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3rd Edition

Panchali Pal

CHAPTER 13

Disorders of Endocrine System

LEARNING OBJECTIVES

After completion of this chapter, students will be able to:

- Provide comprehensive care to a child with Diabetes mellitus.
- Discuss various endocrine disorders.

CHAPTER OUTLINE

- Diabetes Mellitus
- Diabetes Insipidus
- Disorders of Pituitary Gland
- Disorders of Thyroid
- Disorders of Parathyroid Gland
- Congenital Adrenal Hyperplasia
- Childhood Obesity
- Precocious Puberty
- Delayed Puberty
- Phenylketonuria
- Menstrual Abnormalities in Adolescence

KEY TERMS

Hirsutism: Growth of excessive terminal coarse hair in a female with male-like distribution on certain parts of the body.

Ketoacidosis: It is a metabolic state characterized by pathologically high serum and urine concentration of ketone bodies.

Kussmaul respiration: It is an abnormal breathing pattern characterized by rapid deep breathing at a consistent pace associated with severe metabolic acidosis.

Precocious: Exceptionally early physical or mental development hypophysectomy: Surgical removal of hypophysis or pituitary gland.

DIABETES MELLITUS

Diabetes mellitus is a group of metabolic diseases characterized by chronic hyperglycemia resulting from defects in insulin secretion, insulin action, or both. The abnormalities in carbohydrate, fat, and protein metabolism that are found in diabetes are due to deficient action of insulin on target tissues. Diabetes in children accounts for around 5% of the diabetic population. The incidence of diabetes mellitus increases with advancing age of the child with peaks at 5 and 12 years of age.

Classification

- **Type 1 diabetes mellitus or insulin dependent DM:** It is due to β-cell destruction, usually leading to absolute insulin deficiency. It can be immune-mediated or idiopathic.
- **Type 2 diabetes mellitus or noninsulin dependent DM:** It may range from predominantly insulin resistance with relative insulin deficiency to a predominantly secretory defect with or without insulin resistance.
- **Other specific types of DM:** Genetic defects of β-cell function or insulin action, diseases of the exocrine pancreas, drug- or chemical-induced endocrinopathies.
- **Gestational diabetes:** It occurs during pregnancy.

Type 1 diabetes is more common in children but type 2 diabetes is increasing rapidly than type 1 due to obesity and less active lifestyles.

Type 1 Diabetes

Type 1 diabetes is also known as juvenile diabetes. This results when the pancreas is unable to produce and secrete insulin and depends on exogenous insulin to prevent ketosis and preserve life. Genetic, environmental and autoimmune factors are responsible for type 1 diabetes. Polymorphisms in HLA complex, account for most of the genetic risk factors. Environmental factors are viruses, bovine milk protein and nitrosourea compound. It is associated with Hashimoto thyroiditis, Addison's disease and pernicious anemia.

Pathophysiology

Insulin is responsible for uptake, use and storage of glucose, amino acids and fat. In case of deficiency of insulin production, there is decreased glucose uptake and storage as glycogen, and excessive breakdown of fat and glucose from glycogen. This leads to starvation of cells and accumulation of glucose and fat in the blood in the form of free fatty acids and ketone bodies. This results in increased concentration of glucose in the blood, which in turn causes fluid shift from intracellular to extracellular spaces and kidney.

When blood glucose approaches renal threshold, kidney fails to reabsorb all glucose. Therefore, glucose is spilled into urine through osmotic diuresis along with excessive urination to dilute the concentrated glucose. Polyurea leads to dehydration and increased thirst and cellular starvation leads to hunger and excessive eating. Free fatty acids, which are converted by liver to ketone bodies, eventually results in diabetic ketoacidosis.

Clinical Features

- **3 Ps:** Polyurea, polydipsia and polyphagia.
- Weight loss, fatigue and blurred vision.
- **Signs of diabetic ketoacidosis:** Nausea, vomiting, abdominal pain, acetone odor of breath, dehydration, lethargy, Kussmaul respiration and coma.

Diagnosis

According to national diabetes data group and WHO, diagnostic criteria for diabetes mellitus are as following:

- Symptoms of diabetes plus random plasma glucose concentration = 11.1 mmol/L (200 mg/dL).
- Fasting plasma glucose = 7.0 mmol/L (126 mg/dL).
- 2-hour post load glucose = 11.1 mmol/L (200 mg/dL) during an OGTT. (The test should be performed as described by WHO, using a glucose load containing the equivalent of 75 g anhydrous glucose dissolved in water or 1.75 g/kg of body weight to a maximum of 75 g).

Glucose tolerance test is rarely done. The glycosylated hemoglobin is elevated in response to prolonged elevation of blood glucose.

Treatment

The goals of treatment are:

- To control hyperglycemia.
- To facilitate adequate growth.
- To maintain age-appropriate lifestyle.
- To prevent acute complications.

Mainstays of treatment are insulin therapy, nutritional modification, physical activity and glucose monitoring.

Insulin Therapy

Optimal glycemic control in type 1 diabetes mellitus (T1DM) requires intensive insulin therapy. The goal of insulin therapy is to replace the insulin that is not produced in the body.

The choice of insulin types and schedule of injections are determined by child's age, body weight and pubertal status. There are two types of insulin therapies in practice.

1. Conventional therapy, the most commonly used, refers to 1–2 daily insulin injections. The total daily dose is divided into 2/3 prebreakfast and 1/3 predinner.
2. Intensive therapy includes the administration of insulin three times daily by multiple daily injections (MDI) or pen, or an external pump.

Every dose of insulin is adjusted according to the premeal blood glucose value monitored at least four times daily. Total daily dose is divided as follows:

- **Basal dose:** 25–30% of the total dose in toddlers and 40–50% in older children, is given at bedtime. This controls the glucose production between meals and overnight.
- **Bolus doses:** Remaining dose is divided into three premeal doses. The meal time (prandial) doses decrease risk of post-prandial hyperglycemia.

Types of insulin

There are short acting, intermediate or long-acting insulins. Long or intermediate acting insulins are administered to maintain glycemic control during fasting state whereas short acting insulin is useful for glycemic control in postprandial state (Table 13.1).

Two main classes of insulin regimen are—neutral protamine Hagedorn (NPH) with short-acting insulin analogs and long-acting insulin glargine with short acting insulin.

Insulin administration

Insulin is injected into the subcutaneous tissue of the upper arm, anterior and lateral aspects of the thigh, buttocks, and abdomen. Insulin is absorbed more rapidly from the abdomen > arm > thigh > buttock. Rotating within one area is recommended (e.g., rotating injections systematically within the abdomen) rather than rotating to a different area with each injection because it decreases day-to-day variability in absorption. More

TABLE 13.1: Types of insulin

	Insulin	Onset of action	Peak (hours)	Duration (hours)
Short acting	Human regular	30–60 minutes	2–4	6–10
	Lispro, Aspart	5–15 minutes	1–2	4–6
Intermediate acting	NPH, NPL	1–4 hours	5–10	10–16
	Lente	3–4 hours	6–12	12–18
	Ultra Lente	2–4 hours	8–16	16–20
Long acting	Glargine	1–2 hours	Flat	24
	Detemir	1–2 hours	Flat	18–24

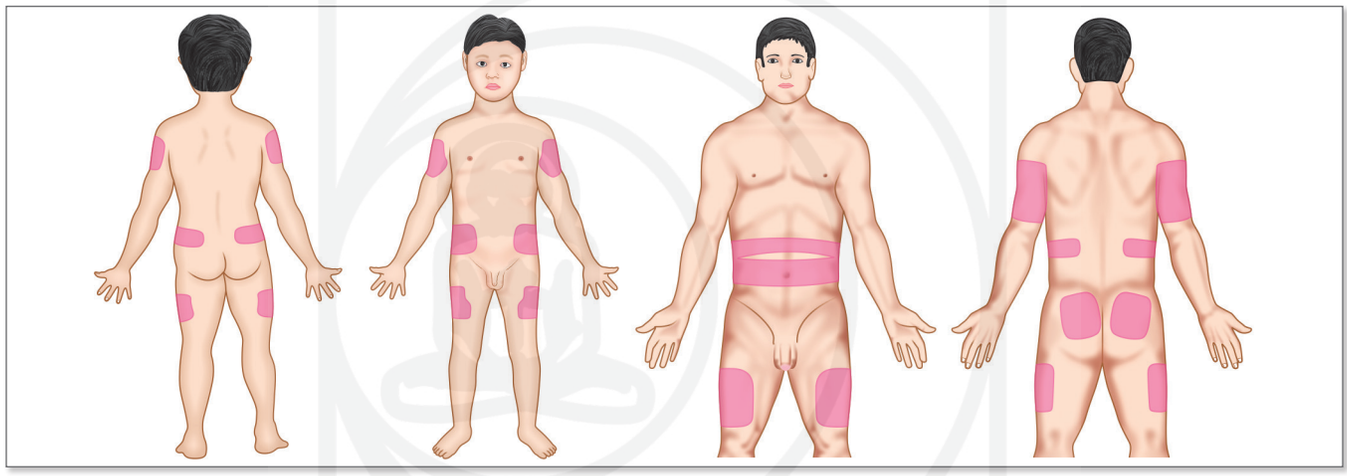

FIGURE 13.1: Sites of insulin administration and rotation of sites

consistency in insulin levels may be achieved by giving all shots in the same part for a week at a time, e.g., in the arm area for a week and then in the leg sites for a week or choose one area for the morning and one for the evening (Fig. 13.1).

Exercise increases the rate of absorption from injection sites; therefore, if one is playing tennis, avoid injecting insulin in that arm. An amount of air equal to the dose of insulin required, is drawn and injected into the vial to avoid creating a vacuum. Air is injected into the long-acting insulin first keeping the vial upright then into the short acting insulin. Then the vial is turned upside down and the short acting insulin is withdrawn first, followed by long-acting insulin. Accidental introduction of long acting insulin into the short acting insulin can increase the duration of short acting insulin effect. Vial should be refrigerated and warmed to room temperature to limit local irritation at the injection site. Extreme temperatures (<36 or >86°F, <2 or >30°C) and excess agitation should be avoided to prevent loss of potency, clumping, frosting, or precipitation.

Modalities of injectable insulin delivery

Insulin can be administered by insulin syringe, insulin pump or pen. Insulin can be administered by continuous subcutaneous insulin infusion *via* insulin pump. There are premixed insulin preparations available in pen syringes, which maintain glycemic control. Intradermal, intranasal and buccal approaches are newer techniques of insulin administration.

Other Therapeutic Measures

- **Nutritional modification:** The goal of the therapy is to match the intake with appropriate insulin. Simple sugars are avoided while food with low glycemic index and fiber are recommended. Limit intake of saturated and trans fat. Timings of meals are adjusted with insulin intake.
- **Physical activity:** Exercise enhances the action of insulin in reducing the blood glucose level. Recommended activities include walking, jogging, swimming and organized sports. Avoid exercise during peak time of insulin action. Add extra 15–30 g carbohydrate snacks for each 45–60 minutes of exercise.
- **Blood sugar monitoring:** Self-monitoring of blood glucose is recommended before meals and before bedtime. Results should be recorded in a diary. Glycosylated hemoglobin is also monitored at regular intervals.

Type 2 Diabetes Mellitus

Type 2 diabetes mellitus is an emerging problem among pediatric population. Familial factors, maternal gestational diabetes, intrauterine growth retardation and obesity may predispose type 2 diabetes in children. Children with type 2 diabetes are typically overweight and have velvety darkening of skin around the neck (acanthosis nigricans). Weight loss and ketoacidosis are often present in type 2 diabetes. Ketosis should be managed with insulin therapy initially and then switched over to oral hypoglycemic agents. Metformin is the drug of choice in these children. Nutritional modification with physical exercise should be continued to achieve near normal glucose level.

Complications

- **Acute:** diabetic ketoacidosis and hypoglycemia.
- **Intermediate:** lipoatrophy, limited joint mobility, growth failure and delayed sexual maturation.
- **Chronic:** retinopathy, peripheral neuropathy, nephropathy and dyslipidemia.

Nursing Management

Assessment

- Monitor blood glucose level, HbA1c level.
- Assess for signs of hyperglycemia and hypoglycemia.

Nursing Diagnosis

- Imbalanced blood glucose level related to lack of insulin production with associated insulin resistance as evidenced by random blood glucose level >200 g/dL.
- Imbalanced nutrition less than body requirement related to insulin deficit.
- Risk for injury related to hypoglycemia or hyperglycemia.
- Deficient knowledge related to unfamiliarity of the treatment plan.
- Ineffective family coping related to chronic illness.

Goal

The child will:

- Maintain near normal blood glucose level.
- Maximize the nutritional status.
- Remain free from injury.

The parents:

- Will develop coping strategies.
- Will demonstrate the self-administration of insulin.

Nursing Interventions

- **Maintaining glucose level:**
  - Monitor blood glucose level premeal and before bed time.
  - Administer insulin as prescribed.
  - Follow the insulin regimen as ordered.
  - If mixed preparations are ordered, withdraw short acting insulin first. Store insulin in a cool dry place, do not heat or freeze. Rotate the insulin site daily.
- **Maintaining nutrition:**
  - Balance the meals and snacks with insulin action.
  - Provide low fat, low glycemic index and high fiber food.
  - Identify favorite foods of the child and incorporate in the meal plan.
  - Provide three meals and two or three snacks.
  - Monitor daily weight.
  - Discuss the relationship of food, insulin and exercise to the parents.
- **Preventing injury:**
  - Teach the family to recognize the signs and symptoms of hypoglycemia and hyperglycemia.
  - Treat hypoglycemia promptly with 15 g of easily digested carbohydrate.
  - Advise to adhere to the treatment regimen.
  - Teach strategies to prevent hypoglycemia such as avoiding missed or delayed meal, excess insulin or excessive exercise.
  - Monitor blood glucose levels at regular intervals.
- **Health education:**
  - Teach the child to self-administer insulin as per direction.
  - Take return demonstration of insulin injection.
  - Advise regarding menu planning as per insulin need.
  - Reinforce the need for regular follow-up.
- **Promoting family coping:**
  - Encourage to express their concerns for their child.
  - Provide explanations of treatment plan.
  - Identify community support service available for the family.
  - Allow the child and family to meet other children with diabetes.
  - Involve the parents in the child care.

Expected Outcome

The child:

- Maintains near normal blood glucose level.
- Maximizes the nutritional status.
- Remains free from injury.

The parents:

- Develop coping strategies and child is demonstrated self-administration of insulin.

DIABETES INSIPIDUS

Diabetes insipidus (DI) is a heterogeneous clinical syndrome of disturbance in water balance, characterized by polyuria (urine output >4 mL/kg/hr), polydipsia (water intake >2 L/m^2/day) and failure to thrive. It is a rare condition in children.

Pathophysiology

Antidiuretic hormone (ADH) is produced by hypothalamus and stored in posterior pituitary. ADH is under control of osmoreceptors in the anterior pituitary. When the osmolality is low, ADH production decreases. This leads to increased urine output normalizing osmolality. Again, when osmolality is high ADH production increases resulting in water retention. In diabetes insipidus, a deficiency of ADH makes body unable to conserve water which causes large volumes of dilute urine. Loss of free water increases serum sodium concentration. Increased oral intake of fluid compensates for large fluid loss.

Three pathophysiologic mechanisms give rise to polydipsia and polyuria:

1. **Central** (vasopressin sensitive, hypothalamic, neurogenic) DI, caused by defective vasopressin synthesis and/or secretion.
2. **Nephrogenic** (vasopressin resistant) DI, caused by defective renal tubular response to vasopressin action.
3. **Primary polydipsia** due to compulsive water drinking (psychogenic) or defective thirst mechanism (dipsogenic).

Etiology

- **Central DI:** Congenital, vasopressinase excess and defect of osmoregulation, primary tumors or metastasis, meningitis, encephalitis, drugs like phenytoin, carbamazepine, trauma or surgery, hypoxic ischemic encephalopathy, etc.
- **Nephrogenic DI:** Congenital, drugs like lithium, hypercalcemia/hypokalemia, polycystic kidney disease and ureter obstruction.
- **Primary polydipsia:** Psychogenic.

Clinical Features

Polyuria, specified as quantified urine output of >4 mL/kg/hr in children (>6 mL/kg/hr in neonates) and polydipsia, referred to as water intake of >2 L/m^2/day (or >5 L/day) and failure to thrive or growth retardation are essential features of DI.

Infants may show symptoms such as vigorous suck, vomiting, recurrent episodes of fever without an apparent cause, excessive crying, irritability, weight loss, constipation, and excessively wet diapers. Younger children often manifest with primary enuresis and difficulty in toilet training. Older children characteristically may have high urine output and nocturia resulting in disturbed sleep and easy fatigability.

Diagnosis

- 24-hr-urine output measurement; urine output >4 mL/kg/hr in infants and children and >6 mL/kg/hr in newborn indicates polyuria.
- Urine analysis and biochemistry.
- Serum potassium and calcium concentrations.

Treatment

Free access to water should be allowed in children with DI. This facilitates maintenance of tonicity if the thirst mechanism is intact preventing dehydration.

- **Dietary modification:** Diet with low sodium, low protein with high calories providing a high calorie: solute ratio is recommended.
- **Drug:** In older children with CDI, aqueous vasopressin, lysine vasopressin, de-amino, D arginine vasopressin (dDAVP) may be used to minimize water excretion. Desmopressin (1-deamino-8-D-arginine vasopressin, dDAVP) is the current drug of choice for long-term therapy of CDI. In nephrogenic DI, hydrochlorothiazide and amiloride are the drugs of choice to reduce urine output.

DISORDERS OF PITUITARY GLAND

The pituitary gland is a tiny organ, the size of a pea, found at the base of the brain, is composed of anterior (i.e., adenohypophysis) and posterior (i.e., neurohypophysis) regions. As the "master gland" of the body, it produces many hormones that travel throughout the body, directing certain processes or stimulating other glands to produce other hormones. The hormones produced from anterior pituitary gland are prolactin, adrenocorticotropin (ACTH), thyroid stimulating hormone (TSH), luteinizing hormone (LH), follicle stimulating hormone (FSH) and growth hormone (GH). Antidiuretic hormone (ADH) also called vasopressin and oxytocin are secreted from posterior pituitary gland.

Hypopituitarism

Hypopituitarism refers to the absence or reduction in function of two or more hormones produced by the pituitary gland. When all pituitary hormones are affected, it is called panhypopituitarism.

- Potential anterior pituitary hormone deficiency is growth hormone deficiency leading to short stature and slow height velocity.
- TSH deficiency leading to hypothyroidism.
- ACTH deficiency causing adrenal deficiency and FSH and LH deficiency leading to delayed puberty.
- Posterior pituitary hypofunction leads to diabetes insipidus.

Causes of Hypopituitarism

- **Congenital:** Birth trauma/asphyxia, midline defect syndromes (e.g., septooptic dysplasia), mutations of genes encoding pituitary transcription factors.
- **Acquired:** Tumors in the hypothalamic-pituitary region (e.g., craniopharyngioma); radiation to the head and neck region, hydrocephalus vascular abnormalities of hypothalamic-pituitary region, major head trauma.

Clinical Features

- Short stature, stunted growth, premature aging, retarded height more than weight with well-nourished appearance and normal skeletal proportion.
- Inactive, less participative in aggressive, sporting activity.
- Delayed eruption of permanent teeth, overcrowded or malpositioned teeth.
- Round faces, depressed nasal bridge, single incisor tooth and micropenis.
- Retarded bone age.
- Slow sexual development; menstrual periods may stop or be irregular.

Diagnosis

- History and physical examination.
- Radioimmunoassay of plasma GH level along with two stimulation tests.
- Serum levels of hormones responsible for growth hormone (like growth factor-1 (IGF-1), and insulin-like binding protein-3).
- Radiographic test of hand and wrist.
- Ophthalmology referral/eye exam.
- Water deprivation.
- Bone age.

Treatment

Treat the underlying cause of the condition and replace the hormones, the body is not making.

Hormone Replacement

Hormone replacement therapy mimics the body's natural production. The medicines should be continued as long as needed, during childhood and adulthood. The following are examples of hormone replacement therapy:

- Levothyroxine for hypothyroidism.
- Synthetic growth hormone for growth deficiency.
- Hydrocortisone for adrenal insufficiency.
- Replacements for sex hormones.
- Replacements for antidiuretic hormone.
- Tumor treatment: Surgery and radiotherapy.

Hyperpituitarism

Hyperpituitarism is the primary hypersecretion of pituitary hormones. It typically results from a pituitary adenoma. There is over secretion of three hormones resulting in the pituitary adenoma which are prolactin, ACTH and growth hormone. The four most common types of hyperpituitarism are:

1. Prolactinoma
2. Corticotropinoma (Cushing's disease)
3. Somatotropinoma (Gigantism)
4. Thyrotropinoma (a rare pituitary cancer)

Excess prolactin may result in **prolactinoma**. Prepubertal children typically manifest with a combination of headaches, visual disturbance, and growth failure. Pubertal females exhibit pubertal arrest or **hypogonadism** due to suppression of **gonadotropin** secretion or local compression of the pituitary. Pubertal males manifest with headaches, visual impairment, and pubertal arrest or growth failure.

Excess ACTH is characterized by weight gain. **Hirsutism** and premature adrenarche may occur in prepubertal children. Pubertal arrest, acne, fatigue, and **depression** are also common.

Excess GH results in **gigantism**. The severity of gigantism depends on whether the **epiphyseal plate** is open. Before epiphyseal closure, excess GH causes proportional overgrowth of long bones accompanied by rapid development of muscles and viscera. If hypersecretion occurs before the epiphyseal closure, acromegaly occurs which is characterized by transverse growth with overgrowth of the head, lips, nose, tongue, jaw, malocclusion of teeth, increased facial hair and thickened, deeply creased skin.

Hypersecretion of posterior pituitary ADH results in syndrome of inappropriate antidiuretic hormone secretion (SIADH) characterized by fluid retention and hypotonicity. It may progress to seizures and stupor if sodium level falls significantly.

Diagnosis

- History and physical examination.
- Radiologic examination.
- Serum hormone levels.

Treatment

- Surgical intervention like cryosurgery or hypophysectomy for pituitary adenoma.
- Hormone replacement with thyroid extract, cortisone and sex hormones.
  - Restrict fluid in SIADH.

DISORDERS OF THYROID

Thyroid disorders are caused by either deficiency or excessive secretion of thyroid hormone. The thyroid hormones are

thyroxine (T_4), triiodothyronine (T_3) and thyrocalcitonin. These hormones are controlled by thyroid stimulating hormone (TSH). Thyroid disorders are hypothyroidism, hyperthyroidism and goiter.

Hypothyroidism

Inadequate secretion of thyroxine can occur at any age and is called hypothyroidism. In childhood, it may be congenital or acquired. It is one of the most common endocrine disorders in childhood.

Causes

- Deficiency of thyrotropin releasing hormone.
- Thyrotropin deficiency and unresponsiveness.
- Defect of fetal thyroid development.
- Defective synthesis of thyroxin.
- Iodine deficiency.
- Maternal drug: Amiodarone.
- Autoimmune problem.
- Irradiation for Hodgkin disease.

Clinical Features

- Enlarged head, prolongation of physiologic jaundice, feeding difficulty.
- Sluggishness, somnolence, choking spell, respiratory distress.
- Subnormal temperature, cold and mottled extremities, narrow palpebral fissure and swollen eyelids.
- Thick, broad protruding tongue.
- Broad hands and short fingers.
- Myxedematous skin changes with dry and scaly skin of eyelids, and back of hands.
- Coarse, brittle and scanty hair.
- Hairline reaches far down the forehead.
- Delayed development.

Diagnosis

- Serum levels of T_3 may be normal but T_4 level is low.
- Serum TSH is elevated, serum prolactin is elevated.
- X-ray skull, ECG and scintigraphy.

Treatment

- Hormone replacement with sodium L thyroxine orally is the mainstay of treatment. It is taken lifelong. Periodic follow-up and growth monitoring is necessary.

Goiter

Goiter is the enlargement of thyroid gland. The goiter may be congenital, acquired, sporadic or endemic. This results from increased secretion of pituitary thyrotropic hormone in response to decreased levels of thyroid hormones. It may also be caused by inflammatory or neoplastic lesion or presence of maternal thyrotropin receptor stimulating antibodies. Thyroid function may be normal, decreased or increased. Enlargement of the thyroid gland causes severe respiratory distress in infants. Thyroid hormone replacement is needed to treat hypothyroidism and reverse the TSH effect on the gland.

Hyperthyroidism (Graves' Disease)

Hyperthyroidism is caused by excessive secretion of thyroid hormone and diffuse toxic goiter (Grave's disease). The peak incidence occurs between 12 and 14 years of age. It has a familial association.

Clinical Features

- **Cardinal signs:** Emotional lability, restlessness, decelerated school performance, voracious appetite and fatigue.
- **Physical signs:** Tachycardia, widened pulse pressure, dyspnea on exertion, exophthalmos, tremor, staring expression with lid lag, warm, moist skin, heat intolerance and systolic murmur.
- **Signs of thyroid storm:** Severe irritability and restlessness, vomiting, diarrhea, hyperthermia, hypertension, tachycardia, prostration, delirium and coma.

Diagnosis

- T_3 and T_4 elevated.
- TSH assay.

Treatment

- **Antithyroid drugs:** Propylthiouracil and methimazole.
- Ablation with radioiodine.
- Subtotal thyroidectomy.
- Beta adrenergic blocking agents along with antithyroid drugs for thyrotoxicosis.
- Restrict vigorous exercises until thyroid levels are normal or near normal.

DISORDERS OF PARATHYROID GLAND

The parathyroid gland secretes parathormone, which along with vitamin D is the principal regulator of calcium homeostasis.

Hypoparathyroidism

Deficiency of parathyroid hormone is known as hypoparathyroidism. There are two classic forms of hypothyroidism observed during childhood. Autoimmune hypoparathyroidism is caused by deficient production of PTH due to

multiorgan failure. Pseudoparathyroidism is characterized by increased PTH production and deficient end organ responsiveness. Idiopathic hypoparathyroidism is a type when no specific etiologic mechanism is found.

Clinical Features

- **Pseudoparathyroidism:** Short stature, round face, short stubby fingers and toes, dimpling of skin over knuckles and mental retardation.
- **Other types of hypoparathyroidism:** Muscle cramps and pain, numbness, stiffness, tingling of the hand and feet, positive Chvostek or Trousseau sign or laryngeal and carpopedal spasms, abdominal pain, tonic rigidity, retraction of head and cyanosis. Teeth eruption is delayed and enamel formation is irregular. Nails of fingers and toes have horizontal groove.

Diagnosis

- Serum calcium level low and phosphorous level elevated.
- Serum alkaline phosphatase is normal or low.
- ECG abnormalities.

Treatment

Treatment of neonatal tetany: IV calcium gluconate 5–10 mL at 0.5–1 mL/min. Calcitriol is additionally given.

- Vitamin D therapy of long-term maintenance dose.
- Oral calcium supplementation.

Hyperparathyroidism

Hyperparathyroidism is caused by excessive production of parathyroid hormone. Primary hyperparathyroidism may be due to adenoma or hyperplasia of the parathyroid gland. The causes of secondary hyperparathyroidism are chronic renal disease, renal osteodystrophies and congenital anomalies of the urinary tract.

Clinical Features

- **GI:** Nausea, vomiting, abdominal discomfort and constipation.
- **CNS:** Delusion, confusion, hallucination, impaired memory, lack of interest.
- **Neuromuscular:** Weakness, easy fatigability, muscle atrophy and tongue twitching.
- **Skeletal:** Vague bone pain, spontaneous fractures.
- **Renal:** Polyurea, polydipsia, renal colic and hypertension.

Diagnosis

- Elevated calcium and lowered phosphorous levels.
- Bone X-ray.
- Electrocardiography.

Treatment

- **Primary hyperparathyroidism:** Surgical removal of the tumor or hyperplastic tissue.
- **Secondary hyperparathyroidism:** Treat underlying cause.

CONGENITAL ADRENAL HYPERPLASIA

Congenital adrenal hyperplasia (CAH) is a group of inherited conditions in which there is an inborn ("congenital") enlargement ("hyperplasia") of the adrenal glands caused by reduced activity of enzymes required for cortisol biosynthesis in the adrenal cortex. It is an autosomal recessive defect which occurs in 1 per 12,000–15,000 births. For a child to have CAH, each parent must either have CAH or carry a genetic mutation. This means that if two parents are CAH carriers, their children have a 25% chance of being affected with CAH. CAH is the most common cause of ambiguous genitalia in females, and can cause acute life-threatening adrenal crisis in both males and females in the neonatal period.

Pathophysiology

The process of making steroid hormones from cholesterol in the adrenal cortex is complex and involves several steps controlled by enzymes. In CAH, a helper enzyme is missing or partly missing. This hinders the production of cortisol and aldosterone. When the pituitary gland senses low levels of cortisol in the bloodstream, it produces ACTH which overstimulates the adrenal cortex causing it to enlarge. This causes the adrenal glands to produce excess androgen, while cortisol and aldosterone levels remain low.

The high level of androgen in girls is manifested as virilized with enlargement of the clitoris and different degrees of partial closure of the vaginal opening. Since boys are already masculinized by their testes, they exhibit no genital abnormality at birth. In many but not all children with CAH, the lack of aldosterone results in excessive loss of salt in the urine after birth. If untreated, this may become critical in the first two or three weeks of life and result in a potentially fatal "salt-losing crisis". The increased ACTH secretion may also cause some pigmentation of the skin. In the most severe cases, cortisol deficiency can lead to low blood sugars.

Types of CAH

- **Classic CAH:** The most common defect is 21-hydroxylase (21-OH) deficiency, which accounts for 90% of all cases of CAH. Clinical consequences of 21-OH deficiency arise primarily from overproduction and accumulation of precursors proximal to the blocked enzymatic step. These precursors are shunted into the androgen biosynthesis pathway, producing virilization in the female fetus or

infant and rapid postnatal growth with accelerated skeletal maturation, precocious puberty, and short adult stature in both males and females. The forms of classic CAH are salt-losing CAH and simple virilization form.

- **Nonclassic (late-onset) CAH:** This form of CAH (also called 'nonclassical CAH') is the mildest form and is almost always due to 21-hydroxylase deficiency. Signs of nonclassical CAH may include rapid early growth and early appearance of pubic hair and acne. Sometimes, the child looks normal until the time of puberty, when excess facial hair, and irregular periods are manifested. Boys often do not require treatment. Girls usually need treatment to suppress their excess androgens.

Clinical Features

- **Girls:** Masculinization, enlarged clitoris, fusion of labia, closed vaginal orifice.
- **Boys:** Precocious genital development, genital enlargement, frequent erections.
- **If untreated:** Early sexual maturation, development of axillary, pubic and facial hair, deepening of voice, no breast development and amenorrhea in girls and small testes in boys.

Diagnosis

- **Newborn screening:** Measurement of 17-OHP in a filter paper blood spot sample obtained by the heel-stick technique.
- **Infant and older children:** Markedly elevated serum levels of 17-OHP, elevated serum concentrations of testosterone in girls and androstenedione in boys and girls, low serum sodium and chloride levels, inappropriately increased urine sodium levels, and elevated levels of serum potassium and serum urea nitrogen in salt loosing form of CAH.

A significant rise in the 17-OHP level 60 minutes after an intravenous bolus of 0.25 mg of ACTH is diagnostic for CAH.

Treatment

Administration of glucocorticoids (hydrocortisone, prednisone, dexamethasone) inhibits excessive production of androgens and prevents progressive virilization. Patients with salt loss need fludrocortisone, a steroid with a salt-retaining action, to replace the missing aldosterone. In the first year of life, children with salt losing CAH may also require salt supplements, usually added to feeds. Patients with nonclassic 21-OH deficiency do not always require treatment.

Treatment is lifelong and compliance with medication and frequent blood monitoring are essential to the child's health, growth and development. After growth is completed, prednisone is given once or twice daily, or dexamethasone, given as a single dose at bedtime.

Surgery may be needed in case of ambiguous genitalia.

CHILDHOOD OBESITY

Childhood obesity is one of the most serious public health challenges of the 21st century. According to WHO, in 2013, the number of overweight children under the age of five, is estimated to be over 42 million globally out of which, 31 million are from developing countries. Overweight is defined as having excess body weight for a particular height from fat, muscle, bone, water, or a combination of these factors. Obesity is defined as having a body mass index (BMI) greater than or equal to the 95th percentile for age and gender in children aged two years and older. Severe obesity is having a BMI greater than or equal to the 120th percentile.

Skin fold thickness and waist circumference are other markers of obesity.

Etiology

Constitutional: Excessive calorie intake, lack of physical activity, unhealthy eating patterns, excessive watching TV.

Pathology

- **Endocrine:** Cushing syndrome, deficiency of growth hormone, hypothyroidism, pseudoparathyroidism.
- **Hypothalamic:** Head injury, infection, brain tumor, radiation after CNS surgery.
- **Genetic:** Hereditary factor, Prader-Willi syndrome, Laurence-Moon-Bardet-Biedl syndrome.
- Carpenter syndrome.
- **Drug:** Antiepileptic, steroids and estrogen therapy.
- **Others:** Polycystic ovarian disease, leptin deficiency, psychiatric disturbances.

Clinical Features

- Height on or above 50th percentile for age.
- Fat deposits under breast area, upper arms and thighs.
- Protuberant abdomen.
- Small genitalia with enlarged breasts.
- Accelerated puberty.

Clinical Features Indicating Pathological Obesity

- Hypogonadism.
- Retinitis pigmentosa.
- Earlobe creases.
- Short hand and feet, almond shaped eyes.
- Buffalo hump and striae.
- Metacarpal shortening.
- Mental retardation.

Complications

- **Central nervous system:** Benign intracranial hypertension.
- **Respiratory:** Obstructive sleep apnea, cor pulmonale.
- **Cardiovascular:** Atherosclerosis, hypertension.
- **Endocrine:** Type 2 diabetes mellitus due to insulin resistance, polycystic ovarian disease.
- **Orthopedic:** Slipped femoral epiphysis, flat feet, blount disease, osteoarthritis.
- **Hepatobiliary:** Fatty infiltration of liver, steatohepatitis cholelithiasis and chronic liver disease.

Diagnosis

- Blood glucose, serum lipid profile, thyroid function test, serum cortisol.
- Bone age estimation.
- MRI, CT scan of brain.
- Pelvic ultrasound.
- Hormonal assay of LH, FSH and testosterone.
- Karyotype.

Management

- **Dietary modification:** Caloric intake of 1200–1800 calories, reduction of junk food, carbonated drinks, refined sugars; following the concept of food pyramid.
- **Lifestyle modification:** Reduction of sedentary lifestyle, increased physical activity, opportunities for play, sports involvement and restricted TV watching and internet surfing.
- **Drugs:** Gastric lipase inhibitor like orlistat, metformin for diabetes; leptin for leptin deficiency and octreotide for hypothalamic obesity.
- **Surgery:** It is the last option in obesity treatment. Laparoscopic gastric binding is usually done to reduce the gastric capacity.

Prevention

Healthy lifestyle habits, such as healthy eating and physical activity, can lower the risk of becoming obese and developing related diseases. The dietary and physical activity of children and adolescents are influenced by many sectors of society, including families, communities, schools, child care settings, medical care providers, faith-based institutions, government agencies, the media, and the food and beverage industries and entertainment industries. Schools play a significantly critical role by establishing a safe and supportive environment with policies and practices that support healthy behaviors. Schools also provide opportunities for students to learn about and practice healthy eating and physical activity behaviors.

PRECOCIOUS PUBERTY

Normal puberty begins between ages 8 and 12 years in girls and between 9 and 14 years in boys. Precocious puberty is defined as signs of puberty that occur before the age of 8 in a girl or 9 in a boy. It occurs much more often in girls than in boys. Thelarche is the onset of female breast development, which is characterized by tender nodules of firm tissue centered on the areolae, which usually are appreciable by palpation before they are by visual inspection. Adrenarche (ad'ren-ar'ke_) is the onset of androgen-dependent signs of puberty (pubic hair, acne, and adult body odor). In females, adrenarche is the result of adrenocortical activity.

In males, either adrenal or gonadal maturation can prompt adrenarche which is also known as pubarche. Menarche is the onset of menstruation.

Causes

- **Adrenal Steroid-dependent precocious puberty:** Adrenal gland tumor, congenital adrenal hyperplasia.
- **Gonadotropin dependent or central precocious puberty:** Idiopathic tumors, infections, head trauma, neurosurgery, arachnoid cyst, hydrocephalus, etc.
- **Gonadotropin independent or peripheral precocious puberty:** Hypothyroidism, ovarian cyst, McCune-Albright syndrome, Gonadal neoplasia, Exogenous steroids (oral contraceptive pills, skin creams, testosterone).
- **Incomplete precocious puberty:** Premature thelarche, premature adrenarche, premature menarche.

Clinical Features

- Linear growth spurt.
- Clitoromegaly, a sign of abnormally high androgen levels in female.
- Thinning of the scrotum and enlargement of the testes in male.
- Growth of the phallus and pubic hair.
- Skeletal growth spurt.
- Testicle volumes greater than 3 mL bilaterally.
- Maturation of the vaginal mucosa: Red glistening mucosa indicates lack of estrogen and pink mucosa with mucus indicates estrogen effect.
- Adrenal, ovarian or testicular tumor.
- Adult body odor.
- Acne, moodiness.

Diagnosis

- Gonadotropin level.
- LH level.
- LH/FSH ratio.
- Random measures of testosterone or estradiol.

- Serum 17-hydroxyprogesterone concentration.
- Ultrasound of abdomen and pelvis.
- Thyroid function test.
- MRI brain, CT scan.
- Bone age assessment.

Treatment

- GnRH agonist (e.g., leuprolide) therapy for central precocious puberty.
- Thyroid replacement for hypothyroidism.
- Aromatase inhibitor or tamoxifen for McCune Albright syndrome.
- Treatment of ovarian, adrenal or testicular tumor.
- Hydrocortisone and fludrocortisone for congenital adrenal hyperplasia.

DELAYED PUBERTY

Delayed puberty is defined as lack of secondary sexual characteristics by the age of 14 years. It is a frequent problem but affects boys more often than girls.

If a girl shows no sign of breast development by the age of 13 or no menstruation by the age of 16 and a boy shows no sign of puberty by the age of 15, it is considered pubertal delay.

Etiology

Girls

- **Hypogonadotropic hypogonadism:** Systemic disorders, malnutrition, hypothyroidism, type I diabetes, genetic, pituitary hormone deficiency, brain tumor, etc.
- **Hypergonadotropic hypogonadism:** Turner syndrome, ovarian insult, gonadotropin resistance, autoimmune ovarian failure.
- **Isolated amenorrhea:** Structural malformation, inefficient androgen action.

Boys

- **Hypogonadotropic hypogonadism:** Constitutional delay, systemic disorders, malnutrition, hypothyroidism, type I diabetes, genetic, dysmorphic syndrome, malformations, pituitary hormone deficiency, surgery and infiltrative disorders.
- **Hypergonadotropic hypogonadism:** Klinefelter syndrome, testicular insult, cryptorchidism, inefficient testosterone action and androgen insensitivity syndrome.

Clinical Features

- **Boys:** Penis and testicles do not enlarge by age 14, short compared with their peers, a delayed growth spurt and features of the systemic disease, brain tumor, hypothyroidism
- **Girls:** Poor smell sensation, amenorrhea, galactorrhea, features of turner syndrome and hypothyroidism.

Diagnosis

- Serum FSH level, prolactin level, LH and testosterone level.
- GnRH stimulation test.
- Thyroid profile.
- Neuroimaging.
- X-ray of the hand and wrist to determine the bone age.

Treatment

- **Girl:** Low dose estrogen, gradually increasing over 3 months till adult dose is achieved; medroxyprogesterone acetate two years after initiation of treatment. Defer hormone replacement till bone age of 12 years.
- **Boys:** Three monthly injection of testosterone enanthate; hormone therapy should be deferred till age of 14 years and bone age of 13.5 years.

PHENYLKETONURIA

Phenylketonuria (PKU) is a genetic metabolic disorder that affects metabolism of phenylalanine due to deficiency of an enzyme phenylalanine hydroxylase (PAH).

Phenylketonuria is an autosomal recessive genetic disease. The most severe form of this condition is known as classic PKU. Other forms are transient, malignant and benign hyperphenylalaninemia.

Clinical Features

- Irreversible intellectual disability.
- Microcephaly.
- Epilepsy.
- Blonde hair, blue eyes and eczema.
- Mousy odor in urine.
- Delayed development and movement disorders.
- Behavioral problems.

Diagnosis

- Newborn screening: Guthrie card bacterial inhibition assay and fluorometric analysis.
- Plasma phenylalanine level >1,000 µmol/L.
- Genetic testing of PAH gene.

Treatment

The main treatment for PKU is a lifelong specialized diet that is very low in phenylalanine started as soon as possible

after birth. Phenylalanine is a component of proteins found in milk, cheese, eggs, meat, fish, and other common foods. A special phenylalanine-free high-protein formula may be given to the babies with PKU. No high-protein animal products are ever allowed. The artificial sweetener aspartame also contains phenylalanine, so "diet" drinks and foods must also be avoided. Adjuvant therapy with single daily dose of 5–10 mg/kg tetrahydrobiopterin may be beneficial. Social support, positive attitude toward treatment and resources to manage the diet lead to greater success in maintaining metabolic control.

MENSTRUAL ABNORMALITIES IN ADOLESCENCE

Dysmenorrhea, dysfunctional uterine bleeding, and amenorrhea are the most common menstrual abnormalities seen among adolescents.

Dysmenorrhea

Dysmenorrhea is the most common menstrual problem that adolescents face. Primary, or functional, dysmenorrhea is pain during menstruation in the absence of pelvic disease, whereas secondary dysmenorrhea is due to a pathologic process.

Primary dysmenorrhea manifests in the second or third pubertal year, when ovulation becomes more regular. Adolescent complains about pain in the lower abdomen, back, or upper thighs which may be associated with headache, nausea, or diarrhea. The symptoms occur due to prostaglandin E2 and F2a secretion in the uterus after an ovulatory cycle, leading to increased uterine contractility and sensitization of pain receptors.

NSAIDs are common drug of choice to treat dysmenorrhea. Rest, exercise and balanced nutrition are advised to reduce this discomfort. Secondary dysmenorrhea is treated by treating the underlying cause.

Dysfunctional Uterine Bleeding

Dysfunctional or abnormal uterine bleeding refers to bleeding that is excessive or occurs outside of normal cyclic menstruation.

Menorrhagia is large quantity of bleeding, metrorrhagia is a term for irregular bleeding, and menometrorrhagia means heavy and irregular bleeding. Treatment options include iron supplementation, oral contraceptives (COCs), progesterone, nonsteroidal antiinflammatory drugs, antifibrinolytics, desmopressin and GnRH analogs.

Amenorrhea

Amenorrhea is the absence of spontaneous menstruation in women of reproductive age. Primary amenorrhea is failure to attain menarche. Secondary amenorrhea is cessation of menstruation for at least three months after attaining menarche, or lack of menstruation for over six months in patients who previously experienced irregular menstruation. Genetic abnormality. hypothalamic-pituitary insufficiency or ovarian problem may cause amenorrhea. Underlying cause needs to be identified and treated.

KEY MESSAGE

- Endocrinal disorders are the significant problems in child growth and development.
- Identification and management are necessary for optimum growth of the child.

SUMMARY

- Juvenile diabetes is a chronic metabolic disorder due to lack of insulin mostly occurring as type 1 diabetes in children.
- Disorders of pituitary gland and thyroid gland should be treated with hormone replacement and surgical intervention.
- Most of the endocrine disorders require life-long treatment.

ASSESS YOURSELF

Long Answer Questions

1. Define juvenile diabetes mellitus. Explain the pathophysiology of type 1 diabetes mellitus in children. Write the nursing process for a child with juvenile diabetes mellitus.
2. Define congenital adrenal hyperplasia. Write the clinical features and management of CAH.

Short Answer Questions

1. Classify diabetes mellitus.
2. Explain the management of diabetes in children.
3. Write short notes on:
   a. Insulin therapy
   b. Hypothyroidism
   c. Diabetes insipidus
   d. Hyperpituitarism

Multiple Choice Questions

1. Juvenile diabetes is:
a. Type 1 diabetes in children
b. Type 2 diabetes in children
c. Gestational diabetes in women
d. Diabetes ketoacidosis in children

2. Example of short acting insulin is:
a. NPH
b. Lente
c. Human regular
d. Glargine

3. Insulin is most rapidly absorbed from:
a. Arm
b. Abdomen
c. Buttocks
d. Thigh

4. If long acting and short-acting insulin need to be administered simultaneously which one should be withdrawn first?
a. Short acting
b. Long acting
c. Any one
d. Should be drawn into two separate syringes

5. Acetone odor of breath is suggestive of:
a. Hypoglycemia
b. Hyperglycemia
c. Diabetes insipidus
d. Diabetic ketoacidosis

6. Diabetic insipidus is caused due to the deficiency of:
a. Insulin hormone
b. Prolactin hormone
c. Antidiuretic hormone
d. Thyroxin

7. Hirsutism is caused by excessive secretion of:
a. Prolactin
b. ACTH
c. Growth hormone
d. ADH

8. Gigantism is a result of hyperpituitarism caused by:
a. Prolactin
b. ACTH
c. Growth hormone
d. ADH

9. Excessive secretion of thyroid hormone causes:
a. Myxedema
b. Goiter
c. Graves' Disease
d. Tetany

10. High protein animal products are not allowed in child with:
a. Congenital hypothyroidism
b. Phenylketonuria
c. Congenital adrenal hyperplasia
d. Diabetic ketoacidosis

ANSWER KEY

1. a **2.** c **3.** b **4.** a **5.** d **6.** c **7.** b **8.** c **9.** c **10.** b

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CHAPTER 23

Fluid, Electrolytes and Acid-Base Imbalances

LEARNING OBJECTIVES

After completion of this chapter, students will be able to:

- Identify fluid and electrolyte imbalances and acid-base imbalance.
- Perform nursing care for fluid electrolyte and acid-base imbalances.

CHAPTER OUTLINE

- Introduction
- Composition of Body Fluids
- Management of Fluid Volume Imbalances
- Management of Electrolyte Imbalances
- Management of Acid-Base Imbalance

KEY TERMS

Acidosis: It is a process that causes increased acidity in the blood and other body tissues.

Alkalosis: An abnormal pathophysiological condition characterized by the buildup of excess base or alkali in the body.

Extracellular fluid: The fluid that is not contained within the cells. Extracellular fluid is made up of interstitial fluid, blood plasma, lymph and transcellular fluid.

Interstitial fluid: It is the body fluid between blood vessels and cells.

INTRODUCTION

Infants and young children have greater risk than adults for disturbance in fluid and electrolyte balance due to different body position, higher metabolic rate and immaturity for physiologic regulation systems. Water and electrolyte imbalances occur more frequently and more rapidly in children and they adjust less promptly to those disturbances.

COMPOSITION OF BODY FLUIDS

Fluid in the body is in a dynamic state. Body fluid is composed of body water that has solutes like electrolytes, dissolved in it. Total body water (TBW) varies with an individual's age and amount of muscle mass and body fat. The more fat an individual has, the smaller is the proportion of body weight due to body water. About 70% of a full-term infant's body weight is attributed to water. By the end of puberty, boys have 60% and girls have 50% total body water.

Total body fluid is located in two major fluid compartments containing two-thirds of total water in the intracellular fluid (fluid inside the cells) and one-third in the extracellular fluid (fluid outside the cells). The extracellular fluid is made up of intravascular fluid or plasma and interstitial fluid (the fluid between the cells and outside the blood and lymphatic vessels). During infancy, a larger proportion of body water is extracellular. Extracellular fluid is rich in sodium, chloride and bicarbonate, while intracellular fluid is low in sodium but rich in potassium, organic phosphates and proteins.

Fluid moves between the intravascular and interstitial compartments by a process called filtration. Water moves in and out of the cells by the process of osmosis. Electrolytes move over cell membranes both by diffusion of particles from a location of greater to lesser concentration and by active transport which is effective even against the concentration gradient (Table 23.1).

TABLE 23.1: Pediatric daily fluid requirements

Age/Weight	Fluid/day
Infants	100–120 mL/kg
<10 kg	100 mL/kg
10–20 kg	1,000 mL + 50 mL/kg >10 kg
>20 kg	1,500 mL + 20 mL/kg >20 kg

MANAGEMENT OF FLUID VOLUME IMBALANCES

The major types of fluid imbalances are extracellular fluid volume deficit (dehydration), extracellular fluid volume excess and interstitial fluid volume excess (edema). Health conditions contributing to fluid imbalance include radiant heat (phototherapy) used to treat hyperbilirubinemia, fever, burns, diarrhea, vomiting or renal problems (Table 23.2).

Extracellular Fluid Volume Deficit (Dehydration)

The state of body water deficit is called dehydration. The three major types of dehydration are:

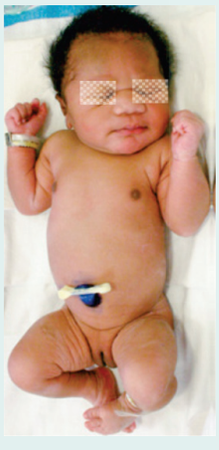
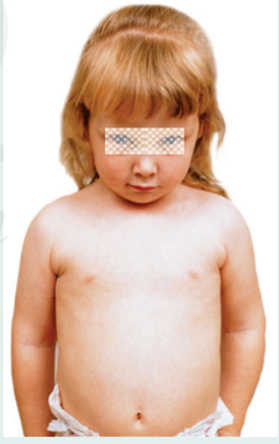
1. **Isotonic dehydration (or isonatremic dehydration):** This occurs when fluid loss is not balanced by intake; and the loss of water and sodium is in proportion. It is commonly manifested in young children through symptoms such as vomiting and diarrhea.
2. **Hypotonic dehydration (or hyponatremic dehydration):** This occurs when fluid loss is characterized by a proportionately greater loss of sodium than water. Severe and prolonged vomiting and diarrhea, burns, renal disease as well as administration of intravenous fluid without electrolytes in treatment of dehydration can lead to this type of dehydration.
3. **Hypertonic dehydration (or hypernatremic dehydration):** This occurs when sodium loss is proportionately less than water loss. Serum sodium is above normal levels. This may be caused by health problems such as diabetes insipidus or administration of intravenous fluid or tube feedings with high electrolyte levels.

The treatment of extracellular fluid volume deficit is administration of sodium containing fluid by oral rehydration therapy or by intravenous fluids.

Extracellular Fluid Volume Excess

Extracellular fluid volume excess occurs when there is too much fluid in the extracellular compartment (vascular and interstitial). Extracellular fluid volume excess is characterized

TABLE 23.2: Levels of fluid and electrolytes differences according to age

Newborn	Infant		Child/Adolescent
Total 75%	Total 65%		Total 50%
Body water	Body water		Body water
ECF-45%	ECF-25%		ECF-10–15%
ICF-30%	ICF-30–40%		ICF-40%
			
Brain and skin occupy a greater proportion of body weight and are high in interstitial fluid	High BSA promotes fluid loss Little fluid reserve in intracellular fluid	5–6 × greater fluid exchange daily High metabolic rate requires generous fluid intake	Kidneys are immature until 2 years and are unable to conserve water and electrolytes or fully assist in acid-base balance

by weight gain. An overload of fluid in the blood vessels and interstitial spaces can cause symptoms such as bounding pulse, distended neck veins in children (not usually evident in infants), hepatomegaly, dyspnea, orthopnea and lung crackles. Edema is the sign of overload of the interstitial fluid compartment. In an infant, edema is often generalized. Edema in children occurs in the dependent parts of the body, i.e., in the parts closest to the ground.

Underlying causes of the disorder should be identified and treated in order to reduce the extracellular fluid volume excess. Diuretics may be administered to remove fluid from the body, thereby reducing the extracellular fluid volume directly.

Interstitial Fluid Volume Excess (Edema)

Edema is an abnormal increase in the volume of the interstitial fluid. Clinical conditions causing edema are:

- **Edema due to increased blood hydrostatic pressure:**
  - **Increased capillary blood flow:** Inflammation, local infection.
  - **Venous congestion:** Extracellular fluid volume excess, right heart failure, venous thrombosis, external pressure on vein muscle paralysis.
- **Edema due to decreased blood osmotic pressure:**
  - **Increased albumin excretion:** Nephrotic syndrome (albumin leaks into urine), Protein-losing enteropathies (excess albumin in feces).
  - **Decreased albumin synthesis:** Kwashiorkor (low-protein, high-carbohydrate starvation diet provides too few amino acids for liver to make albumin), liver cirrhosis (diseased liver unable to make enough albumin).
- **Edema due to increased interstitial fluid osmotic pressure:**
  - Increased capillary permeability—inflammation, toxins, hypersensitivity reactions, burns.
- **Edema due to blocked lymphatic drainage:**
  - Tumors
  - Goiter
  - Parasites that obstruct lymph nodes
  - Surgery that removes lymph nodes

Edema causes localized or generalized swelling. In a child who is walking, dependent edema is observed in the ankles; in a child who is supine in bed, it is seen in the sacral area. The main focus of management for edema is to treat the underlying condition that caused the edema.

MANAGEMENT OF ELECTROLYTE IMBALANCES

Electrolytes are normally gained and lost in relatively equal amounts so that body electrolyte balance is maintained (Table 23.3). However, when a child has an abnormal process loss, such as vomiting, wound drainage or nasogastric suction, electrolyte balance can be impaired.

TABLE 23.3: Normal serum values of electrolytes in children

Electrolyte	Newborn	Infant and child
Sodium	131–144 mmol/L	132–141 mmol/L
Potassium	• Premature 4.5–7.2 mmol/L • Term 3.2–5.7 mmol/L	• 3.3–4.7 mmol/L
Calcium	• Premature 3.5–4.5 mEq/L (1.7–2.3 mmol/L) • Term 4–5 mEq/L (2–2.5 mmol/L)	• 4.4–5.3 mEq/L (2.2–2.7 mmol/L)
Magnesium	• 1.3–2.7 mg/dL (0.5–1.1 mmol/L)	• 1.6–2.7 mg/dL (0.7–1.1 mmol/L)

Hyponatremia

A serum sodium level below 135 mmol/L in children (133 mmol/L in newborn) is called hyponatremia.

Causes

- **Hypovolemic hyponatremia:** Osmotic diuresis, adrenal insufficiency, diarrhea, vomiting, drains, cystic fibrosis, ascitis, burns.
- **Normovolemic hyponatremia:** Syndrome of inappropriate antidiuretic hormone secretion (SIADH), meningitis, tumors, severe asthma, pneumonia, postoperative period, hypothyroid.
- **Hypovolemic hyponatremia:** Congestive heart failure, cirrhosis, renal failure, nephritic syndrome, excessive intravenous D5W (5% dextrose in water) rather than isotonic fluids.

Clinical Manifestations

- Manifestations of hyponatremia are anorexia, nausea, vomiting, confusion, headache, respiratory distress, muscle weakness, decreased deep tendon reflexes, agitation, lethargy or confusion.
- The condition can further progress to respiratory arrest, seizures, dilated pupils, decorticate posturing, coma, cardiac arrhythmias, myocardial ischemia, brain ischemia and death.

Treatment

- Correction of intravascular volume depletion with isotonic fluid (NS or Ringer lactate).
- Replace sodium over 24–48 hours after estimating the sodium deficit.

- Fluid restriction only in case of SIADH; diuretics may be given.

Hypernatremia

A serum sodium level above 148 mmol/L in children (146 mmol/L in newborns) is diagnostic of hypernatremia.

Causes

- **Hypotonic loss:** Diabetes insipidus, diarrhea or vomiting without fluid replacement, excessive sweating without fluid replacement, high solute intake without adequate water.
- **Hypertonic sodium gain:** Saline infusion, improper formula or ORS, boiled skimmed milk, hypertonic dialysis, Cushing syndrome.

Clinical Manifestations

- Thirsty child with a shriveled tongue and rough feeling to skin.
- A decreased level of consciousness manifested by confusion, lethargy, or coma resulting from shrinking of the brain cells.
- Seizures in case of rapid or severe hypernatremia.
- Hypertonicity, hyperreflexia.

Treatment

- If a child is dehydrated, isotonic fluids may be infused first to replenish the volume, followed by hypotonic fluid to correct the osmolality.
- Fluids to be infused: D5 0.25 NS or D5 0.5 NS.
- In seizures, use hypertonic (3%) saline at 5–6 mL/kg infusion over 1–2 hours.
- Treat the underlying causes.

Hypokalemia

Serum potassium level below 3.5 mmol/L in children (3.7 mmol/L for newborn) is called hypokalemia.

Causes

Hypokalemia is caused by conditions contributing to increased potassium excretion, decreased potassium intake, shift of potassium from the extracellular fluid into cells, and loss of potassium by an abnormal route.

- **Increased potassium excretion through the gastrointestinal tract:** Vomiting and diarrhea (gastroenteritis), nasogastric suctioning and intestinal decompression.
- **Increased urinary potassium excretion:** Osmotic diuresis, hypomagnesemia, hypercalcemia, increased aldosterone (hyperaldosteronism, congestive heart failure, nephrotic syndrome, cirrhosis) and increased cortisol (Cushing syndrome).
- **Decreased potassium intake:** Malnutrition, potassium less parenteral nutrition, anorexia nervosa.
- **Shift of potassium from the extracellular fluid into cells:** Alkalosis, hypothermia due to certain medications, refeeding syndrome, hypokalemic periodic paralysis.

Clinical Manifestations

- Gastrointestinal smooth muscle activity is slowed, resulting in diminished bowel tones, abdominal distension, constipation or paralytic ileus.
- Skeletal muscles are weak and unresponsive to stimuli; deep tendon reflexes are diminished; and weakness may progress to flaccid paralysis.
- The respiratory muscles may be impaired.
- Cardiac arrhythmias particularly a prolonged QT interval, depressed ST segment, and flat or inverted T waves can occur.
- Polyuria, polydipsia and decreased urine specific gravity.

Treatment

- **Replacement of potassium:** Intravenously or orally—2–4 mEq/kg/day in 3–4 divided doses orally or 0.5–1 mEq/kg over 1 hour.
- Treat the cause of imbalance.

Hyperkalemia

Hyperkalemia, an excess of potassium in the blood, is defined by a level above 5.8 mmol/L in children or above 5.2 mmol/L in newborns.

Causes

Hyperkalemia is caused by conditions that involve increased potassium intake, shift of potassium from cells into the extracellular fluid and decreased potassium excretion.

- **Increased potassium intake:** Intravenous potassium overload, exchange transfusions in newborn or multiple blood transfusions after a serious injury or in surgery.
- **Shift of potassium from cells into the extracellular fluid:** Massive cell death, as with a crush injury, sickle-cell anemia, chemotherapy for a malignancy, drugs like beta blockers, metabolic acidosis.
- **Decreased potassium excretion:** Acute or chronic oliguria during renal failure, severe hypovolemia, lead poisoning, Addison disease and hypoaldosteronism.

Clinical Manifestations

- **Gastrointestinal smooth muscle:** Hyperactivity causes intestinal colic, cramping and diarrhea in some children.

- **Skeletal muscles:** Leg weakness, flaccid paralysis, lethargic
- **Dysfunction of cardiac muscle:** Cardiac arrhythmias, heart failure and cardiac arrest, ECG changes (prolonged QRS complex, a peak in T waves, and prolonged PR intervals).
- **Renal:** Oliguria and anuria.

Treatment

- Prompt discontinuation of potassium containing fluids or medications.
- Prevent cardiac arrhythmia by infusing 10% IV calcium gluconate 0.5 mL/kg over 5–10 minutes.
- Increase cellular uptake of potassium by administering regular insulin, glucose, sodium bicarbonate, salbutamol.
- Eliminate body sodium by administering sodium polystyrene sulfonate (kayexalate), loop diuretics, peritoneal dialysis or hemodialysis.

Hypocalcemia

Hypocalcemia is a deficiency of serum calcium that is below 4.4 mEq/L in children or 4 mEq/L in newborns.

Causes

- **Decreased calcium intake or absorption:** Chronic generalized malnutrition, or a diet low in vitamin D and calcium, high phosphate intake, chronic diarrhea and steatorrhea.
- **Shift of calcium to a physiologically unavailable form:** Hypoparathyroidism, DiGeorge syndrome (congenital absence of the parathyroid glands), hypomagnesemia and alkalosis.
- **Increased calcium excretion:** Steatorrhea, acute pancreatitis.
- **Loss of calcium by an abnormal route:** Burn or wound drainage or acute pancreatitis, prolonged therapy with frusemide, corticosteroid.

Clinical Manifestations

- Increased muscular excitability (tetany), twitching and cramping, tingling around the mouth or in the fingers, carpal spasm and pedal spasm.
- Laryngospasm, seizures and cardiac arrhythmias in severe cases.
- Congestive heart failure in neonates.

Treatment

- Oral or intravenous administration of calcium.
- Magnesium replacement, if hypomagnesemia.
- High calcium foods.

Hypercalcemia

Hypercalcemia refers to a plasma level of calcium that is above 5.3 mEq/L in children or 5 mEq/L in newborns.

Causes

Hypercalcemia is caused by conditions contributing to increased calcium intake or absorption, shift of calcium from bones into the extracellular fluid and decreased calcium excretion.

- **Increased calcium intake or absorption:** Infant with increased intake of chicken liver, excessive vitamin D or A doses, children receiving total parenteral nutrition with high doses of calcium, adolescents taking calcium-rich foods concurrently with antacids (milk-alkali syndrome).
- **Shift of calcium from bones into the extracellular fluid:** Excessive amounts of parathyroid hormone produced in hyperparathyroidism, prolonged immobilization, malignancies such as leukemias, bone tumors and chemotherapy
- **Decreased calcium excretion:** Thiazide diuretics, lithium and theophylline.

Clinical Manifestations

- Decreased neuromuscular excitability.
- Constipation, anorexia, nausea and vomiting.
- Fatigue with skeletal muscle weakness.
- Confusion, lethargy and decreased attention span.
- Cardiac arrhythmias and arrest.
- Flaccid muscles and failure to thrive.
- Polyuria and polydipsia.

Treatment

- Increase urinary excretion of calcium by administering fluid or diuretics.
- Decrease intestinal absorption of calcium—glucocorticoids
- Dialysis may be needed.
- Treat the underlying causes.

Hypomagnesemia

Hypomagnesemia is defined as a plasma magnesium concentration that is below 1.5–1.7 mg/dL.

Causes

- **Decreased magnesium intake or absorption:** Prolonged intravenous therapy without magnesium, chronic malnutrition, chronic diarrhea, short bowel syndrome, malabsorption syndromes and steatorrhea.
- **Shift of magnesium to a physiologically unavailable form:** Transfusion of excessive citrated blood products, liver transplant patients with impaired citrate metabolism.

TABLE 23.4: Summary of assessment of electrolyte imbalances

System of assessment	Specific assessments	Changes with electrolyte imbalances
• Skeletal muscle function	• Muscle strength	• **Weakness, flaccid paralysis:** Hyperkalemia; hypokalemia
• Neuromuscular excitability	• Deep tendon reflexes • Chvostek sign • Trousseau sign • Paresthesia • Muscle cramping or twitching	• **Depressed:** Hypercalcemia; hypermagnesemia • **Hyperactive:** Hypocalcemia; hypomagnesemia • **Positive:** Hypocalcemia; hypomagnesemia • **Positive:** Hypocalcemia; hypomagnesemia • **Digital or perioral:** Hypocalcemia • **Present:** Hypocalcemia; hypomagnesemia
• Gastrointestinal tract function	• Bowel sounds • Elimination pattern	• **Decreased or absent:** Hypokalemia • **Constipation:** Hypokalemia; hypercalcemia • **Diarrhea:** Hyperkalemia
• Cardiac rhythm	• Arrhythmia • Electrocardiogram	• **Irregular:** Hyperkalemia; hypokalemia; hypercalcemia; hypocalcemia; hypermagnesemia; hypomagnesemia • **Abnormal:** Hyperkalemia; hypokalemia; hypercalcemia; hypocalcemia; hypermagnesemia; hypomagnesemia
• Cerebral function	• Level of consciousness	• **Decreased:** Hyponatremia; hypernatremia

- **Increased magnesium excretion:** Diuretic therapy, acute renal failure, diabetic ketoacidosis and hyperaldosteronism.
- **Loss of magnesium by an abnormal route:** Prolonged nasogastric suction and through sequestration of magnesium in acute pancreatitis, medications like antineoplastic agents, systemic antifungals, aminoglycoside antibiotics and laxatives without magnesium.

Clinical Manifestations

- Neuromuscular excitability (tetany).
- Hyperactive reflexes, skeletal muscle cramps, twitching, tremors and cardiac arrhythmias.
- Seizures.

Treatment

- Administration of magnesium.
- Treat the underlying cause of imbalance.

Hypermagnesemia

Hypermagnesemia refers to the plasma magnesium concentration that is above 2.4 mg/dL.

Causes

- **Decreased magnesium excretion:** Oliguric renal failure and adrenal insufficiency.
- **Increased magnesium intake:** Hypermagnesemia in the newborn due to magnesium sulfate administered to treat eclampsia in the mother before delivery, magnesium containing enemas, laxatives, antacids, and intravenous fluids, aspiration of seawater, as in near-drowning, Addison disease.

The assessment of electrolyte imbalances has been summarized in Table 23.4.

Clinical Manifestations

- Decreased muscle irritability, hypotension, bradycardia, drowsiness, lethargy and weak or absent deep tendon reflexes.
- Flaccid muscle paralysis, fatal respiratory depression, cardiac arrhythmias and cardiac arrest occur in severe cases.

Management

- Treat the underlying cause.
- Monitor ECG.
- Administer Ca gluconate.
- Increase fluid intake (except in oliguric renal failure) and administration of diuretics.
- Dialysis may be required.

MANAGEMENT OF ACID-BASE IMBALANCE

Physiology of Acid-Base Balance

Maintenance of acid-base balance is vital for normal functioning of cells. Acidity is expressed in terms of pH (the negative logarithm of the hydrogen ion concentration). The range of possible pH values is 1–14. A pH of 7 is neutral. Lower the pH, more acidic the solution. A pH above 7 is basic or alkaline.

The pH of the blood influences the pH inside the cells (Table 23.5). Acidemia refers to a blood pH below normal levels, whereas alkalemia is an increased blood pH. If the

TABLE 23.5: Normal blood pH and gases in children

	Infants	Children	Adolescents
Arterial blood pH	7.18–7.50	7.27–7.49	7.35–7.41
Arterial blood pO_2	60–70 mm Hg	80–108 mm Hg	80–100 mm Hg
Arterial blood pCO_2	27–41 mm Hg	32–48 mm Hg	32–48 mm Hg
Arterial blood HCO_3^- (bicarbonate)	19–24 mmol/L	18–25 mmol/L	20–29 mmol/L

pH inside the cells becomes too high or too low, then the speed of chemical reactions becomes inappropriate for cell function. Thus, acid-base imbalances result in clinical signs and symptoms.

Three systems regulate acid-base balance interdependently. They are the buffer, respiratory and renal systems. Cells produce two kinds of acids: carbonic acid (H_2CO_3) and metabolic (noncarbonic) acid. These acids are released into the extracellular fluid which has to be neutralized or excreted from the body to prevent dangerous accumulation. They can be neutralized to some degree by the buffers in body fluids.

A buffer is a compound that binds hydrogen ions when their concentration rises and releases them when their concentration falls. The most important buffer is the bicarbonate-carbonic acid (HCO_3^-–H_2CO_3) pair, which is responsible for buffering ECF. Hemoglobin and oxyhemoglobin pair is also an important buffer of carbonic acid.

The lungs excrete excess carbonic acid from the body. A child breathes out carbon dioxide and water, the components of carbonic acid, with each breath. An indirect laboratory measurement of carbonic acid is pCO_2, the partial pressure of carbon dioxide in arterial blood.

Metabolic acids are excreted by the kidneys. They reabsorb filtered bicarbonate to prevent its loss through urine and they regenerate bicarbonate when required to restore balance.

Metabolic Acidosis

Metabolic acidosis is a condition characterized by a decrease in serum pH (<7.35) due to decrease in plasma bicarbonate or increase in hydrogen ion concentration.

Causes

- **Bicarbonate loss:** Proximal renal tubular acidosis, diarrhea, fistula or drainage of small bowel or pancreas.
- **Increased acid production:** Oliguria (e.g., renal failure), distal renal tubular acidosis, hyperalimentation, diabetic ketoacidosis, starvation, ketoacidosis, inborn errors of metabolism (e.g., maple syrup urine disease), tissue hypoxia (lactic acidosis).

Clinical Manifestations

- Decreased blood pH and decreased HCO_3^- and pCO_2.
- Tachypnea, Kussmaul respirations.
- Tachycardia.
- Cardiac dysrhythmias.
- Hypotension.
- Poor perfusion with a grayish pallor.
- Decreased peripheral pulses.
- Increased capillary refill time.
- Pulmonary edema.
- Decreased level of consciousness, headache, lethargy.
- Drowsiness.
- Confusion.
- Apathy.
- Unresponsiveness.
- Seizures.
- Nausea and vomiting.
- Abdominal distension and pain.

Treatment

- Identification and treatment of the underlying cause.
- Treatment of renal failure or renal tubular acidosis with oral alkali therapy or dialysis.
- Intestinal fistula is repaired.
- Insulin therapy in diabetic ketoacidosis.
- In severe metabolic acidosis, intravenous sodium bicarbonate may be used to elevate the pH and prevent cardiac arrhythmias.

Metabolic Alkalosis

Metabolic alkalosis is an excess of bicarbonate in extracellular fluid caused by conditions leading to excess base because of loss of hydrogen ion, reabsorption of bicarbonate or loss of other ions (i.e., chloride, sodium).

Causes

- Vomiting, chloride diarrhea.
- Gastrointestinal suctioning.
- Diuretics.
- Hypokalemia, hypocalcemia and hypochloremia.
- Exogenous alkali intake: HCO_3^-, citrate, lactate, acetate.
- Excessive steroid use.
- Renal failure.
- Extracellular fluid volume depletion.
- Cystic fibrosis.
- Excess mineralocorticoid.

- Hyperaldosteronism, Cushing syndrome.
- Bartter syndrome.

Clinical Manifestations

- Elevated blood pH, bicarbonate and pCO_2.
- Increased neuromuscular excitability.
- Muscle cramps, twitching, tetany.
- Hypoventilation.
- Confusion.
- Lethargy.
- Unresponsiveness.
- Hyperreflexia.
- Seizures.
- Nausea, vomiting and diarrhea.

Treatment

- Treat the underlying cause of the condition.
- Mild cases rarely require correction.
- Discontinue diuretics.
- Volume resuscitation with normal saline.
- Acetazolamide increases renal excretion of bicarbonate.
- Cautious use of HCl or ammonium chloride.
- In case of renal failure, hemodialysis may be required.

Respiratory Acidosis

Respiratory acidosis is an excess of ECF carbonic acid that is caused by conditions resulting in hypoventilation and CO_2 retention. pCO_2 increases and the pH of the blood begins to decrease.

Causes

- **Hypoventilation:** Acute paralysis of respiratory muscles, acute or chronic lung parenchymal and airway disease, progressive neuromuscular disease, restrictive lung disease.
- **CO_2 retention:** Burns, malignant hyperthermia, fever.

Clinical Manifestations

- Increased pCO_2, pH can be decreased or normal.
- Central nervous system depression, as evidenced by confusion, lethargy, headache, increased intracranial pressure and even coma.
- Tachycardia, cardiac arrhythmias, hypotension.

Treatment

- Reestablish effective ventilation and treat the underlying problem.
- Bronchodilators for bronchospasm, mechanical ventilation for neuromuscular defects.
- Decrease sedative use.
- Surgery for kyphoscoliosis.

Respiratory Alkalosis

Respiratory alkalosis is a deficit of carbonic acid in extracellular fluid caused by conditions leading to alveolar hyperventilation and CO_2 deficit.

Causes

- **Pulmonary disorders:** Pneumonia, pulmonary edema, pulmonary emboli and interstitial lung disease.
- **Increased metabolic rate:** Fever, hyperthyroidism, exercise, anemia and sepsis.
- **Increased intracranial pressure:** Meningitis, encephalitis, head trauma, stroke, brain lesions.
- **Intoxications:** Alcohol, salicylate, paraldehyde, xanthine.
- **Miscellaneous:** High altitude, hepatic failure, congestive heart failure with hypoxemia and mechanical ventilation.

Clinical Manifestations

- Decreased pCO_2, elevated blood pH.
- Breathlessness.
- Extremity and perioral paresthesias.
- Vertigo and syncope.
- Anxiety, nervousness, confusion, dizziness.
- Hyperreflexia.
- Muscle cramps, twitching, tetany.
- Seizures.

Treatment

- Restoration of effective ventilation and treatment of the underlying cause.
- Sedation, breathing exercises, and relaxation with controlled breathing.
- Administration of 3–5% CO_2 and neuromuscular paralysis with intubation and mechanical ventilation may also be required in severe respiratory alkalosis and when other measures are ineffective.

KEY MESSAGE

- Fluid and electrolyte as well as acid-base balance regulate key functions of the body.
- Nurses need to be keen observers to detect cues for fluid electrolyte and acid-base imbalances in children.

SUMMARY

- Hyponatremia and hypokalemia should be treated with administration of sodium chloride infusion and potassium chloride infusion or orally.
- ABG analysis should be done for suspected acidosis and alkalosis patients. Identification and treatment of underlying cause should be done.

ASSESS YOURSELF

Long Answer Questions

1. What are the types of dehydration or extra cellular fluid volume deficit?
2. Describe the causes, clinical manifestations and treatment of sodium and potassium deficiency in children.
3. How will you manage hyponatremia and hypokalemia?

Short Answer Questions

1. What are the causes of hypocalcemia?
2. What are the clinical manifestations of hypomagnesemia?
3. Write a short note on:
   a. Metabolic acidosis
   b. Respiratory alkalosis

Multiple Choice Questions

1. The most common cause of hyponatremia in children is:
a. Cystic fibrosis
b. Meningitis
c. Diarrhea
d. None of these

2. Tetany is caused due to deficiency of:
a. Sodium
b. Magnesium
c. Calcium
d. Both b and c

3. Kussmaul respiration is seen in case of:
a. Metabolic acidosis
b. Metabolic alkalosis
c. Respiratory acidosis
d. Respiratory alkalosis

4. Deficit of carbonic acid in extracellular fluid causes:
a. Metabolic acidosis
b. Metabolic alkalosis
c. Respiratory acidosis
d. Respiratory alkalosis

5. pH of blood above 7 indicates that the blood is:
a. Acidic
b. Basic
c. Neutral
d. None of these

ANSWER KEY

1. c **2.** d **3.** a **4.** d **5.** b

FURTHER READINGS

- *Alterations In fluid, Electrolyte, And Acid-base balance. http://www.omkarmin.com/pdf/alterations-in-fluid-electrolyte-and-acid-base-balance-51638.pdf*
- *Basic Fluid and Electrolytes http://www.cs.amedd.army.mil/borden/FileDownloadpublic.aspx?docid=ebc1c8ba-4ac9-4106-b97e-a27a805f8881*
- *Bhatt Swarna Rekha. Achar's Textbook of Pediatrics. Hyderabad: University Press (India) Pvt Ltd; 2009*
- *Dutta Parul. Pediatric Nursing. New Delhi: Jaypee Brothers Medical Publishers (P) Ltd; 2009.*
- *Gupte Suraj. The Short Textbook of Pediatrics. New Delhi: Jaypee Brothers; 2004*
- *Hockenberry Marlin J. Wong's Essentials of Pediatric Nursing. St Louis: Mosby; 2005*
- *James Rowen Susan and Ashwill Jean Weiler. Nursing Care of children: Principles and Practice. St. Louis: Mosby;2009*
- *Kathryn E. Roberts Pediatric Fluid and Electrolyte Imbalances: A Case Study Approach www.pediatricnursing.net/conf/handouts/2010/session_211_roberts.pdf*
- *Marlow R Dorothy and Redding A Barbara. Marlow's Textbook of Pediatric Nursing. South Asian edition. New Delhi: Elsevier; 2013*
- *Paul Vinod K and Bagga Arvind. Ghai Essential Pediatrics. New Delhi: CBS Publishers and Distributors Pvt. Ltd; 2013*
- *Powers A Acid-Base Balance. repository.upenn.edu/cgi/viewcontent.cgi?filename=8&article...*
- *Yadav Manoj. A Text Book of Child Health Nursing with Procedures. Jalandhar City: S. Vikas and Company; 2013.*

Textbook of

Pediatric Nursing for BSc Nursing Students

Salient Features

- Written in adherence with the revised INC syllabus for BSc Nursing also useful for Post-Basic BSc and MSc Nursing.
- Recent updates covered as per the advancement in the field of pediatric Practice and New diseases/conditions added.
- Algorithms and Protocols for the management of neonatal problems included extensively.
- Special emphasis has been given on growth and development of children of different age groups.
- Contains diagrammatically illustrated pathophysiology for easy grasp of the pathophysiological features of the respective disease conditions.
- Includes Nursing Processes of various disease conditions according to Latest NANDA Diagnosis 2021-23.
- Detailed coverage of Drugs in Pediatrics to facilitate safe medication practice by students and nurses.
- 13+ Pediatric Procedure videos covered.

Learning Objectives enlist what the students will learn after studying the entire chapter.

LEARNING OBJECTIVES

After completion of this chapter, students will be able to:
- Understand the development of child health and its modern concept.
- Differentiate between adult and child.
- Identify the rights of the children.

Chapter Outline provides a quick glance of the entire chapter in one go.

CHAPTER OUTLINE
- Introduction
- Historical Development of Child Health
- Philosophy and Modern Concept of Child Care

Important Key Terms have been added in the beginning of every chapter to get a quick and easy understanding of an important term in one go.

KEY TERMS

Child welfare: It is a continuum of services that are designed to ensure the safety and wellbeing of every child in the country.

Pediatrics: A branch of medicine that deals with diagnosis, treatment and prevention of diseases in children.

Must Know boxes provide highly useful additional information to enhance the knowledge of the students.

Must Know

Pediatric Nurse Practitioner Competencies
- Assessment of health status
- Diagnosis of health status
- Plan of care and implementation of treatment
- Creating a climate of mutual trust and establishing

35+ Child Health Nursing Procedures in the separate section.

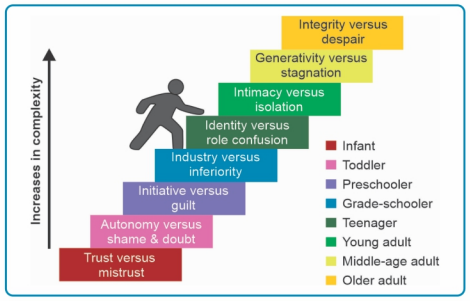
Procedure 1 TEMPERATURE RECORDING IN NEWBORN

Normal temperature in newborn is 36.5°–37.5°C. Accurate temperature recording should be done if the baby is:
- Preterm/low birth weight or sick.
- Admitted in hospital.
- Susceptible to develop hypothermia or hyperthermia.

Giving extra edge to book from the practical point of view with **20+ OSCE** stations checklist.

Sl. no.	Steps	Score	Student's score
1.	Advise mother to sit or lie in a comfortable position and help the mother to continue breastfeeding	1	
2.	Describe and ensure correct position: • Baby's body is well-supported. • The head, neck and the body of the baby are kept in the same plane. • Entire body of the baby faces the mother. • Baby's abdomen touches mother's abdomen.	4	
3.	Describe and ensure good attachment: • Baby's mouth is wide open. • Lower lip is turned out. • Chin is touching her breast. • Larger area of the areola is visible above than below.	4	
4.	Check for effective suckling—slow, deep sucks with pauses.	1	
	Total	10	

Illustrations and Tables are used to make learning easy for students.



Evolving conceptual details for application in clinical situations are depicted in **Nursing Implication** boxes.

Nursing Implications

Five key aspects of effective communication with a hospitalized child are: eye contact, good posture, speaking concisely, thorough explanation and summarization.

Text integrated with **Case Scenarios** to understand the topic with applied approach.

CASE SCENARIO

Rahul, an 8-year-old school boy, suddenly developed high fever, skin rash and joint pain. Fever was not controlling with antipyretics. The child had positive tourniquet test. On laboratory investigation, platelets were remarkably low and

Detailed **Assess Yourself** exercises in each and every chapter will facilitate structured learning and revision of the material provided in the respective chapters.

ASSESS YOURSELF

Long Answer Questions
1. Discuss pediatric nursing.
2. Explain the role of a pediatric nurse.
3. Define evidence-based practice and explain the steps of evidence-based practice in pediatric nursing.

The **Appendices** included in the book provide extra information to students apart from their regular syllabus-based study.

DOMAIN 1. HEALTH PROMOTION

Class 1: Health Awareness
- Decreased diversional activity engagement
- Readiness for enhanced health literacy

About the Author

Panchali Pal *PhD, MSc (N), MA (Public Administration), BSc (N),* is currently working as Professor cum Principal at Kothari Institute of Nursing, Kothari Medical Centre, The West Bengal University of Health Sciences, Kolkata and completed PhD from West Bengal University of Health Sciences, Kolkata. She has completed BSc (Hons) Nursing from AIIMS, New Delhi and MSc Nursing from SNDT Women's University, Mumbai. The author has published and presented numerous papers in national and international journals and conferences. She is the recipient of Asian Patient Safety Award for best poster presentation in Asian Patient Safety Congress.

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