, involved in calcium absorption through the apical membrane of enterocytes in small intestine. It also promotes re-absorption of calcium in the kidneys.
- Magnesium also plays an important role in calcium absorption by bones. Release of calcium from the sarcoplasmic reticulum is inhibited by magnesium. Thus hypomagnesemia results in an increased intracellular calcium level. This inhibits the release of parathyroid hormone, which can result in hypoparathyroidism and hypocalcemia. Furthermore, it makes skeletal and muscle receptors less sensitive to parathyroid hormone.

7.4.5.2 Potential negative interactions

- Unesterified long-chain saturated fatty acids, i.e. palmitic acid, have a melting point above body temperature and, with sufficient calcium in the intestinal lumen, form insoluble calcium soaps.
- Sodium binding to calcium
- Phytic acid binding to calcium
- Oxalic acid binding to calcium
- Cortisol binding to calcium
- Low pH food and proteins (the latter promotes gastric acid)

7.5 LIPID PROFILE

Lipid profile or lipid panel, is a panel of blood tests that serves as an initial broad medical screening tool for abnormalities in lipids, such as cholesterol and triglycerides. The results of this test can identify certain genetic diseases and can determine approximate risks for cardiovascular disease, certain forms of pancreatitis, and other diseases.

7.5.1 Components

The lipid profile typically includes:

- Low-density lipoprotein
- High-density lipoprotein
- Triglycerides
- Total cholesterol

Using these values, a laboratory may also calculate:

- Very low-density lipoprotein
- Cholesterol:HDL ratio

7.5.2 Procedure

Traditionally, most laboratories have required patients to fast for 9–12 hours before screening. However, recent studies have questioned the utility of fasting before lipid panels, and some diagnostic labs now routinely accept non-fasting samples.

7.5.3 Implications

This test is used to identify hyperlipidemia (various disturbances of cholesterol and triglyceride levels), many forms of which are recognized risk factors for cardiovascular disease and rarely pancreatitis. A total cholesterol reading can be used to assess an individual's risk for heart disease; however, it should not be relied upon as the only indicator. The individual components that make up total cholesterol reading LDL, HDL, and VLDL are also important in measuring risk. The lipid profile includes total cholesterol, HDL-cholesterol (often called good cholesterol), LDL-cholesterol (often called bad cholesterol), and triglycerides. Sometimes the report will include additional calculated values such as the Cholesterol/HDL ratio or a risk score based on lipid profile results, age, sex, and other risk factors. The lipid profile is used to guide providers in deciding how a person at risk should be treated. The results of the lipid profile are considered along with other known risk factors of heart disease to develop a plan of treatment and follow-up.

7.5.4 Normal range

LDL	:	60–130 mg/dL
HDL	:	> 40 mg/dL
Total cholesterol	:	< 200 mg/dL
Triglycerides	:	10–150 mg/dL
VLDL	:	2–38 mg/dL



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7.6 URINE LEVELS OF SUGAR

7.6.1 Glycosuria

Glycosuria or glucosuria is the excretion of glucose into the urine. Ordinarily, urine contains no glucose because the kidneys are able to reclaim all of the filtered glucose back into the bloodstream. Glycosuria is nearly always caused by elevated blood glucose levels, most commonly due to untreated diabetes mellitus. Rarely, glycosuria is due to an intrinsic problem with glucose reabsorption within the kidneys themselves, a condition termed renal glycosuria. Glycosuria leads to excessive water loss into the urine with resultant dehydration, a process called osmotic diuresis.

7.6.2 Pathophysiology

Blood is filtered by millions of nephrons, the functional units that comprise the kidneys. In each nephron, blood flows from the arteriole into the glomerulus, a tuft of leaky capillaries. The Bowman's capsule surrounds each glomerulus, and collects the filtrate that the glomerulus forms. The filtrate contains waste products (e.g. urea), electrolytes (e.g. sodium, potassium, chloride), amino acids, and glucose. The filtrate passes into the renal tubules of the kidney. In the first part of the renal tubule, the proximal tubule, glucose is reabsorbed from the filtrate, across the tubular epithelium and into the bloodstream.

The proximal tubule can only reabsorb a limited amount of glucose. When the blood glucose level exceeds about 160–180 mg/dl, the proximal tubule becomes overwhelmed and begins to excrete glucose in the urine. This point is called the renal threshold of glucose (RTG). Some people, especially children and pregnant women, may have a low RTG (less than ~7 mmol/L glucose in blood to have glucosuria). If the RTG is so low that even normal blood glucose levels produce the condition, it is referred to as renal glycosuria. Glucose in urine can be identified by Benedict's qualitative test. A urine dipstick can show a false-positive glucosuria if someone is taking Pyridium standard, medications that relieve symptoms of urinary tract infection.

7.6.3 Diagnosis

A doctor normally can diagnose renal glycosuria when a routine urine test (Urinalysis) detects glucose in the urine, while a blood test indicates that the blood glucose level is normal. In most affected individuals, the condition causes no apparent symptoms (asymptomatic) or serious effects. When renal glycosuria occurs as an isolated finding with otherwise normal kidney function, the condition is thought to be inherited as an autosomal recessive trait.



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7.7 URINE LEVELS OF CREATININE

Creatinine is a breakdown product of creatine phosphate in muscle and is usually produced at a fairly constant rate by the body (depending on muscle mass). Serum creatinine (a blood measurement) is an important indicator of renal health because it is an easily-measured by-product of muscle metabolism that is excreted unchanged by the kidneys. Creatinine itself is produced via a biological system involving creatine, phosphocreatine (also known as creatine phosphate), and adenosine triphosphate (ATP, the body's immediate energy supply). Creatine is synthesized primarily in the liver from the methylation of glycoamine by S-adenosyl methionine. It is then transported through blood to the other organs, muscle, and brain where, through phosphorylation, it becomes the high-energy compound phosphocreatine. During the reaction, creatine and phosphocreatine are catalyzed by creatine kinase, and a spontaneous conversion to creatinine may occur.

7.7.1 Creatinine clearance

Creatinine is removed from the blood chiefly by the kidneys, primarily by glomerular filtration but also via proximal tubular secretion. There is little or no tubular reabsorption of creatinine. If the filtration in the kidney is deficient, creatinine blood levels rise. Therefore, creatinine levels in blood and urine may be used to calculate the creatinine clearance (CrCl), which correlates with the glomerular filtration rate (GFR). Blood creatinine levels may also be used alone to calculate the estimated GFR (eGFR).

The GFR is clinically important because it is a measurement of renal function. However, in cases of severe renal dysfunction, the creatinine clearance rate will overestimate the GFR because hypersecretion of creatinine by the proximal tubules will account for a larger fraction of the total creatinine cleared. Ketoacids, cimetidine, and trimethoprim reduce creatinine tubular secretion and, therefore, increase the accuracy of the GFR estimate, in particular in severe renal dysfunction. An alternate estimation of renal function can be made when interpreting the blood (plasma) concentration of creatinine along with that of urea. BUN-to-creatinine ratio (the ratio of blood urea nitrogen to creatinine) can indicate other problems besides those intrinsic to the kidney; for example, a urea level raised out of proportion to the creatinine may indicate a pre-renal problem such as volume depletion. Each day, 1-2% of muscle creatine is converted to creatinine. Men tend to have higher levels of creatinine than women because, in general, they have a greater mass of skeletal muscle. Increased dietary intake of creatine or eating a lot of meat can increase daily creatinine excretion.

7.7.2 Diagnostic use

7.7.2.1 Serum creatinine

Measuring serum creatinine is a simple test, and it is the most commonly used indicator of renal function. A rise in blood creatinine level is observed only with



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marked damage to functioning nephrons. Therefore, this test is unsuitable for detecting early-stage kidney disease. A better estimation of kidney function is given by calculating the estimated glomerular filtration rate (eGFR). eGFR can be accurately calculated using serum creatinine concentration and some or all of the following variables: sex, age, weight, and race.

7.7.2.2 Urine creatinine

Creatinine concentration is also checked during standard urine drug tests. Normal creatinine levels indicate the test sample is undiluted, whereas low amounts of creatinine in the urine indicate either a manipulated test or low individual baseline creatinine levels. Random urine creatinine levels have no standard reference ranges. They are usually used with other tests to reference levels of other substances measured in the urine. Diuretics, such as coffee and tea, cause more frequent urination, thus potentially decreasing creatinine levels. A decrease in muscle mass will also cause a lower reading of creatinine, as will pregnancy.

7.7.3 Interpretation

The typical human reference ranges for serum creatinine are 0.5 to 1.0 mg/dl (about 45-90 $\mu\text{mol/l}$) for women and 0.7 to 1.2 mg/dl (60-110 $\mu\text{mol/L}$) for men. While a baseline serum creatinine of 2.0 mg/dl (150 $\mu\text{mol/l}$) may indicate normal kidney function in a male body builder, a serum creatinine of 1.2 mg/dl (110 $\mu\text{mol/l}$) can indicate significant renal disease in an elderly female. Creatinine levels may increase when ACE inhibitors (ACEI) or angiotensin II receptor antagonists (or angiotensin receptor blockers, ARBs) are taken. Using both ACEI and ARB concomitantly will increase creatinine levels to a greater degree than either of the two drugs would individually. An increase of <30% is to be expected with ACEI or ARB use.

7.7.4 Normal Results

Urine creatinine (24-hour sample) values can range from 500 to 2000 mg/day. Results depend greatly on your age and amount of lean body mass.

Another way of expressing the normal range for these test results are:

- 14 to 26 mg per kg of body mass per day for men
- 11 to 20 mg per kg of body mass per day for women

7.7.5 Abnormal Results Mean

Abnormal results of urine creatinine are nonspecific, but may be due to any of the following conditions such as, glomerulonephritis, high meat diet, kidney infection (pyelonephritis), kidney failure, muscular dystrophy (late stage), myasthenia gravis, prerenal azotemia, reduced kidney blood flow (as in shock or congestive heart failure), urinary tract obstruction.

7.8 URINE LEVELS OF PROTEINS

7.8.1 Proteinuria

Proteinuria means the presence of an excess of serum proteins in the urine. The excess protein in the urine often causes the urine to become foamy, although foamy urine may also be caused by bilirubin in the urine (bilirubinuria), or drugs such as pyridium. Up to 150 mg a day of protein may be excreted by a normal person. Proteinuria can also be caused by certain biological agents, such as Avastin used in cancer treatment, or by excessive fluid intake (drinking in excess of 4 litres of water per day).

7.8.2 Causes

There are three main mechanisms to cause proteinuria:

- Due to disease in glomerulus
- Because of increased quantity of proteins in serum (overflow proteinuria)
- Due to low reabsorption at proximal tubule (Fanconi syndrome)

7.8.3 Measurement

Conventionally, proteinuria is diagnosed by a simple dipstick test, although it is possible for the test to give a false negative reading, if the protein in the urine is composed mainly of globulins or Bence-Jones proteins. Alternatively the concentration of protein in the urine may be compared to the creatinine level in a spot urine sample. This is termed the protein/creatinine ratio (PCR). Proteinuria is defined as a protein/creatinine ratio greater than 45 mg/mmol (which is equivalent to albumin/creatinine ratio of greater than 30 mg/mmol or approximately 300 mg/g) with very high levels of proteinuria being for a PCR greater than 100 mg/mmol.

7.8.4 Associated conditions

Proteinuria may be a sign of renal (kidney) damage. Since serum proteins are readily reabsorbed from urine, the presence of excess protein indicates either an insufficiency of absorption or impaired filtration. Diabetics may suffer from damaged nephrons and develop proteinuria.

7.8.5 Conditions with proteinuria as a sign

Proteinuria may be a feature of the following conditions such as, nephrotic syndromes (i.e. intrinsic renal failure), toxic lesions of kidneys, amyloidosis, collagen vascular diseases (e.g. systemic lupus erythematosus), dehydration, glomerular diseases, strenuous exercise, stress, IgA nephropathy, IgM nephropathy, diabetes mellitus (diabetic nephropathy), drugs (e.g. NSAIDs, nicotine, penicillamine, lithium carbonate, antibiotics, or opiates (especially heroin),



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infections (e.g. HIV, syphilis, hepatitis, poststreptococcal infection), aminoaciduria, hypertensive nephrosclerosis, sickle cell disease, hemoglobinuria, multiple myeloma, myoglobinuria, organ rejection (Kidney transplant patients may have gamma-globulins in their urine if the kidneys start to reject), systemic lupus erythematosus, rheumatoid arthritis, glycogen storage disease type 1, and urinary tract infection which has spread to the kidney(s). Conditions with proteinuria consisting mainly of Bence-Jones proteins as a sign are Waldenström's macroglobulinemia, chronic lymphocytic leukemia, amyloidosis, malignancies (e.g., lymphoma, other cancers), multiple myeloma.

7.8.6 Treatment

Treating proteinuria mainly needs proper diagnosis of the cause. The most common cause is diabetic nephropathy; in this case, proper glycemic control may slow the progression. Medical management consists of angiotensin converting enzyme (ACE) inhibitors, which are typically first-line therapy for proteinuria. In patients whose proteinuria is not controlled with ACE inhibitors, the addition of an aldosterone antagonist or angiotensin receptor blocker may further reduce protein loss. Caution must be used if these agents are added to ACE inhibitor therapy due to the risk of hyperkalemia. Proteinuria secondary to autoimmune disease should be treated with steroids or steroid-sparing agent plus the use of ACE inhibitors.



INTEXT QUESTIONS 7.1

- I. Choose the best answer
- Measuring glucose levels before the first meal of the day is termed as
 - Post prandial blood glucose
 - Fasting blood glucose
 - Normal blood glucose
 - None
 - The catabolic hormone which increases blood glucose level is
 - Glucagon
 - Insulin
 - Histamine
 - None
 - The coenzyme acts as blood clotting factor is
 - Magnesium
 - Iron
 - Calcium
 - Lead

4. The vitamin D-dependent calcium-binding protein that actively transports calcium into the body
 - (a) Calbindin
 - (b) Calmodulin
 - (c) Transferrin
 - (d) Globulin
5. Good cholesterol is termed for
 - (a) LDL-cholesterol
 - (b) VLDL-cholesterol
 - (c) HDL-cholesterol
 - (d) Triglycerides

II. Fill in the blanks

6. Excretion of glucose into the urine is called as
7. is the by-product of muscle metabolism
8. The protein in the urine is composed mainly of globulins is termed medically as
9. The two most common employed enzymes in the blood glucose measurement are
10. increases if glomerular filtration rate and blood volume decrease.

III. Match the following

- | | |
|----------------------------|---------------------------------|
| 11. Bone formation | (a) LDL |
| 12. Bad cholesterol | (b) Calcium |
| 13. Glycosuria | (c) Diabetes mellitus |
| 14. Bilirubin in the urine | (d) Benedict's qualitative test |
| 15. Metformin | (e) Bilirubinuria |

**WHAT HAVE YOU LEARNED**

- Glucose is the primary source of energy for the body's cells. Its level should be between 70 and 100 mg/dL for normal person.
- Anabolic hormone (insulin) decreases blood glucose and catabolic hormones (such as glucagon, cortisol and catecholamines) increase blood glucose.
- More precise blood glucose measurements are performed in a medical laboratory, using hexokinase, glucose oxidase or glucose dehydrogenase enzymes.
- The liver produces urea in the urea cycle as a waste product of the digestion of protein.
- Derangements of the calcium mechanism lead to hypercalcemia or hypocalcemia, both of which can have important consequences for health.

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- Calbindin is a vitamin D-dependent calcium-binding protein inside intestinal epithelial cells actively transports calcium into the body.
- Hyperlipidemia is recognized as a risk factor which leads to cardiovascular disease.
- HDL-cholesterol is often called good cholesterol and LDL-cholesterol is often called as bad cholesterol.
- Glycosuria is nearly always caused by elevated blood glucose levels. Glucose in urine can be identified by Benedict's qualitative test.
- Serum creatinine is an important indicator of renal health because it is an easily-measured by-product of muscle metabolism. Creatinine is removed from the blood chiefly by the kidneys.
- Proteinuria means the presence of an excess of serum proteins in the urine.
- The three main mechanisms that cause proteinuria are disease in glomerulus, increased quantity of proteins in serum, and low reabsorption at proximal tubule (Fanconi syndrome).



TERMINAL QUESTIONS

1. Write short note on Laboratory tests of Blood glucose
2. Write short note on Lipid profile
3. Write short notes on urine levels of creatinine



ANSWERS TO INTEXT QUESTIONS

- I. 1. (b) 2. (a) 3. (c) 4. (a) 5. (c)
- II. 6. Glycosuria,
7. Creatinine,
8. Bence-Jones proteins,
9. Glucose oxidase and Hexokinase,
10. Blood urea nitrogen,
- III. 11. (b) 12. (a) 13 (d) 14. (e) 15. (c)