III
Semester

Textbook of

Adult Health Nursing

(MEDICAL SURGICAL NURSING)

with Integrated Pathophysiology and Evidence-based Practice

As per the Revised Indian Nursing Council Syllabus (2021–22)

Volume
I

Special Features

- Authored, edited & reviewed by top nursing faculties PAN India
- **1200+** figures, illustrations and tables covered
- First ever book developed in the Indian context
- An exclusive book conforming to the latest INC syllabus
- Emphasis on critical thinking and clinical judgement through various case scenarios
- Enriched with evidence-based information, nursing care plan, nursing implication and clinical procedures

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- Procedures demonstrated by clinical specialists working at NABH/JCI accredited multispeciality hospitals
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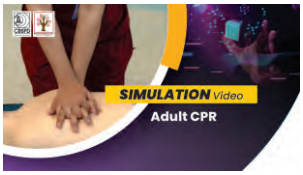
- Demonstrate common clinical errors
- Semester wise and subject wise (as per new INC Syllabus)
- Procedures are demonstrated as per the Indian context



Adult CPR

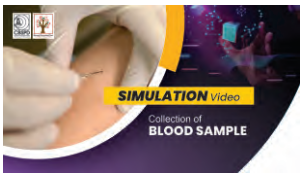
Duration 00:10:29

The video explains the steps of adult CPR following the guidelines of American Heart Association (2020). The well-defined steps and use of AED are easy to understand and recall.

Airway Adjuvants

Duration 00:11:50

This video will enable you to understand different types of airway adjuncts whether it is advanced airway (Endotracheal tube, tracheostomy tube, laryngeal mask airway) or basic airway (oropharyngeal tube or nasopharyngeal tube)



Collection of Blood Sample

Duration 00:11:55

With the help of this video, you will be able to understand how to take the blood sample in a step by step manner and also to learn about the order of draw sequence.

Drug Calculation

Duration 00:11:53

This video helps you to understand different formulas used in drug calculation. The video covers the drug calculation for tablets, injections and drop factor.

Intradermal Injection

Duration 00:07:49

This video explains different aspects of intradermal injection, purposes, steps of procedure, after care and also common errors committed by health care staff during administration of intradermal injection.

Nasogastric Feeding

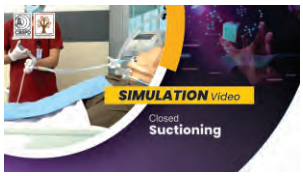
Duration 00:19:37

The video focuses on feeding the patient with the help of nasogastric tube. Clinical importance has been explained with the help of procedure demonstration which is easy to understand.

Oxygen Devices

Duration 00:12:57

In this video, you will understand the various aspects of Oxygen devices, methods of delivering oxygen devices, their uses and contraindications. The oxygen devices demonstrated in this video are-nasal cannula, simple face mask, venturi mask, rebreather mask and non rebreather mask.



Closed Suctioning

Duration 00:13:19

This video focuses on the core concept of closed suctioning with the use of closed suction catheter. This procedure is common in critical care units and through this video, it will be easy to understand the suctioning procedure and the process of hyperventilation through ventilator.

ECG Leads Placement

Duration 00:06:37

The 12-ECG leads represent heart's electrical activity recorded from electrodes on the body surface. This video demonstrates the correct placement of 12-ECG leads on the patient's chest and extremities.

Incentive Spirometry

Duration 00:05:40

This video shows the procedure of incentive spirometry to patient. The patient is instructed to hold the spirometer in an upright position, exhale normally, and then place the lips tightly around the mouthpiece.

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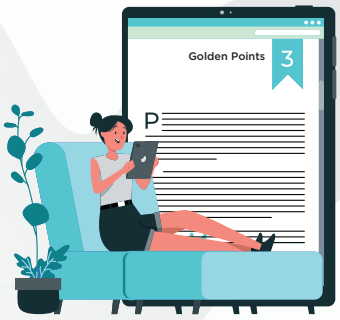

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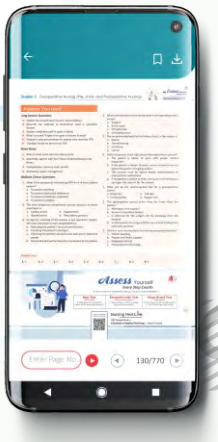
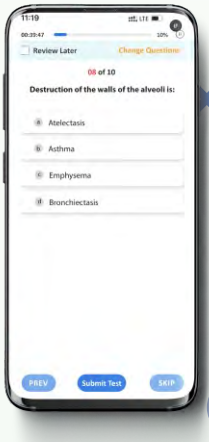


Learn

ii. Solved Exercises

Subjective and Objective exercises of the book have been given with their solution (in PDF) to evaluate and assess the complete chapter knowledge

- **100+** Long Answer Questions
- **100+** Short Answer Questions
- **500+** Multiple Choice Questions



Practice

iii. MCQs

200+ Chapter-wise Multiple Choice Questions in Practice and Review mode to provide in-depth concept clarity

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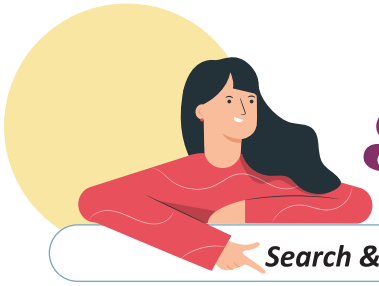

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- Get 100+ Important topics of the book at one click
- 1000+ Key Terminologies with their meaning in searchable format

VOLUME I

Chapter 1: Introduction to Medical Surgical Nursing

- Evolution and Trends of Medical and Surgical Nursing
- Health Care Models
- Roles and Responsibilities of a Nurse in Medical Surgical Nursing Settings
- Medical Surgical Asepsis
- Inflammation, Infection & Immunity

Chapter 2: Perioperative Nursing *(Pre-, Intra- and Postoperative Nursing)*

- Organization and Physical Setup of the Operation Theater
- Principles of Operation Theater Attire
- Types of Anesthesia
- Surgical Instruments
- Cardiopulmonary Resuscitation Guidelines
- Pain Management

Chapter 3: Nursing Care of Patient with Common Signs and Symptoms and Management

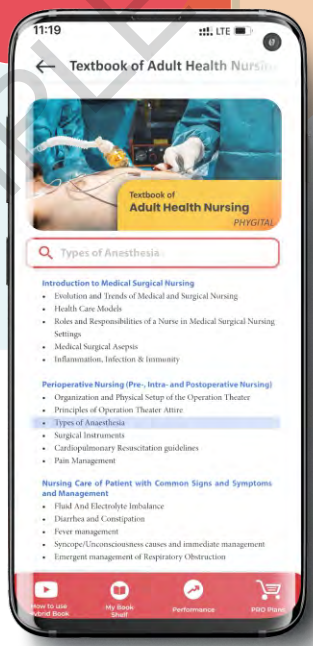
- Fluid and Electrolyte Imbalance
- Diarrhea and Constipation
- Fever Management
- Syncope/Unconsciousness Causes and Immediate Management
- Respiratory Obstruction

Chapter 4: Nursing Management of Patient with Respiratory System Disorders

- Diseases of the Upper Respiratory Tract
- Pathophysiology and Symptoms of Cystic Fibrosis
- Nursing Management for the Patient with Lung Transplantation

and Many more...

Important Topics



VOLUME II

Chapter 12: Nursing Management of Patients with Disorders of Ear, Nose and Throat

- Causes and Symptoms of Otalgia
- Pathophysiology and Symptoms of Otitis Externa
- Management of Vertigo
- Types and Pathophysiology of Rhinitis
- Management of Epistaxis
- Causes and Symptoms of Sinusitis
- Pathophysiology and Treatment of Chronic Pharyngitis
- Pathophysiology and Treatment of Acute Tonsillitis

Chapter 13: Nursing Management of Patients with Disorder of Eye

- Refractive Error
- Treatment of Conjunctivitis
- Surgical Management of Cataract
- Types and Symptoms of Glaucoma
- Pathophysiology of Retinal Detachment
- Eye Banking
- Role of Nurse in Communication with Visually Impaired Patient
- Nursing Management Principles in Eye Care

Chapter 14: Nursing Management of Patients with Genitourinary Disorders

- Causes and Pathophysiology of Glomerulonephritis
- Pathophysiology of Nephrotic Syndrome
- Treatment of Renal Calculus
- Symptoms of Acute Renal Failure
- Nursing Management of Patient with Chronic Renal Failure (End Stage Renal Failure)
- Hemodialysis
- Causes and Treatment of Polycystic Kidney Disease
- Kidney Transplantation
- Symptoms of Urinary Tract Infection

and Many more...

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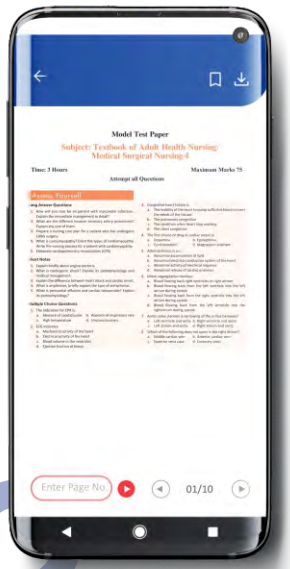

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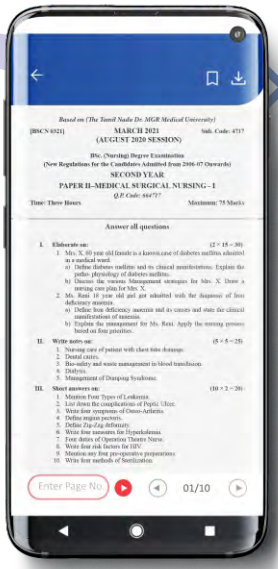

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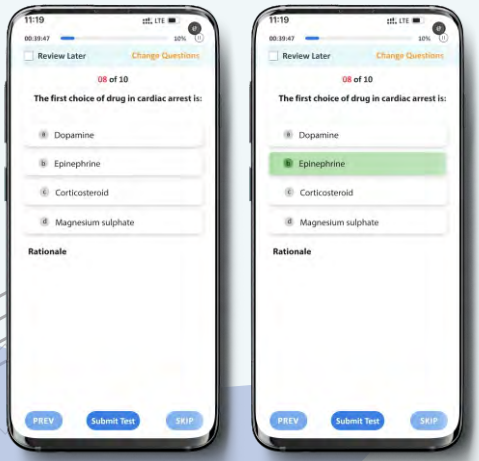

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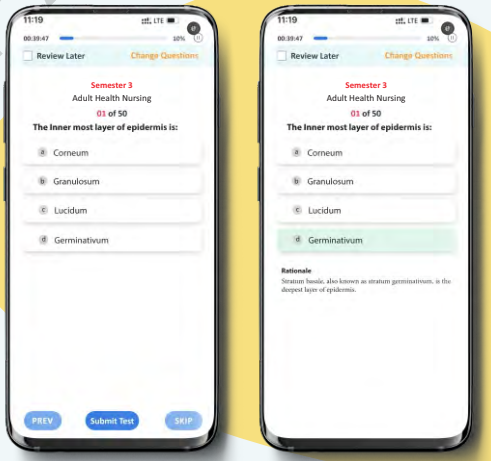
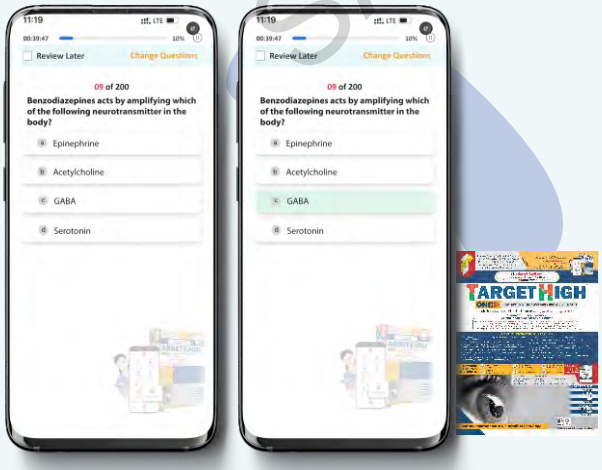
6 Tests covering **300** Questions with rationale (in Practice/Review mode) of Adult Health Nursing Subject covering all important topics



ii. Semester-wise Test

(All Semester Subjects)

2 Tests covering **200+** MCQs based on important topics of 2nd Year (3rd and 4th Semesters)



iii. Mega Grand Test *(All subjects)*

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- Get **500+** Topic-wise Selective Images & Tables with their descriptions for LMR and Quick reference, based on the topics of University examination
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List of **High Yield** Topics

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Chapter 1: Introduction to Medical Surgical Nursing

Chapter 2: Perioperative Nursing (Pre-, Intra- and Postoperative Nursing)

Chapter 3: Nursing Care of Patient with Common Signs and Symptoms and Management

and Many more...

VOLUME II

Chapter 12: Nursing Management of Patients with Disorders of Ear, Nose and Throat

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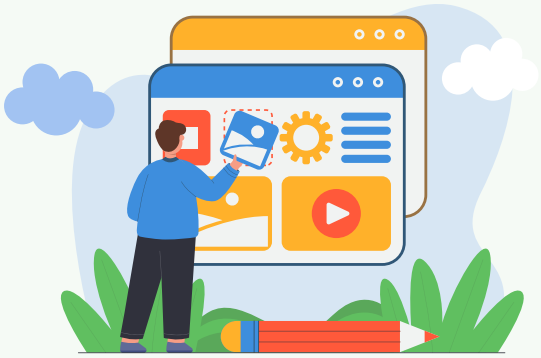

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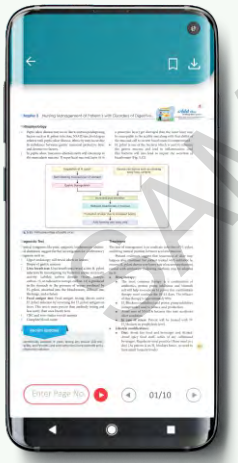
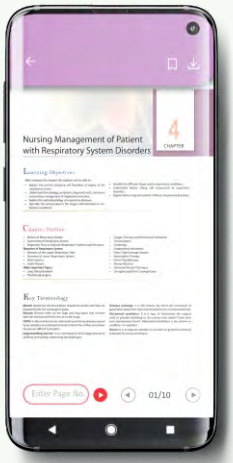


ii. Recent Update

Regular updates related to
Recent advancement and **Book Errata**



iii. E-book

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Volume I

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Preface

The **Textbook of Adult Health Nursing** book is organized into two volumes. These two volumes carry 23 chapters strictly designed according to the new undergraduate Nursing Syllabus (2021–22) prescribed by the Indian Nursing Council (INC).

- Chapters 1–3 cover the common medical surgical conditions and concept of medical surgical nursing
- Chapters 4–23 cover the system wise disease conditions with their medical, surgical and nursing management.

The new INC syllabus bridges the gap between theory and practice, similarly this book has been designed in such a manner that it covers both the theory as well as practical portions. This is for the first time that any medical surgical text book brings case scenarios and list of nursing procedures related to the disease condition. The case scenarios not only help nursing students to improve critical thinking but they also help the nursing instructors to prepare similar scenarios for clinical judgment exercises. The list of clinical procedures in the each chapter helps the teachers and students to correlate with the clinical postings and also reminds to learn the procedure.

Apart from this, the book contains practice-oriented features, such as operation theater instruments and preparation of sets for common surgical procedures, position and draping for common surgical procedures. While studying medical surgical nursing or any disease condition, the nursing students always face challenges in understanding pathophysiology, and this book has made it easy as the pathophysiology of all disease conditions has been explained through flowcharts.

This book covers detailed nursing care plan for every disease and more on nursing care point of view with nursing care plans and focus on critical thinking.

Students always prefer to study in concise manner, therefore, the content is presented using bullet points and diagrams to make it easy-to-grasp. Causes, risk factors along with signs and symptoms are presented with bullet points to make them easy to notice.

An easy-to-use format includes subheadings for every disease condition:

- Definition
- Risk factors/Causes
- Signs & Symptoms
- Pathophysiology
- Assessment
- Diagnostic Tests
- Medical/Surgical Management
- Nursing Care Plan
- Alternative Therapies

All types of questions—long answer questions, short answer questions and multiple choice questions—are added at the end of every chapter for self-evaluation and practice.

We believe that this book will be the most preferred book for nursing students and faculty.

Special Features

- Authored, edited & reviewed by top nursing faculties PAN India
- 1200+ figures, illustrations and tables covered
- First ever book developed in the Indian context
- An exclusive book conforming to the latest INC syllabus
- Emphasis on critical thinking and clinical judgment through various case scenarios
- Enriched with evidence-based information, nursing care plan, nursing implication and clinical procedures

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Acknowledgments

First of all, I would like to express my gratitude to Almighty Lord Jesus, Who gives me strength and directions in all walks of my life.

My deep heartfelt thanks are due to my parents who guided me and opened my door of opportunity in education. I am pretty sure that they really feel proud for my professional accomplishment.

I would like to pen my sincere thanks to my loving wife Shweta for her light-hearted and positive attitude and also her continuous encouragement and cooperation in bringing out this book.

I express my gratefulness to my sweet daughter Saesha, who is always lucky for me. I signed for this publication in the same month she born.

I am also thankful to my sisters Joyce and Sonia who are wonderful nurses too and who have always encouraged me with their positive attitude.

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I wish to acknowledge my gratitude to my nursing mentors and teachers for being the source of inspiration. My special thanks go to Mrs Smiti Victor, Dhamtari Christian Hospital College of Nursing who was my first medical surgical nursing subject teacher.

Special thanks to Ms Soumya for her tremendous help throughout the preparation of this publication.

My sincere appreciation and warmest gratitude to all individual who have contributed to my success in making my professional career.

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We extend our special thanks to **Mr Satish Kumar Jain** (Chairman) and **Mr Varun Jain** (Managing Director), M/s CBS Publishers and Distributors Pvt Ltd for their wholehearted support in publication of this book. We have no words to describe the role, efforts, inputs and initiatives undertaken by **Mr Bhupesh Aarora** [Sr. Vice President – Publishing and Marketing (Health Sciences Division)] for helping and motivating us.

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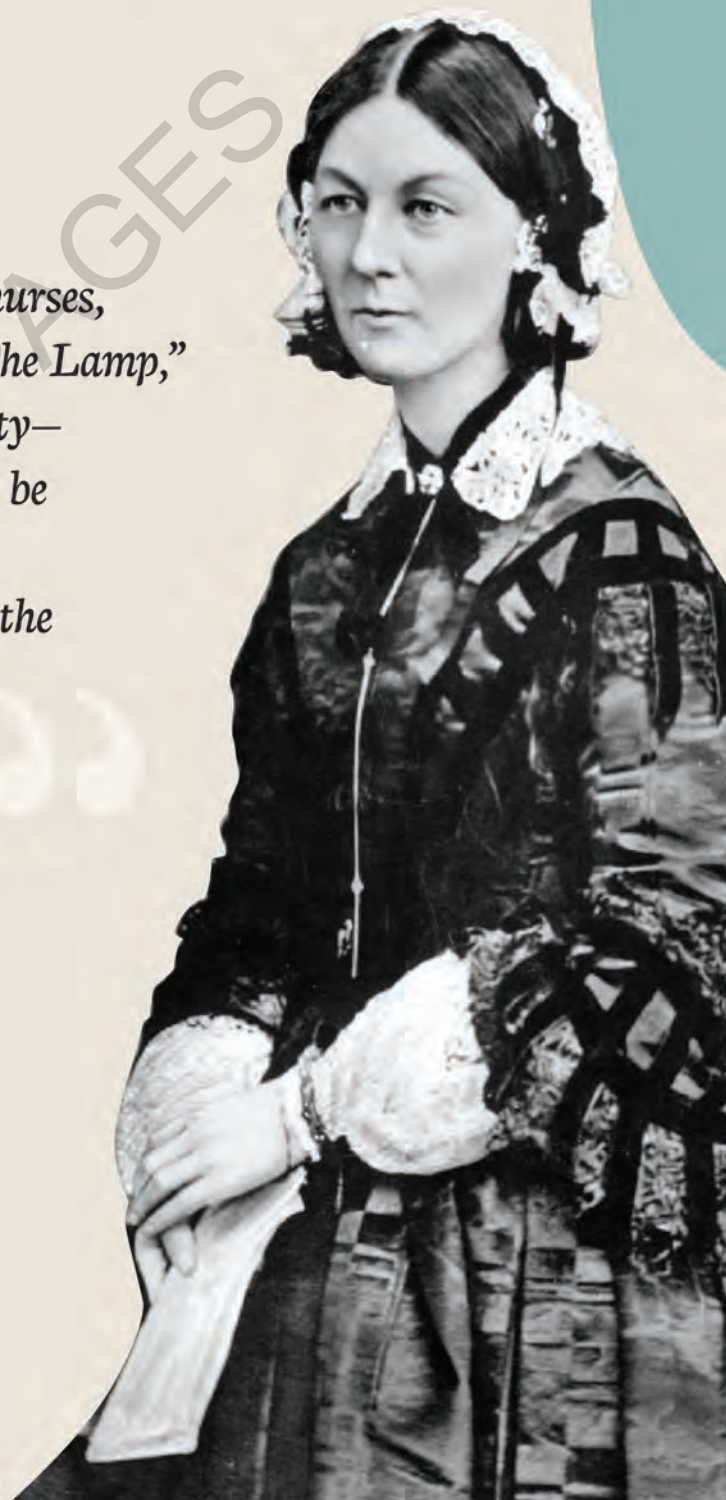
Extends its Tribute to

Florence Nightingale

For glorifying the role of women as nurses,
For holding the title of " The Lady with the Lamp,"
For working tirelessly for humanity–
Florence Nightingale will always be
remembered for her
selfless and memorable services to the
human race.



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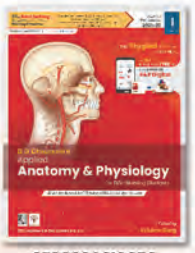

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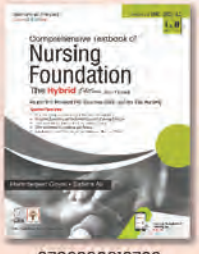

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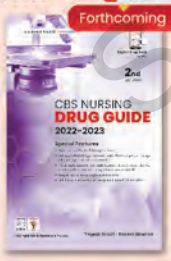

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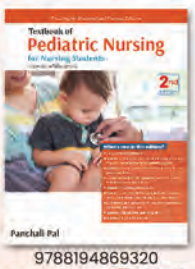

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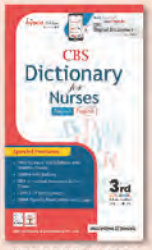

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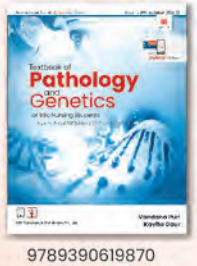

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The last two years were the most crucial phase when the entire world stood still due to adversity of COVID-19. The normal life was in turmoil, and people had no idea what would be their next step and how long this crisis would persist. In the midst of all, a few things which nobody could stop is 'Change', which is inevitable. During the last two years, we have done a lot of innovations and put our best efforts in implementing those innovations to bring quality education and make sure that every person should have access to best possible education.

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NN Social, a community of 20K+ professionals, is an initiative of Nursing Next Live as India's knowledge-sharing network platform for the nursing segment. Nursing Next Social is curated with the aim to bring all the nursing faculty members across the nation closer and together on a single platform. Through **NN Social**, we aim to connect the sharp minds across the nation to use their knowledge for the better future of Nursing Profession. With NN Social **India's top-notch societies, like TNAI, SOCN, NTA, KINS, etc. are associated with us.** Apart from this, NN Social has a strong network of 100+ authors, 500+ reviewers and contributors. They all are dedicated and committed as we are, toward imparting quality nursing education.

In the era of digitalization, to make study interactive and convenient, we have conceptualized the idea of **Hybrid Edition of the books**. In this series, our many bestselling titles are available in the hybrid form. This hybrid learning is a blended learning wherein printed booklets are thoughtfully integrated with the digital support to reconceptualize the learning method in a more interactive manner with added values to knowledge. Hybrid edition is an endeavor to facilitate the next level of preparation for any nursing competitive exams through quality content, flexibility, customization and engaging interactive learning experiences.

We have also increased our **social media presence** through meaningful and innovative ideas and are committed to assist the nursing professionals in gaining and sharing the knowledge. We have taken the initiative to learn from the experience of the others and started **NNL Talks**. It is a platform where every nursing professional who has done exceptionally well in his/her career, toppers of any Nursing Exams and those who manage themselves in all the odds and stand firm and determined and succeed in his/her life, can share the success journey. We aim to motivate, educate and encourage the nursing professionals through various activities and posts on our social media platform.

Whatever initiative we take, we always make sure that it is for a noble cause of promoting the quality education accessible to everyone.

Today we can say this with confidence, we "CBS Publishers and NNL" have an edge over all other Indian and International Publishers. Our Approach, Vision, Mission, Concept, Content, Reach, Ideas all have a single goal that is better nursing education can lead to a better healthcare system.

Long way to go…. Together!

Looking forward to invite more young and experienced minds who can join us as Authors, Reviewers, Contributors, and Faculties and accomplish our mission of providing quality nursing education to all.

With Best Wishes
Mr Bhupesh Aarora
Sr. Vice President – Publishing and Marketing
(Health Sciences Division)

Special Features of the Book

Learning Objectives

After studying this chapter, the students will be able to:

- Summarize the history of Medical Surgical Nursing.
- Discuss the trends in Medical Surgical Nursing.
- Describe the concepts of innate immunity and acquired immunity.

Learning Objectives given in the beginning of each chapter enable the student to know what he/she will learn after reading it.

Chapter Outline

- Introduction
- Evolution and Trends of Medical and Surgical Nursing
- Telehealth and Chatbot Facilities for Easy Access to Patient Care
- Concept of Health and Disease
- International Classification of Diseases (ICD)

Chapter Outline will summarize what the students will learn after studying the chapter.

Key Terminology

Acute: It is characterized by having sudden onset with very serious effect.
Aging: It is the process of becoming older.
Amputation: Refers to the removal of an extremity or any other body part due to disease condition.
Anatomy: Refers to the science the deals with the structure of body.

Important terms of the respective chapters have been summarized in beginning of chapter under **Key Terminology**.

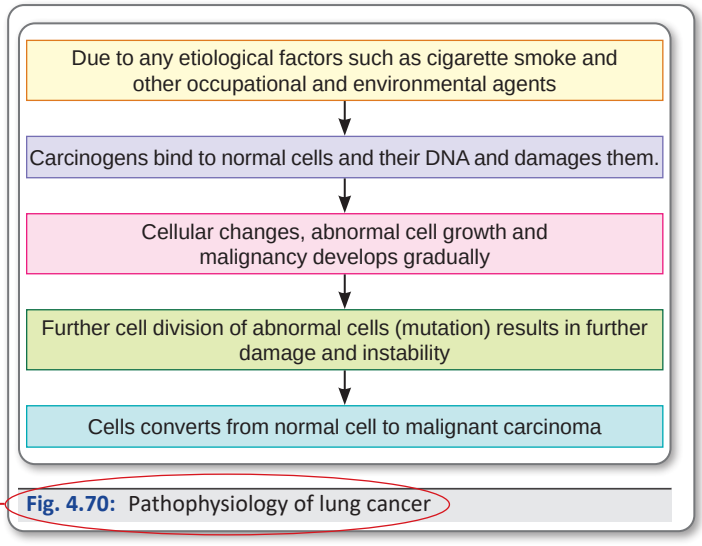

Fig. 4.70: Pathophysiology of lung cancer

All disease conditions pathophysiology have been explained through attractive **Flowcharts**.

Clinical Skills

1. Gastrointestinal assessment
2. Colostomy care
3. NG tube insertion and feeding
4. NG aspiration
5. Oral care
6. Peg feeding
7. Stool culture
8. Assisting in patient with Ultrasonography
9. Assisting in patient with upper GI series
10. Duodenostomy, jejunostomy feeding
11. Gastric lavage
12. Gastric gavage

CASE SCENARIOS RELATED TO NURSING MANAGEMENT OF PATIENT WITH DISORDER OF DIGESTIVE SYSTEM

CASE 1

After having a serious loss in business, Mr Naveen, 52-year-old patient, attempts suicide by ingesting 4 × 350 mL bottles of insecticide. The patient demonstrates a cholinergic toxidrome and requires large doses of atropine for reversal. 2 PAM is also antidotal.

- **Speech:** Patient is conscious but altered mental status and patient becomes unconscious after about 5 minutes.
- **Medication:** None
- **Weight:** 72 kg

- What advice you will give to the patient whenever she is pla to travel?

CASE 3

Mr Sudhir, a 52-year-old patient, came to the nearby local h with a chief complaint of a sudden onset of acute upper abdominal pain radiating to his back. The pain began shortl his morning meal and he vomited several times, without re his pain.

Each and every chapter is begining with **Clinical Skills** covering respective system case presentations.

High Yield Points

- Sullivan's index is an expectation of life free of disability.
- HALE is the equivalent number of years in full health that a newborn can be expected to live based on the current rates of ill health and mortality.

Important and must know points have been covered under **High Yield Points** boxes.

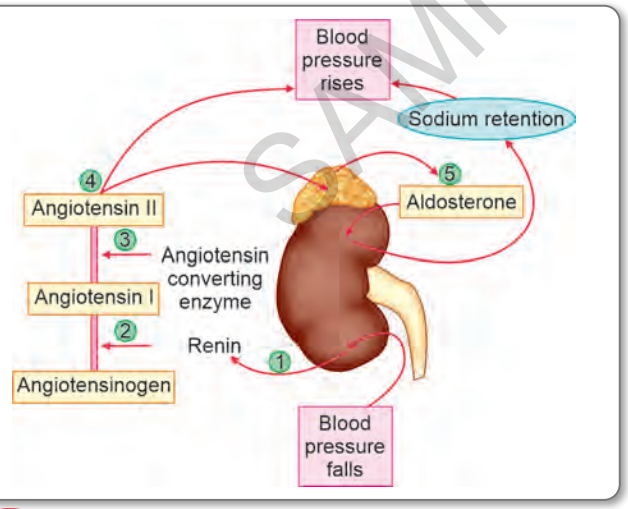

Fig. 3.4: Renin-angiotensin-aldosterone system

Important concept topics have been supplemented with numerous **Figure/Illustration/Tables** for easy understanding.

Principal facts have been covered under **Must Know** boxes.

Must Know

Chlorhexidine + detergent (Hibiscrub, Savlon): useful skin 'disinfectant'. Very active against Gram +ve organisms.

PROCEDURE

1. Gather all necessary equipment. Place it within easy reach.
2. Explain the procedure to the patient. Secure a signed consent.
3. Position the client as mentioned above.
4. Expose the puncture site by removing the gown on the affected side. Put rubber protector below point.

Throughout the book **Nursing Procedures** have been covered in separate boxes.

Throughout the book **Evidence-Based** facts have been covered.

Evidence-Based Practice

A meta-analysis study conducted with an aim to examine the impact of antipyretic therapy on mortality in critically ill septic adults and results revealed that Antipyretic treatment does not significantly improve 28-day/hospital mortality in adult patients with sepsis.

Nursing Care Plan

Gastritis

Assessment	Nursing diagnosis	Goal	Interventions	Rationale	Evaluation
Subjective data: Patient complains of burning kind of pain in stomach. **Objective data:** Physical appearance tells restlessness and pain measurement noted 5/10	**Acute pain:** Gastric related to increased gastric acid concentration	To reduce the level of pain and to promote patient comfort.	• Assessing patient condition and note report of pain on 10 point scale. • Report and observe all non-verbal pain cues such as diaphoresis, restlessness, and dull looks, vital alteration like increased blood pressure, pulse or respiration.	• To identify characteristics of pain such as location, kind and duration of pain • Assess the ongoing level of pain level • To identify any alarming signs of vitals • Calm environment will avoid irritation and keep patient on	Pain level is reduced as reported by visual pain scale.

Nursing Care Plan for each and every disease condition have been covered in tubular format extensively with their diagnosis, goal, interventions, rationals and evaluation.

Nurses Roles in respective clinical practices have been highlighted.

Nurses Role

Role of Nurse While Handling Suture

- Nurse should be responsible for keeping sutures sterile in tray
- Do not expose suture for a long time
- Do not open all the sutures at one time

Critical Thinking and Clinical Judgment

Which are the client's presurgery laboratory results which would indicate a need to contact the surgeon?

On the first postoperative day after a colon resection which activities would the nurse plan for immobility?

If In the recovery room, the postoperative client becomes cyanotic. Discuss the nursing quick nursing action for the same?

Critical Thinking and Judgment aspects have been covered at places from clinical practice point of view.

Each and every disease/condition have been covered with their relevant **Nursing Management** consideration.

Nursing Management

- Treatment of underlying cause
- The immediate treatment goal is to restore normal fluid volume and osmolality.
- If symptoms are mild and serum sodium is greater than 125 mEq/L/day (125 mmol/L), the only treatment may be restriction of fluids to 800–1000 mL/day.
- This restriction should result in gradual, daily reductions in weight, a progressive rise in serum sodium concentration and osmolality, and symptomatic improvement.
- In cases of severe hyponatremia (<120 mEq/L), especially in the presence of neurologic symptoms such as seizures, intravenous hypertonic saline solution (3–5%) may be administered.

Nursing Alert

Patient should begin diuresis within 30 minutes after receiving IV furosemide. If not than another dose has to be given

Nursing Alert from clinical consideration point of view have been covered.

Summary

- The protective zone, which separates the clean area of the suite from the less clean part of the hospital area, is located outside the clean zone.
- The aseptic zone is constructed around the clean zone. Only staff members who have changed out of their outerwear in the protective zone are allowed entry to this area.
- The disposal zone is the hallway where used equipment, soiled sheets, and operating room waste are removed. Each OR has disposal areas that lead to the disposal zone.
- A hospital's operating theater is a location where surgical procedures are performed in an aseptic setting.
- Major OTs are used for major surgeries such as orthopedic, neurosurgery, cardiac surgery, gastro surgery. It is attached with recovery rooms

Each and every chapter is ending with **Summarized one-liner** for quick glance over the chapter.

Important subjective and objective questions under **Assess Yourself** have been added in the last of every chapter to help student assess their learning.

Assess Yourself

Long Answer Questions

1. Explain the preoperative nurse's responsibilities.
2. Describe the methods of sterilization used in operation theater.
3. Explain anesthesia and its types in detail.
4. What is suture? Explain the types of suture in detail.
5. Prepare a case presentation for patient who received CPR.
6. Conduct hands-on practice on CPR.

Short Notes

1. Role of scrub nurse and circulatory nurse
2. Anesthetic agents with their route of administration and doses
3. Postoperative recovery room period
4. Biomedical waste management

Multiple Choice Questions

1. What is the purpose of maintaining NPO for 6–8 hours before surgery?
   a. To prevent vomiting
   b. To prevent electrolyte imbalance
   c. To prevent pulmonary aspiration
   d. To prevent urination
2. The most dangerous complication during induction of spinal anesthesia is:
   a. Cardiac arrest
   b. Hypotension
   c. Hyperthermia
   d. Respiratory paralysis

ADULT HEALTH NURSING–I
(With Integrated Pathophysiology)

Placement: III SEMESTER
Theory: 7 Credits (140 hours)
Practical: Lab/Skill Lab (SL) – 1 Credit (40 hours) Clinical – 6 Credits (480 hours)

Course Content **T– Theory, L/SL–Lab/Skill Lab**

Unit	Time (Hrs)	Learning Outcomes	Content	Teaching/Learning Activities	Assessment Methods
I	6 (T) 4 (L/SL)	• Narrate the evolution of medical surgical nursing • Apply nursing process in caring for patients with medical surgical problems • Execute the role of a nurse in various medical surgical setting • Develop skills in assessment and care of wound • Develop competency in providing pre- and postoperative care	**Introduction** • Evolution and trends of medical and surgical nursing • International classification of diseases • Roles and responsibility of a nurse in medical and surgical settings ▪ Outpatient department ▪ In-patient unit ▪ Intensive care unit • Introduction to medical and surgical asepsis ▪ Inflammation, infection ▪ Wound healing—stages, influencing factors ▪ Wound care and dressing technique • Care of surgical patient ▪ Preoperative ▪ Postoperative • Alternative therapies used in caring for patients with medical surgical disorders	• Lecture cum discussion • Demonstration and practice session • Role play • Visit to outpatient department, in patient and intensive care unit	• Short answer • OSCE
II	15 (T) 4 (L/SL)	• Explain organizational set up of the operating theater • Differentiate the role of scrub nurse and circulating nurse • Describe the different positioning for various surgeries • Apply principles of asepsis in handling the sterile equipment • Demonstrate skill in scrubbing procedures	**Intraoperative care** • Organization and physical set up of the operation theater ▪ Classification ▪ OT design ▪ Staffing ▪ Members of the OT team ▪ Duties and responsibilities of the nurse in OT • Position and draping for common surgical procedures • Instruments, sutures and suture materials, equipment for common surgical procedures	• Lecture cum discussion • Demonstration, practice session, and case discussion • Visit to receiving bay	• Caring for patient intra-operatively • Submit a list of disinfectants used for instruments with the action and precaution

Contd...

Unit	Time (Hrs)	Learning Outcomes	Content	Teaching/Learning Activities	Assessment Methods
		• Demonstrate skill in assessing the patient and document accurately the surgical safety checklist • Develop skill in assisting with selected surgeries • Explain the types, functions, and nursing considerations for different types of anesthesia	• Disinfection and sterilization of equipment • Preparation of sets for common surgical procedures • Scrubbing procedures—gowning, masking and gloving • Monitoring the patient during the procedures • Maintenance of the therapeutic environment in OT • Assisting in major and minor operation, handling specimen • Prevention of accidents and hazards in OT • Anesthesia—types, methods of administration, effects and stages, equipment and drugs • Legal aspects		
III	15 (T) 4 (L/SL)	• Identify the signs and symptoms of shock and electrolyte imbalances • Develop skills in managing fluid and electrolyte imbalances • Perform pain assessment and plans for the nursing management	**Nursing Care of Patients with Common Signs and Symptoms and Management** • Fluid and electrolyte imbalance • Shock • Pain	• Lecture, discussion • Demonstration • Case discussion	• Short answer • MCQ • Case report
IV	18 (T) 4 (L)	• Demonstrate skill in respiratory assessment • Differentiates different breath sounds and lists the indications • Explain the etiology, pathophysiology, clinical manifestations, diagnostic tests, and medical, surgical, nutritional, and nursing management of common respiratory problems • Describe the health behaviour to be adopted in preventing respiratory illnesses	**Nursing Management of Patients with Respiratory Problems** • Review of anatomy and physiology of respiratory system • Nursing assessment—history taking, physical assessment and diagnostic tests • Common respiratory problems: ▪ Upper respiratory tract infections ▪ Chronic obstructive pulmonary diseases ▪ Pleural effusion, empyema ▪ Bronchiectasis ▪ Pneumonia ▪ Lung abscess ▪ Cyst and tumors ▪ Chest injuries ▪ Acute respiratory distress syndrome ▪ Pulmonary embolism • Health behaviours to prevent respiratory illness	• Lecture, discussion, • Demonstration • Practice session • Case presentation • Visit to PFT Lab	• Essay • Short answer • OSCE

Contd...

Unit	Time (Hrs)	Learning Outcomes	Content	Teaching/Learning Activities	Assessment Methods
V	16 (T) 5 (L)	• Explain the etiology, pathophysiology, clinical manifestations, diagnostic tests, and medical, surgical, nutritional, and nursing management of gastrointestinal disorders • Demonstrate skill in gastrointestinal assessment • Prepare patient for upper and lower gastrointestinal investigations • Demonstrate skill in gastric decompression, gavage, and stoma care • Demonstrate skill in different feeding techniques	**Nursing Management of Patients with Disorders of Digestive System** • Review of anatomy and physiology of GI system • Nursing assessment—history and physical assessment • GI investigations • Common GI disorders: ▪ Oral cavity: Lips, gums and teeth ▪ GI: Bleeding, infections, inflammation, tumors, obstruction, perforation and peritonitis ▪ Peptic and duodenal ulcer ▪ Malabsorption, appendicitis, hernias ▪ Hemorrhoids, fissures, fistulas ▪ Pancreas: Inflammation, cysts, and tumors ▪ Liver: Inflammation, cysts, abscess, cirrhosis, portal hypertension, hepatic failure, tumors ▪ Gallbladder: Inflammation, cholelithiasis, tumors • Gastric decompression, gavage and stoma care, different feeding techniques • Alternative therapies, drugs used in treatment of disorders of digestive system	• Lecture, discussion • Demonstration, • Role play • Problem-based learning • Visit to stoma clinic	• Short answer • Quiz • OSCE
VI	20 (T) 5 (L)	• Explain the etiology, pathophysiology, clinical manifestations, diagnostic tests, and medical, surgical, nutritional, and nursing management of cardiovascular disorders • Demonstrate skill in cardiovascular assessment • Prepare patient for invasive and non-invasive cardiac procedures • Demonstrate skill in monitoring and interpreting clinical signs related to cardiac disorders • Complete BLS/BCLS module	**Nursing Management of Patients with Cardiovascular Problems** • Review of anatomy and physiology of cardiovascular system • Nursing assessment: History and physical assessment • Invasive and noninvasive cardiac procedures • Disorders of vascular system: Hypertension, arteriosclerosis, Raynaud's disease, aneurysm and peripheral vascular disorders • Coronary artery diseases: coronary atherosclerosis, angina pectoris, myocardial infarction • Valvular disorders: Congenital and acquired • Rheumatic heart disease: Pericarditis, myocarditis, endocarditis, cardiomyopathies • Cardiac dysrhythmias, heart block • Congestive heart failure, corpulmonale, pulmonary edema, cardiogenic shock, cardiac tamponade • Cardiopulmonary arrest	• Lecture, discussion • Demonstration • Practice session • Case discussion • Health education • Drug book/ presentation • Completion of BCLS module	• Care plan • Drug record • BLS/BCLS evaluation

Contd...

Unit	Time (Hrs)	Learning Outcomes	Content	Teaching/Learning Activities	Assessment Methods
VII	7 (T) 3 (L)	• Explain the etiology, pathophysiology, clinical manifestations, diagnostic tests, and medical, surgical, nutritional, and nursing management of hematological disorders • Interpret blood reports • Prepare and provides health education on blood donation	**Nursing Management of Patients with Disorders of Blood** • Review of Anatomy and Physiology of blood • Nursing assessment: History, physical assessment and diagnostic tests • Anemia, polycythemia • Bleeding disorders: Clotting factor defects and platelets defects, thalassemia, leukemia, leukopenia, agranulocytosis • Lymphomas, myelomas	• Field visit to blood bank • Counseling	• Interpretation of blood reports • Visit report
VIII	8 (T) 2 (L)	• Explain the etiology, pathophysiology, clinical manifestations, diagnostic tests, and medical, surgical, nutritional, and nursing management of endocrine disorders • Demonstrate skill in assessment of endocrine organ dysfunction • Prepare and provides health education on diabetic diet • Demonstrate skill in insulin administration	**Nursing Management of Patients with Disorders of Endocrine System** • Review of anatomy and physiology of endocrine system • Nursing assessment—history and physical assessment • Disorders of thyroid and parathyroid, adrenal and pituitary (hyper, hypo, tumors) • Diabetes mellitus	• Lecture, discussion • Demonstration • Practice session • Case discussion • Health education	• Prepare health education on self-administration of insulin • Submits a diabetic diet plan
IX	8 (T) 2 (L)	• Explain the etiology, pathophysiology, clinical manifestations, diagnostic tests, and medical, surgical, nutritional, and nursing management of disorders of integumentary system • Demonstrate skill in integumentary assessment • Demonstrate skill in medicated bath • Prepare and provide health education on skin care	**Nursing Management of Patients with Disorders of Integumentary System** • Review of anatomy and physiology of skin • Nursing assessment: History and physical assessment • Infection and infestations; dermatitis • Dermatoses; infectious and noninfectious • Acne, allergies, eczema and pemphigus • Psoriasis, malignant melanoma, alopecia • Special therapies, alternative therapies • Drugs used in treatment of disorders of integumentary system	• Lecture, discussion • Demonstration • Practice session • Case discussion	• Drug report • Preparation of home care plan

Contd...

Unit	Time (Hrs)	Learning Outcomes	Content	Teaching/Learning Activities	Assessment Methods
X	16 (T) 4 (L)	• Explain the etiology, pathophysiology, clinical manifestations, diagnostic tests, and medical, surgical, nutritional, and nursing management of musculoskeletal disorders • Demonstrate skill in musculoskeletal assessment • Prepare patient for radiological and nonradiological investigations of musculoskeletal system • Demonstrate skill in crutch walking and splinting • Demonstrate skill in care of patient with replacement surgeries • Prepare and provide health education on bone healing	**Nursing Management of Patients with Musculoskeletal Problems** • Review of anatomy and physiology of the musculoskeletal system • Nursing assessment: History and physical assessment, diagnostic tests • Musculoskeletal trauma: Dislocation, fracture, sprain, strain, contusion, amputation • Musculoskeletal infections and tumors: Osteomyelitis, benign and malignant tumor • Orthopedic modalities: Cast, splint, traction, crutch walking • Musculoskeletal inflammation: Bursitis, synovitis, arthritis • Special therapies, alternative therapies • Metabolic bone disorder: Osteoporosis, osteomalacia and Paget's disease • Spinal column defects and deformities—tumor, prolapsed intervertebral disc, Pott's spine • Rehabilitation, prosthesis • Replacement surgeries	• Lecture/discussion • Demonstration • Case discussion • Health education	• Nursing care plan • Prepare health teaching on care of patient with cast
XI	20 (T) 3 (L)	• Explain the etiology, pathophysiology, clinical manifestations, diagnostic tests, and medical, surgical, nutritional, and nursing management of patients with communicable diseases • Demonstrate skill in barrier and reverse barrier techniques • Demonstrate skill in execution of different isolation protocols	**Nursing Management of Patients with Communicable Diseases** • Overview of infectious diseases, the infectious process • Nursing assessment: History and physical assessment, diagnostic tests • Tuberculosis • Diarrhoeal diseases, hepatitis A-E, Typhoid • Herpes, chicken pox, smallpox, Measles, mumps, influenza • Meningitis • Gas gangrene • Leprosy • Dengue, plague, malaria, chikungunya, swine flu, filariasis • Diphtheria, pertussis, tetanus, poliomyelitis • COVID-19 • Special infection control measures: Notification, isolation, quarantine, immunization	• Lecture, discussion, demonstration • Practice session • Case discussion/ seminar • Health education • Drug book/ presentation • Refer TB control and management module	• Prepares and submits protocol on various isolation techniques

Contents

Volume I

Volume II

Chapter 17 Nursing Management of Patients with Neurological Disorders 1237–1319

Chapter 18 Nursing Management of Patients with Immunological Problems 1321–1352

Chapter 19 Nursing Management of Patients with Oncological Conditions 1353–1421

Chapter 20A Nursing Management of Patients in Emergency Situations 1423–1463

Nursing Management of Patient with Disorders of Endocrine System

8
CHAPTER

Learning Objectives

After studying this chapter, the students will be able to:

- Explain anatomy and physiology of endocrine system.
- Describe techniques of assessment of endocrine glands.
- List the various investigations and tests done to evaluate endocrine disorders.
- Discuss disorders of thyroid gland. Hypothyroidism—its clinical manifestations, diagnostic tests and management.
- Explain hyperthyroidism—incidence, etiology, pathophysiology, clinical manifestations, diagnostic tests.
- Write management of hyperthyroidism—medical, surgical and nursing management.
- Describe disorders parathyroid gland—hyperparathyroidism—its clinical manifestation, diagnostic evaluation and nursing care plan.
- Discuss hypoparathyroidism—etiology, pathophysiology, clinical manifestation and medical nursing management.
- Explain disorders of pancreas—diabetes mellitus—etiology, pathophysiology, clinical manifestation, diagnostic evaluation, management and complications.
- Write about disorders of adrenal gland—adrenal tumor—types, management and nursing care plan.
- List pituitary disorders—like gigantism, acromegaly, dwarfism, acromicria, Simmonds' disease, SIADH, dystrophia adiposogenitalis, diabetes insipidus.
- Describe etiology, pathophysiology, clinical manifestations, diagnostic tests and management of pituitary disorders.

Chapter Outline

- Review of Anatomy and Physiology of Endocrine System
- Assessment and Diagnostic Tests

Disorders of Endocrine System

- Pituitary Gland
- Pituitary Disorders

Disorders of Thyroid Gland

- Hypothyroidism
- Hyperthyroidism (Thyrotoxicosis)
- Thyroiditis
- Goiter

Disorders of Parathyroid Gland

- Parathyroid Disorders

Disorders of Pancreas

- Diabetes Mellitus

Disorders of Adrenal Gland

- Addison's Disease
- Adrenal Tumors
- Tumors of Adrenal Glands

Key Terminology

Acromegaly: It is defined as an endocrine disorder that is characterized by progressive enlargement of body parts due to excessive secretion of growth hormone.

Adrenal gland: It is defined as a pair of suprarenal endocrine gland consists of adrenal cortex and medulla to regulate metabolic pathways.

Aldosterone: It is defined as mineral corticoid hormone secreted by adrenal cortex to promote the absorption of sodium and excretion of potassium by kidneys.

Cortisol: Also called stress hormone and defined as steroid hormone which is produced by adrenal cortex and perform functions of inflammation, immune function and metabolism.

Cushing syndrome: It is defined as a group of symptoms that appear due to excessive secretion of adrenocorticotropic hormone and characterized by features of truncal obesity, moon face, buffalo hump, Hypertension, etc.

Diabetes insipidus: An endocrine disorder occurs due to deficiency of vasopressin and characterized by excretion of large volumes of diluted urine.

Diabetes mellitus: A collection of metabolic illnesses known as diabetes mellitus causes elevated blood glucose level as a result of abnormalities in insulin secretion, insulin action, or both.

Ductless glands: The glands present in the human body which secrete their products directly into the bloodstream and are also called as endocrine glands.

Endocrine: It is defined as simply hormone secretion by a ductless gland in the body or something that is internally secreted.

Gigantism: It is an excessively large growth that occurs in childhood before the growth plates close due to an over secretion of growth hormone.

Glucose tolerance test: It is also known as oral glucose tolerance test, the glucose tolerance test finds abnormalities with how your body processes glucose after eating, frequently before your fasting blood glucose level does.

Glycosylated hemoglobin: Glycosylated hemoglobin level is the indicator of how effectively blood sugar has been managed over a longer period of time, every 2–3 months.

Gonads: A gonad is a reproductive gland. Male gonads are the testes, whereas female gonads are the ovaries.

Hashimoto thyroiditis: The autoimmune thyroid illness Hashimoto thyroiditis is characterized by the destruction of thyroid cells by a variety of cell- and antibody-mediated immunological mechanisms.

Hormone: Defined as special type of messengers consists of chemical transmitters that are secreted by one organ of body and carried to target site by bloodstream.

Hypothalamus: It is defined as part of brain which is located inferior to thalamus, serves and a linking structure between nervous and endocrine system and maintains homeostasis of body.

Insulin: Also called investment hormone and is defined as a peptide hormone produced by β cells of pancreas to regulate glucose levels in blood.

Nephropathy: Long-standing history of diabetes and persistent albuminuria lasting 3–6 months in at least 2 out of 3 consecutive urine collections.

Neuropathy: A descriptive word for a demonstrable illness that is either clinically obvious or subclinical and only manifests in peripheral neuropathy when there is diabetes mellitus present.

Pancreas: It is defined as elongated heterocrine gland which consists of digestive exocrine gland and hormone producing endocrine gland located behind the stomach.

Parathyroid gland: An endocrine gland located in neck and embedded posterior to thyroid gland regulates calcium and phosphorus metabolism.

Pineal gland: Also called as epiphysis gland located above hypothalamus behind 3rd ventricle and produces melatonin to regulate circadian rhythm and influences sleep—wakes cycles.

Pituitary gland: Also called hypophysis or master gland of the endocrine system that is round in structure, located in sella turcica and controlled by hypothalamus.

Radiosensitive iodine: Radiosensitive iodine is radioactive form of iodine that is used for imaging test to treat cancer mainly thyroid cancer.

Retinopathy: It is a retinal disease caused by microangiopathy, a long-term impact of diabetes that results in gradual retinal damage and blindness.

Clinical Skills

1. Assessment of endocrine system
2. RBS monitoring
3. Insulin administration

CASE SCENARIOS RELATED TO NURSING MANAGEMENT OF PATIENT WITH ENDOCRINE DISORDER

CASE 1

A 67-year-old postmenopausal woman whose name was Meena was diagnosed to have T2DM 4 years back and is currently on dosage of metformin. Her glycemic levels for the past few weeks have not been controlled well and she visited doctor. She also diagnosed osteoporosis and she had a fragility fracture recently and is taking calcium, Vitamin D, and bisphosphonates for prevention of further fractures. Meena stays alone in a flat and manages her daily activities by herself.

- Discuss the case history and present complaint.
- What precautions or treatment she has to take according to her current situation?
- Prepare the nursing care plan.

CASE 2

Mrs Sushma, 50-year-old housewife came to emergency department with complains of progressive weight gain of 17 kg in 6 months, she also tells that she is feeling dizziness, fatigue and experience loss of memory, slow speech. Sushma was telling to a nurse in ER that she also has dry skin, constipation, and cold intolerance.

Findings of physical examination are:

- Temperature 96.8°F
- Pulse 58/minute and regular
- BP 110/60.

On examination, nurse found that her weight is 92 kg and has a puffy face, with pale, cool, dry, and thick skin.

- Enlist the signs and symptoms.
- What will the diagnosis and what physical findings supported the diagnosis?
- On the basis of initial assessment, prepare the nursing care plan.

CASE 3

Ms Shreya, 23-year-old was observed in April 2022 complaining of polydipsia, polyuria, nocturia and weight loss since November 2021. Doctors suspect Diabetes Insipidus and therefore Diabetes mellitus (DM) was excluded and she was admitted for study of possible diabetes insipidus for various investigations. Water deprivation test was suggestive of Central Diabetes Insipidus. MRI report showed infundibular hypophysitis and no hyperintense signal in the neurohypophysis. She started her therapy with oral desmopressin with clinical improvement.

- Discuss management of diabetes insipidus.
- On the basis of assessment, prepare the nursing care plan.

REVIEW OF ANATOMY AND PHYSIOLOGY OF ENDOCRINE SYSTEM

The endocrine system consists of organ systems that participate in communication in the human body through the secretion of ductless glands, called hormones. Nervous system communicates through its electrical and chemical signaling between neurons and target cells. In this way, endocrine system and nervous system maintain the homeostasis in the human body. There are two types of glands: endocrine glands and exocrine glands. Major differences between endocrine and exocrine glands are described in Table 8.1.

TABLE 8.1: Differences between endocrine and exocrine glands

Endocrine glands	Exocrine glands
• Secrete hormones into the surrounding fluids, which enters into blood and lymph	• Secrete substances directly to target tissues
• Secrete hormones	• Don't secrete hormones
• Ductless glands	• May or may not have ducts
• Example: Pituitary gland, Thyroid gland, ovaries	• Example: Salivary glands, sweat glands, gastric glands

The hormone is secreted by endocrine gland in the extracellular fluid. From there, hormone diffuses into the bloodstream and is transported throughout the body. The hormone, after reaching to the organ, binds to receptor on target cells and induces its specific responses. The endocrine system regulates growth, reproduction, fluid and electrolyte balance, body metabolism and sexual development. In normal healthy individual, the level of a hormone is constant. If hormone concentration in the body increases, its production is inhibited and if its concentration in the body decreases, body produces more hormone for regulation of its concentration in the bloodstream. This mechanism is called negative and positive **feedback control** respectively**.** The hormone levels can be affected by various factors. The nervous system can control the level of hormones by releasing and inhibiting action of hypothalamus. For example,

- Hypothalamus stimulates anterior pituitary to produce TSH
- TSH stimulates thyroid gland to produce T3 and T4
- Nutrition can also affect the level of hormones. For example, iodine is required for production of thyroid hormones T3 and T4.

Classification of Hormones

Hormones are classified into 2 major categories on the basis of their chemical structure or morphology, as Water-soluble hormones and lipid-soluble hormone (Table 8.2).

TABLE 8.2: Differences between water-soluble and lipid-soluble hormones

Water-soluble hormone	Lipid- soluble hormone
• Hormones are soluble in water • Unable to enter into the cell through phospholipid of the plasma membrane • Binds with the receptors tissue on the surface of cell • **Reaction:** Amino-tri-phosphate synthesized into Amino-mono-phosphate inside the cell • **Hormones:** Insulin, epinephrine, HGH and Oxytocin	• Hormones are soluble in lipids • Passes into the plasma membrane • Bind is directly to receptors into the cell nucleus • **Reaction:** cell growth and function through production of RNAs from DNA of the cell. • **Hormones:** Estrogen, Testosterone, Glucocorticoids

Endocrine Glands

The endocrine glands are:

- Pineal gland
- Hypothalamus and Pituitary gland
- Thyroid gland
- Parathyroid glands
- Pancreas
- Adrenal glands
- Gonads (Ovaries and Testes)

The location of the gland is shown in Figure 8.1.

Pineal Gland

The pineal gland, is located in the center of the head also called the pineal body or epiphysis, hormone secreted by the pineal gland is melatonin, a hormone produced at night and related to the regulation of circadian rhythm (or the circadian cycle, the wakefulness-sleep cycle). Melatonin also regulates body's functions related to the night-day cycle (Fig. 8.2).

Hypothalamus and Pituitary Gland

The pituitary gland, also called as hypophysis, is situated in the skull below the hypothalamus. It is often called "Master Gland" because it produces largest number of hormones that control functions of other endocrine glands of the body (Fig. 8.3).

Pituitary Gland has two parts: Anterior Pituitary (or adenohypophysis) and the posterior pituitary (or neurohypophysis) (Fig. 8.4).

Pituitary gland is connected by the hypophyseal stalk to the hypothalamus. The hypothalamus is situated on the dorsal side of the pituitary gland. The pituitary gland has two parts. The anterior (or front) pituitary produces hormones that affect the

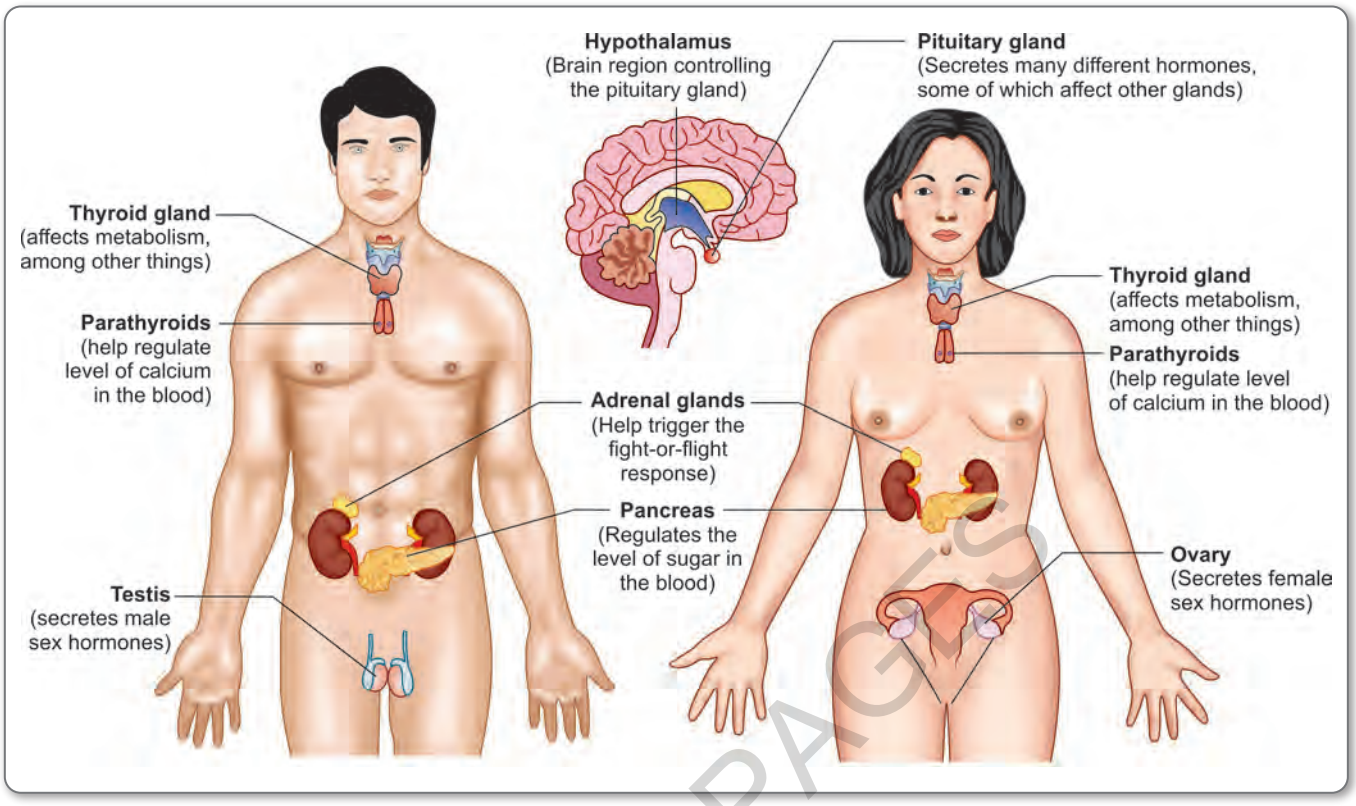

Fig. 8.1: Location of the endocrine glands

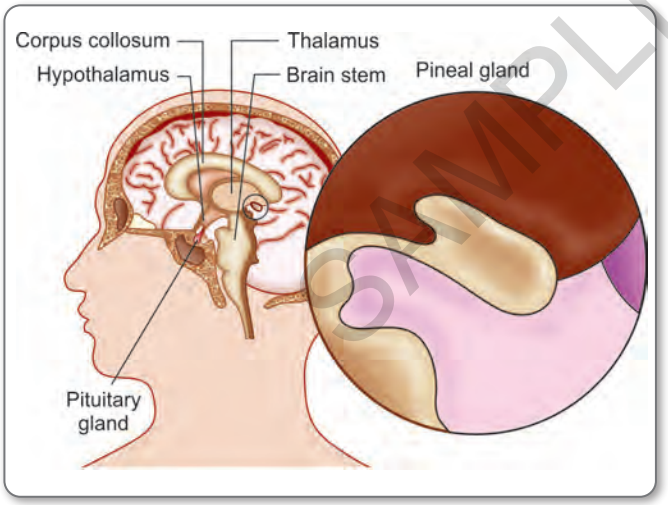

Fig. 8.2: Pineal gland

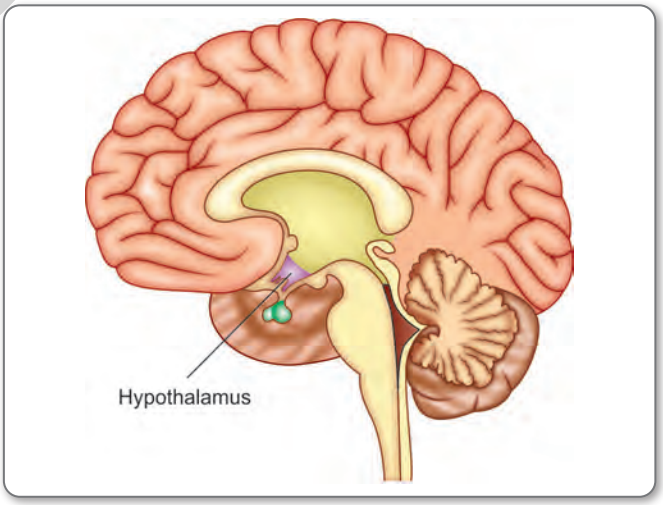

Fig. 8.3: Hypothalamus

breasts, adrenals, thyroid, ovaries and testes, as well as several other hormones. The main glands affected by the posterior (or rear) pituitary are the kidneys. The hormones produced by the anterior pituitary gland are ACTH (adrenocorticotropic hormone), FSH (follicle-stimulating hormone), GH (growth hormone), LH (luteinizing hormone), PRL (prolactin), TSH (thyroid-stimulating hormone. Posterior pituitary gland secretes ADH (antidiuretic hormone, or vasopressin) and Oxytocin. Pituitary hormone secretion (Fig. 8.5) is controlled by hypothalamus by secreting releasing factor.

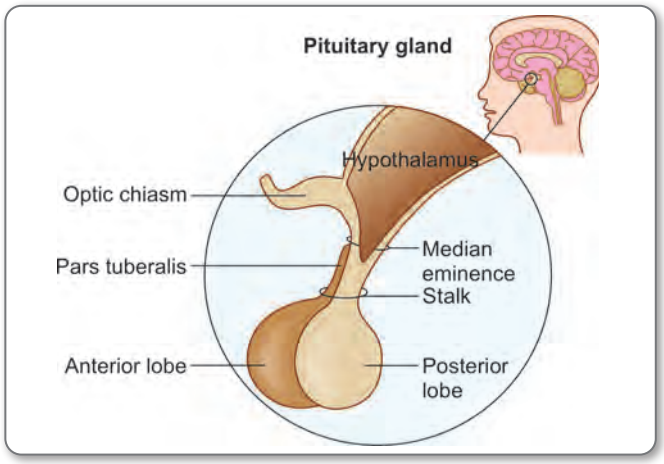

Fig. 8.4: Pituitary gland

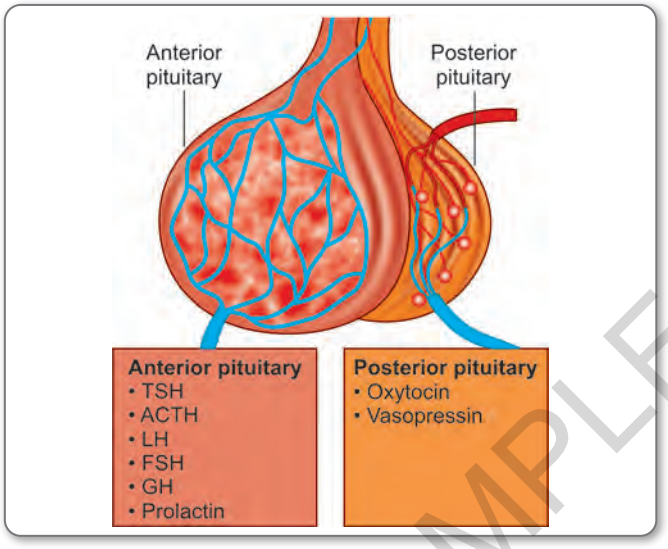

Fig. 8.5: Hormones secreted by pituitary gland

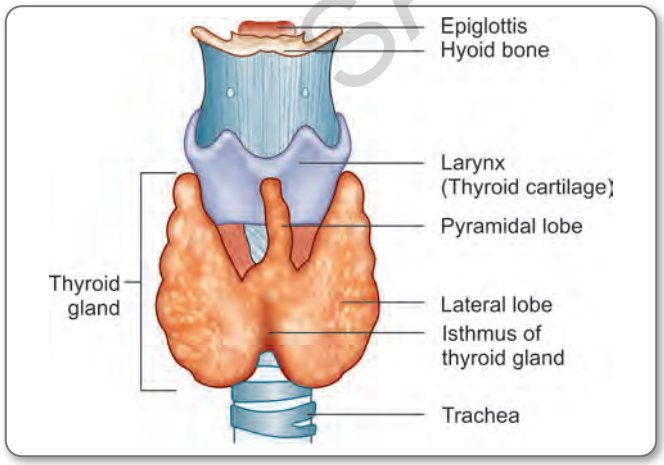

Fig. 8.6: Thyroid gland

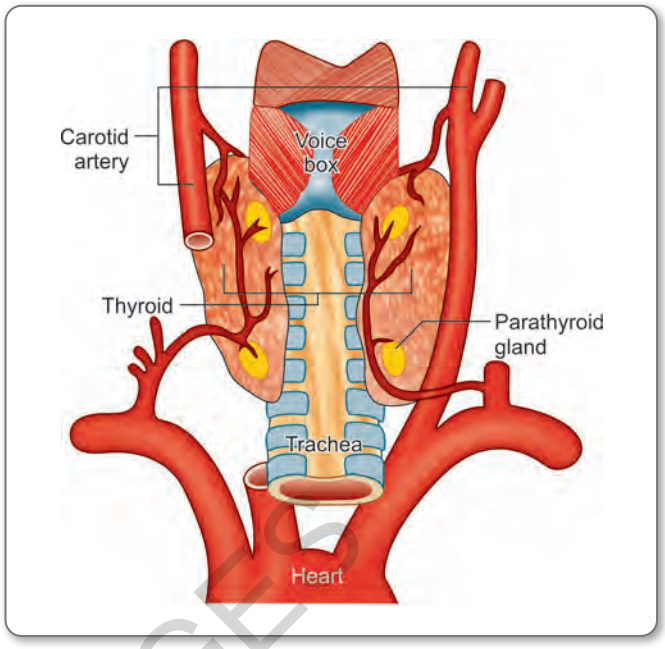

Fig. 8.7: Location of parathyroid gland

Thyroid Gland

The thyroid gland (Fig. 8.6) is located at the anterior and upper part of the trachea. The gland resembles a butterfly in shape. It has two lobes, which are connected by isthmus. Hormones secreted by thyroid gland are called thyroid hormones (TH) and include T3 (triiodothyronine) and T4 (thyroxine). Thyroid hormone increases the metabolism in adults. Calcitonin is also secreted by thyroid gland which decreases excess level of calcium in the body.

Parathyroid Glands

There are four to six parathyroid glands embedded in the posterior lobes of thyroid gland. Hormone secreted by parathyroid gland is called parathyroid hormone (PTH) or parathormone. PTH hormone controls phosphate metabolism. Vitamin D in normal amounts is necessary for PTH effect (Fig. 8.7).

www.parathyroid.com/parathyroid.htm

Pancreas

Pancreas (Fig. 8.8) is situated in the abdomen behind the stomach between spleen and duodenum. It performs both endocrine and exocrine functions. Endocrine cells of pancreas produces hormones (glucagon and insulin) while exocrine secretion includes digestive enzymes (pancreatic polypeptides).

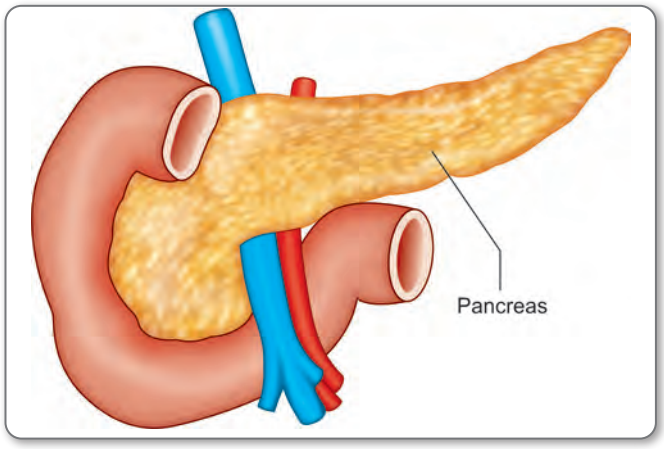

Fig. 8.8: Pancreas

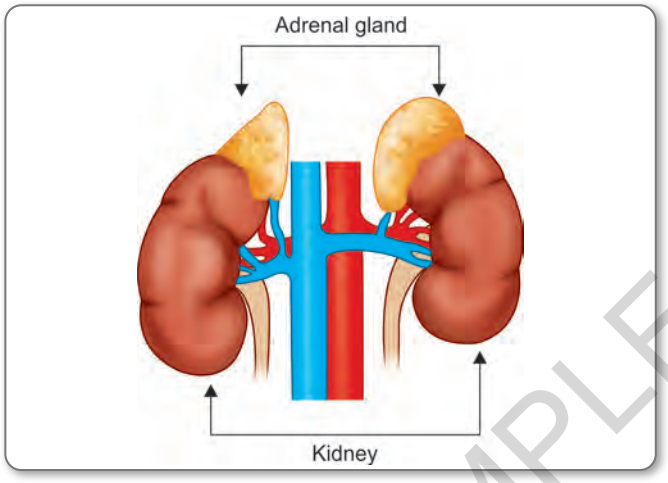

Fig. 8.9: Adrenal gland

Adrenal Glands

Adrenal glands (Fig. 8.9) are located on the upper portion of the kidney. They are two in number. Each gland has two parts; inner part is medulla and outer part is cortex. Adrenal medulla secretes catecholamine and adrenal cortex secretes steroid hormones.

The catecholamine: epinephrine (adrenaline) and nor epinephrine (noradrenaline)

Epinephrine stimulates and pituitary to secrete ACTH. Norepinephrine acts on heart it increases the heart rate and force of cardiac contraction. It also causes vasoconstriction throughout the body.

Gonads

The testes in man and ovaries in female are gonads (Fig. 8.10). Gonads secrete sex hormones, which regulate the growth and promote the onset of puberty. Testis in man produces androgen

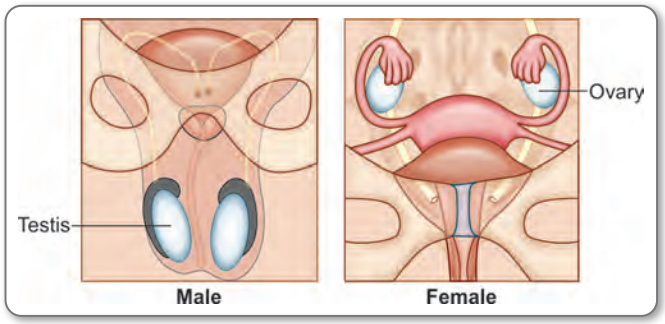

Fig. 8.10: Gonads

(primarily testosterone); in women, ovaries secretes estrogen and progesterone to maintain reproductive functioning.

Evidence Based Practice

A Study conducted by Ed Susman March 07, 2015 revealed— Men with borderline low testosterone levels who are referred to an endocrinologist often show signs of clinical depression or depressive symptoms, researchers reported that out of 200 men in a study, 56% had significant depressive symptoms, had a known diagnosis of depression and/or were using antidepressants when they were seen by Michael Irwig, MD, associate professor of medicine and director of the Center for Andrology at George Washington University, Washington.

ASSESSMENT AND DIAGNOSTIC TESTS

History taking—Nurses must have an adequate knowledge regarding anatomy and physiology of endocrine system, which consists normal function of endocrine glands or their hormones. She will be able to identify the altered functions in the endocrine disorder

Assessment of Endocrine Disorders

History of Past Illness

- Childhood health problems
- Assess an abnormal growth pattern, e.g., head circumference, size of the hand and feet.
- Any history of hair growth on face in women, decreased hair growth in men.
- Take history of immunization.
- History of hospitalization, head trauma (may lead to pituitary dysfunction), past surgical history.

Family History

Family history is important, as many endocrine disorders run in the family. A well collected history will help in diagnosis and management of the disease.

History of exercise, nutrition and habits:

- Assess exercise, food intake, sleep and rest pattern.
- Also explore use of alcohol; alcohol may cause liver and pancreas abnormality.
- Collect demographic data of the client—Age, gender, geographical residence,
- Medical history of some disorders, e.g., Diabetes mellitus, hepatitis, and disorder of gallbladder.

Physical Examination

Nurse will use various methods of physical examination which includes vitals measurement, systematic inspection/head-to-toe inspection, the endocrine glands should be inspected and palpated routinely in all suspected patients. It is important to examine physical, psychological and behavioral changes. Patients suffering with endocrine disorder may exhibit following changes:

Assess clients' chief complaints by system wise assess-ment:

- **Integumentary system**
  - Hyper pigmentation or hypo pigmentation may be due to Addison's disease.

  Changes in hair distribution amount or texture, etc.
- **Cardiovascular system**
  - Circulation (Palpitation, chest pain, dysrhythmias, gallop rhythm, etc.)
  - Kussmaul's respiration (Deep rapid breathing is due to diabetic ketoacidosis.
- **Neurological system**
  - Nervousness, increased irritability, insomnia, fatigue, muscle weakness, etc.
- **Elimination**
  - Polyuria, diarrhea, stool changes anorexia, etc.
  - Food and Fluid (Weight loss/gain, increased thirst, nausea, vomiting, etc.)
- **Musculoskeletal**
  - Generalized weakness
  - Decreased muscle mass
  - Enlargement of bones and cartilage
- **Neurosensory**
  - Speech alteration, mental status changes, etc.
- **Pain**
- **Ego integrity**
  - Anxiety, depression, emotional liabilityetc.
- **Safety**

  Heat/cold intolerance, excessive sweating, exophthalmos, elevated temperature, etc.
- **Reproductive system**
  - Decreased libido, hypomenorrhea, impotence, etc.
  - Fluid deprivation-weight loss/gain
- **Head and Neck**
  - Pituitary gland enlargement or tumor leads to pressure on optic nerve which causes decreased visual acuity.
  - In hyperthyroidism fluid accumulation in eye and retro-orbital tissue can cause eyeball protrusion from orbit.
  - In Cushing Syndrome increased cortical secretion cause moon face and facial fullness
  - In hyperthyroidism patient may have puffiness, periorbital edema,
  - Goiter can be found out due to iodine deficiency

Clinical Manifestation

The clinical manifestation of endocrinal diseases are tabulated as follows (Table 8.3).

TABLE 8.3: Clinical manifestation of Endocrine diseases

- Lethargy
- Muscle hypertonia (Fig. 8.11)

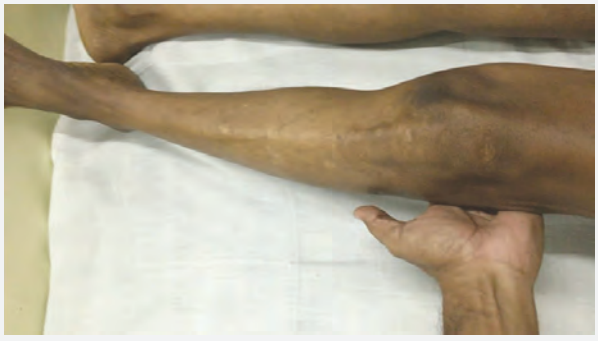

Fig. 8.11: Muscle hypertonia of lower limb

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- Mask-like face (Fig. 8.12)

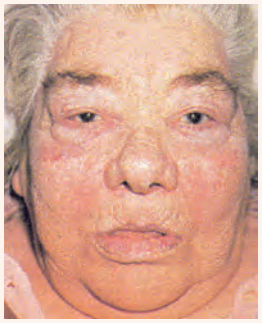

Fig. 8.12: Mask like face as seen in Parkinson's disease

- Vision change (Fig. 8.13)

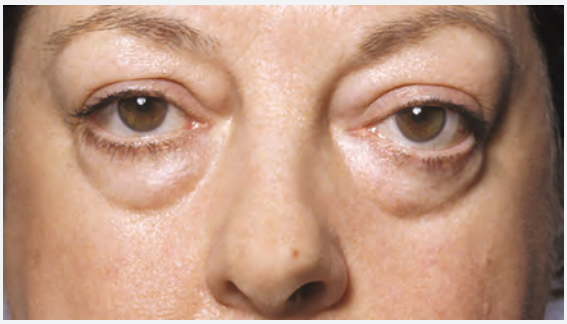

Fig. 8.13: Vision changes in thyroid disease.

- Dull mental process
- Emaciation (Fig. 8.14)

Fig. 8.14: Emaciation

- Exophthalmoses (Fig. 8.15)

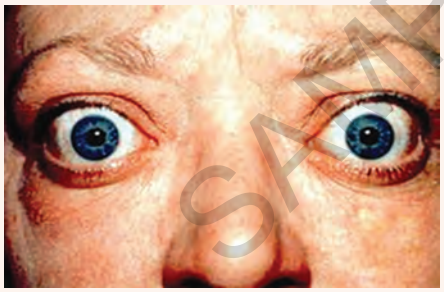

Fig. 8.15: Exophthalmos as seen in Graves disease

- Growth of hair on female face (Fig. 8.16)

Fig. 8.16: Growth of hair on female face

- History of emotional reaction
- Buffalo hump, moon face (Fig. 8.17)

Fig. 8.17: Moon face as evident in Cushing syndrome

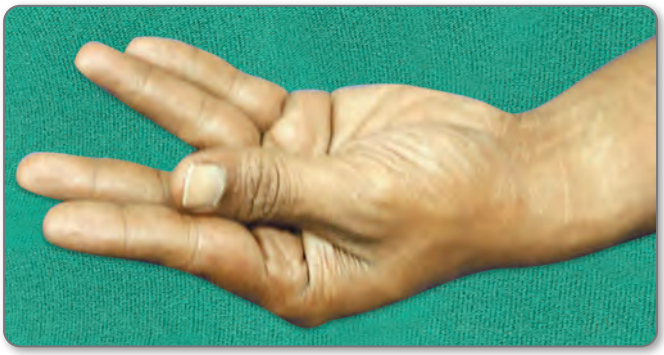

Fig. 8.18: Carpopedal spasm as evident in hypocalcemia

- Measurement of blood calcium, phosphate or serum cortisol level
- Chvostek's sign (the twitching of the facial muscles in response to tapping over the area of the facial nerve)
- Trousseau's sign (carpopedal spasm caused by inflating the blood-pressure cuff to a level above systolic pressure for 3 minutes.) (Fig. 8.18)

Common manifestations during assessment which suggest the endocrine disorders are as heat/cold intolerance, altered energy level, weight gain/loss, growth of hair on female face, alteration in secondary sexual characteristics, vision changes, joint pain, etc. Nurse will document all this collected history in sequence, so that it helps to diagnose the disease (Table 8.4).

Diagnostic Tests

TABLE 8.4: Diagnostic tests to assess endocrine function

Name of the Test	Indication	Normal Value	Patient Preperation
Growth hormone (GH), Human Growth Hormone (hGH)	To assess GH levels to identify GH deficiency (dwarfism) or GH excess (gigantism, acromegaly).	Men: <5 ng/mL Women: <10 ng/mL	Client should not eat or drink anything 8–10 hours prior to having blood drawn. Instruct client to rest for 30 to 60 minutes before blood is drawn
Somatomedin C (Insulin- Like Growth Factor or IGF-1)	This test used to evaluate secretion of growth factor and to identify GH deficiency or excess (as above).	125–250 ng/mL	Fasting previous night of the investigation preferred but not necessary
Water Deprivation Test	This combination of blood and urine test is used to identify causes of polyuria (increased urine output), syndrome of inappropriate antidiuretic hormone, psychogenic polydipsia, ADH or vasopressin is given IM or subcutaneously		Instruct not to smoke, eat or drink after midnight, duration of test will be 8 hours. Every hour assess weight, take postural BP (lying and standing measures separated as ordered) assess urine for volume and specific gravity, and send samples of urine to the lab for osmolality. Blood samples for osmolality are taken when urine samples are collected and when client demonstrates orthostatic hypotension.
Magnetic Resonance Imaging (MRI)	This radiographic study is done to identify tumors of the hypothalamus or pituitary gland.		Explain client that he has to lie still during the examination. Assess for any metallic implants (such as pacemakers, body piercings, shrapnel). If present, test is not performed.
Thyroid–stimulating TSH	In this blood test, TSH and T_4 levels are measured to differentiate pituitary from thyroid causes of hypothyroidism.	<3 ng/mL	Client should not eat shellfish for several days before the test. Evaluate medications: TSH value may be increased by aspirin, steroids, dopamine, and heparin; and decreased by lithium and potassium iodide.
Thyroxine (T_4)	To determine thyroid function and aid in the diagnosis of hyperthyroidism and hypothyroidism.	Free T_4 1.0–2.3 ng/mL	Assess medication: Value of T4 may be altered by chlorpromazine (Thorazine), phenytoin (Dilantin), heparin, lithium, sulfonamides, reserpine (Serpasil), testosterone, propranolol (Inderal), tolbutamide (Orinase), and salicylates in high doses. oral contraceptives, estrogen, clofibrate, and perphenazine (Trilafon).

Contd...

Name of the Test	Indication	Normal Value	Patient Preperation
Triiodothyronine (T_3)	To diagnose hyperthyroidism and to compare T_3 with T_4 for diagnosis of thyroid disorder	80–200 ng/dL	Evaluate medications: Value can be altered by propylthiouracil, methimazole, lithium, phenytoin (Dilantin), propranenololreserpine, large doses of aspirin, steroids, and sulfonamides. Estrogen, progestins, oral contraceptives, T_3 and methadone.
Triiodothyronine Resin Uptake (T_3RU)	This test is an indirect measure of free thyroxine (T_4). The client's blood is mixed with radioactive T_3 and synthetic resin, and the radioactive T_3 will bind with available thyroid-binding globulin sites. In hyperthyroidism there are few binding sites left, more T_3 is taken up by the resin, and a high T_3 resin uptake results. The opposite occurs in hypothyroidism.	25–35% uptake	No special preparation is needed.
Thyroid Antibodies (TA)	To assess thyroid immune disease (Graves disease, chronic thyroiditis, Hashimoto's thyroiditis).	Antithyroglobulin: negative to titer <1:20 Antimicrosomal: negative to titer <1:100	Assess for recent viral infection (which could trigger autoimmune disease).
Radioactive Iodine Uptake (RIA)	This test provides a direct measure of thyroid activity and is useful in evaluating the activity of solitary thyroid nodules. Based on the rationale that the thyroid gland takes up iodine in any form, radioactive iodine is given orally or intravenously, and the thyroid gland uptake is measured with a scanner at several hourly intervals and at 24 hours.	2–4 hours: 3–19% 24 hours: 11–30%	Nothing orally mouth for 6 to 8 hours before the test. Tell clients not to take supplemental iodine several weeks before the test and thyroid medications should be discontinued.
Thyroid scan	This radiologic study evaluates thyroid nodules. Radioactive isotopes are given orally and a scanner is passed over the thyroid to make a graphic record of the radiation emitted.	A normal thyroid scan has a homogenous pattern of radiation with symmetric lobes.	No special preparation is needed.
Parathyroid hormone (PTH)	A blood test done to identify hypoparathyroidism or hyperparathyroidism; also used to monitor response to PTH therapy.	Intact PTH: 11–54 pg/mL	Do not to eat or drink for 8 hours before the test.
Calcium (Ca)	To check for serum calcium excess or deficit in parathyroid and bone disorders; and to monitor calcium levels.	9.0–11.0 mg/dL, 4.5–5.5 mEq/L, or 2.3-2.8 mmol/L (SI units)	Observe for manifestations of tetany, including positive Chvostek's and Trousseau's signs, if hypocalcemia.
Cortisol	To measure amount of total cortisol in the serum and evaluate adrenal cortex function. It is decreased in Addison's disease and hypothyroidism; increased in Cushing's syndrome and hyperthyroidism.	8 a.m. – 10 a.m.: 138–635 nmol 4 p.m.–6 p.m.: 83–359 nmol <100 mcg/24 hours	Instruct client not to eat or drink and to rest for 2 hours before the test. Evaluate medications: Cortisol is decreased by androgens, phenytoin (Dilantin), and increased by oral contraceptives, estrogen, spironolactone (Aldactone) and Triparanol.

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Name of the Test	Indication	Normal Value	Patient Preperation
Aldosterone	To assess a deficit or an excess of aldosterone; and to compare blood and urine levels with other lab data to evaluate overhydration with increased sodium and adrenal malfunction. A 24-hour urine test is considered a more reliable measure of aldosterone than a random aldosterone test.	<16 mcg/dL (fasting) 6-25 mcg/24 hours	Assess diet and lab results: Levels are increased by hyponatremia, hyperkalemia, and a low-salt diet. Values are increased by diuretics, hydralazine (Apresoline), diazoxide (Hyperstat), nitroprusside, and oral contraceptives.
Adrenocorticotropic Hormone (ACTH)	To determine if a decreased plasma level of cortisol is due to adrenal cortex hypofunction or pituitary hypofunction.	7 a.m. – 10 a.m.: 8-80 pg/mL 4 p.m.: 5-30 pg/mL 10 p. m. – 12 p. m.: < 10 pg/mL	Instruct the client that food and fluids may be restricted, and to eat a low-carbohydrate diet for 24 hours prior to the test. Assess medications: ACTH values may be increased by metyrapone, vasopressin and insulin; and decreased by steroids, estrogen amphetamines and alcohol.
ACTH Stimulation	Done to check for pituitary hypofunction. The drug metyrapone (Metopirone) is given to block the production of cortisol, thus causing an increased ACTH secretion. If the ACTH level does not increase, the problem is pituitary insufficiency.		Ask medications as for ACTH test.
ACTH Suppression	Done to check for the origin of the condition. The drug dexamethasone (Decadron) is given to suppress ACTH production. If an extremely high dose is needed, the cause is of pituitary origin; if the plasma cortisol continues to be high with ACTH suppression, the cause could be adrenal cortex hyperfunction (Cushing's syndrome). Normally the plasma cortisol level should double in 1 hour.		Tell client to avoid tea, caffeinated coffee, and chocolates; no other food or fluid restriction is needed. Assess medications: False positives may be caused by phenytoin, barbiturates, meprobamate, and carbamazepine. If dexamethasone causes gastric irritation, milk or antacids may be required.
17-Ketpsteroids	This 24-hour urine test is done to measure metabolites in urine and evaluate adrenal cortex function.	Men: 5-25 mg in 24 hours Women: 5-15 mg in 24 hours	Instruct client how to save urine (urine must contain a preservative and be refrigerated). Assess medications and refer to information about the test. Levels are affected by a variety of drugs; if possible, these should be discontinued for 48 hours before the test. Women cannot have the test while menstruating because blood can cause a false-positive finding.
Computerized Tomography (CT) of the Abdomen	This radiologic study is used to assess the adrenal gland for tumors (including size and metastasis).		Determine if contrast medium will be used; if so, assess client for allergy to iodine (shellfish).
Fasting Blood Sugar	This test of serum or plasma is used to identify or confirm a diagnosis of diabetes mellitus. A finding of greater than 125 mg/dL might indicate diabetes.	Serum/plasma: 70-100 mg/dL	Tell the client not to eat or drink for 12 hours before the test. Do not administer insulin until blood specimen is taken. Assess medications: FBS may be increased by cortisone, diuretics, ACTH, levodopa, epinephrine, anesthetics and phenytoin (Dilantin).

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Name of the Test	Indication	Normal Value	Patient Preperation
Oral Glucose Tolerance Test (OGTT)	If previous fasting blood sugar (FBS) results are elevated or inconsistent, diabetes mellitus should be diagnosed.		Instruct the client to fast for 12 hours prior to the test. Inform the client that they cannot consume any food or liquids during the test, excluding water. Evaluate the medications: Steroids, oral contraceptives, estrogens, thiazide diuretics, and salicylates are medications that may raise OGTT levels. Inform the client that throughout the test, he or she might feel lethargic and perspire; in this case, the nurse should be informed. Although they are typically transient, they could be signs of hyperinsulinism.
Glycosylated Hemoglobin (HbA1c)	The efficacy of treating diabetes mellitus is evaluated using this serum test. The results show the average blood sugar level over a 1 to 4 month period; a high level denotes uncontrolled diabetes mellitus and a higher risk for complications.	5.5–9% of total high	Chronic renal failure, long-term blood loss, and anemias can all lead to decreased levels. Hyperglycemia, alcohol consumption, pregnancy, hemodialysis, and continuous cortisone use can all cause levels to rise.
Computed Tomography (CT) of the abdomen	This radiographic examination is used to find pancreatic cysts or malignancies.		If a contrast medium is given, check for iodine allergies (shellfish).

Desmopressin Response Test

Desmopressin is also known as DDAVP (1-deamino-8-D-arginine Vasopressin). It is a synthetic hormone that temporary boost level of clotting factor in bloodstream. This hormone can be used before any surgery or invasive procedure to stop excessive bleeding.

It also helps to release Von Willebrand factor and factor VIII (clotting factor). Von Willebrand factor is a protein which sticks to the areas of blood vessels that is damaged and helps the bleeding to stop. Less Willebrand factor in the blood cause bleeding for longer period.

Recommendation of Test

The test is carried out for:

- Mild Hemophilia
- VonWillebrand Disease
- Platelet Function Defect

Precautions

It is important to ask the patient before test for:

- Medication for heart problem
- History of high blood pressure
- Pregnancy

Test Procedure

- Before the patient taking DDAVP, nurse must take blood sample to check the factor level and also record blood pressure and pulse
- If readings are normal, give quick injection of DDAVP under skin at upper arm. Patient may feel stinging sensation as the medicine is injected
- After 90 minutes second blood sample should be taken
- After 4 to 6 hours third blood sample should be taken to monitor the increase of factor level in the bloodstream

Side Effects of DDAVP

- Redness on chicks
- Low blood pressure
- Nausea
- Headache
- Allergic reaction at injection site
- Warmth around the face

DAAVP can cause body to retain fluid therefore it is advised to the patient not to drink more than 1 L of fluid during the 24 hours after the injection

DISORDERS OF ENDOCRINE SYSTEM

PITUITARY GLAND

Pituitary gland which is located in the brain is called master gland. It regulates the function of various hormone releasing glands such as thyroid, ovaries, testes and adrenal glands (Fig. 8.19). When the pituitary gland produces an excessive amount or insufficient amount of a certain hormone, pituitary diseases result. A pituitary tumor is the most frequent cause of these conditions.

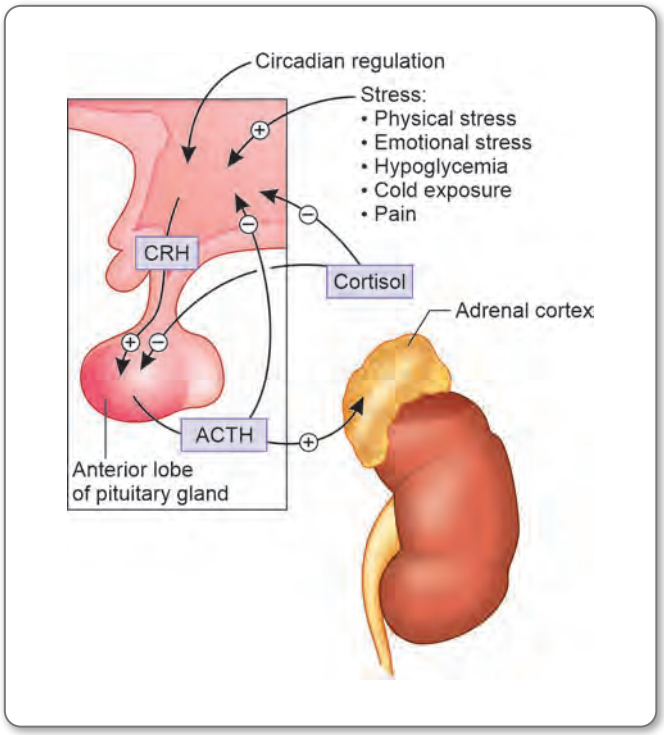


Fig. 8.19: Function of pituitary gland

Causes of Disorder of Pituitary Gland

Pituitary functions may be altered by over secretion or under secretion of the hormones released by the gland. These disorders occur independently in anterior and posterior lobe.

Mainly of two reasons are there:

1. Hyperactivity
2. Hypoactivity

Types of Pituitary Disorders (Table 8.5)

TABLE 8.5: Types of pituitary disorders

Part Involved	Hyperactivity	Hypoactivity
Anterior pituitary	1. Gigantism 2. Acromegaly 3. Acromegalic gigantism 4. Cushing's Syndrome	1. Dwarfism 2. Acromicria 3. Simmonds' disease
Posterior pituitary	Syndrome of Inappropriate ADH (SIADH)	Diabetes insipidus
Anterior and posterior pituitary	—	Dystrophia adiposogenital

PITUITARY DISORDERS

Simmonds' Disease

In 1939, the German Medical Society proposed renaming anterior pituitary deficiency Simmonds Disease after Maurice Simmonds. Pituitary cachexia is another name for Simmonds' disease. It is attributed to hypophysis (anterior portion of pituitary gland) destruction or physiology exhaustion. Tumor, embolic infarction, tuberculosis, and syphilis cause destruction. Simmonds' disease symptoms include premature senility, gonad and genital atrophy, amenorrhea, breast atrophy, loss of pubic hair, loss of libido, constipation, hypotension, and muscular weakness.

SIADH

The syndrome of inappropriate antidiuretic hormone secretion (SIADH) occurs when the body produces excessive levels of antidiuretic hormones, causing the body to retain water and sodium in the blood.

SIADH is most commonly caused by hypothalamic disease such as cancer, meningitis (inflammation of the meninges, the membranes that cover the brain and spinal cord), encephalitis (brain inflammation), brain tumors, psychosis, lung diseases, head trauma, Guillain-Barré syndrome, and certain medications, as well as damage to the hypothalamus or pituitary gland during surgery (Fig. 8.20). In more severe cases of SIADH, the following symptoms may occur: nausea or vomiting, cramps or tremors, depression, memory impairment, irritability, personality changes such as combativeness, confusion, and hallucinations, seizures, and stupor or coma. A complete medical history and physical examination, as well as blood and urine tests to measure sodium, potassium, and osmolality (concentration of solution in the blood and urine), will be required to confirm SIADH. Specific treatments for SIADH include medications that inhibit the action of ADH (also known as vasopressin), surgical removal of an ADH-producing tumor, and other medications to help regulate body fluid volume.

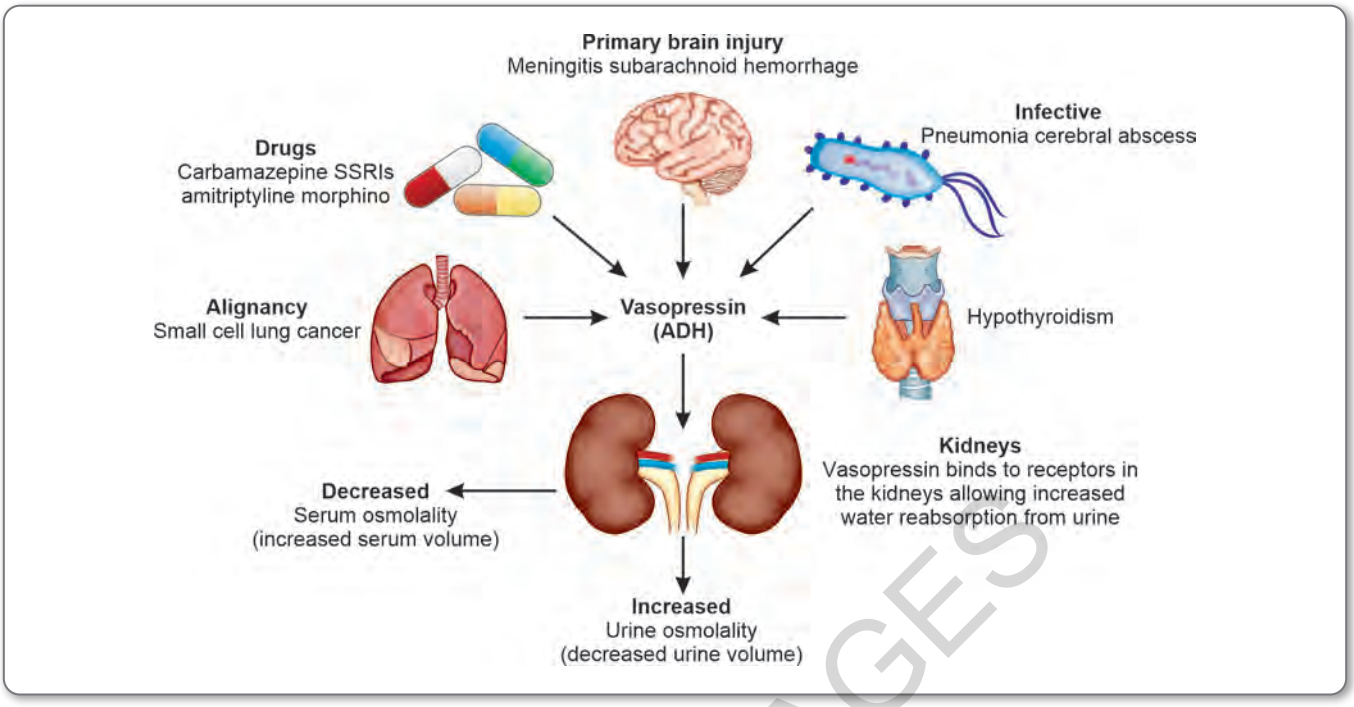


Fig. 8.20: Causative factors associated with SIADH

Pathophysiology

Pathophysiology of SIADH has been depicted in Figure 8.21.

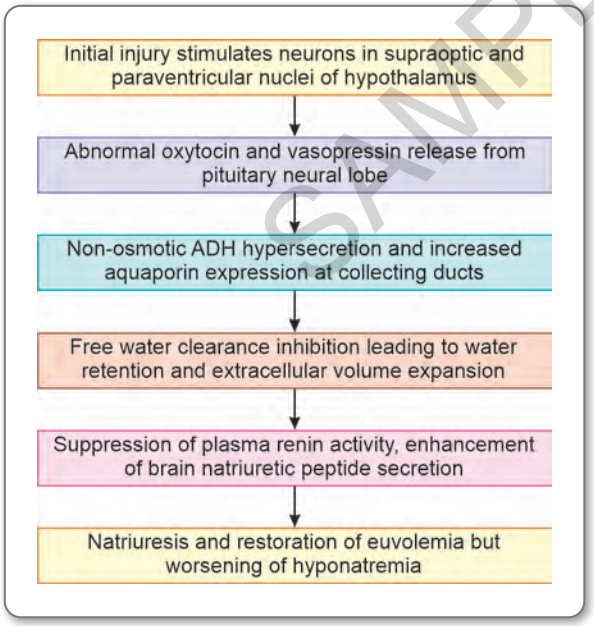


Fig. 8.21: Schematic diagram explaining pathophysiology of SIADH

Management of SIADH (Figs 8.22 and 8.23)

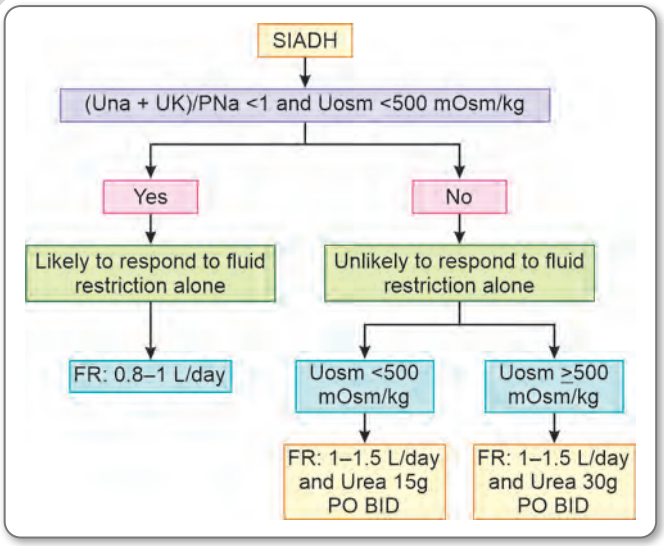


Fig. 8.22: Flowchart explaining management of SIADH

Indirect Modalities

- Fluid restriction
- Treatment of underlying pathology

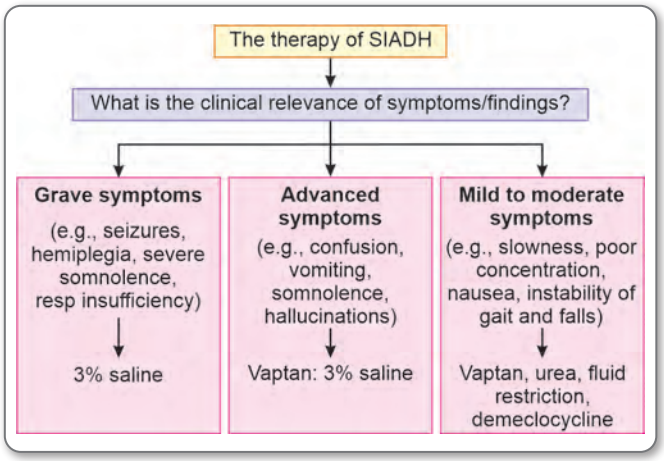


Fig. 8.23: Recommended fluid therapy for SIADH

- Hypertonic saline
- Loop Diuretics
- Urea
- Demeclocycline, lithium
- Hemodialysis, CVVH, SLEDD

Direct Modalities

- **Vaptan treatment:** Vaptans are a new class of pharmaceuticals that have been developed to treat hypervolemic and euvolemic hyponatremia. These medications are nonpeptide vasopressin antagonists that compete with V2 receptors in the kidney to inhibit the hormone's antidiuretic effect. This blockage causes water diuresis (aquaresis), which reduces body water content and raises plasma sodium levels if not offset by increased fluid intake. As a result of this increase in plasma sodium, thirst and plasma vasopressin concentrations rise, ostensibly limiting the effects of vasopressin antagonists. Nonetheless, vaptans are especially useful in treating hypervolemic hyponatremia caused by severe congestive heart failure or chronic liver failure, because the only other treatments currently available, such as fluid restriction and diuretics, are slow-acting and ineffective. Vaptans can also be used to treat euvolemic hyponatremia caused by the syndrome of inappropriate antidiuretic hormone (SIADH), especially when it is chronic and/or symptomatic. Vaptans, on the other hand, are less useful than hypertonic saline infusion in cases of acute, severe, and symptomatic hyponatremia because their effects vary unpredictably from patient to patient. Vaptan therapy is completely contraindicated in hypovolemic hyponatremia (low total body water) and in the vasopressin-independent form of inappropriate antidiuresis caused by constitutive activating V2 receptor mutations.

Anterior Pituitary Disorders

Gigantism and Acromegaly

Gigantism and acromegaly are the disorders of abnormally hypersecretion of growth hormone. Tall stature is a hallmark of gigantism, which develops when growth hormone hypersecretion takes place prior to the union of the long bone epiphysis. Acromegaly develops when the epiphysis fuses and GH hypersecretion continues, resulting in large extremities and a distinctive facial appearance (Fig. 8.24).

Etiology and Risk Factors

- Pituitary adenomas that secrete growth hormone are the primary cause of acromegaly and gigantism in around 95% of cases.
- Acromegaly can be due to an ectopic secretion of growth hormone-releasing hormone (GHRH) release from neuroendocrine tumors in the pancreas or lungs.
- Acromegaly can occasionally be caused due to an ectopic growth hormone release by abdominal and hemopoietic cancers.
- The genetic syndromes such as multiple endocrine neoplasia-1 (MEN-1), neurofibromatosis are linked to gigantism and acromegaly.

Clinical Manifestations

- Enlargement of the hands and feet is seen due to the bony expansion and soft tissue swelling.
- Hyperhidrosis and skin tags are present in about 98% of cases of acromegaly. Skin tags are due to the epithelial cell hyperproliferation induced by GH.
- Acromegalic facies: Prominent supraorbital ridge, broad nose, acne, large lips, overbite, prognathism, tongue enlargement, and coarsening of facial features form the characteristic acromegalic facies.

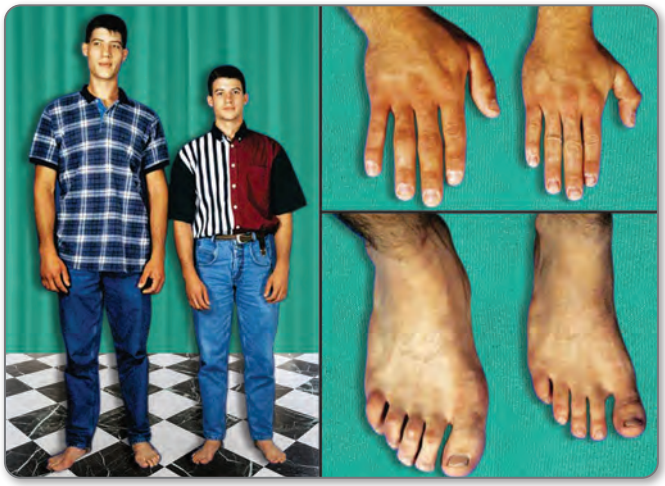


Fig. 8.24: Pituitary gigantism

- Generalized weakness and lethargy are common symptoms.
- Elongation of the jaw can lead to teeth malocclusion and temporomandibular joint pain.
- Abnormal curvature of spine from side to side and from front to back also known as kyphoscoliosis.
- Carpal tunnel syndrome due to the compression of median nerve by overgrown tissues.
- Delayed puberty or hypogonadotropic hypogonadism is also evident.
- Also, the heart, liver, kidneys, spleen, thyroid gland, parathyroid glands, colon, and pancreas are larger than normal.

Diagnostic Investigations

- A thorough clinical examination, a detailed patient history and specialized procedures like blood tests, a glucose tolerance test, magnetic resonance imaging (MRI), or computed tomography are used to confirm the diagnosis.
- Cortical thickening, enlargement of the frontal sinuses, and enlargement and erosion of the sella turcica are evident on CT, MRI, or skull x-rays.
- X-rays hands shows tufting, and the soft tissue thickening.
- Patients with suspected acromegaly should undergo monitoring for serum Insulin growth factor-1 levels, because they are often markedly high (3-fold to 10-fold).
- Normal plasma GH levels are high. Blood should be drawn before the patient consumes breakfast (basal state).
- Additional testing can be done, such as echocardiography for determining the involvement of heart.
- Prolactin levels should be checked, especially if the patient presents with galactorrhea or hypogonadism.

Management

Surgery, pharmacological management, and radiation therapy are the three main therapeutic modalities for acromegaly.

Surgical Management

Transsphenoidal surgery is typically used to treat acromegaly, which involves removing all or part of a pituitary tumor. Surgery frequently produces a quick therapeutic response, alleviating pressure on nearby brain structures immediately and reducing growth hormone levels. Surgery is more successful for people with microadenomas than it is for people with larger tumors (macroadenomas).

As long as the tumor is still accessible, surgery is also the treatment of choice in the event of a recurrence.

Diabetes insipidus and deficits in the anterior pituitary are examples of post-operative consequences.

Pharmacological Therapy

Acromegaly patients are frequently treated with three major kinds of drugs. These include dopamine antagonists, growth hormone receptor antagonists, such as pegvisomant, and analogs of the somatostatin hormone, such as octreotide, lanreotide, and pasireotide.

Radiation Therapy

Radiation therapy is used as a treatment for the patients in whom the surgery was ineffective to lower the hormone levels or tumor size. It is administered over the course of four to six weeks and daily treatments are often required for conventional fractionated radiation therapy.

Complications

The following complications arise due to disease conditions or treatment modalities:

- Cardiomyopathy
- Hypertension
- Obstructive sleep apnea
- Hypopituitarism
- Arthropathy
- Colon polyps

Cushing Syndrome

Cushing syndrome is a hormonal disorder results from excessive or high level of adrenocorticol activity. Cushing syndrome is also known as "Hypercortisolism" commonly caused by over use or continuous use of corticosteroid medications which lead to overproduction of corticosteroids from adrenal gland. Functions of cortisol

- Maintain Blood Pressure
- Maintain blood sugar level by balancing the effect of insulin
- Lower inflammation
- Convert food into energy
- Responding to stress

Cushing syndrome is more common in women than men. Sometimes malignancies such as bronchogenic carcinoma can cause Cushing Syndrome through ectopic production of ACTH.

Causes and Risk Factors

Over production of cortisol in the body causes Cushing Syndrome.

- Long-term use for corticosteroids medications such as prednisone

- Malnutrition
- Alcoholism
- Depression
- Panic disorders
- Athletic training
- High stress level
- Tumors produces too much ACTH such as – Pituitary gland tumors, ectopic tumors and adrenal gland tumors

Pathophysiology of Cushing Syndrome

When activated by ACTH, the adrenal gland releases cortisol and other steroid hormones as shown in Figure 8.25. In response to stimulation by corticotropin-releasing hormone (CRH) from the hypothalamus, the pituitary gland produces and releases ACTH into the petrosal venous sinuses. Just before waking up, ACTH is released at its peak, and throughout the day, levels decrease. Cortisol's negative feedback mechanism keeps CRH and ACTH secretion under control.

Cushing's disease adenomas secrete excessive amounts of ACTH and retain some negative feedback responsiveness to high glucocorticoid doses.

Symptoms

- Weight gain, especially in upper body parts
- Mood shaped face
- Rounded, puffy face
- Thinning of skin
- Tiredness
- Fatigue
- Weakness

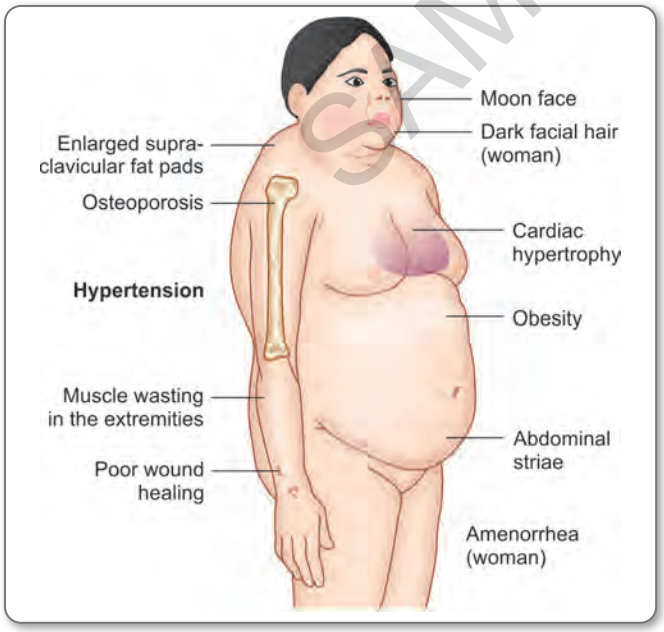


Fig. 8.25: Cushing's syndrome

- Acne
- Buffalo Hump (fatty deposits between the shoulder and upper back)
- High Blood sugar level
- High Blood pressure level
- Osteoporosis
- Depression
- Sleep Problem

Additional symptoms are

In children

- Obesity
- Slow Growth
- High Blood Pressure

In women

- Develop extra facial and body hair
- Irregular menstruation
- Decreased fertility

In men

- Erectile dysfunction
- Low sex drive or interest
- Decreased fertility

Diagnostic Investigations

- **Physical examination and history:** Sometimes Cushing Syndrome can be difficult to diagnose by physical examination itself, therefore history collection is must. Nurse should ask the following questions
  - What symptoms have you experienced or notice?
  - When did you first notice them?
  - Does anything cause symptoms worst or better?
  - What medications are you taking?
- **Laboratory test**
  - **24-hours urinary free cortisol test:** In this test patient will be asked to collect urine for 24 hours to measure the level of cortisol.
  - **Salivary cortisol level:** In normal person physiologically cortisol level drops in the evening. This test measures the level of cortisol in saliva. The saliva sample must be collected in the night to see if the levels are high or low.
  - **Dexamethasone suppression test:** In this test a low dose steroid (Dexamethsone) pill will be given to the patient at late night. The blood will be tested in the next morning to check the level of cortisol. Dexamethasone drops the cortisol level. In Cushing Syndrome patient, it won't occur.
  - **Blood ACTH (Adrenocorticotropin Hormone) Test**: To confirm the tumor is the cause for Cushing Syndrome a blood test is recommended to check the level of ACTH. Low level of ACTH indicates an

adrenal tumor is likely cause and high level of ACTH indicates pituitary or ectopic tumor

- **Petrosal sinus sampling:** In this test blood samples takes from a vein near the pituitary and also from a vein away from pituitary. Patient will get a shot of corticotrophin-releasing Hormone (CRH). High level of ACTH in blood sample taken from near the pituitary vein indicates a pituitary tumor and similar level of ACTH from both the veins sample indicates an ectopic tumor.

- **Imaging studies:**
  - To visualize the tumors in the adrenal and pituitary glands imaging test like CT scan and MRI can be done.

Treatment

The treatment of Cushing Syndrome depends upon the cause of excessive or high level of cortisol.

Elaborate Treatment of Cushing syndrome

The goal of Cushing syndrome treatment is to reduce the body's high cortisol levels. The treatment options are determined by the causative factors.

Reducing Corticosteroid Use

The dosage of the medication will be gradually decreased over time if prolonged usage of corticosteroids is the cause of Cushing syndrome. Patients are not allowed to stop or reduce the dosage of corticosteroid medications by themselves. It is always advisable to gradually taper off the corticosteroid medications, as it enables the body to produce cortisol normally.

Surgery

If a tumor is the cause of Cushing syndrome, surgical removal of the tumor or removal of the adrenal gland, known as "adrenalectomy," is recommended.

Radiation Therapy

Radiation therapy is recommended for a patient whose tumor cannot be completely removed, Patient who is not surgically suitable.

Radiation can be administered in small doses using a technique known as stereotactic radiosurgery. The latter procedure involves delivering a large, one-time dose of radiation to the tumor.

Medications

Cortisol production is controlled with medications.

In people who have become very sick with Cushing syndrome, medications are sometimes used before surgery to improve signs and symptoms and reduce surgical risk. There are medications available to control excessive cortisol production by the adrenal gland. Some of which are included below:

- Ketoconazole
- Mitotane (Lysodren)
- Metyrapone (Metopirone)
- Pasireotide (Signifor)
- Osilodrostat (Isturisa)
- Mifepristone (Korlym, Mifeprex) for patient who have type 2 diabetes or glucose intolerance.

These medications can cause fatigue, nausea, vomiting, headaches, muscle aches, high blood pressure, low potassium, and swelling as side effects.

Lifestyle Modification

Dietary component is an important component of keeping cortisol levels at normal and to prevent the complications of Cushing Syndrome

- **Restrict calorie intake:** Weight gain is the main symptom of Cushing Syndrome, Have food which does not increase weight
  - *Avoid drinking alcohol:* Alcohol consumption cause increased level of cortisol
  - *Blood sugar monitoring:* Cushing Syndrome condition may lead to high blood sugar level
  - *Low sodium intake*: Hypertension or High Blood pressure is a symptom of Cushing Syndrome
- **Take enough calcium and vitamin D:** Cushing Syndrome can weaken the bones which can lead to have patient more prone to fractures

Complications

- Frequent Infections
- Heart Attack
- Stroke
- Hypertension
- Depression
- Enlargement of Tumor
- Type 2 Diabetes
- Cognitive Difficulties
- Bone Loss or Fracture

Dwarfism

The term "dwarfism" in medicine refers to small stature. It is defined as height-vertex below two standard deviations (–2 SD) or in the third percentile for a given age and sex.

As the term suggests, proportional short stature (PSS) refers to a person's proportionately short limbs and trunk. Disproportionately short stature (DSS), on the other hand,

denotes that the person's trunk or extremities are small and there is a big disparity between their sitting and standing heights.

Etiology

Either an underlying medical condition or factors leading to abnormal growth can account for the short stature. Dwarfism has several pathological causes, including:

- Familial Short Stature (FSS): Familial short stature can be identified by a favorable family history and the absence of underlying pathological causes of dwarfism.
- This pattern of small stature depends on heredity, malnutrition during pregnancy or childhood, or both.
- Deficiency of growth hormone: Lack of growth hormone is a common contributor to dwarfism. GH stimulates bone elongation and the growth of soft tissue and cartilage, its deficiency causes dwarfism.
- Achondroplasia is an autosomal dominant genetic disorder, which occurs due to the mutation in the Fibroblast growth factor receptor-3 (FGFR-3) gene. It limits the conversion of cartilage to bone.
- Genetic diseases such as Down's, Turner's, Prader-Willi syndrome can also lead to short stature
- Systemic diseases: Undernutrition, juvenile idiopathic arthritis, inflammatory bowel disease (IBD), celiac disease, chronic kidney disease (CKD) are some systemic conditions that have a secondary impact on growth.

Clinical Manifestations

- Stunted skeletal growth, marked by maximum height approximately 3 feet
- Head becomes slightly larger in relation to the body, and flat nasal bridge is evident.
- Short and wide hands and feet
- Mental activity is normal without any abnormality
- Bowing of legs that progressively worsen over time.

Extra Edge

Simmonds' Disease

Simmonds' disease is a term used to describe panhypopituitarism that results from the destruction of pituitary gland by tumors, infiltrative processes (such as lymphocytic), or trauma.

Simmonds' disease has a subclassification called Sheehan's syndrome that only occurs during the peripartum stage.

Disorders of Posterior Pituitary Gland

Posterior pituitary gland secretes the hormone like antidiuretic hormone (ADH), disorder may be there if ADH secretion is increased or decreased. Destruction of posterior pituitary gland by any disease is rare, if the gland is surgically removed, then also there may not be deficiency of hormone because hypothalamus continues to synthesize oxytocin and ADH.

Disorder of posterior lobe is known as syndrome of inappropriate ADH secretion and diabetes insipidus.

Syndrome of Inappropriate Hypersecretion of Antidiuretic Hormone

Syndrome of inappropriate antidiuretic hormone secretion (SIADH) is characterized by persistent production or activity of arginine vasopressin (AVP) in the presence of normal or elevated plasma volume.

In hospitalized patients, SIADH is the most frequent cause of euvolemic hyponatremia.

Etiology and Risk Factors

- Ectopic production of ADH as seen in brain related diseases such as stroke, head injury and meningitis
- **Drug-induced:** Some drugs of class antidepressants, anticonvulsants, antipsychotic have propensity to cause SIADH.
- **Neoplastic disorders:** Tumors of lungs (meosthelioma, small cell lung cancer), Gastrointestinal (pancreatic cancer, duodenal cancer), and genitourinary system (cancer cervix) can cause SIADH.
- **Pulmonary disease:** pneumonia, TB and cancer also increases the risk of SIADH.
- **Surgery:** ADH hypersecretion is frequently linked to surgical procedures.

Note: Mnemonic for remembering causes of SIADH

Mnemonic

MAD CHOP
M – Major Surgery
A – ADH production by tumors (Ectopic)
D – Drugs (antidepressants, psychotropics)
C – CNS disorders
H – Hormone deficiency
O – Others (Guillain-Barre Syndrome, HIV)
P – Pulmonary disorders (TB, Pneumonia)

Pathophysiology

ADH maintains plasma tonicity by altering the water balance. The Osmoreceptors in the hypothalamus detects change in the plasma osmolality. A decrease in tonicity inhibits ADH release and subsequent water retention. And, ADH release is stimulated when tonicity rises,

In SIADH, levels of ADH are high even in the presence of decreased plasma osmolality or hyponatremia, which causes excessive water absorption leading to increased blood volume.

Clinical Manifestations

Hyponatremia and decreased ECF osmolality can induce water to move into the cells, leading to cerebral edema.

- Nausea and malaise are some of the first clinical signs of acute hyponatremia, which occurs when the serum sodium concentration falls below 125 to 130 mEq/L,
- Headache, drowsiness, obtundation, and eventually seizures can develop with a more severe and sudden reduction in sodium concentration.
- If the serum sodium level drops below 115 to 120 mEq/L, coma and respiratory arrest may occur.
- Chronic hyponatremia causes the brain to adapt, and the individuals remain asymptomatic even when their serum salt level falls below 120 mmol/L. Chronic hyponatremia can lead to various non-specific symptoms such as: cramping, nausea, vomiting, abnormal gait, memory loss, and cognitive difficulties.

Diagnostic Investigations

- **History taking:** Patient should be enquired about past head injuries, smoking, weight gain, pulmonary symptoms, drug use, or substance abuse (particularly heroin).
- **Physical examination:** Physical examination should include checking intake-output, blood pressure and skin turgor. Euvolemia is often indicated by moist mucous membranes without jugular venous pulsation or edema.

Investigations for SIADH

- **Serum osmolality and serum sodium:** Hyponatremia (i.e., serum Na^+ <135 mmol/L) is a defining feature of SIADH.
- **Urinary osmolality:** The urine osmolality is typically submaximally diluted (i.e., >100 mOsm/kg) in SIADH patients.
- **Imaging studies:** Chest radiography can reveal an underlying pulmonary etiology of SIADH. In some cases, A head MRI or CT scan may reveal the signs of cerebral edema or other cerebral conditions such as brain tumor.

Complications

- Brain Herniation
- Decreased Consciousness

Management

Treatment for SIADH includes correcting underlying problems such hypothyroidism, lung infection, or CNS infection as well as maintaining appropriate salt levels. More than 130 mEq/L is the desired sodium correction level.

Emergent Care

- For people with moderate-to-severe hyponatremia that has a recorded duration of less than 48 hours, urgent therapy should be carefully considered.
- Serum Na^+ levels should be maintained at a maximum level of 125–130 mEq/L, by raising serum sodium by 0.5–1 mEq/h.
- Along with treating hyponatremia, caution should be taken to avoid developing severe hypokalemia.

Acute Setting

Patients presenting with moderate symptoms such as confusion, delirium, disorientation, nausea, and vomiting, should be treated with 3% hypertonic saline, loop diuretics with saline, vasopressin-2 receptor antagonists, and water restriction.

Chronic Setting

Fluid restriction and Vasopressin receptor antagonists are the main treatment choices for asymptomatic patients with persistent SIADH. Other therapy approaches include chronic loop diuretics with increased salt intake, urea, mannitol, and demeclocycline.

Diabetes Insipidus

Diabetes insipidus (DI) is characterized by the passing of significant amounts of diluted urine (>3 L/24 h). It is primarily caused due to the deficiency in arginine vasopressin.

There are two types of diabetes insipidus, i.e., central and nephrogenic diabetes insipidus.

Nephrogenic DI is caused by kidney resistance to the hormone arginine vasopressin (AVP), whereas central DI is caused by a lack of AVP hormone in the pituitary gland or the hypothalamus.

Etiology and Risk Factors

- Malignant and benign tumor of brain such as craniopharyngiomas
- Head injury or cranial surgery
- Subarachnoid hemorrhage
- Hypoxic brain injury
- Multiple drug treatments, including lithium, antibiotics, antifungals, and antineoplastic agents.
- Familial and hereditary inheritance as evident in case of nephrogenic DI.

Pathophysiology

Central DI

Traumatic or pathological injuries to the posterior pituitary gland or hypothalamus results in damage to hormone-

secreting cells in those regions, which interferes with the normal secretion and release of ADH. A diuretic phenomenon results from the renal collecting ducts' loss of capacity to accomplish enough water reabsorption, which is necessary for maintaining the body's volume.

Nephrogenic DI

The malfunction of the kidneys' ADH receptors is the cause of nephrogenic DI. AVPR1 and AVPR2 are two receptors that often react to rising ADH levels in the bloodstream. Vasoconstriction and prostaglandin release are mediated by AVPR1, whereas antidiuretic response and some coagulation factors (factor VIII and von Willebrand's factor) are mediated by AVPR2 receptors.

Clinical Manifestations (Fig. 8.26)

- The three main symptoms of diabetes insipidus are polyuria, polydipsia, and nocturia. The daily urine volume varies greatly from patient to patient, ranging from 3 to 20 L, but it is generally consistent for each patient.
- Children may have severe dehydration, constipation, vomiting, fever, irritability, failure to thrive, and growth retardation as nonspecific symptoms.
- Patients with diabetes insipidus may also experience weakness, lethargy, fatigue and myalgias.
- The findings of the exam may be completely normal.

Note: Mnemonic for manifestations of DI

Mnemonic

DDD
D – Diabetes insipidus
D – Deficient ADH (Central DI) or ADH Doesn't work (Nephrogenic DI)
D – Dilute urine (<300 mOsm/L)

Polyuria

- Excretion of large quantity of dilute urine with increased frequency of voiding is called polyuria. Daily output is 4 to 12 liters.
- Due to absence of ADH, the epithelial cells of distal convoluted tubule in the nephron and the collecting duct of the kidney becomes impermeable to water

Polydipsia

Intake of excess water. Because of polyuria, thirst center in hypothalamus stimulate which results in intake of large quantity of water.

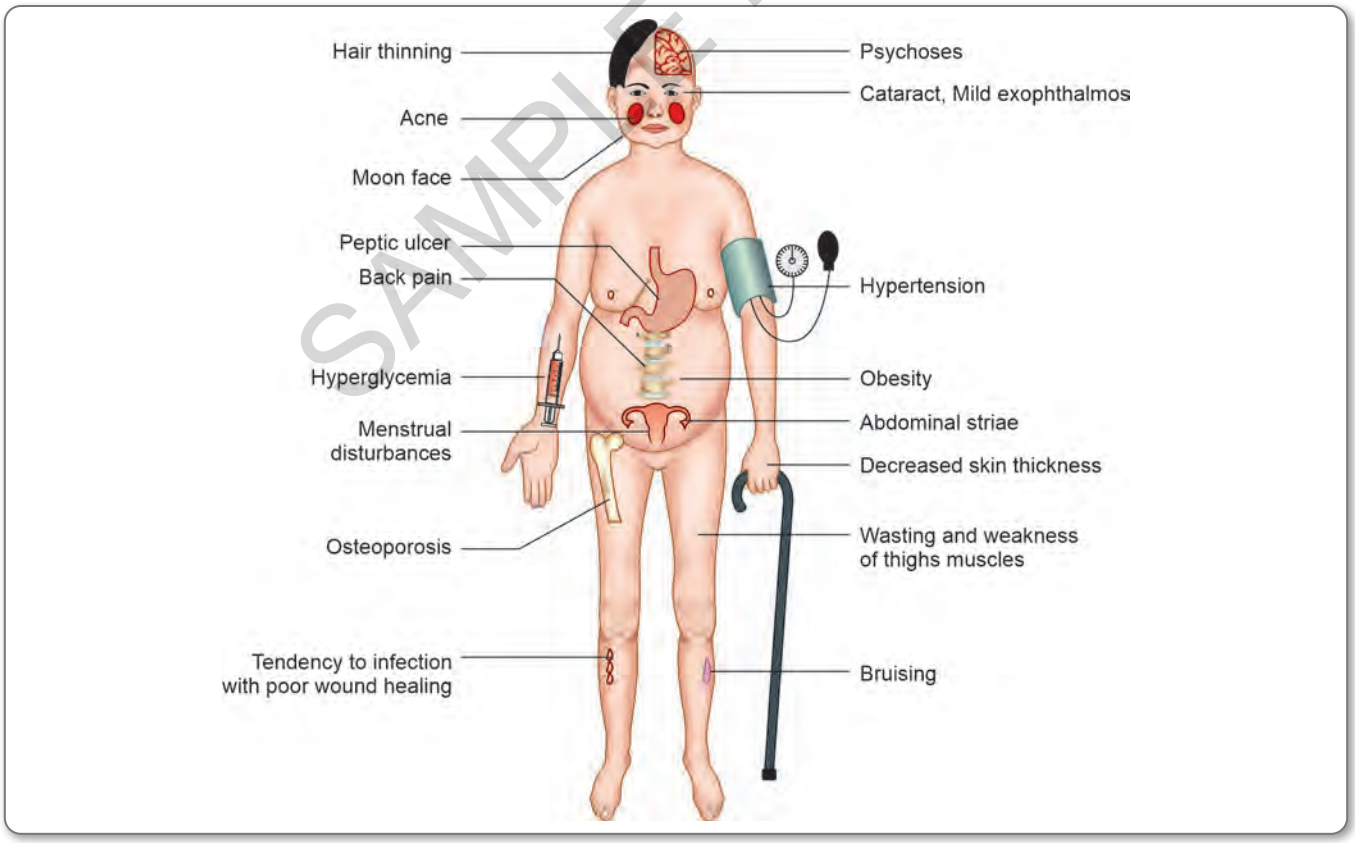


Fig. 8.26: Signs and symptoms of diabetes insipidus

Dehydration

In some cases, the thirst center in the hypothalamus is also affected by the lesion. Therefore, water intake decreases in these patients and, the loss of water through urine is not compensated.

Diagnostic Investigations

- 24-hour urine collection (volume)—typically 3–20 L of urine per day
- Urine osmolality—low results <300 mmol/kg
- Serum osmolality—normal or elevated
- Urinalysis—to rule out other causes of manifestations such as Diabetes mellitus (DM)
- Serum glucose—for determining coexisting

Water Deprivation Test/Dehydration Test

This is done to differentiate between central and nephrogenic DI. If the response occurs to synthetic form of AVP (i.e., desmopressin), the type is central DI (marked by reduction in urine output and increase urine osmolality of >50%.

Management

Hypernatremia management

- IV hypotonic fluids (5% dextrose and 0.45% sodium chloride)
- Frequent monitoring of electrolytes

Central diabetes insipidus

- Desmopressin
- Oral or IV fluid replacement (only in acute settings)

Nephrogenic diabetes insipidus

- Maintenance of adequate per oral fluid
- High dose desmopressin
- Sodium restriction
- Hydrochlorothiazide
- Treat underlying cause: Such as uncontrolled diabetes, chronic hypercalcemia or hypokalemia

Nursing Management

Monitor intake and output, assess for thirst, urination, assess urine specific gravity, teach the patient and family.

Disorders of Both Anterior and Posterior Pituitary

Froehlich Syndrome

Adiposogenital dystrophy, another name for the constellation of endocrine disorders known as Froehlich syndrome, is thought to be the result of injury to the hypothalamus, a region of the brain that connects the neurological system to the endocrine system through the pituitary gland.

Symptoms of the Froelich syndrome include increased or excessive eating that results in obesity, undersized testes, and a delayed onset of puberty. Delays in physical development and the emergence of secondary sexual traits are also frequent in children with Froehlich syndrome.

Pituitary Tumor

The majority of pituitary tumors are benign. However, a tumor that develops on or close to the pituitary gland may:

- Cause changes in hormone production leading to weight gain, excessive or stunted development, high blood pressure, decreased sex drive, or mood swings.
- Tumor mass can also compress optic nerve leading to vision loss and frequent headaches.
- Releasing excess of one or more hormone.
- Not releasing any hormones
- Causing pressure on nearby structures (Example- blurred vision due to the pressing on the nerve of the eye

Pituitary gland diseases are diagnosed using brain imaging (CT and MRI scan) and blood studies. Treatment for pituitary abnormalities depends upon the underlying cause. Hormone insufficiency may need to be corrected with lifelong hormonal supplements, while abnormal cancerous growth requires resection.

Nelson Syndrome

Dr Don J Nelson first reported Nelson syndrome (NS), also known as post adrenalectomy, in 1958. This is a potentially fatal condition that develops following a therapeutic bilateral adrenalectomy. The time between bilateral adrenalectomy and NS diagnosis ranges from 0.5 to 24 years. The loss of feedback inhibition of the hypothalamic-pituitary-adrenal axis is thought to lead to the development of an adrenocorticotropic hormone (ACTH) secreting pituitary tumor. The surgical removal of the tumor is the recommended treatment for Nelson syndrome.

DISORDERS OF THYROID GLAND

HYPOTHYROIDISM

Introduction

Hypothyroidism is a deficiency of thyroid hormone resulting in slowed body metabolism, decreased heat production and decreased oxygen consumption by the tissues.

Incidence and prevalence: Prevalence rates are greater in elderly women (10%) than in elderly men (2.3%). Even though highest incidence is in adults between 30–65 years, it is seen in newborn also.

Definition: Hypothyroidism is a condition in which the thyroid gland does not make enough thyroid hormone. The most common cause of hypothyroidism is inflammation of the thyroid gland, which damages the gland's cells.

Types of Hypothyroidism

- **Primary hypothyroidism:** TH levels are low and TSH levels are elevated indicating that pituitary is attempting to stimulate the secretion of thyroid hormones but the thyroid is not responding. It is most frequently autoimmune in origin but can also be related to iodine deficiency.
- **Secondary hypothyroidism**: Secondary hypo-thyroidism, which occurs in only 5% of cases, is caused by a failure of the pituitary gland to stimulate the thyroid gland or a failure of the target tissues to respond to the thyroid hormones resulting in decreased TSH levels. For example, the pituitary gland and hypothalamus produce hormones that trigger the release of thyroid hormone. A problem with one of these glands can make your thyroid underactive.
- **Tertiary or central hypothyroidism**: It develops if the hypothalamus cannot produce TRH and subsequently does not stimulate pituitary to secrete TSH. It may be due to a tumor, other destructive lesion in hypothalamic region. When this occurs, both TSH and TH levels are again low in serum.
- **Hashimoto's disease**: comes under primary hypothyroidism classified as an autoimmune disorder in which antibodies develop that destroys thyroid tissue. Functional thyroid tissue is replaced with fibrous tissue and TH level decreases. This decreasing level of TH prompts gland to enlarge to compensate, causing a goiter.
- **Cretinism:** It is caused by thyroid hormone deficiencies during fetal or early neonatal life. It can be caused by maternal iodine deprivation or congenital thyroid abnormalities.
- **Iatrogenic hypothyroidism:** It is caused in process of treating hyperthyroidism with radioactive iodine treatment and radiation therapy results in over destruction of thyroid tissue.

Causes of Hypothyroidism (Table 8.6)

TABLE 8.6: Causes of hypothyroidism

- **Autoimmune or Hashimoto's thyroiditis:** In which the immune system attacks the thyroid gland, is the most common example. With Hashimoto's, your body produces antibodies that attack and destroy the thyroid gland. Thyroiditis may also be caused by a viral infection.
- **Postpartum thyroiditis:** Some women develop hypothyroidism after pregnancy

Contd...

- **Radiation therapy:** Radiation to the neck as in case of lymphoma damages cells of the thyroid gland and predisposes a patient to develop hypothyroidism.
- **Radioactive iodine treatment:** Radioactive iodine therapy is prescribed to the patients with overactive thyroid gland. It can destroy the cells of thyroid glands and can predispose a person for hypothyroidism.
- **Medications:** Certain drugs used to treat cancer, psychological disorders, and heart diseases can have an impact on thyroid hormone production. These include lithium, interferon alpha-amiodarone (Cordarone), and interleukin-2.
- **Thyroid surgery:** Following a complete thyroidectomy, patients develop hypothyroidism and need lifelong thyroid hormone medication.
- **Iodine deficiency:** Iodine deficiency impairs thyroid hormone production, which in turn has an impact on the developing brain, liver, kidney, heart, and muscle. Iodine deficiency disorders arise when iodine levels in the soil are low, food products have low iodine concentrations and intake of iodine is poor. The thyroid gland may no longer be able to produce enough thyroid hormone if iodine requirements are not met.
- **Problems with the thyroid at birth:** Some infants could be born with a thyroid gland that did not function properly or developed inadequately. The condition is known as congenital hypothyroidism.
- **Pituitary gland damage or disorder:** A pituitary gland disease can prevent the thyroid hormone from being produced. Thyroid-stimulating hormone (TSH), which is produced by the pituitary gland, has a stimulating effect on the thyroid gland.
- **Disorder of the hypothalamus:** If the hypothalamus does not produce enough thyroid-releasing hormone (TRH). TRH influences thyroid-stimulating hormone (TSH) secretion, a hormone produced by the pituitary gland.

Clinical Manifestations (Fig. 8.27)

Fatigue/Feeling Tired

Fatigue is one of the most common symptoms of hypothyroidism. Thyroid hormone controls energy balance and

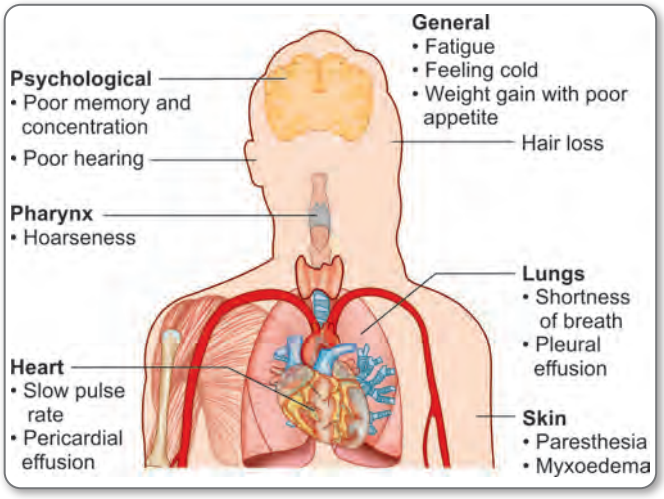


Fig. 8.27: Signs and symptoms of hypothyroidism

metabolism and low thyroid hormone level cause exhausted and sluggish. Patient with hypothyroidism feel un-rested, even though they may be sleeping more.

Weight Gain

Unexpected weight gain is also a symptom of hypothyroidism. When thyroid levels are low, metabolic changes occurs and instead of burning calories for growth and activity, the amount of energy use at rest decreases and as result it tends to store more calories from the diet as fat. The low thyroid hormone levels cause weight gain. Hypothyroidism signals to eat more and burn few calories which lead to weight gain.

Feeling Cold

Heat is a byproduct of burning calories. In cases of hypothyroidism, basal metabolic rate decreases, reducing the amount of heat generate.

That's why low levels of thyroid hormone cause to feel colder. Low thyroid hormone slows body's heat production and cause feeling cold.

Weakness and Aches in Muscles and Joints

Low thyroid hormone flips the metabolic switch toward catabolism and during catabolism, muscle strength decreases, potentially leading to feelings of weakness. Low levels of thyroid hormone slow down metabolism and cause painful muscle breakdown.

Hair Loss

The hair follicles are regulated by thyroid hormone. Low thyroid hormone causes hair follicles to stop regenerating and affects rapidly growing cells (hair follicles), resulting in hair loss.

Itchy and Dry Skin

Like hair follicles, skin cells are characterized by rapid turnover. In Hypothyroidism the dead skin may take longer to shed, leading to flaky, dry skin. Myxedema is specific to thyroid problems, which shows dry skin and red swollen rash on skin.

Feeling Depressed

Hypothyroidism cause depression and anxiety, the cause is still unknown but it is recommended to consult to a physician or therapist.

Trouble Concentrating or Remembering

Many patients with hypothyroidism complain of mental concentrating. Sudden or severe episodes of difficulties in memory or concentration could be a signal of hypothyroidism.

Constipation

Low thyroid levels put the brakes on colon and cause constipation.

Heavy or Irregular Periods

Thyroid hormone interacts with the hormones that control the menstrual cycle, and abnormal levels of it can disrupt their signals.

Risk Factor

- Gender- Women are more likely to develop hypothyroidism than men.
- Type I Diabetes, Multiple Sclerosis, Rheumatoid arthritis, Celiac Disease, Pernicious Anemia or Vitiligo.
- Genetic defects resulting in faulty metabolism of iodine and ingestion of large amount of medical goitrogens (Adrenergic antagonists, glucocorticoids, dopamine, lithium, rifampin, carbamazepine, propylthiouracil and thiocarbamides, aminothiazonate) or nutritional goitrogens (cabbage, soybean, peanuts, peaches, peas, strawberries, spinach and radishes).
- Family history of thyroid disease.
- Iodine deficient area (endemic goiter area).
- Pregnant and lactating women.
- Elderly men receiving aminoglutethimide (1,000 mg day or greater) for prostate cancer.

Pathophysiology

Iodine is required for the synthesis and secretion of thyroid hormone. The hypothalamus regulates the pituitary secretion of TSH by negative feedback system. The hypothalamic–pituitary–thyroid axis plays a key role in maintaining thyroid hormone levels within normal limits. Production of TSH by the anterior pituitary gland is stimulated in turn by thyrotropin-releasing hormone (TRH), released from the hypothalamus (Fig. 8.28). Production of TSH and TRH is decreased by thyroxine by a negative feedback process.

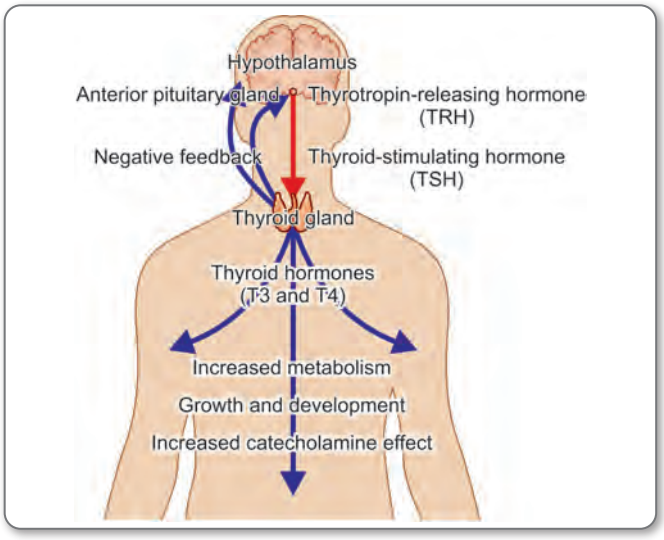


Fig. 8.28: Normal physiology of thyroid hormone production

TABLE 8.6: Thyroid function test (TFT) includes T3, T4 and TSH level in plasma

Test	Normal Value	Significance
Thyroid-stimulating hormone (TSH)	0.5–5.0 U/mL	Increased in primary hypothyroidism, decreased in primary hyperthyroidism
Triiodothyronine (T3)	80–200 ng/100 mL	Decreased in hypothyroidism Increased in hyperthyroidism
Thyroxine (T4)	4–12 µg/100 mL	Decreased in hypothyroidism Increased in hyperthyroidism

When thyroid hormone level is decreased, then basal metabolic rate become slow, which leads to slowing of all body processes. Most important is the change in lipid metabolism. There will be an increase in serum cholesterol and triglycerides with increased atherosclerosis and coronary heart disease in hypothyroidism.

Diagnostic Tests

- Thyroid function test (TFT) (Table 8.6)
- Basal metabolic rate (BMR)
- Protein binding iodide (PBI)
- Radioactive Iodide uptake test (RAIU test)
- Ultrasonography (USG) of thyroid
- Electrocardiogram (ECG) reveals low voltage, T wave abnormalities.
- Other scanning procedures like CT scan, MRI scan.

Management

Goal: To return the patient to the euthyroid (normal) state and to prevent complications.

Medical Management

Correct Thyroid Hormone Deficiency

- Synthetic Levothyroxine (Synthroid or Levothroid) is the preferred preparation for treating hypothyroidism and suppressing nontoxic goiters. Its dosage is based on age, general medical condition and patient's serum TSH concentration. If replacement therapy is adequate, the symptoms of myxedema disappear and normal metabolic activity is resumed.
- **Dosage:** Initially low dose, i.e., 25–50 mcg/day, may adjust dose by 12.5–25 mcg q6–8 weeks depending on the lab reports.

Prevention of Cardiac Dysfunction

Long-term hypothyroidism almost always results in increased blood cholesterol, atherosclerosis, and coronary artery disease. When thyroid replacement is started, angina or dysrhythmias may develop because thyroid hormones boost catecholamine's cardiovascular effects. Thyroid hormone administration must be stopped right once if angina or dysrhythmias happen, and when it is safe to restart, it should be done carefully at a lower dosage while being closely monitored by a doctor.

Supportive Therapy

Maintaining vital functions is part of the management of severe hypothyroidism and myxedema coma. Due to the risk of water intoxication, fluids should be given with caution. If myxedema has progressed to myxedema coma, thyroid hormone is administered intravenously until consciousness is restored.

Prevention of Medication Interactions

Because thyroid hormones and other drugs may interact, care must be exercised during treatment.

Thyroid hormones can raise blood sugar levels, therefore, requires dose modification in diabetic patients. Insulin dosage or use of oral antidiabetic medications is modified. The pharmacologic actions of digitalis glycosides, anticoagulants, and indomethacin may all be enhanced by thyroid hormones.

Tricyclic antidepressants and phenytoin both have the potential to enhance the effects of thyroid hormone.

Dietary Management

Adequate nutrition (well balanced) diet is promoted for patients with hypothyroidism. Patients are advised to follow prescribed calorie intake to achieve goals of weight loss. If iodine deficiency or excessive intake of goitrogenic foods has been identified, then instruct patient to avoid such foods. In severe hypothyroidism apathy, anorexia and self-care deficit may occur which limit food intake so attention must be taken to achieve adequate intake. Food low in calories, high in fiber, and high in protein should be taken. As the metabolic rate rises, the caloric content can be increased. The patient's intolerance to cold may extend to cold foods, making meal planning more difficult. Fluid restriction and occasionally sodium modifications are necessary in severe hyponatremia.

Nursing Care Plan

Hypothyroidism

Assessment	Nursing Diagnosis	Goal	Implementation	Scientific Principles	Evaluation
Inactive, lethargic. Not interested in communication	Activity intolerance related to fatigue	Increase activities and increase independence	Assist with self-care activities Provide stimulation through conversation	Help patient to participate in self-care activities Promote interest.	Participates in self-care activities
Cold skin, subnormal body temperature	Risk for impaired thermoregulation	Maintenance of normal body temperature	Provide extra layers of clothing Avoid use of external heat source	Minimize heat loss Detect body temperature	Maintain baseline body temperature
Not passed motion	Impaired bowel motility: constipation related to depressed gastrointestinal function	Return of normal bowel function	Encourage increased fluid intake within limits of fluid restriction Provide foods high in fiber. Encourage patient to use lamonexative and enema	Promote passage of soft stools Increase bulk of stools and more frequent bowel movement	Reports normal bowel function Drinks recommended amount of fluid each day
Respiration is slow and shows an abnormal pattern	In effective breathing pattern related to depressed ventilation	Improved respiratory status and maintenance of normal breathing pattern	Monitor respiratory rate, depth, pattern pulse oximetry and arterial blood gases Encourage deep breathing, coughing and use of incentive spirometry	Identify baseline to assess changes and evaluate effectiveness of interventions Prevents atelectasis and promotes adequate ventilation	Shows improved respiratory status and maintenance of normal breathing pattern Demonstrates normal respiratory rate, depth and pattern
Client wants to know about disease condition and treatment	Deficit knowledge about therapeutic regimen	Knowledge and acceptance of treatment	Explain rationale for thyroid hormone replacement	Provide rationale for patient to use thyroid hormones	Describe therapeutic regimen correctly
Client is disoriented about time, place, date and event around him or her	Disturbed thought process related to depressed metabolism and altered cardiovascular and respiratory status	Improve thought process	Orient client to time, place, date and event around him or her	Orientation to time, place and person will help in reducing alteration in thought process	Shows improved cognitive functioning Identifies time, place, date and events correctly
Potential to develop complications like myxedema and myxedema coma	Risk for injury	Minimized risk for injury	Monitor client for increasing severity of signs and symptoms of hypothyroidism	Extreme hypothyroidism may lead to myxedema and myxedema coma	Exhibits reversal of myxedema and myxedema coma

Nursing Alert

Medications are administered to the patient with hypothyroidism with extreme caution because of the potential for altered metabolism and excretion and depressed metabolic rate and respiratory status.

Modifying Activity

The patient with hypothyroidism experiences decreased energy level and lethargy. The patient's ability to exercise and participate in activities is further limited by the changes in cardiovascular and pulmonary status secondary to hypothyroidism. A major role of the nurse is assisting with care and hygiene while encouraging the patient to participate in activities within established tolerance levels to prevent the complications of immobility.

Promoting Physical Comfort

The patient often experiences chilling and extreme intolerance to cold. So, extra clothing and blankets are provided. Electric blankets and heating pads shouldn't be used because they increase the risk of vascular collapse, peripheral vasodilation, and further heat loss. In addition, the patient may be burned by these objects without realizing it due to delayed reactions and diminished mental status.

Providing Emotional Support

The hypothyroid patient may respond emotionally strongly to changes in look and body image. The nurse reminds the patient and family that the condition itself includes the failure to notice symptoms, which is a common symptom. To deal with the resulting emotional concerns and reactions, the patient and family may need support and counseling.

Teaching Self-Care to Patients

The majority of hypothyroidism treatment is carried out at home, thus the nurse educates the patient regarding the intended actions, scheduling, side effects, and medication administration at home. The nurse provides written instructions and guidelines for the patient and family. It is often reassuring to the patient and family to be informed that many of the symptoms will disappear with effective treatment. The nurse reinforces periodic follow-up, testing and instructs the patient and family members about the signs of over-medication and under-medication.

Complications

Myxedema Coma

It is the most severe form of hypothyroidism. It is caused from uncontrolled low thyroid production usually due to illness, abrupt stopping thyroid replacement medication, or removal of thyroid gland.

Signs and symptoms (Fig. 8.29):

- Subnormal temperature
- Bradycardia
- Low blood pressure
- Low blood sugar and sodium levels
- Unresponsiveness and extreme drowsiness

Monitor for myxedema coma:

- Vital signs: HR, BP, ECG
- Weight
- Keep patient warm and assess for constipation and encourage fluids

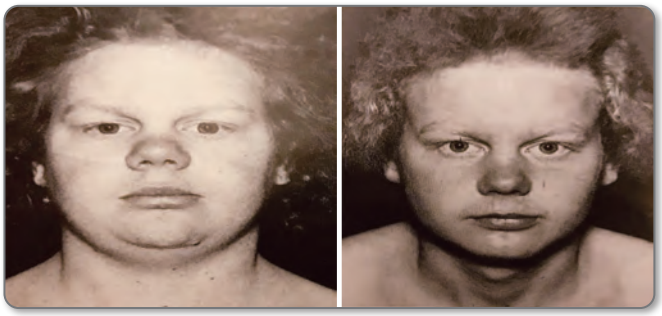


Fig. 8.29: Myxedema

Patient education

- Watch for signs and symptoms of toxicity which would present as signs and symptoms of hyperthyroidism (fast heart, feeling hot, sweating)
- Teach the patient that new cardiac or hyperthyroidism symptoms need to be reported immediately.
- The patient should report any abnormal weight gain or loss or change in bowel elimination.

High Yield Point

The effects of analgesic agents, sedatives, and anesthetic agents in hypothyroidism patients are prolonged, therefore be cautious when administering to elderly patients because of concurrent changes in liver and renal function. Thyroid hormone replacement should be started with low doses and gradually increased to prevent serious cardiovascular and neurologic side effects, such as angina. Regular testing of serum TSH is recommended for people older than 60 years.

Treatment of Myxedema Coma

- Myxedema coma is a medical emergency. For a successful outcome, early diagnosis, prompt thyroid hormone treatment, and adequate supportive measures are necessary.
- Thyroid hormone administration is crucial, and treatment should begin intravenously. Usually, a substantial intravenous loading dosage of 300–500 g T4 may be administered, followed by daily doses of 1.6 g/kg (initially intravenously, and orally when feasible). T3 should be added if no response is received within 24 hours.
- Glucocorticoids should be given (such as 100 mg of hydrocortisone intravenously every eight hours.)
- In case of co-existing myxedema and obesity condition, mechanical ventilation may be necessary.
- Heated blankets may be used to maintain body heat, as the cutaneous blood flow is significantly decreased in severe hypothyroidism.
- Volume expansion is typically necessary case of hypotension. If fluid therapy does not improve circulation, dopamine should be added.

- Monitoring serum glucose is necessary. If adrenal insufficiency is present, supplemental glucose may be required.
- It is necessary to assess for precipitating events, in many cases, there may be no signs of infection, such as fever, tachycardia, or leukocytosis. Antibiotics for prevention are advised until infection has been ruled out.

Cretinism

Hypothyroidism in children is called cretinism. Etiology is associated with endemic goiter. Heredity is the factor causing cretinism. Other causes may be maldevelopment of thyroid, atrophic changes in the fetal gland resulting from acute thyroiditis and sequence of infection in mother during pregnancy.

Causes

Cretinism occurs due to thyroid gland failure to produce thyroid hormone from birth or before birth, which is known as congenital cretinism or congenital iodine deficiency syndrome. It can lead to severe and irreparable mental defects, stunted growth of the child.

Clinical symptoms

Clinical symptoms are striking disturbances of growth, mental retardation and delay in sex development. Growth disturbances, that is disproportionate dwarfism limb measurement is unduly short when compared with trunk. Retardation of bone development specially long bones. Puberty may be delayed. Secondary sex characters fail to appear. Skin dry, tongue broad,large and thick (Fig. 8.30). Extremities are cold. Basal metabolic rate reduces to 50%.

Fig. 8.30: Cretinism

Treatment and Prognosis

Result of treatment in cretinism depends on early diagnosis and institution of thyroid hormone at the earliest possible stage. Result of thyroid therapy on the growth defect in cretinism is most satisfactory provided treatment begins early. It is difficult to measure the mental improvement. Defect in sex development can be corrected by thyroid therapy.

HYPERTHYROIDISM (THYROTOXICOSIS)

Introduction

When the tissues are exposed to and respond to excess thyroid hormone, the condition is known as hyperthyroidism. The excess hormone secreted by thyroid gland increases the metabolic rate (Fig. 8.31). Hyperthyroidism affects all major organs of the body.

Definition: Hyperthyroidism is over production of hormone by the thyroid gland. Hyperthyroidism is the hyper metabolic, clinical and biochemical state caused by excessive production of thyroid hormone. Hyperthyroidism (overactive thyroid) causes sudden weight loss, a rapid or irregular heartbeat, sweating and nervousness or irritability.

Incidence and Prevalence

Hyperthyroidism is a disorder that predominantly affects women (female to male ratio 4:1) especially women between 20 and 40 years. Peaks incidence in the third to fifth decades. Incidence rate is 0.02% to 0.06.

High Yield Point

About 42 million people in India suffer from thyroid disease. Prevalence of thyroid function abnormalities found is 971. Prevalence of hyperthyroidism is 1.6% in community based study and 0.6% in hospital based study.

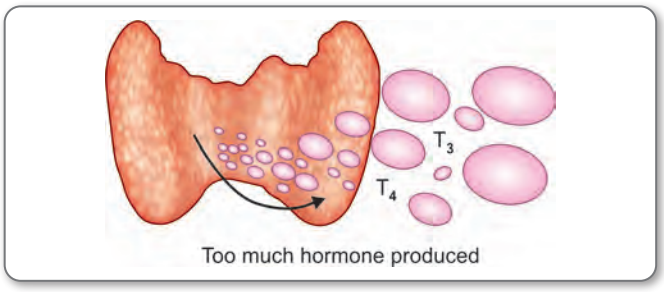


Fig. 8.31: Excess secretion of thyroid hormone by thyroid gland

Etiology

Hyperthyroidism may be due to over functioning of the entire gland. The causes are:

- Auto immune disorder or **Graves' disease** (toxic diffuse goiter): In this, serum of the patients has an antibody that binds to Thyroid Stimulating Hormone (TSH) receptors in the thyroid follicles and causes the thyroid cells to hyper function. Or we may say that development of antibodies against various antigens within the thyroid gland, by insufficient iodine supply, infection, stress leads to Graves' Disease (Fig. 8.32).
- Toxic multinodular goiter (Plummer's disease) common in iodine deficient area.
- Toxic adenoma.
- Thyroiditis: Is an inflammatory process in the thyroid by bacterial or fungal infection.
- T3 thyrotoxicism: T3 level gets elevated but cause is unknown.
- Hyperthyroidism caused by metastatic thyroid cancer.
- Pituitary hyperthyroidism: Rare pituitary adenomas (Fig. 8.33).
- Iodine induced hyperthyroid, over production by administration of supplemental iodine to a person with endemic goiter.
- Ingestion of thyroid hormone and ingestion of Amiodarone hydrochloride (Thyrotoxicosis factitia)
- Family history and Smoking cigarettes is also counted as a risk factor.

Pathophysiology

There is disturbance in normal regulation of thyroid hormone (TH) secretion. Due to increase TH secretion, there will be stimulation of cardiac function and increase in the number of β adrenergic receptors, it leads to tachycardia and increased cardiac output, stroke volume. Metabolism leads to a negative nitrogen balance, lipid decreased, and less nutrition causing weight loss.

Hyperthyroidism also affects the hormone secretion and metabolism of hypothalamic, pituitary and gonadal hormones. There will be delayed onset of puberty, irregular menstrual cycle and infertility in women. In males, decreased libido, delayed puberty.

Excessive TH will induce hypermobility of gut, which causes diarrhea. And eventually, over stimulation of levator palpebrae superioris of the eye will result in wide, gaze, staring and lid lag of the eyes. Over stimulation of neuromuscular will lead to nervousness, irritability and tremor (Fig. 8.34).

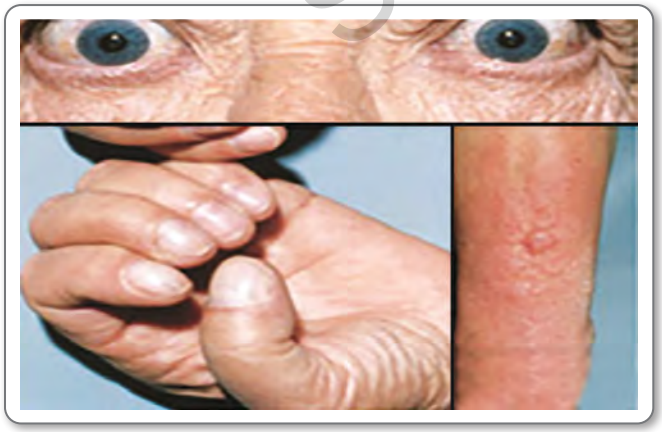


Fig. 8.32: Graves disease

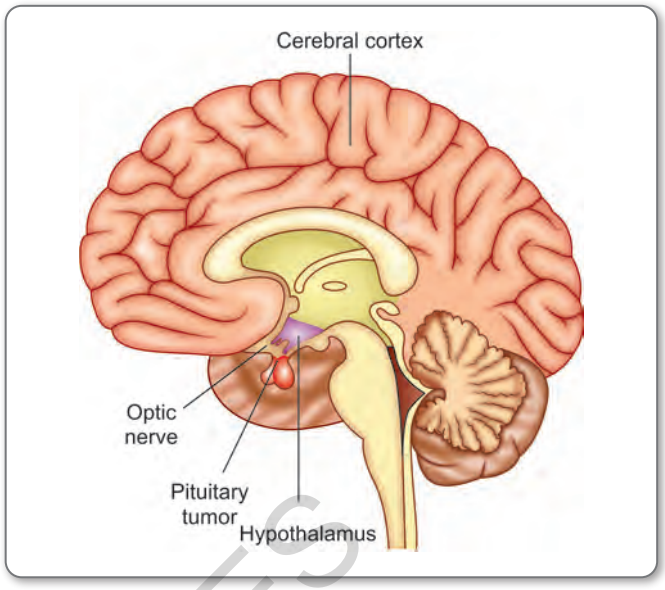


Fig. 8.33: Pituitary adenoma

Clinical Manifestations in Different Systems

Clinical manifestations have been summarized in Figure 8.35.

Cardiovascular

Hypertension, increased rate and force of cardiac contractions, rapid pulse, increased cardiac output, arrhythmias, palpitations, angina, blood pressure systolic increased and diastolic decreased.

Respiratory

Increased respiratory rate, dyspnea on mild exertion.

Musculoskeletal

Fatigue, muscle weakness, dependent edemas, osteoporosis.

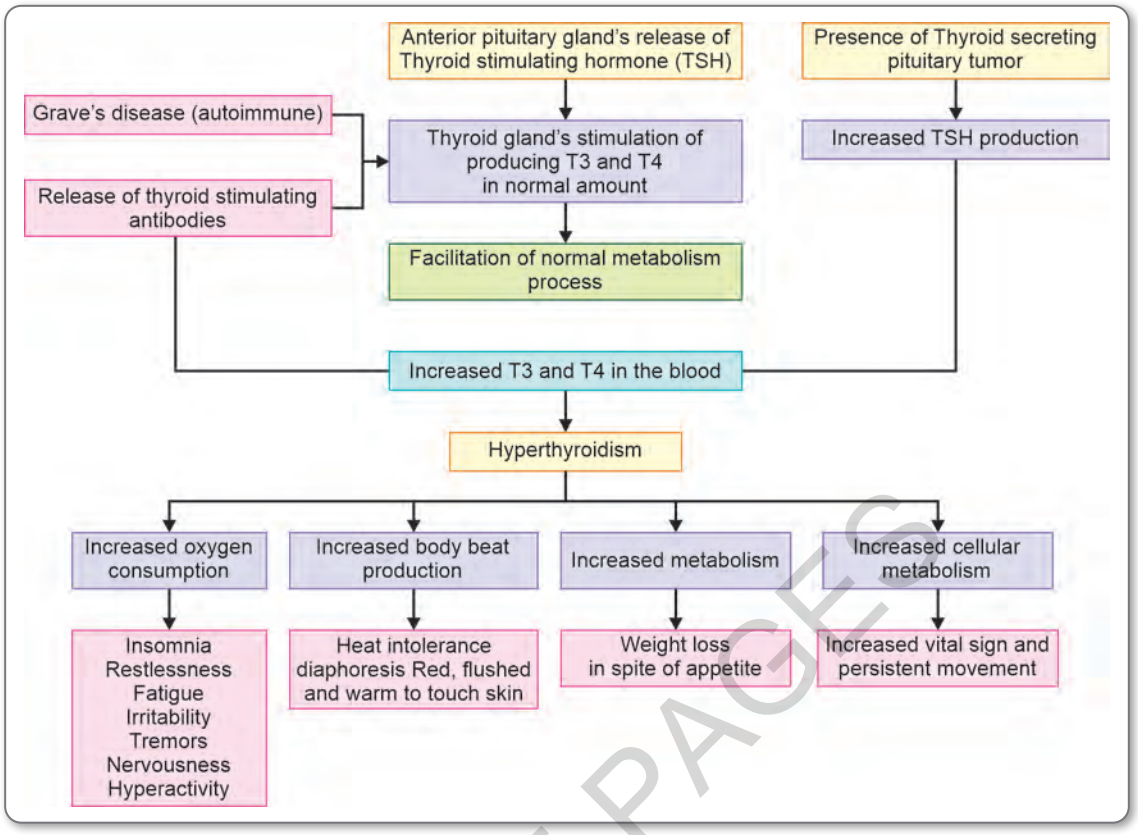


Fig. 8.34: Pathophysiology of hyperthyroidism

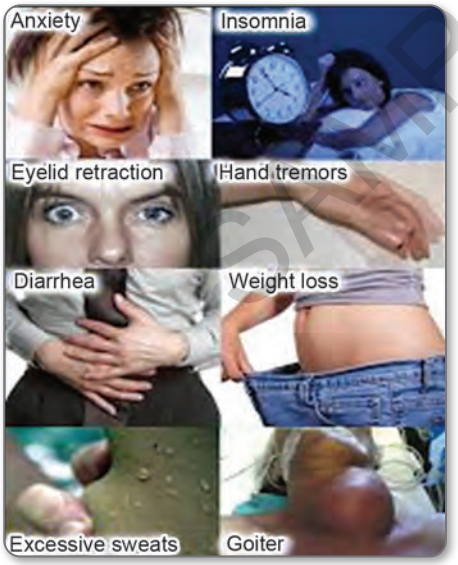


Fig. 8.35: Clinical manifestations of hyperthyroidism

Gastrointestinal

Increased appetite, thirst, weight loss, increased peristalsis, diarrhea, increased bowel sound, splenomegaly, hepatomegaly.

Integumentary

Warm, smooth, moist skin; thin brittle nails, thyroid acropachy of nails which includes clubbing and osteoarthropathy of nails, onycholysis which means detachment of nails, hair loss, palmer erythema; fine silky hair, thyroid dermopathy which includes fine and skin colors papules and plaque on shin (front part of leg below knee joint)

Nervous System

Difficulty in focusing eyes, nervousness, fine tremor (of fingers and tongue), insomnia, liability of mood, restlessness, personality changes, depression, fatigue, apathy, lack of ability to concentrate, stupor, coma.

Reproductive

Menstrual irregularities, oligomenorrhea, amenorrhea, decreased libido.

Ophthalmologic Changes

Soft tissue involvement: Lacrimation, Redness, Burning sensation, Photophobia, Gritty sensation. Proptosis (exophthalmoses) and lagophthalmos, keratitis, Extraocular muscle dysfunction, diplopia, optic neuropathy, blindness.

Evidence Based Practice

"Abnormalities in thyroid function can have an adverse effect on reproductive health and result in reduced rates of conception, increased miscarriage risk and adverse pregnancy and neonatal outcomes," said study co-author Amanda Jefferys in a journal news release. She is a researcher from the Bristol Center for Reproductive Medicine at Southmead Hospital in Bristol, England.

Diagnostic Evaluation

The values of hormones are compared with the normal values so that an accurate results are obtained (Table 8.7).

TABLE 8.7: Normal values of thyroid hormone

Hormone	Normal Values
TSH	0.3–4.5 Mu//L
T3	3.1–7.8 pmol/L
T4	9–23 P pmol/L
Thyroglobulin	Up to 45 pmol/L (30 ng/mL)

Blood Test

- Markedly suppressed TSH (<0.05 μIU/mL) and also decreased TRH level
- Elevated serum T4 and T3
- Thyroid antibodies—Thyroid stimulating immunoglobulin (TSI)

Thyroid Scan

It shows how and where iodine is distributed in thyroid which helps to identify cause of hyperthyroidism through images of nodules and other possible thyroid irregularities. It is done by injecting radioactive isotope intravenously then images of thyroid by special scanner using computer screen are taken in lying down position of patient.

Radioactive Iodine Uptake Test

It measures amount of iodine thyroid collects from bloodstream. Patient is given small oral dose of radioactive iodine. As we know that iodine collects in thyroid gland to manufacture TH. Patient will be checked after 2, 6 or 24 hours and sometimes after all three time periods to determine how much iodine thyroid gland has absorbed. Low level of iodine uptake is indication for hypothyroidism and high level uptake for hyperthyroidism.

Nuclear Scintigraphy

It is a form of test used in nuclear medicine, wherein radioisotopes are taken internally and the emitted radiation is captured by external detectors' in the form of two dimensional images.

Management of Hyperthyroidism

Medical Management

Treatment of hyperthyroidism is directed toward reducing thyroid hyperactivity to relieve symptoms and remove the cause of important complications. Treatment depends on the cause of the hyperthyroidism and may require a combination of therapeutic approaches.

- **Antithyroid or thionamide:** Inhibits the biosynthesis of thyroid hormone, stops the production step of iodination, e.g., propylthiouracil (PTU) and methimazole are most frequently used drugs.
- **Iodides:** Iodides reduce the metabolic rate rapidly because they block the release and synthesis of thyroid hormone, e.g., Lugol's solution and saturated solution of potassium iodide are used.
- **Alternative to iodide:** Lithium: Ability to block the release of thyroid hormone
  - **Dexamethasone:** Effective in blocking hormone release in patients with Graves disease
- **Thyroid blockers or antagonists:** Treat the symptoms from increased sympathetic nervous system stimulation in hyperthyroidism such as tachycardia, dysrhythmias, and angina, e.g., atenolol, Inderal, propranolol, metoprolol.

Nursing Responsibility

Patients receiving these medications should be observed for the development of goiter and should be cautioned against use of over the counter medication that contain iodides and increase the response into iodide therapy. Cough medications expectorants, bronchodilators, and salt substitutes may contain iodide and should be avoided by the patent receiving iodide therapy.

Radioactive (Iodine) Isotope Therapy

Radioactive Iodine (^{131}I) is given to those patients who are middle aged and older. Radiotherapy should not be given to pregnant women. Radioactive iodine is given by mouth which gets into bloodstream and is absorbed by overactive thyroid cells and thyroid cells will die as a result of poisoning due to radioactive iodine.

Nursing Alert

Care of the patient receiving radioactive iodine

When giving direct care, keep your contact time with the patient to a minimum and keep a safe distance. It is not advisable for expectant mothers to care for patients receiving radioactive iodine. Body fluids such as urine, vomit, and others should be disposed of in accordance with hospital protocol since they are polluted. Following the disposal of hazardous material, flush the toilet twice.

The patient should be advised to wash their hands thoroughly after urinating and to avoid close contact with relatives for

about a week while at home. Avoiding oral contact with others and washing eating utensils well with soap and water are both recommended. if the medication is being given.

Inform the patient that symptoms of hyperthyroidism will subside in around 6 to 8 weeks. Rare side effects could include nausea or a sore throat. Encourage the patient to drink plenty of fluids to help remove the radioactive iodine (RAI) from the body.

Surgical Management

Indication for Surgery

Client should be of young age and free from diabetes, heart disease, renal disease, drug allergies.

Thyroidectomy

In this either total or partial removal of thyroid gland is performed. In case of cancer of thyroid gland, total thyroidectomy is done. Partial or subtotal thyroidectomy performed to treat hyperthyroidism and extreme cases of simple goiter. After total thyroidectomy hormone replacement therapy is given lifelong.

Preoperative Preparation

Patient must be euthyroid (normal thyroid function) before surgery. Patient's given antithyroid drugs before surgery. Iodine preparation also given preoperatively to reduce the size and vascularity of thyroid organ.

Dietary Management

- High calorie diet—4,000 to 5,000 cal daily given to satisfy hunger and to prevent tissue breakdown.
- High protein, vitamins and minerals—1–2 g/kg body weight, minerals and particularly vitamin A, B_6 and C.

Nursing Care Plan

Hyperthyroid Patient

Assessment	Nursing Diagnosis	Goal	Implementation	Scientific Principles	Evaluation
Hot skin, Warm to touch skin.	Altered body temperature related to hyper metabolic state	Maintain normal body temperature	Monitor temperature. Temperature may be elevated Maintain environment cool and comfortable for patient Bedding and clothing should be changed as per need Apply cooling blanket as ordered Administer antipyretics as per prescription Reason for patient's discomfort and importance of providing cool environment should be explained to the family members also	One of the first manifestations of thyroid storm is elevated temperature. Cool baths and cold fluid may provide relief.	Maintains normal body temperature
Diarrhea	Disturbances in normal bowel pattern related to increase in peristalsis	Reduce patient discomfort and maintain fluid and electrolyte balance	Provide a low-fiber diet. Provide small frequent meals of bland foods (bananas, rice, apple, sauce) that are less likely to worsen diarrhea. To reduce diarrhea highly seasoned foods and stimulants like coffee, tea, cola, alcohol are discouraged. Monitor electrolytes, especially sodium and potassium. Replace fluid loss as diarrhea causes fluid loss. Keep skin clean and dry; apply barrier cream to protect skin from injury from stool.	Fiber can increase peristalsis and stools. Diarrhea can cause electrolyte loss. Monitor for dehydration.	Diarrhea controlled.
Weight loss, Fatigue	Imbalanced nutrition less than body requirements related to increased metabolic needs and excessive appetite	Improve nutritional status and maintain current activity level.	Assess the weight of the patient weekly Help to plan high calorie, high protein high carbohydrate diet with selection from all food groups Suggest small and frequent diet like six small meals per day or between meal snacks Serve food according to his or her choice	High calorie high protein diets fulfill the increased metabolic need.	The weight is maintained.

Contd...

Assessment	Nursing Diagnosis	Goal	Implementation	Scientific Principles	Evaluation
Irritable and emotionally unstable.	Ineffective individual coping R/T irritability, hyper excitability, apprehension and emotional instability	Enhancing coping measures	Discuss the underlying cause for this type of symptom both with patient and family and reassure them that the symptoms can be subsided with appropriate treatment. Maintain calm, relaxed environment Stressful experiences are minimized and relaxing activities are encouraged	Relaxing activities help in reducing stress	Demonstrate effective coping in dealing with others
Unable to close eye lids.	Sensory/ perceptual changes (potential visual) related to infiltrative changes associated with hyperthyroidism	Improve the vision and to prevent worsening of the vision.	Assess visual activity, ability to close eyes, photophobia Protect the eyes Use patches or glasses when in high wind Use artificial tears as prescribed Instruct the patient not to lie prone. Teach patient about correct method of eye drops instillation.	Covering eyes relieves discomfort	Relieve discomfort Eyes are protected
	Disturbance in self-esteem R/T changes in appearance, excessive appetite, and weight loss –	To improve self-esteem	Patient and family should be informed that these changes are due to dysfunction of thyroid gland and out of patient control If changes in appearance are very disturbing to patient then cover or remove mirrors Provide privacy while eating as patient will get annoyed on any comment about his need of large amount of meal	Help in improving coping and relief of stress	Achieves increased self-esteem
Confused regarding disease and treatment.	Knowledge deficit related to disease condition, treatment and prognosis.	Improve knowledge level.	Assess the knowledge of the patient Clear all his doubts Impart education about disease and treatment Adequate teaching should be given related to home care and follow up care	Health education will improve the knowledge of disease and its treatment	Patient is confident and relaxed
Assess for postoperative complications	Collaborative problems/ potential complications	Absence of complications.	For Thyrotoxicosis or thyroid storm- It is a potentially fatal, acute episode of increased thyroid activity Sign and symptoms are high fever, severe tachycardia, delirium, and extreme irritability. Causing factors are untreated hyperthyroidism, infection, thyroid ablation, metabolic catastrophes, surgery, trauma, labor and delivery, MI, pulmonary embolus, medication overdoses, etc. It is a clinical diagnosis also called as thyroid crisis. Cardiac and respiratory functions are assessed by measuring vital signs and cardiac output, ECG monitoring, arterial blood gases, and pulse oximetry. Oxygen is administered to prevent hypoxia, to improve tissue oxygenation, and to meet the high metabolic demands. Intravenous fluids may be necessary to maintain blood glucose levels and to replace lost fluid. Antithyroid medications with adjuvant therapy should also be continued If shock develops, treatment strategies must be implemented	After thyroidectomy hemorrhage and respiratory obstruction may develop	Not develop any complication

Promoting Home and Community-Based Care

Teaching Patients Self-Care

The nurse teaches the patient how and when to take prescribed medication and provides instruction about the essential role of the medication in the broader therapeutic plan. The type and amount of information given depends on the patient's stress and anxiety levels. The patient and family members receive verbal and written information about the actions and possible side effects of the medications. If patient has Graves ophthalmology, he may need to use eye drops to moisten eyes and wear sunglasses to protect eyes.

Continuing Care

The nurse reinforces to the patient and family the importance of long-term follow-up because of the risk for hypothyroidism after thyroidectomy or treatment with antithyroid medications or radioactive iodine. Besides this,

- Avoid stress
- Avoid caffeine
- Avoid smoking

THYROIDITIS

Inflammation of thyroid gland is known as thyroiditis. Acute suppurative thyroiditis, which is caused due to a bacterial infection, subacute thyroiditis, which occurs from a viral infection of the gland, and chronic thyroiditis, which is typically autoimmune in nature, are various inflammatory conditions affecting the thyroid gland.

Hashimoto Thyroiditis

Hashimoto thyroiditis is a type of autoimmune thyroid disease which is characterized by the immune system's different cell- and antibody-mediated immunological mechanisms destroying thyroid cells. Hypothyroidism has an insidious onset with mild sign and symptoms that may progress to severe symptoms over the course of months or years.

Patients with hypothyroidism may present subclinical, symptoms, and their condition may be identified with routine thyroid function testing. Such patients could experience vague symptoms that are challenging to link to thyroid issues.

Hashimoto thyroiditis or a variant of it can be diagnosed when anti-TPO and anti-Tg (anti-thyroglobulin) antibodies are present; however, 10–15% of people with Hashimoto thyroiditis may not have these antibodies.

Thyroid hormone replacement is the preferred method of treatment for Hashimoto thyroiditis Levothyroxine sodium, taken orally, is the preferred medication, typically for life.

De Quervain's Thyroiditis

Subacute granulomatous thyroiditis, also known as De Quervain thyroiditis, is a type of self-limited subacute thyroiditis that is frequently accompanied with an upper respiratory tract viral infection like the measles, mumps, coxsackievirus, adenovirus, or influenza viruses.

The majority of patients complain of neck pain in addition to thyrotoxicosis symptoms and signs include tachycardia, hot flushes, heat intolerance, and palpitations.

Aspirin is typically used as a pain reliever together with bed rest. Occasionally, in severe situations, cortisone and thyroid hormone may be administered. After a few weeks or months, almost all patients recover, and their thyroid glands return to normal functioning.

Once the inflammation has subsided, a few patients will develop hypothyroidism and require lifelong thyroid hormone replacement. Recurrences don't happen often.

Silent Thyroiditis

The third and least frequent kind of thyroiditis is silent thyroiditis.

This form of thyroiditis resembles a combination of De Quervain's and Hashimoto's thyroiditis. The majority of patients are young female following pregnancy.

In most cases, there is no need for medication, and after three months, the thyroid gland returns to normal in 80% of patients. The condition's milder symptoms resemble Graves' disease.

The most common form of treatment for palpitations is bed rest combined with β blockers. Treatment with radioactive iodine, surgery, or antithyroid drug is not required.

A few patients require thyroid hormone therapy because they had developed persistent hypothyroidism.

Acute and Subacute Thyroiditis

Acute infectious thyroiditis is also known as suppurative thyroiditis.

Caused by radiation-induced and traumatic thyroiditis, as well as bacterial or fungal infection (most frequently resulting from a piriform sinus tract fistula or hematogenous dissemination).

Both subacute and acute thyroiditis are characterized by painful anterior neck swelling and constitutional symptoms.

Acute infectious thyroiditis is treated with surgical drainage and systemic antibiotic therapy. Treatment for subacute thyroiditis is not usually necessary, β-blockade can be used to relieve symptoms of thyrotoxicosis and nonsteroidal anti-inflammatory medications (NSAIDs) or glucocorticoids can be used to treat the painful thyroiditis. To ensure recovery, thyroid function monitoring is advised.

GOITER

Goiter is an abnormal enlargement of the thyroid gland. Although goiters are usually painless, an abnormally large enlargement of thyroid gland can be diffuse or nodular.

Pathophysiology

Chronic absence of T3 and T4 causes elevated level of TSH, which then lead to diffuse homogenous hypertrophy and hyperplasia of follicular cells colloid in efforts to produce more thyroid hormone. This is a reversible change. Enlarged thyroid hyper involuted with colloid is called colloid goiter. Differential response to TSH leads to formation of nodules with in the gland. Several nodule may coalesce and leads to formation of multinodular goiter. The nodules may undergo secondary changes central necrosis, cystic degeneration, hemorrhage, calcification, and malignant changes.

Clinical Manifestations (Fig. 8.36)

Not all goiters cause signs and symptoms, but when they do, it may have the following signs and symptoms:

- Swelling at the base of the neck
- Coughing
- Tightness in the chest
- Hoarseness, stridor, Difficulty in swallowing occurs due to local compression
- Pain due to hemorrhage, inflammation, necrosis, or malignant transformation

Etiology and Risk Factors

- Iodine deficiency due to lack of intake of iodine rich diet
- Inflammatory disorders of the thyroid gland such as autoimmune thyroiditis, silent thyroiditis, radiation thyroiditis, subacute thyroiditis, and suppurative thyroiditis.

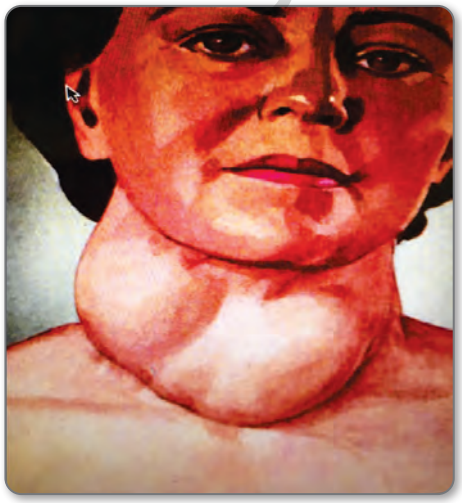


Fig. 8.36: Simple goiter

- Thyroid cancer and granulomatous and infiltrative diseases of the thyroid

Diagnostic Investigations

Physical examination: Inspection should be done by asking the patient to sip some water. When swallowing, the thyroid gland should move.

With the neck relaxed and not hyperextended, the goiter is palpated either from in front of or behind the patient.

A hard thyroid gland indicates malignancy, while a firm, rubbery thyroid suggests Hashimoto thyroiditis.

Following laboratory tests are conducted to diagnose goiter

- Thyroid hormone assessment
- Antibody test to check for abnormal antibodies such as: antithyroglobulin
- Ultrasonography to reveal the size, consistency and nodularity of the thyroid gland.
- Thyroid scan to check the nature and size of thyroid
- CT scan can be used to identify size and goiter extent.
- Radionuclide scanning-is done to assess the thyroid functioning determined on the basis of radionuclide uptake.
- Biopsy to obtain a tissue or fluid for sampling

An enlarged thyroid gland simply by feeling neck and having the Patient swallow during a basic physical exam indicate goiter.

Following laboratory tests are conducted to diagnose goiter

- Thyroid hormone assessment
- *Antibody test* to check for abnormal antibodies
- *Ultrasonography* to reveal the size of the thyroid gland and whether the gland contains nodules
  - *Thyroid scan* to check the nature and size of thyroid
  - *Biopsy* to obtain a tissue or fluid for sampling

Management

Medical Management

- Treating small benign euthyroid goiters is not necessary. Goiters that are large may need both medical and surgical intervention.
- Levothyroxine suppressive therapy has the potential to shrink the size of a benign euthyroid goiter.

Surgical Management

Surgery is recommended for large compressive goiter. Surgery such as total thyroidectomy is necessary for multinodular goiter. Hemodynamic monitoring is a crucial part of preoperative management. And, monitoring of serum calcium levels is another important aspect of postoperative care.

DISORDERS OF PARATHYROID GLAND

PARATHYROID DISORDERS

Parathyroid glands are four small endocrine glands. Parathormone (PTH) regulates the amount of calcium and phosphorus that is stored in the bones and circulated in the blood. A low blood calcium level in the blood causes the parathyroid gland to secrete parathyroid hormone, which is essential for controlling the levels of calcium in the bones and blood.

Hyperparathyroidism (Overactivity of the Parathyroid Gland)

Hyperparathyroidism is a disease characterized by excessive secretion of parathyroid hormone. Primary hyperparathyroidism is characterized by an uncontrolled overproduction of parathyroid hormone (PTH), which causes improper calcium homeostasis. In 85% of the cases, it is seen in case of solitary adenoma.

Secondary hyperparathyroidism (CKD) is characterized by an overproduction of parathyroid hormone as a result of hypocalcemia, often brought on by vitamin D insufficiency and/or chronic kidney disease.

Chronic stimulation of the parathyroid gland to release parathyroid hormone causes secondary hyperparathyroidism.

Tertiary hyperparathyroidism is a condition of excessive parathyroid hormone secretion that causes hypercalcemia. It is characterized by autonomous parathyroid hormone hypersecretion that results in hypercalcemia (Fig. 8.37).

Incidence

Parathyroidism usually occurs at the age of 60 years and is more common in women as compared to men. Incidence is 27 per 1000,000.

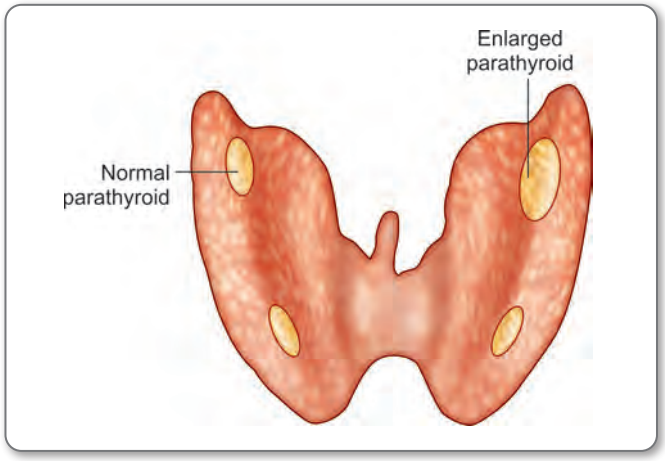


Fig. 8.37: Enlarged parathyroid gland

Etiology and Risk Factors

- Adenoma or hyperplasia of the gland
- As a result of renal failure there may be hyperplasia of the gland.
- Osteogenesis imperfecta, multiple myeloma or carcinoma with bone metastasis
- Autonomous production of parathyroid hormone (PTH).

Pathophysiology

Normally PTH increases the bone reabsorption of calcium there by maintain proper balance of calcium and phosphorus ion in the blood. Excessive PTH causes bone damage, Hypercalcemia, and kidney damage

Approximately 3% or 4% of all patients with primary hyperparathyroidism will have an enlargement of all 4 parathyroid glands, a term called parathyroid hyperplasia. In this instance, all of the parathyroid glands become enlarged and produce too much parathyroid hormone. This is a much less common scenario, but the end results on the tissues of the body are identical.

An even rarer situation occurs in less than 1% of the people who have two parathyroid adenomas while having two normal glands. This is very uncommon.

Evidence Based Practice

A scientific study of 6,331 patients with primary hyperparathyroidism examined how many glands typically go bad in this disease. This study included data collected on a portion of patients with this disease over 10 years (1987–1997). The complete data and statistical analysis was published in March 1998 by Dr James Norman's group in the Journal of the American College of Surgeons They concluded between 94% and 96% of all patients with hyperparathyroidism have one bad gland and three normal glands.

Clinical Manifestations

Hyperparathyroidism sometimes may not have any symptom. But it may manifest symptoms of skeletal, renal, gastrointestinal disorder and neurological disorders as depicted in Figure 8.38.

In severe condition, bones can lose calcium and become very brittle and break off leading. This condition is more commonly seen in older patients.

Persistently elevated calcium levels can precipitate pancreatitis and gastric ulcer. Also, it can lead to the formation of kidney stones.

Patients also experiences bone pain, pruritus, fatigue and lethargy due to the hypercalcemia and hyperphosphatemia.

Diagnostic Evaluation

Diagnosis of hyperparathyroidism is confirmed by X-ray findings, increased serum calcium levels, decreased serum

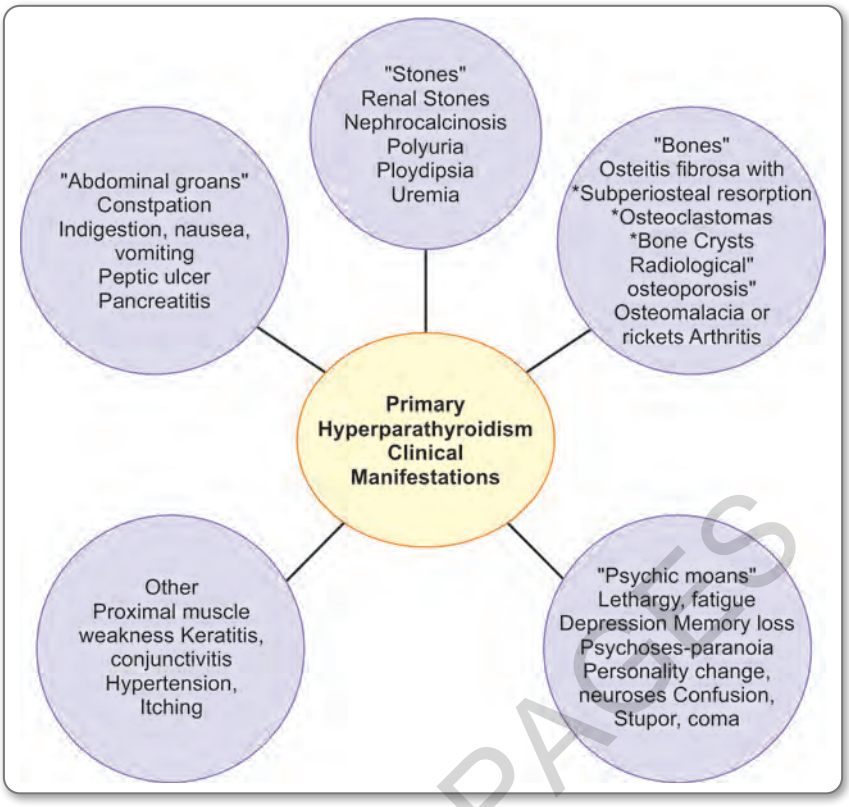


Fig. 8.38: Line diagram of clinical manifestations of hyperparathyroidism

phosphate levels, and level of urine calcium and phosphorus increased.

Diagnostic comparison of hyperparathyroidism and hypoparathyroidism (Table 8.8)

TABLE 8.8: Diagnostic comparison of Hyperparathyroidism and hypoparathyroidism

Hyperparathyroidism	Hypoparathyroidism
Increased bone resorption	Decreased bone resorption
Elevated serum calcium levels	Depressed serum calcium level
Depressed serum phosphate level	Elevated serum phosphate level
Hypercalciuria, hyperphosphaturia	Hypocalciuria, hypophosphaturia
Decreased neuromuscular irritability	Increased neuromuscular irritability, which may progress to tetany

Management

Medical Management

The aim of medical management is lowering increased serum calcium level and drugs are given to increase the bone reabsorption of calcium.

To lower the calcium level—measures are—Hydration by infusion of normal saline solution, because normal saline expands the volume and also inhibits the reabsorption of calcium by kidney. Diuretics like furosemide can be given after the patient is rehydrated to promote calciuria. Thiazide diuretics are not given to the patient to lower calcium level as thiazide diuretics promote calcium retention in the kidneys. Also, the patient with hypercalcemia should be given a diet low in calcium and vitamin D.

Pharmacological Management

Drugs like plicamycin (mithracin), gallium nitrate (Ganite), phosphates and calcitonin which inhibit bone reabsorption are given. Glucocorticosteroids may be used to reduce hypercalcemia.

Surgical Management

Parathyroidectomy: Surgical removal of parathyroid gland.

Nursing Management

Physical assessment and history taking is very important to prepare a nursing care plan for the client under medical management.

Nursing Care Plan

Hyperparathyroidism

Assessment	Nursing Diagnosis	Goal	Implementation	Scientific Principles	Evaluation
Potential risk for fracture	Risk for injury related to demineralization of bone resulting in pathologic fracture	Prevent fracture	Protect the client from accidents Keep the client's bed in the low position and use side rails. Help in ambulation	To provide safety for the client	Absence of accidents and fracture
Complain of Painful urination, hematuria	Impaired urinary elimination related to renal involvement secondary to hypercalcemia and hyperphosphaturia resulting in urolithiasis	To resume a normal urination	Encourage fluid at least 3,000 mL per day	Dehydration is dangerous because it increases serum calcium level and promotes the formation of renal stone	Prevent formation of renal stone
Anorexia, weight loss	Imbalanced nutrition less than body requirement related to anorexia and nausea, decreased food intake	Client will have adequate food intake	Encourage low calcium diet. Avoid milk and milk products	To correct hyper-calcemia. Milk and milk products alleviate distressing gastrointestinal manifestations.	Absence of nausea. Maintain ideal body weight
Complained of not assessed motion	Constipation related to decreased frequency of stools and painful defecation	Maintain a normal bowel pattern	Prevent constipation and fecal impaction. Increase fluid intake and amount of fiber in diet	Relieve constipation by softening the stools	Normal bowel pattern, daily bowel movement

Hypoparathyroidism

Hypoparathyroidism is a disorder in which parathyroid glands secrete abnormally less hormone (PTH). PTH regulates and maintains a balance of calcium and phosphorus in the body. In hypoparathyroidism, serum calcium levels are abnormally low, and serum phosphate levels are abnormally high. Patient may present with neuromuscular irritability (tetany).

Incidence is more in female than male.

Etiology

Factors that can cause hypoparathyroidism include:

- **Acquired hypoparathyroidism:** Due to accidental damage, or removal of the parathyroid glands during thyroid surgery or surgery for throat cancer or neck cancer.
- **Autoimmune disease:** The immune system creates antibodies against the parathyroid tissues, trying to reject them as if they were foreign bodies and parathyroid gland does not produce PTH.
- **Hereditary:** A family history may be responsible for hypoparathyroidism.
- **Exposure to radiation:** It can result in destruction of your parathyroid glands, as can radioactive iodine treatment for hyperthyroidism, occasionally.
- **Low levels of magnesium:** In blood, they affect the function of parathyroid glands. Normal serum magnesium levels are required for optimum secretion of parathyroid hormone.

Pathophysiology

Due to etiological factor such as tumor of thyroid gland causes inadequate secretion of PTH and reabsorption of Ca in GI tract. When blood calcium falls to low level, it leads to muscular hyperirritability and uncontrolled spasm.

Clinical Manifestations

Signs and symptoms of hypoparathyroidism can include:

- Tingling or burning (paresthesias) fingertips, toes and lips
- Cramps in legs, feet
- Muscles spasm
- Fatigue or weakness
- Painful menstruation

- Loss of hair, e.g., thinning of eyebrows
- Dry, coarse skin
- Brittle nails
- Headaches
- Depression, mood swings
- Memory problems

Management

Medical Management

Hypocalcemia is corrected by giving calcium and vitamin D to the patients. Patients with primary hypoparathyroidism are advised to take diet rich in calcium content such as milk based products. A pure form Synthetic parathyroid hormone is now available.

Acute emergency due to elevated serum calcium level should be managed to prevent laryngeal spasm and respiratory obstruction. Immediately 10% calcium gluconate is administered intravenously. Instruct client to breathe into a paper bag, so carbon dioxide inhaled this way will elevate the amount of ionized calcium in the blood. Oral calcium salts are given when danger of tetany has passed. Vitamin D is also given to maintain serum calcium level.

Nursing Management

Assess the client suffering with Hyperparathyroidism for change of the body like dry skin and hair. Assess for cataract, Parkinson's like symptoms, respiratory problem, e.g., laryngospasm then go for nursing care planning.

Nursing Care Plan

Hypoparathyroidism

Assessment	Nursing Diagosis	Goal	Implementation	Scientific Principles	Evaluation
Potential for spasm of larynx	Risk for injury—muscle spasm Tetany related to decreased serum calcium levels	Return to normal calcium level	Always be prepared for laryngeal spasm and respiratory obstruction Endotracheal tube, laryngoscope and tracheostomy tray should be available near client's bed	Safety precautions to prevent laryngeal spasm	Maintain normal serum calcium level
Client having fatigue	Activity intolerance related to hypermetabolic state	Maintain usual routine, decreased performance emotional liability/ irritability	• Monitor vital signs, noting pulse rate at rest and when active • Provide for quiet environment; cool room, decreased sensory stimuli, soothing colors, quiet music	• Pulse is typically elevated and, even at rest, tachycardia (up to 160 beats/min) may be noted • Reduces stimuli that may aggravate agitation, hyperactivity, and insomnia	Maintain usual routine, decreased performance
Dizziness, orthostatic hypotension	Risk for decreased cardiac output	Maintain adequate cardiac output	• Monitor BP lying, sitting, and standing, if able. Note widened pulse pressure. • Monitor central venous pressure (CVP), if available	• General or orthostatic hypotension may occur as a result of excessive peripheral vasodilation and decreased circulating volume • Provides more direct measure of circulating volume and cardiac function	A risk diagnosis is not evident by signs and symptoms, as the problem has not occurred and nursing interventions are directed at prevention
Weight loss, anorexia	Risk for imbalanced nutrition: Less then body requirements	Mainntain normal body weight and laboratory values and be free of signs of malnutrition	• Monitor daily food intake. Weigh daily and report losses. • Encourage patient to eat and increase number of meals and snacks. Give or suggest high-calorie foods that are easily digested	• Continued weight loss in face of adequate caloric intake may indicate failure of antithyroid therapy • To promote weight gain	Demonstrate stable weight with normal laboratory values and be free of signs of malnutrition

Complications

Hypoparathyroidism may result in numerous complications.

Reversible Complications

Low calcium levels can lead to the following manifestations:

- **Tetany:** It is characterized by severe, protracted cramp-like spasms. Tetany can also cause twitches or spasms in the face, mouth, or arms. It can also cause soreness in the muscles. These throat spasms might hinder breathing and can even precipitate an emergency condition.
- **Paresthesia:** The sensory signs of these include tingling or pins-and-needles sensations in the lips, tongue, fingers, and feet.

Irreversible Complications

Hypoparathyroidism-related problems may be avoided with an accurate diagnosis and prompt treatment.

- Slow mental development (or mental retardation) in children
- Stunted growth (short height)
- Cataracts, and
- Calcium deposits in the brain that can cause balancing problems and seizures.

DISORDERS OF PANCREAS

DIABETES MELLITUS

Diabetes mellitus is a group of metabolic diseases characterized by hyperglycemia resulting from defects in insulin secretion, insulin action, or both. [American Diabetes Association (ADA) Expert Committee on the Diagnosis and Classification of Diabetes Mellitus, 2003]. The chronic hyperglycemia of diabetes causes long-term complications such as dysfunction, and failure of various organs, especially the eyes, kidneys, nerves, heart, and blood vessels (Fig. 8.39). Client sometimes call it as "high sugar" hyperglycemia. This glucose is derived from absorption of food from gastrointestinal tract and formation of glucose in the liver.

Insulin secreted by the β cells of pancreas controls the glucose level in the blood. In diabetes, cells do not respond to insulin or pancreas stops producing insulin, this leads to hyperglycemia.

Classification of Diabetes

There are two main categories of DM—type 1 and type 2

- **Type 1 diabetes:** In this type of diabetes, pancreatic β cells are destroyed which lead to absolute insulin deficiency. About 10% of all diabetes patients come under type 1 diabetes usually diagnosed before the age of 30 years.
- **Type 2 diabetes:** It is most common form of diabetes, 90%–95% diabetes patients are of this type. Commonly type 2 diabetes diagnosed after 40 years of age.

Incidence

Diabetes is fast gaining the status of a potential epidemic in India with more than 62 million diabetic individuals currently diagnosed with the DM type 1, 2. In 2000, India (31.7 million) topped the world with the highest number of people with diabetes mellitus followed by China (20.8 million) with the United States (17.7 million) in second and third place, respectively.

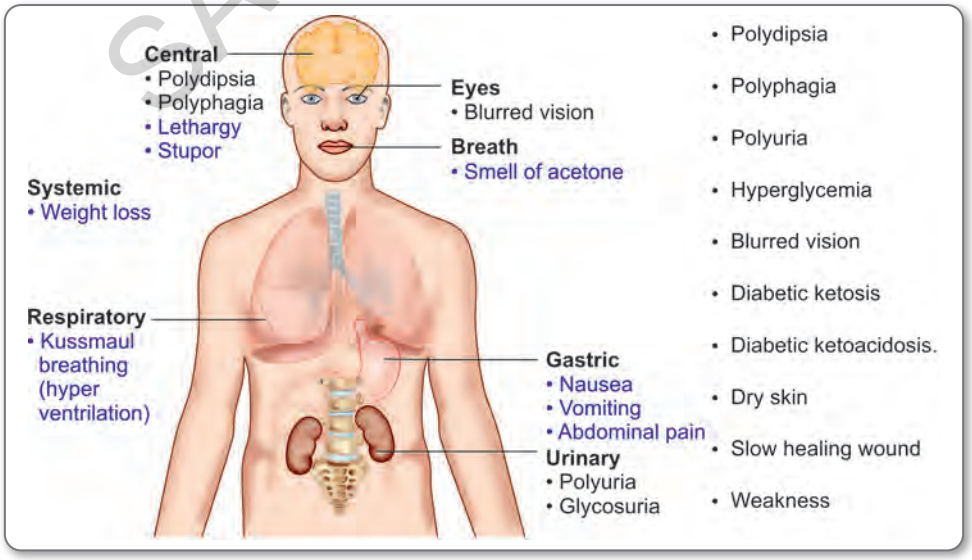


Fig. 8.39: Clinical manifestations of diabetes mellitus

Evidence Based Practice

According to Wild et al., the prevalence of diabetes is predicted to double globally from 171 million in 2000 to 366 million in 2030 with a maximum increase in India. It is predicted that by 2030 diabetes mellitus may afflict up to 79.4 million individuals in India, while China (42.3 million) and the United States (30.3 million) will also see significant increase in those affected by the disease. India currently faces an uncertain future in relation to the potential burden that diabetes may impose upon the country.

Etiology and Risk Factor

- **Type 1 DM** (juvenile-onset or insulin-dependent), there is no insulin produced by pancreas because of
- Autoimmune pancreatic β-cell destruction. It develops in childhood or adolescence before age 30.

However, it can also develop in adults (latent autoimmune diabetes of adulthood, which often initially appears to be type 2 DM).

The pathogenesis of the autoimmune β-cell destruction involves may be an interaction between susceptibility genes, autoantigens, and environment. Some populations have susceptible gene, they show higher prevalence of type 1 DM, e.g., Scandinavians, Sardinians. Diet may also be a factor. Exposure of infants to dairy products (especially cow's milk and the milk protein β casein), high nitrates in drinking water, and low vitamin D consumption have been linked to increased risk of type 1 DM.

Pathophysiology

Insulin is a hormone. Its secretion increases when a person eats meal so that glucose can move from blood in to muscles, liver, and fat cells, where insulin transports and metabolizes glucose for energy. Insulin also inhibits the breakdown of protein and fat.

Type 2 DM (previously called adult-onset or non–insulin-dependent), Inadequate secretion of insulin in the body or patients develops resistance to insulin. The disease generally develops in adults and becomes more common with increasing age.

Peripheral insulin resistance and insufficient insulin production by pancreatic β cells are two traits that define type 2 diabetes. Reduced glucose transport into muscle cells, increased hepatic glucose synthesis, and accelerated fat breakdown are all caused by insulin resistance, which has been linked to higher plasma levels of free fatty acids and proinflammatory cytokines.

Type 1 diabetes mellitus: A condition known as type 1 diabetes mellitus (T1DM) is caused due to the autoimmune destruction of endocrine pancreatic cells. Insulin secretion decreases due to the loss of β-cell mass until there is insufficient insulin left to sustain normal blood glucose levels. Hyperglycemia develops and diabetes may be diagnosed once 80–90% of the beta cells have been damaged. To correct this catabolic situation, avoid ketosis, reduce hyperglucagonemia, and restore normal lipid and protein metabolism in patients, exogenous insulin is required. Patients who have autoimmune conditions such Graves disease, Hashimoto thyroiditis, and Addison disease are more likely to develop type 1 diabetes.

Diagnostic Evaluation

Diagnostic Criteria for Diabetes Mellitus and Impaired Glucose Regulation (Table 8.9)

TABLE 8.9: Diagnostic criteria for diabetes mellitus and impaired glucose regulation

Test	Normal	Impaired Glucose Regulation	Diabetes
FPG [mg/dL (mmol/L)]	<100 (< 5.6)	100–125 (5.6–6.9)	≥126 (≥7.0)
OGTT [mg/dL (mmol/L)]	< 140 (<7.7)	140–199 (7.7–11.0)	≥200 (≥ 11.1)
HbA1c(%)	< 5.7	5.7–6.4	≥6.5

FPG = Fasting plasma glucose; HbA1c = glycosylated Hb; OGTT = Oral glucose tolerance test, 2-h glucose level.

Clinical Manifestations

The most common symptoms of DM are:

- Polyuria, polydipsia, and polyphagia, together with lassitude, nausea, and impaired vision, are the most typical signs of type 1 diabetes mellitus (DM).
- Osmotic diuresis as a result of hyperglycemia leads to polyuria.
- Muscle wasting from the catabolic state of insulin insufficiency, hypovolemia, and hypokalemia can result in fatigue and weakness
- Patients with new-onset type 1 DM will gradually lose weight as a result of water loss, a catabolic state with decreased glycogen, proteins, and triglycerides, and water loss.

Management

Aim of treatment is to normalize the blood glucose level and insulin availability (Fig. 8.40). The diabetes management included following strategies.

- Nutritional management
- Exercise
- Pharmacological management
- Lifestyle modification by education.

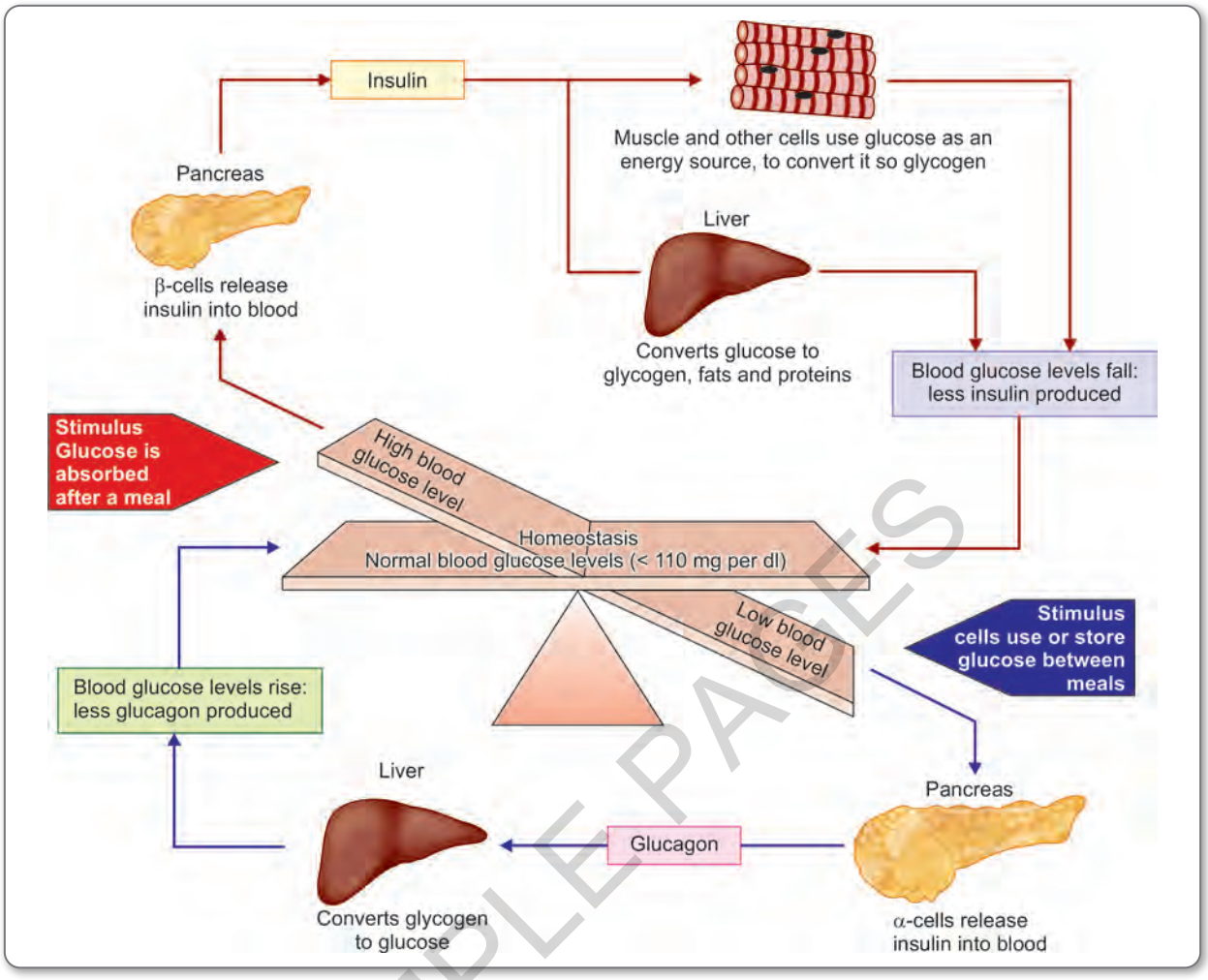


Fig. 8.40: Mechanism to maintain normal blood glucose level

Nutritional Management

Nutritional management is most important step in diabetes, control the calorie intake to achieve targeted body weight and blood glucose level. Food should contain all essential vitamins and minerals. Patients on insulin therapy should control blood glucose level to normal, should have consistency in amount of calorie and carbohydrate intake. Also consistency in meal time should be maintained. Long term patients should be educated regarding diet, life style and counseling about nutrition should be encouraged.

Planning a Meal

While meal is planned, give preference to patient's food preferences, usual eating time and food culture (Fig. 8.41). Take a complete diet history of the patient. Nurse should communicate patient's dietary preferences to the dietitian. Patient has to be educated regarding diet, importance of consistence eating habits the relationship of food and insulin and need for individual meal plan. Initially patient may have

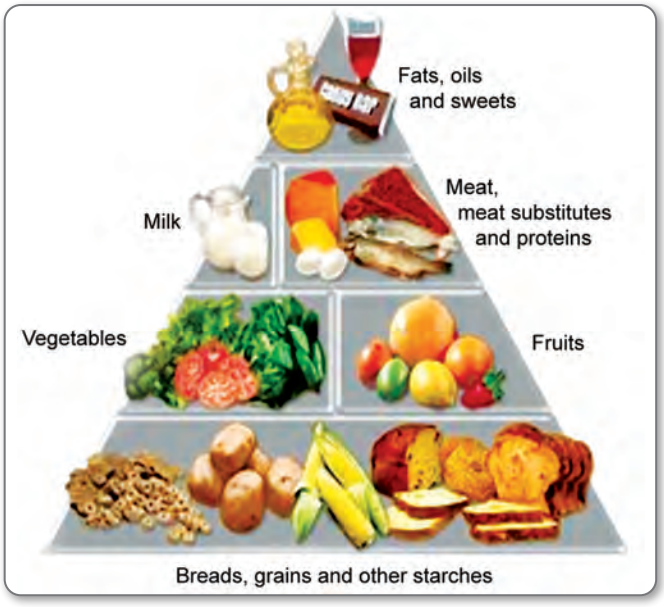


Fig. 8.41: Food pyramid for diabetes patients

difficulty in learning food exchange system. It is important to give education in simple form and at their understanding level.

Caloric Requirement

Calorie requirement is calculated based on age, gender, height and weight. The calorie is divided into carbohydrates, proteins, and fats. Meal is planned according to patient's lifestyle and food preferences. For all patients, individual patient should work closely with registered dietitian to achieve individualized goal. ADA recommended 50–60% calorie from carohydrates, 20–30% from fat, and remaining 10–20% from protein. All carbohydrates should be eaten in moderate amount.

- **Fat:** Nutrition guide line recommended that 30% of the total calorie should be derived from fat. Saturated fat is limited. Only 10% of total calorie is taken from saturated fat.
- **Protein:** The meal plan may include some nonanimal sources of protein(legume, whole grain). Protein in food is restricted for patients showing signs of renal disease.
- **Fiber:** High fiber diet is recommended. Increased fiber included in diet may improve level of glucose in the blood. Fibers should be included in the diet plan slowly, sudden increase in fiber in diet may require adjustment in insulin dose to prevent hypoglycemia. Fiber rich food should be chosen from vegetable, fruit, and starch

Exercise

Regular exercise is very important part of diabetes management. Exercise helps cells to be more sensitive to insulin so it can work more efficiently. So, exercising consistently can lower blood glucose and improve A1c diabetics range (Fig. 8.42).

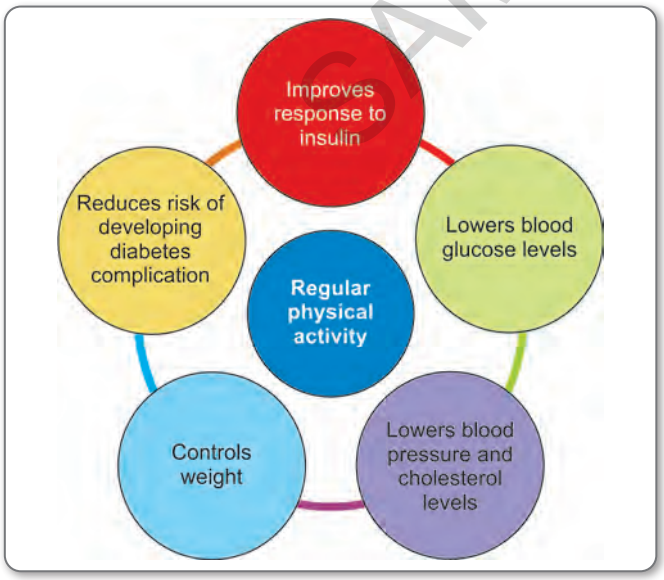


Fig. 8.42: Benefits of regular exercise in diabetes

Evaluation of the patient before exercise: Before starting exercise program, clients with diabetes mellitus have a detailed medical evaluation with appropriate diagnostic studies. Client should be evaluated for the presence of macro- and microvascular complications, Identification of areas of concern will allow the design of an individualized exercise prescription that can minimize risk to the patient.

Guide to diabetes and exercise are: A careful medical history and physical examination should focus on the symptoms and signs of disease affecting the heart and blood vessels, eyes, kidneys, feet, and nervous system.

Preparing for exercise

- It is generally advised that physical exercise should be followed by a suitable warm-up and cool-down period.
- A warm-up should last 5–10 minutes and involve low-intensity aerobic exercise (walking, cycling, etc.).
- The purpose of the warm-up session is to get the heart, lungs, and skeletal muscles ready for gradually increasing the intensity of the exercise.
- Muscles should be gradually stretched for further 5–10 minutes after a brief warm-up.
- Stretching should focus primarily on the muscles engaged during the vigorous physical activity session, but warming up all muscle groups is ideal.
- Maintaining proper hydration is also important, since dehydration can negatively impact heart health and blood sugar levels.
- For maintaining or improving upper body strength in nearly all diabetic individuals, moderate weight training programs with light weights can be performed.

Exercise and type 2 diabetes

Numerous type 2 diabetes patients may benefit from physical exercise, and new research has emphasized the value of long-term physical activity programs for the treatment and prevention of this widespread metabolic disorder and its complications.

Evidence Based Practice

Numerous long-term studies have shown that regular physical activity training consistently improves insulin sensitivity and glucose metabolism, effects that can last for at least 5 years. The physical activity regimen utilized in these investigations were at a maximum intensity of 50–80% three to four times a week for 30–60 minutes per session. 10–20% improvement in HbA1c was evidenced from the baseline and was noticeable in type 2 diabetic patients with mild disease.

Exercise and Type 1 Diabetes

People with type 1 diabetes who have good blood glucose control and do not have complications can engage in all types of physical activity, including recreational sports, competitive professional performance, and leisure activities.

Following is a summary of general recommendations that may be useful in controlling the glycemic response to physical activity:

- Exercise should be avoided if fasting glucose levels are above 250 mg/dL and ketosis is present; if these levels are above 300 mg/dL but ketosis is not, exercise should be avoided.
- If blood glucose levels are less than 100 mg/dL, consume additional carbohydrates.
- Monitoring blood sugar levels before and after the exercise can help to determine whether a patient needs to modify the diet.
- Recognize the glycemic reaction.

Patient should take additional carbohydrates to prevent hypoglycemia, carbohydrate-based foods should be readily available during and after physical activity.

Exercise in the Elderly

Evidence suggests that regular physical activity can promote fitness, improves muscle mass, and strength that occurs with ageing. A number of recent studies of exercise training have included significant numbers of older diabetes patients. These patients have done well with good training and metabolic responses, levels of adherence and prevent incidence of complications.

Monitoring

Monitoring of blood glucose is very essential and important for diabetes management. Client should be educated to monitor blood glucose.

- **Self-monitoring of blood glucose (SMBG)** has proved beneficial for blood glucose level control. If patient can measure blood glucose and able to respond to the result they will be able to adjust treatment regimen to obtain optimal blood glucose control. Client will be able to detect and prevent hypoglycemia and hyperglycemia. This will reduce the development of long term complication of hyperglycemia.

  For all patients on insulin therapy SMBG is recommended four times a day (before meal and bed time). Patents on oral hypoglycemic drugs should assess their blood glucose at least two or three times per week.
- **Continuous glucose monitoring system (CMS):** In this method, continuously blood glucose is monitored. A sensor attached to an infusion set, similar to an infusion set, is inserted subcutaneously in the abdomen and connected to the device worn on a belt. After 72 hours, download the data from the device and blood glucose levels are analyzed.
- **Glycated hemoglobin or glycosylated hemoglobin—HbA1c, or A1c:** This is a blood test where we can assess average blood glucose levels over a period of 2- 3 months. In hyperglycemia, glucose molecules bind to hemoglobin. As long as the blood glucose level is high more glucose molecules bind to hemoglobin and value of A1c will be higher. Normal range of Hb AIc is 4–6%. Normal range indicates consistently near normal blood glucose.
- **Urine glucose testing:** Previously urine testing for glucose was only test for diabetes for daily assessment. Urine glucose testing is no longer used.
- **Testing for ketones:** Ketone in urine is a high risk for DKA (Diabetes Ketoacidosis). Most commonly ketones are assessed by using a dipstick. If a patient with type 1 diabetes having glycosuria or persistently high blood glucose (more than 240 mg/dL). One should perform urine testing for ketone.

Pharmacologic Therapy

Oral Anti-Diabetic Agent

There are currently four classes of oral antidiabetic agents:

1. Biguanides
2. Insulin Secretagogues—Sulfonylureas;
   Insulin Secretagogues—Non-sulonylureas
3. α-glucosidase inhibitors
4. Thiazolidinediones (TZDs)

Oral agent monotherapy

If glycemic control is not achieved (HbA1c >6.5% and/or; Fasting plasma glucose >7.0 mmol/L or; Random plasma glucose >11.0 mmol/L) with lifestyle modification within 1–3 months, oral anti-diabetic agent should be initiated.

In the presence of marked hyperglycemia in newly diagnosed symptomatic type 2 diabetes (HbA1c >8%, FPG >11.1 mmol/L, or RPG >14 mmol/L), oral anti-diabetic agents can be considered at the outset together with lifestyle modification. Metformin is the drug of choice in overweight/obese patients. TZDs and acarbose should be advised in the patients who are intolerant to metformin.

Combination oral agents

Combination oral agents are recommended in newly diagnosed symptomatic patients with HbA1c >10 and patients who are unable to achieve target objective of HbA1c after 3 months on monotherapy.

- **Combination of oral agents and insulin:** Consider adding intermediate acting or long-acting insulin if targets are not met after receiving combination therapy at the recommended dose for 3 months.
- Combining oral antidiabetic medications with insulin has been demonstrated to enhance glycemic control in people who are unable to reach their target blood sugar levels despite taking the highest dosage possible.

Diabetes Management Algorithm

The management of diabetes is shown as an algorithm in Figure 8.43 and oral hypoglycemic medications are completed in Table 8.10.

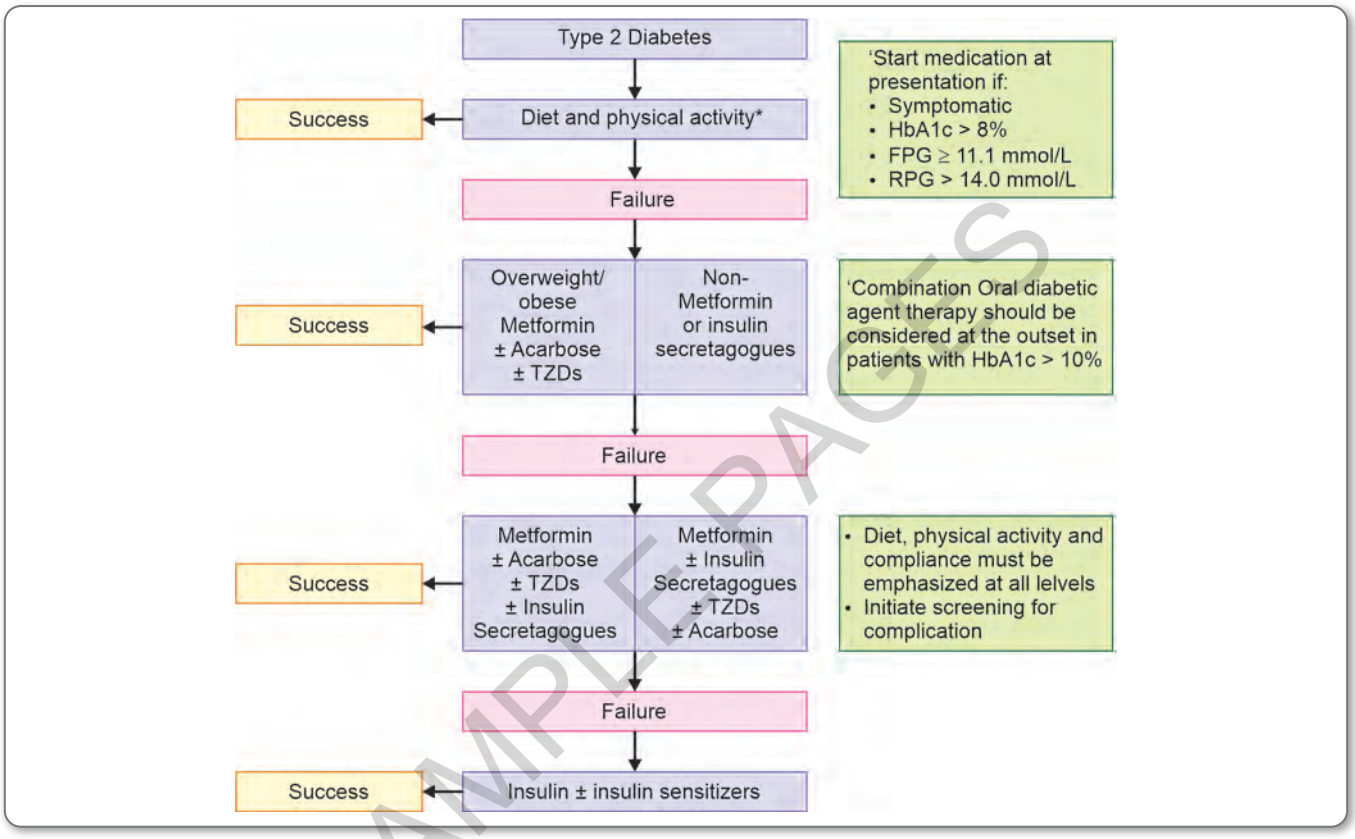


Fig. 8.43: Oral antidiabetic drugs

Oral Hypoglycemic Medications (Table 8.10)

TABLE 8.10: Hypoglycemic agents and their actions

Drug Class	Drug Name	Brand Class	Mechanism of Action
Biguanides	Metformin	Glucophage®	Inhibits glucose production by the liver
Sulfonylureas (second-generation)	Glimepride Glipizide Glyburide	Amaryl® Glucotrol® Diabeta® Glynase PresTab® Micronase®	Increases insulin secretion by pancreatic beta cells
Meglitinides	Repaglinide Nateglinide	Prandin® Starlix®	Increases insulin secretion by pancreatic beta cells
Thiazolidinediones (TZDs)	Pioglitazone Rosiglitazone	Actos® Avandia®	Increases glucose uptake by skeletal muscles
α-glucosidase inhibitors	Acarbose Miglitol	Precose® Glyset®	Inhibits carbohydrate absorption in the small intestine

General recommendation for use of oral antidiabetic agent in diabetes

- Long-acting sulfonylureas should be avoided in older non-obese patients, although short acting insulin secretagogues can be started.
- Monitoring of renal function is necessary. Diabetes during pregnancy is not advised to be treated with oral anti-diabetic medications.
- When diabetes is discovered after a stressful event, such as an infection, oral anti-diabetic medications are typically not the primary line of treatment. Both of the aforementioned conditions require insulin therapy.
- For all age groups, there are targets for control of blood glucose levels. Targets should be individualized and are tailored for patients with comorbidities.
- When necessary, start with a low dose of an oral anti-diabetes medication while emphasizing the need of a healthy diet and regular exercise. To establish steady state blood glucose management, a suitable interval between increments (2–16 weeks, depending on the agents utilized) should be provided.

Insulin Therapy

Insulin metabolizes glucose when glucose increases in blood after meal. In type 1 diabetes body is unable to produce insulin, hence insulin has to give be from outside lifelong.

Use of Insulin

Short-term use

- Acute illness, surgery, stress and emergencies
- Pregnancy
- In cases of severe metabolic decompensation (diabetic ketoacidosis, hyperosmolar nonketotic coma, lactic acidosis, and severe hypertriglyceridemia), significant hyperglycemia, and hyperosmolar nonketotic coma, insulin may be administered as the first line of treatment for type 2 diabetes.

Long-term use: Consider changing to multi-dose insulin therapy if objectives are not met after the best dose of combination therapy or BIDS. Insulin secretagogues should be stopped before starting this, although insulin sensitizers like Metformin or TZDs can still be used.

Insulin regimens: If effective glycemic control is to be accomplished, the majority of patients will need more than one daily injection. However, some people may benefit from a once-daily injection of a medication with an intermediate acting insulin. A common treatment is a twice-daily combination of short- and intermediate-acting insulin. A combination of short- and intermediate-acting insulin may be administered in the morning.

Short-acting insulin doses are administered, along with an evening dosage of intermediate-acting insulin just before bed. When strict glycemic control is required, a routine of repeated short-acting insulin injections before the main meals and a suitable amount of intermediate-acting insulin should be administered at bedtime.

Insulin is classified in several groups depending on onset, peak and duration of action (shown in Table 8.11)

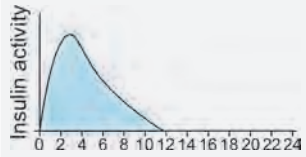
TABLE 8.11: Various types of insulin

Insulin Type/Action (Appearance)	Brand Names (Generic Name in Brackets)	Basal/Bolus	Dosing Schedule
Rapid-acting analog (clear) Onset; 10–15 minutes Peak 60–90 minutes Duration: 4-5 hours	Humalog® (Insulin lispro) NovoRapid® (insulin aspart)	Bolus	Usually taken right before eating or to lower high blood glucose
Short-acting (clear) Onset: 0.5–1 hour Peak 2–4 hours Duration: 5–8 hours	Humulin®-R Novolin®ge Toronto	Bolus	Taken about 30 minutes before eating. or to lower high blood glucose
Intermediate-acting (cloudy) Onset: 1–3 hours Peak 5–8 hours Duration: up to 18 hours	Humulin®–N Novolin®ge NPH	Basal	Often taken at bedtime. or twice a day (morning and bedtime)
Extended long-acting analog (Clear and colorless) Onset: 90 minutes Peak none Duration: 24 hours	Lantus® (Insulin glargine) Levermir® (insulin detemir)	Basal	Usually taken once or twice a day
Premixed (cloudy) A single vial contains a fixed ratio of insulins (the numbers refer to the ratio of rapid- or fast-acting to intermediate-acting Insulin in the vial)	Humalog® Mix 25th. Humulin® (20/80.30/40) Novolin®ge (10/90, 20/80, 30/70, 40/60, 50/50)	Combination of basal and bolus Insulin	Depends on the combination

Overview of Insulin and Action (Fig. 8.44)

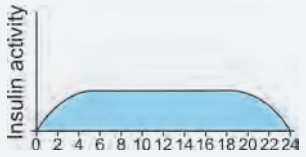
Soluble human insulin: Actrapid, Humulin S

Onset: 30 minutes
Peak: 2–4 hours
Duration: 6–8 hours



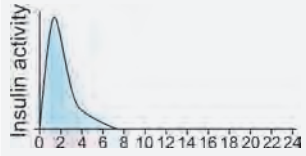
Long-acting basal analogs: Glargine (Lantus), detemir (Levemir)

Onset: ~ 2 hours
Peak: None
Duration: 18–24 hours



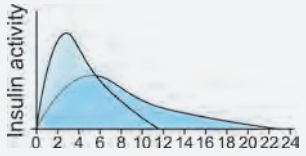
Rapid-acting insulin analog: NovoRapid aspart, human lispro, Apidra

Onset: 0–15 minutes
Peak: 1–2 hours
Duration: 3–5 hours



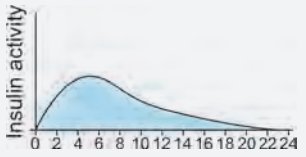
Pre-mixed human soluble/isophane: Mixtard 30, humulin M3, etc.

Onset: See above
Peak: See above
Duration: See above
Mixtard 30, M3 refers to % of soluble insulin, i.e., 30% soluble 70% isophane



Intermediate human isophane insulin's Insulatard, humulin I

Peak: 4–8 hours
Duration: 14–16 hours



Pre-mixed human soluble/isophane: Mixtard 30, humulin M3 etc

Onset: See above
Peak: See above
Duration: See above
NovoMix 30, Humalog Mix50/ Mix25 refers to % of rapid acting analog insulin

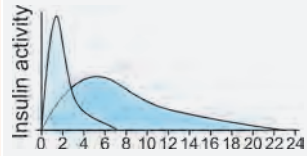


Fig. 8.44: An overview of insulin

Nursing Alert

Read the level before administering insulin to avoid mistake.

The insulin is injected in the sites shown in Figure 8.45

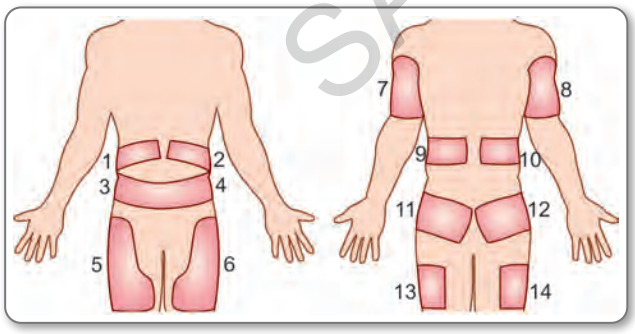


Fig. 8.45: Sites for insulin injection

Methods of Administering Insulin

Insulin is usually given subcutaneously by single-use syringes with needles, an insulin pump, or by repeated-use insulin pens with needles (Figs 8.46 to 8.49). Patients who wish to reduce repeated skin puncture of insulin injections often use an injection port in conjunction with syringes.

Administration schedules often attempt to mimic the physiologic secretion of insulin by the pancreas. Hence, both long-acting insulin and short-acting insulin are typically used.

Insulin Pump

Insulin pumps offer improved control over basal doses calculated to fractions of a unit, background or "basal" insulin dosage, and internal calculators may be useful for calculating "bolus" infusion dosages. Insulin pumps may resemble "electrical injectors" attached to a catheter or cannula that has been temporarily implanted.

Compared to traditional (or jet) injection, a pump makes it easier to establish sufficient glucose control.

In addition to the risk of infection and ulceration posed by indwelling catheters, certain patients may also experience lipodystrophy as a result of infusion settings. Infusion sites can frequently be kept clean to reduce these risks. The proper use of an insulin pump requires caution and effort.

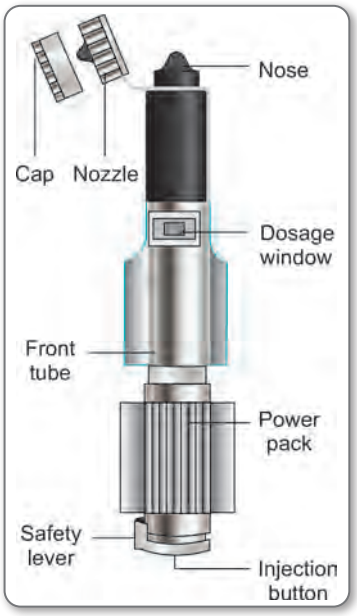


Fig. 8.46: Insulin pen

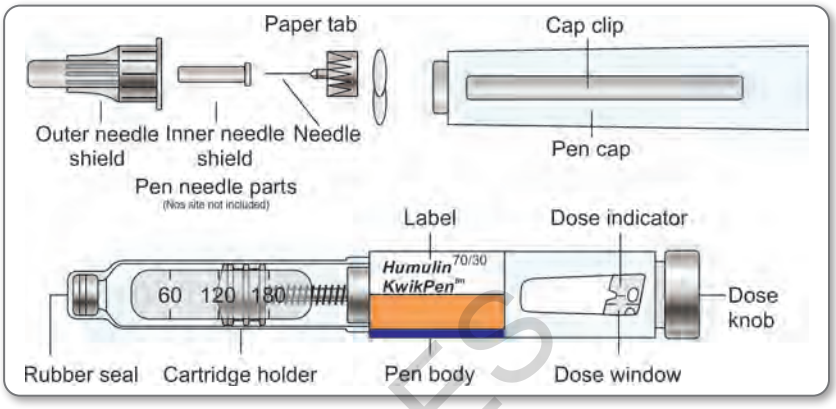


Fig. 8.47: Jet injection

Fig. 8.48: Insulin syringe

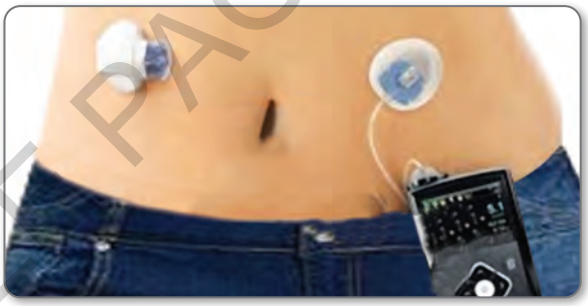


Fig. 8.49: Insulin pump

Nursing Care Plan

Client with Diabetes

Assessment	Nursing Diagnosis	Goal	Implementation	Scientific Principles	Evaluation
Assess the level of knowledge and ability to perform self-care	Deficient knowledge related to disease condition, exercise regimen and dietary management.	Patient will be willing to learn about self-care exercises and dietary management	• Assess clients understanding of diabetes management • Explain to the client and family basic pathophysiologic mechanism of diabetes and how to manage.	Client will be able to correlate pathophysiology with management of problem.	Client will plan a diet plan, exercise plan after explanation about diabetes management.
Assess for dietary pattern and preferences	Imbalanced nutrition less than body requirement related to metabolic disorder	To improve the nutritional pattern of the patient.	• Diet should be planned in consultation with dietitian to control the glucose levels • Keep in mind clients food preference. • Clients nutrition intake is monitored carefully along with blood glucose, urine ketone and daily weight		Achieve blood glucose control

Assessment	Nursing Diagnosis	Goal	Implementation	Scientific Principles	Evaluation
Assess for skin integrity	Risk for impaired skin integrity related to sensory impairment over the bony prominences secondary to neuropathy.	Maintenance of skin integrity	• Skin should be assessed frequently for dryness and break. The feet should be cleaned with warm water and soap. • Excessive soaking of the feet should be avoided. The feet are dried thoroughly, especially between the toes, and lotion is applied to the entire foot. Except between the toes.		• No signs of impaired skin integrity will be visible on assessment. • Patient will be able to understand and demonstrate foot care
Assess the level of knowledge about complication	Risk for injury related to complications such as diabetes ketoacidosis, hyperglycemia	Patient will be willing to learn the ways of preventing complications.	• Monitor blood glucose level and insulin should be administered to Control blood glucose level. • Treatment is given for hypoglycemia (with oral glucose), or hyperglycemia (with supplemental regular insulin often than every 3–4 hours.		• Achieve optimal control of blood glucose • Avoids extreme of hypoglycemia and hyperglycemia.
Assess the risk of long term micro vascular complication	Risk for injury related to potential macrovascular complications such as retinopathy, nephropathy and neuropathy	Patient will be willing to learn the ways of preventing complications.	• Explain yearly or more frequent eye examination, early treatment along with good control of blood glucose and blood pressure may prevent visual loss from retinopathy.		

Complications of Insulin Administration

Hypoglycemia

Hypoglycemic reactions, the most common complication of insulin therapy, may result from delay in taking a meal or unusual physical exertion. With more type 1 patients attempting "tight" control, this complication has become even more frequent. In older diabetics, in those taking only longer-acting insulins, and often in those attempting to maintain euglycemia on infusion pumps, autonomic counter regulatory responses are less readily elicited during hypoglycemia, and central nervous system dysfunction may occur, i.e., mental confusion, bizarre behavior, and ultimately coma.

Insulin Allergy

Insulin allergy, also known as immediate-type hypersensitivity, is a rare disorder in which tissue mast cells that have been sensitized by anti-insulin IgE antibodies release histamine, causing local or systemic urticaria. Anaphylaxis occurs in severe cases. Extremely few people have an allergy to insulin. Antihistamines, corticosteroids, and even desensitization may be needed for treatment, particularly in cases of systemic hypersensitivity.

Lipodystrophy at Injection Site

Subcutaneous fat tissue may atrophy at the injection site, resulting in disfiguring excavations and depressed areas. The complications develop due to an immune reaction, which is rare in case of pure insulin preparations.

Restoration of normal contours frequently occurs when these preparations are injected directly into the atrophic region.

Lipohypertrophy results from insulin's pharmacologic actions being repeatedly deposited in the same area. Rotation of injection site will prevent lipohypertrophy.

Complications of Diabetes Mellitus

In patients with diabetes mellitus (DM), years of poorly controlled hyperglycemia lead to multiple, primarily vascular, complications that affect small vessels (microvascular), large vessels (macrovascular), or both.

Immune dysfunction is another major complication and develops from the direct effects of hyperglycemia on cellular immunity (Fig. 8.50).

- Microvascular complications are:
  - Retinopathy

Fig. 8.50: Foot ulcer

- Nephropathy
- Neuropathy

Microvascular disease may also impair skin healing, so that even minor breaks in skin integrity can develop into deeper ulcers and easily become infected, particularly in the lower extremities. Intensive control of plasma glucose can prevent or delay many of these complications but may not reverse them once established.

Macrovascular complications are:

- Angina pectoris and myocardial infarction
- Transient ischemic attacks and strokes
- Peripheral arterial disease

Another serious issue is immune dysfunction, which results from the direct impact of hyperglycemia on cellular immunity. Patients with diabetes are more prone to bacterial and fungal infections.

Diabetic Retinopathy

Diabetic retinopathy is the most frequent cause of adult blindness. Initial symptoms include retinal capillary microaneurysm, which is followed by neovascularization (proliferative retinopathy) and macular edema. However, focal blurring, vitreous or retinal detachment, and partial or complete vision loss ultimately appear (Fig. 8.51). There are no early symptoms or indicators.

To prevent eyesight loss, early detection and treatment is essential. Patients with both type 1 and type 2 diabetes should have regular (often annual) retinal examinations for screening and diagnosis. Blood pressure and glucose control should be done very strictly to prevent complications. Pan retinal laser photocoagulation or, less frequently, vitrectomy may be necessary for more severe retinopathy. Inhibitors of vascular endothelial growth factor (VEGF) are promising novel treatments for proliferative retinopathy and retinal edema.

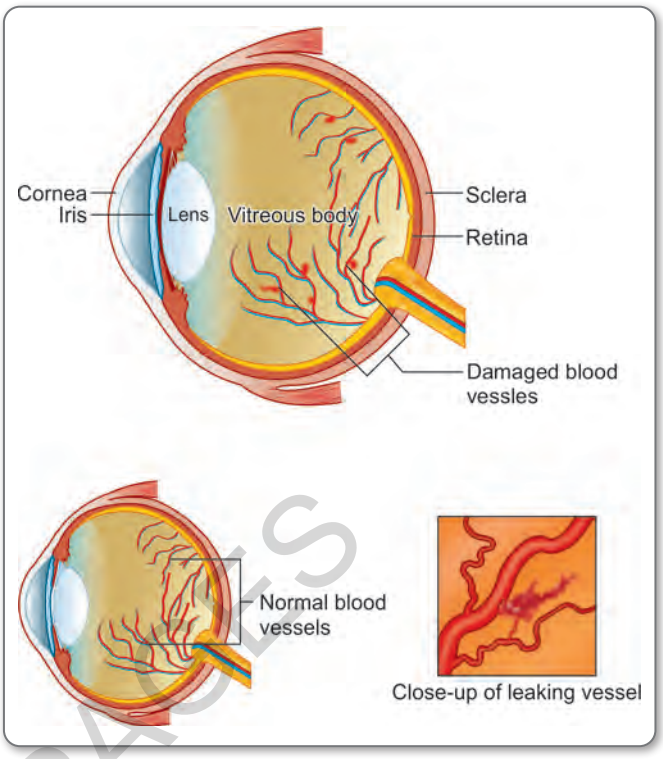


Fig. 8.51: Diabetic retinopathy

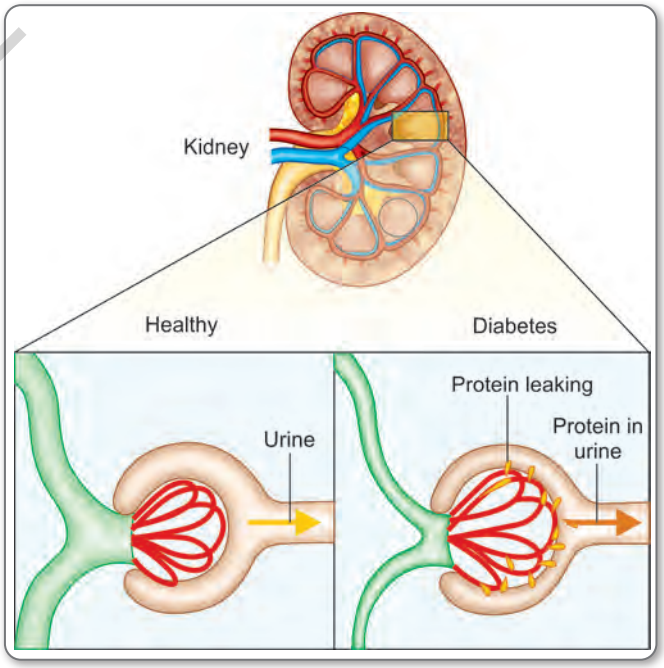


Fig. 8.52: Diabetic nephropathy

Diabetic Nephropathy

Diabetic nephropathy is a leading cause of chronic kidney disease among patients with diabetes mellitus (Fig. 8.52). It is characterized by thickening of the glomerular basement

membrane, mesangial expansion, and glomerular sclerosis. These changes cause glomerular hypertension and progressive decline in glomerular filtration rate.

Diagnosis is by detection of urinary albumin. Once diabetes is diagnosed (and annually thereafter), urinary albumin level should be monitored so that nephropathy can be detected early. A spot urine sample's albumin:creatinine ratio or a 24-hour collection's total urinary albumin might be used for monitoring. Early diabetic nephropathy and moderately elevated albuminuria (formerly known as microalbuminuria) are indicated by ratios >30 mg/g or albumin excretion of 30 to 300 mg/day. Severely elevated albuminuria, also known as macroalbuminuria, or overt proteinuria, is defined as an excretion of albumin greater than 300 mg/day and denotes more severe diabetic nephropathy. A urine dipstick is often only considered positive if the protein excretion exceeds 300–500 mg/day.

Treatment is rigorous, focusing on both blood pressure and glucose control. When albuminuria first appears, an ACE inhibitor or an angiotensin II receptor blocker should be used to treat hypertension and limit the progression of renal disease because these medications lower intraglomerular blood pressure and have renoprotective benefits. These medications have not been demonstrated to be helpful for initial prevention (i.e., in patients who do not have albuminuria).

Diabetic Neuropathy

The causes of diabetic neuropathy include intracellular metabolic alterations that affect nerve function, nerve ischemia brought on by microvascular illness, and direct effects of hyperglycemia on neurons (Fig. 8.53). There are numerous types, such as:

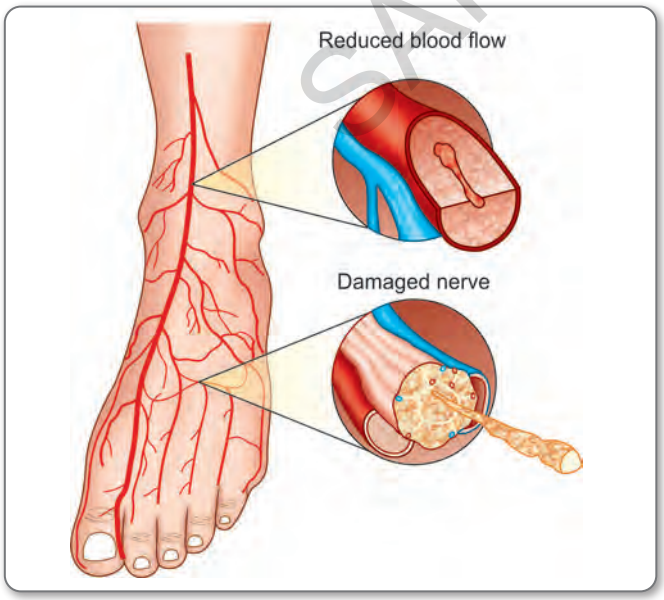


Fig. 8.53: Diabetic neuropathy

- Symmetric polyneuropathy (with small- and large-fiber variants)
- Autonomic neuropathy
- Radiculopathy
- Cranial neuropathy
- Mononeuropathy

The distal feet and hands (stocking-glove distribution) are the most frequently affected by symmetric polyneuropathy, which presents as paresthesias, dysesthesias, or a painless loss of touch, vibration, proprioception, or temperature sensation. Because of inappropriate footwear and abnormal weight bearing, these symptoms in the lower extremities may mask foot injuries, which may result in foot ulcers and infection, fractures, subluxations, dislocations, or the breakdown of healthy foot architecture (Charcot joint).

Pain, numbness, and a loss of temperature perception with intact vibration and positional sensitivity are the hallmarks of small-fiber neuropathy. Patients frequently develop autonomic neuropathy, foot ulceration, and neuropathic joint deterioration. Muscle weakness, loss of vibration and position awareness, and absence of deep tendon reflexes are characteristics of predominant large-fiber neuropathy. Foot drop and atrophy of the intrinsic muscles of the feet are prevalent.

Autonomic neuropathy can cause orthostatic hypotension, exercise intolerance, resting tachycardia, dysphasia, nausea and vomiting (due to gastroparesis), constipation and diarrhea (including dumping syndrome), fecal incontinence, urinary retention and incontinence, erectile dysfunction and retrograde ejaculation, and reduced vaginal lubrication.

Radiculopathies typically damage the proximal L2 through L4 nerve roots, which can result in lower extremity discomfort, weakness, and atrophy (diabetic amyotrophy), or the proximal T4 through T12 nerve roots, which can result in abdominal pain (thoracic polyradiculopathy).

When they impact the third cranial nerve, cranial neuropathies result in diplopia, ptosis, and anisocoria; when they affect the fourth or sixth cranial nerves, they result in motor palsies.

Finger numbness and weakness (median nerve) or foot drop are symptoms of neuropathies (peroneal nerve). Additionally, nerve compression illnesses including carpal tunnel syndrome are more common in DM patients. Multiple locations can simultaneously have mononeuropathies (mononeuritis multiplex). All have a tendency to mostly affect elderly people and typically fade away on their own over several months.

It is possible to identify symmetric polyneuropathy by examining for sensory impairments and weakened ankle reflexes. Patients with the highest risk of developing foot ulcers are those who are unable to perceive the mild touch of a nylon monofilament. As an alternative, vibratory sensation on the first toe's dorsum can be tested with a 128-Hz tuning fork.

For all types of neuropathy, electromyography and nerve conduction investigations may be required. They are also occasionally used to rule out other potential causes of neuropathic symptoms, such as nondiabetic radiculopathy and carpal tunnel syndrome.

Managing neuropathy requires a multifaceted strategy that includes pain management, frequent foot care, and glycemic control. Strict glucose management may reduce neuropathy. Tricyclic antidepressants and topical capsaicin cream are two treatments for symptom relief (e.g., amitriptyline), serotonin-norepinephrine reuptake inhibitors (e.g., duloxetine), and anticonvulsants (e.g., pregabalin, gabapentin). Patients with sensory loss should examine their feet daily to detect minor foot trauma and prevent it from progressing to limb-threatening infection.

Macrovascular Disease

Large-vessel atherosclerosis is a result of the hyperinsulinemia, dyslipidemias, and hyperglycemia. Manifestations are:

- Angina pectoris and myocardial infarction
- Transient ischemic attacks and strokes
- Peripheral arterial disease

The history and physical examination are used to make the diagnosis; screening techniques, like the coronary calcium score, are expanding in their use. Treatment involves strict atherosclerotic risk factors control, factors, such as stabilisation of plasma lipids, glucose, and blood pressure together with daily exercise, cessation of smoking and consumption of ACE inhibitors and aspirin. A multifaceted strategy involves controlling blood sugar, hypertension and dyslipidemia, which could be useful in lowering the rate of heart-related events.

Cardiomyopathy

Multiple factors, such as epicardial atherosclerosis, hypertension, left ventricular hypertrophy, microvascular disease, endothelial dysfunction, obesity, and metabolic abnormalities are known to cause diabetic cardiomyopathy. Patients are more prone to get heart failure following myocardial infarction. Heart failure is caused by deterioration in left ventricular systolic and diastolic function.

Infection

Because hyperglycemia has damaging consequences on granulocyte and T-cell functioning, patients with poorly controlled diabetes mellitus are more likely to contract bacterial and fungal infections. Diabetes patients are more susceptible to mucocutaneous fungal infections (such as oral and vaginal candidiasis) and bacterial foot infections (including osteomyelitis), which are frequently made worse by lower extremity vascular insufficiency and diabetic neuropathy. These infections can also cause mucocutaneous fungal infections (such as oral and vaginal candidiasis) and bacterial foot infections (such as amputation-related necrosis).

A well-known risk factor for surgical site infections is hyperglycemia.

Other Complications

Diabetic foot complications include: skin changes, ulceration, infection, and gangrene. These are caused by vascular disease, neuropathy, and immunosuppression. Also, various rheumatologic conditions, such as muscle infarction, carpal tunnel syndrome, Dupuytren contracture, adhesive capsulitis, and sclerodactyly, are more likely to develop in people with diabetes mellitus.

Patients of diabetes are also at risk of ophthalmologic conditions unrelated to diabetic retinopathy (such as cataracts, glaucoma, corneal abrasions, and optic neuropathy), hepatobiliary conditions (such as cirrhosis, gallstones, and nonalcoholic fatty liver disease), and dermatological conditions (such as tinea infections, lower-extremity ulcers, diabetic dermopathy). Dementia and depression are also prevalent.

Diabetic Ketoacidosis

Diabetic ketoacidosis (DKA) is an acute and life-threatening complication of diabetes, which is characterized by hyperglycemia, ketoacidosis, and ketonuria.

DKA primarily affects people with type 1 diabetes; however, it can also occur in people with type 2 diabetes. The condition is characterized by blood sugar levels more than 250 mg/dL, a blood pH below 7.3 and a spike in serum ketone levels greater than 5 mEq/L. Ketonemia and ketonuria are characteristic manifestations of DKA.

Etiology and Risk Factors

- Underlying concomitant infection (UTI)
- Poor compliance and missed insulin treatment
- Newly diagnosed diabetes mellitus
- Psychological, surgical stress
- Medications (steroids)

Pathophysiology

DKA usually occurs secondary to absolute or relative insulin deficiency which is accompanied by an increase in counter-regulatory hormones (i.e., glucagon, cortisol, growth hormone, epinephrine). This particular hormonal imbalance promotes hepatic lipolysis, glycogenolysis, and gluconeogenesis. Severe hyperglycemia is caused by an excessive amounts of hepatic gluconeogenesis, glycogenolysis, and counter-regulatory hormones, while lipolysis raises blood free fatty acids, which get accumulated. Ketonemia is the initial result of the gradual rise in these acidic organic material concentrations in the blood. Ketones overflow into urine when they aren't removed by the body quickly enough (i.e., ketonuria).

Metabolic acidosis may result if it is not addressed in a timely manner (Fig. 8.54).

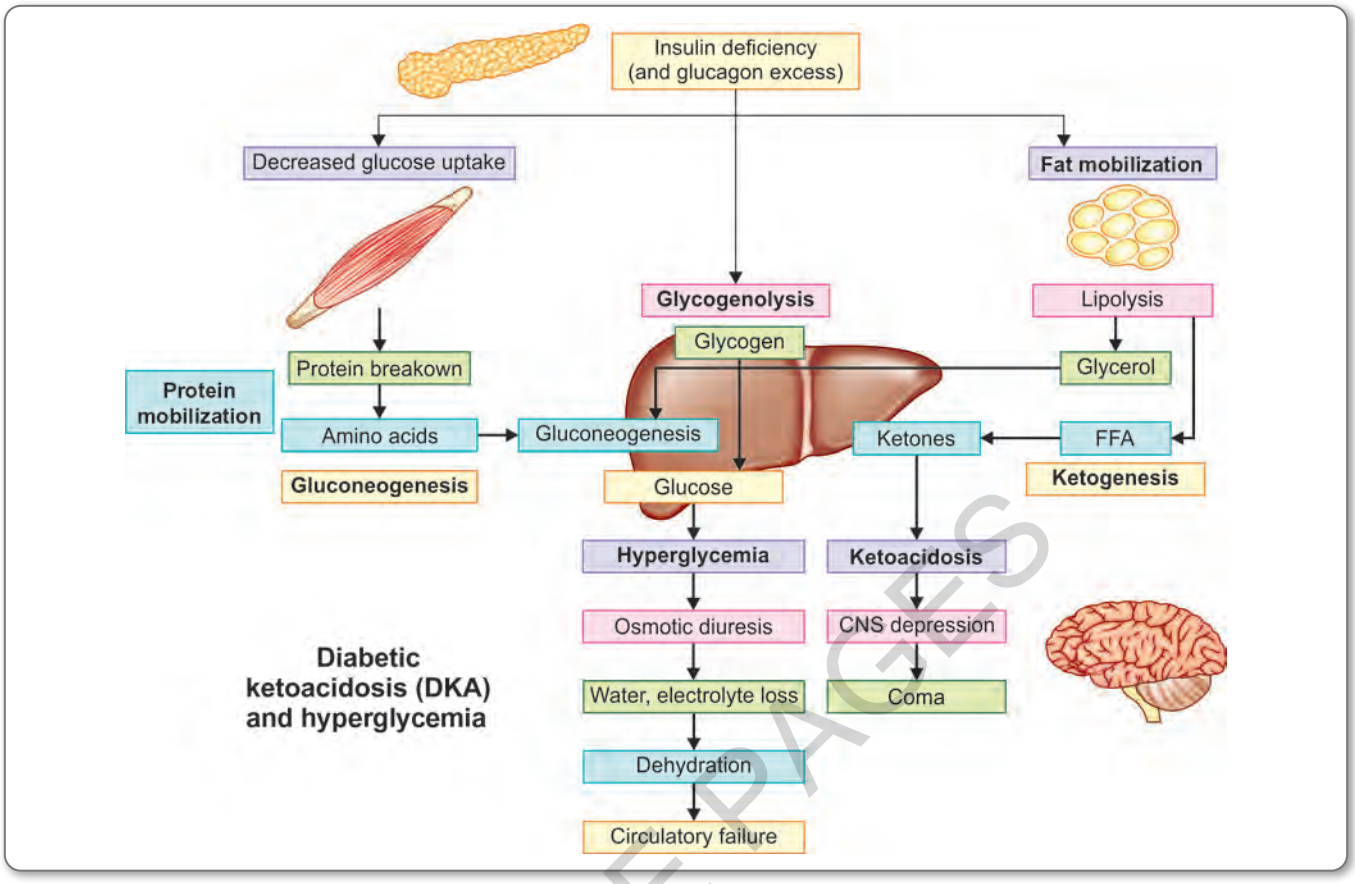


Fig. 8.54: Pathophysiology of DKA

Clinical Manifestation

Diabetic ketoacidosis manifestations include hyperglycemia symptoms with nausea, vomiting, and—especially in children—abdominal pain.

The signs of a more severe condition include lethargy and somnolence. Patients may have high blood pressure and tachycardia from dehydration and acidity, which causes rapid and deep breathing (Kussmaul respirations). Due to the acetone they exhale, they could also have fruity breath. Fever, if present, indicates an underlying infection and is not a sign of DKA itself. DKA worsens without prompt medical attention, eventually leading to coma and death.

Acute cerebral edema, which affects 1% of DKA patients, mostly affects children and occurs less frequently in teens and young adults. Some individuals experience headaches and changes in their state of consciousness as the first signs of this condition, but for others, respiratory arrest is the first symptom.

Although the exact etiology is unknown, it may be linked to abrupt decreases in serum osmolality or brain ischemia. When DKA is the first sign of diabetes mellitus, it is most likely to occur in children under the age of five. The children who are at greatest risk at presentation have the lowest Paco2 and highest blood urea nitrogen levels. Additional risk factors include the use of bicarbonate during the treatment of DKA and delays in the correction of hyponatremia.

Diagnosis

- **Laboratory studies for DKA includes:** Electrolyte abnormalities such as hyponatremia, elevated serum creatinine, and elevated plasma osmolality can be seen in lab studies.
- **Blood tests for glucose every 1-2 h until patient is stable:** Patients with DKA typically have blood sugar levels higher than 250 mg/dL.
- **Urine dipstick test:** The urine dipstick test is positive for both glucose and ketones in DKA patients.
- **Serum electrolyte determinations:** In patients with DKA, serum potassium levels are initially high or within the normal ranges. This is caused by the extracellular transfer of potassium in exchange for hydrogen that accumulates in acidosis. Patients that are affected typically have low serum sodium levels. Additionally, these patients always have low levels of serum phosphate and chloride.

To determine the level of acidosis that is present, bicarbonate levels and the anion gap should be measured together.

- **Initial arterial blood gas (ABG) measurements:** ABG analysis from DKA patients frequently displays the classic signs of metabolic acidosis, low bicarbonate levels, and low pH (less than 7.3).

Prognosis

The fatality rate for diabetic ketoacidosis is less than 1% overall, but it is higher in the elderly and in people who have other serious conditions. A worse prognosis is indicated by shock or coma upon admission. Infection, hypokalemia, and circulatory collapse are the three main causes of mortality.

Strategies for Management of DKA

- IV 0.9% saline
- Correction of hypokalemia
- IV insulin (as long as serum potassium is ≥ 3.3 mEq/L)
- Rarely IV sodium bicarbonate (if pH <7 after 1 h of treatment)

Rapid intravascular volume replacement, correction of hyperglycemia, acidosis and prevention of hypokalemia are the most urgent treatment goals for managing diabetic ketoacidosis.

Intensive care facilities should be utilized for treatment because patient requires intensive clinical and laboratory assessment as well as close monitoring.

Volume Repletion

To increase blood pressure and maintain glomerular perfusion, intravascular volume should be quickly restored. Once intravascular volume is restored, remaining total body water imbalances are corrected more gradually, often over the course of around 24 hours. Adults often achieve initial volume replenishment with rapid IV infusions of 1–3 L of 0.9% saline solution, followed by saline infusions at 1 L/h or faster as indicated to elevate blood pressure, treat hyperglycemia, and maintain adequate urine output. Over the course of the first 5 hours, adults with diabetic ketoacidosis often require at least 3 L of saline. Normal saline is replaced with 0.45% saline when blood pressure is steady and urine flow is satisfactory.

A change to 5% dextrose in 0.45% saline should be made to the IV fluid when plasma glucose decreases to less than 200 mg/dL (11.1 mmol/L).

Correction of Hyperglycemia and Acidosis

Regular insulin is administered as a 0.1% saline solution continuous IV infusion after an initial 0.1% unit/kg IV bolus to treat hyperglycemia. Preflushing the IV tubing with insulin solution helps reduce the unpredictable effects that insulin adsorption can have. Insulin doses should be doubled if plasma glucose does not decrease by 50–75 mg/dL (2.8–4.2 mmol/L) within the first hour.

Children should receive a 0.1 unit/kg/h or higher continuous IV insulin infusion, with or without a bolus.

Adults should be administered 5% dextrose in IV fluids when their plasma glucose falls below 200 mg/dL (11.1 mmol/L) to lower their risk of hypoglycemia. Once the anion gap has reduced and blood and urine consistently test negative for ketones, the amount of regular insulin given intravenously can be decreased to 0.02–0.05 units/kg/h. Then, regular insulin 5–10 units SC q 4–6 hours may be used as an alternative to insulin replacement. Once the patient is stable and able to eat, a basal-bolus or split-mixed insulin regimen is started. Following the administration of the first dosage of subcutaneous insulin, IV insulin should be continued for 1–4 hours.

Hypokalemia Prevention

Serum potassium should be maintained between 4 and 5 mEq/L by replacing 20–30 mEq of potassium in each liter of intravenous fluid. Insulin should be withheld if serum potassium is below 3.3 mEq/L, and potassium should be given at a rate of 40 mEq/h until blood potassium is below 3.3 mEq/L. If serum potassium is higher than 5.3 mEq/L, potassium supplementation should be withheld.

Initial serum potassium levels that are normal or increased could signify changes in intracellular potassium levels in response to acidemia and mask the true potassium deficits that practically all patients with diabetic ketoacidosis have. In the early phases of treatment, levels should be examined regularly or every other hour because insulin replacement quickly shifts potassium into cells.

Other Measures

During the course of treating DKA, hypophosphatemia frequently arises; however, phosphate replacement is typically of questionable use.

Potassium phosphate, 1–2 mmol/kg of phosphate, can be administered over 6–12 hours if necessary (for example, if rhabdomyolysis, hemolysis, or neurologic deterioration ensues). The serum calcium level typically decreases when potassium phosphate is administered; therefore, this should be watched for.

Hypoglycemia (Low Blood Glucose)

A decrease in plasma glucose concentration that causes symptoms or signs including impaired mental status and/or sympathetic nervous system stimulation is known as hypoglycemia.

Etiology

People with type 1 diabetes and type 2 diabetes frequently experience low blood sugar. A person with type 1 diabetes may encounter up to two mild low blood glucose episodes per week on average.

In comparison to NPH (isophane insulin) and conventional insulin, newer insulins are recommended because they are less likely to result in low blood glucose levels, especially overnight. Risk of hypoglycemia may also be decreased with insulin pumps.

A change in food, an infection, a medicine overdose, a change in exercise level, or a change in metabolism can all cause hypoglycemia. In addition to other diverse etiologies, additional causes include digestive issues, idiopathic causes, fasting, insulinoma, endocrine issues, extrapancreatic causes, hepatic illness, and bariatric surgery.

Low blood glucose can also be caused by accidentally injecting the incorrect insulin type, administering excessive amounts of insulin, or injecting insulin into the muscle rather intradermally.

Clinical Manifestation

Symptoms of hypoglycemia may be categorized as neurogenic (adrenergic) or neuroglycopenic.

Sweating, trembling, tachycardia, anxiety, and a sense of hunger are all symptoms of sympathetic activation. Symptoms of neuroglycopenia include weakness, exhaustion, or vertigo, inappropriate behavior (which is occasionally misdiagnosed as drunkenness), difficulties concentrating, disorientation, impaired vision, and, in severe cases, coma and death.

In the morning before eating or throughout the day, especially in the afternoon if meals are skipped or postponed, fasting hypoglycemia usually occurs.

2–4 hours after a meal, postprandial hyperglycemia frequently develops, especially if the meal contains a lot of simple carbohydrates.

Hypoglycemia causes nonspecific physical symptoms that are typically linked to the central and autonomic nervous systems.

Patient should be assessed for hypothermia, tachypnea, tachycardia, hypertension, and bradycardia.

Treatment

Glucose is the cornerstone of hypoglycemia treatment. Depending on the underlying cause or the associated symptoms, other drugs might be given.

- **Fasting low blood sugar:** Dietary therapy may be useful for treating people with fasting hypoglycemia's symptoms. Complex carbohydrates are preferred at frequent meals and snacks, especially at night.

  Intravenous (IV) glucose infusion can be used as part of medical treatment for patients with fasting hypoglycemia if nutritional therapy is insufficient. The endogenous insulin secretion can be effectively suppressed by IV octreotide.
- **Reactive hypoglycemia:** In patients with reactive hypoglycemia, refined carbohydrates should be restricted. Simple sugars should be avoided, meal frequency should be increased, and meal size should be decreased for patients. Patients might need six small meals and two to three snacks each day. A meal with more protein and fiber may be useful.
- **The 15-15 rule:** The 15-15 rule states that patient with hypoglycemia can be given 15 g of carbohydrate and blood glucose should be checked after 15 minutes of providing carbohydrate. Patient should be given another serving if blood glucose level is still under 70 mg/dL.

  Once the blood glucose levels are normalized, patients should be instructed to eat a meal or snack to ensure that it does not fall.

Management of Severe Hypoglycemia

When blood glucose levels are too low, the pancreatic hormone glucagon urges the liver to release stored glucose into the bloodstream. When a diabetic patient's blood glucose level is too low to be treated with the 15-15 rule, injectable glucagon is used to treat them. By prescription, glucagon kits are available in the market.

Prevention of Hypoglycemia

The best strategy to prevent hypoglycemia is to manage the diabetes adequately and develop the ability to recognize it at the earliest so that it can be treated—before it get worsened.

The practical way for avoiding hypoglycemia is to frequently check blood glucose levels using a glucose monitor (CGM). Studies demonstrate that a person's risk of hypoglycemia decreases the more frequently blood sugar levels are checked. This is due to the fact that decline in the blood glucose levels can be detected correctly, before it becomes too low.

Blood glucose levels should be checked at the following moments:

- Before and after meals
- Before and after exercise, or during the exercise if it's an intense session
- Before going to the bed
- When following a different work schedule, an increase in physical activity or travel across time zones.

Home and Community-Based Care

Teaching Self-Care to Patients

Assess the client's knowledge even if the patient has had diabetes for many years, it is important to plan and implement a teaching plan that includes basic information about diabetes, its cause and symptoms, and acute and chronic complications

and their treatment. The nurse should give repeated return demonstrations of skills that were not performed correctly during the initial assessment. Self-care is very important act for the prevention of long-term complications, including foot care, eye care, and risk factor management. Instruct the client and family about the importance of health promotion activities and recommended health screening.

Continuing Care

The client with diabetes whenever admitted to the hospital for whatever problem, the home care nurse can use this opportunity to assess the patient's knowledge about diabetes management and the patient's and family's ability to carry out that management to reinforce client's knowledge and skills.

Also when the nurse goes for home visit find out the signs and symptoms of long-term complications and assess the patient's and family's techniques in blood glucose monitoring, insulin administration, and food selection. Explain the client and family, the importance of participating in health promotion activities as well as recommended health screening.

DISORDERS OF ADRENAL GLAND

ADDISON'S DISEASE

Addison's disease is a primary adrenal insufficiency that is caused by an autoimmune disease. It is caused by the bilateral destruction of the adrenal cortex, which results in a reduction in adrenocortical hormones such cortisol, aldosterone, and androgens.

The insidious progression of Addison disease typically begins with glucocorticoid deficit and progresses to mineralocorticoid deficiency.

Etiology and Risk Factors

Adrenal insufficiency is classified as primary or secondary.

- Primary adrenal insufficiency can be caused by any illness that directly damages the adrenal cortex. some causes are:
- Autoimmune polyglandular endocrinopathies causes the destruction of adrenal glands.
- Infections: This includes HIV, CMV, TB, and sepsis.
- Bilateral adrenal hemorrhages may be caused by DIC, trauma, meningococcemia, or neoplastic processes, among other things.
- Sarcoidosis, lymphoma, and hereditary conditions are among more causes.
- Certain medications, including ketoconazole and etomidate, can result in adrenal insufficiency.
- The most frequent cause of secondary insufficiency is exogenous steroid treatment, which suppresses ACTH production.

Pathophysiology

In Addison's disease, the lack of negative feedback inhibition causes the production of cortisol, aldosterone, and eventually adrenocorticotropic (ACTH) and melanocyte-stimulating hormone (MSH) hormones to diminish as a result of adrenal insufficiency.

Clinical Manifestations

Typically, Addison disease presents as a insidious, gradual onset of vague symptoms.

- Fatigue, generalized weakness, weight loss, nausea, vomiting, stomach discomfort, dizziness, tachycardia, and/or postural hypotension are some of the initial symptoms.
- Almost all patients experience hyperpigmentation, which is a common manifestation. It is typically widespread and most pronounced in pressure and sun-exposed locations. The vermilion border of the lips, gingival mucosa, elbows, knuckles, posterior neck, breast areola, nipples, and nail beds are areas where hyperpigmentation is more noticeable.
- Unusual tiredness, poor appetite, persistent gastrointestinal pain and weight loss are few common manifestation.
- Female patients may have reduced axillary, pubic hair and body hair, which is due to loss of the adrenal androgens.

Diagnostic Evaluation

Low levels of cortisol and aldosterone, high levels of renin, and a blunt cortisol response to ACTH stimulation are used to confirm the diagnosis. The approach of evaluation is as follows:

- **Rapid adrenocorticotrophic hormone test:** Following synthetic ACTH injection, blood is collected in 2 separate tubes for baseline cortisol and aldosterone value testing.
- Hyponatremia, hyperkalemia, and a mild nonanion-gap metabolic acidosis are the three most notable findings in the comprehensive metabolic panel.
- CBC count shows a normocytic normochromic anemia
- Thyroid-stimulating hormone—patients with Addison disease may experience elevated thyroid-stimulating hormone (TSH), low thyroxine, thyroid autoantibodies in addition to or instead of hypothyroidism symptoms.
- Although an abdominal CT scan may be normal, it may also reveal bilaterally enlarged adrenal glands.

Treatment

For adrenal insufficiency to be managed effectively, early detection is essential. The Addison crisis is a serious endocrine emergency that needs to be identified and treated right away.

Acute Phase

- Fluid resuscitation to restore the intravascular volume with intravenous (IV) normal saline is necessary for patients with an adrenal crisis. -A normal saline bolus is given as the first fluid replacement, and then 5% glucose in isotonic saline is given.
- Correction of glucocorticoid and mineralocorticoid hormone deficiency: The administration of hydrocortisone is the initial hormonal treatment. 100 mg IV bolus is the initial dose for adrenal crisis, followed by 50–100 mg IV every 6 hours for a total of 24 hours.

Maintenance Phase

Hormone replacement therapy must be used as a lifelong treatment. Maintaining a normal level of glucocorticoids and mineralocorticoids is the goal of maintenance therapy. These are the typical dosages:

- Glucocorticoids: 5–25 mg/day of hydrocortisone (can be divided into 2 or 3 doses)
- Prednisone 3–5 mg each day

Extra Edge

The hydrocortisone dosage should be increased in order to account for a potential stress reaction when there is a fever, infection, or other disease present.

Note: Mnemonic for remembering manifestations of Addison's disease

Mnemonic

STEROID
S – Sugar and sodium low
T – Tired and muscle weakness
E – Electrolyte imbalance of high potassium and high calcium
R – Reproductive change
O – Low blood pressure
I – Increased pigmentation of the skin
D – Diarrhea and nausea, depression

PRACTICE PEARLS

Addisonian Crisis: Remember the 5 S's
1. Sudden pain in stomach, back and legs
2. Syncope (unconsciousness)
3. Shock
4. Super low blood pressure
5. Severe vomiting, diarrhea and headache

Information on Cushing's disease is given on page 628.

Note:
HYPERsecretion of CORTISOL
"Cushion is similar to cushion (extra padding)"
HypOsecretion of Aldosterone and Cortisol
"ADDison = ADDed an extra steroid hormone (Aldosterone and cortisol)

ADRENAL TUMORS

Normal cell division is organized and regular; body cell will grow and divide to replace old or damaged cells. Normal cells will stop dividing after production of cells required by the body. Tumors occur when there is an error in this process, and cells continue to grow in an uncontrolled manner. Tumors can either be benign or malignant. Although benign tumors can grow in an uncontrolled way, they do not metastasize. Malignant tumors will grow uncontrolled in such a way that they invade and damage other tissues around them. About 8% of people worldwide develop benign adrenal tumors. Malignant tumors are very rare; occurring in two out of every one million people worldwide.

Anatomy and Physiology of Adrenal Gland

There are two adrenal glands, each attached to the upper portion of a kidney (Fig. 8.55). They have a flattened pyramidal shape. In an adult, each adrenal gland is 3–5 cm in height, 2–3 cm in width, and a little less than 1 cm thick. Its adult mass is 3.5–5 g. The adrenal glands differentiate into two regions: adrenal cortex and adrenal medulla.

Adrenal Cortex

The outer part of the adrenal gland called the adrenal cortex produces steroid hormones that are involved in regulating a

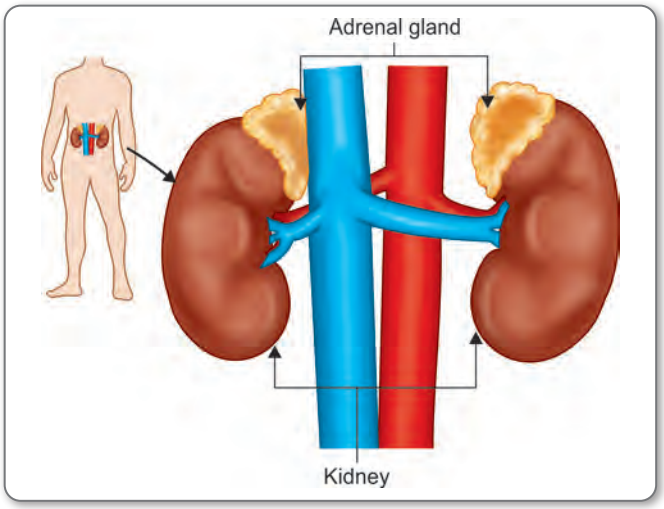


Fig. 8.55: Adrenal gland

number of different body functions. Adrenocortical secretions make it possible for the body to adapt to stress of all kinds. The secretion of hormones from the adrenal cortex is regulated by the hypothalamic-pituitary-adrenal axis. The three types of steroid hormones produced by the adrenal cortex are glucocorticoids, mineralocorticoids, and androgens.

Glucocorticoids: The glucocorticoids, which regulate metabolism and resistance to stress, include cortisol, (hydrocortisone), corti-costerone, and cortisone.

Mineralocorticoids: Aldosterone is the major mineralocorticoid. It regulates homeostasis of sodium ions and potassium ions and helps adjust blood pressure and blood volume. It also promotes excretion of H^+ in the urine; this removal of acids from the body can help prevent acidosis.

Androgens: Also called the Adrenal sex hormones having effects similar to male sex hormones. In both males and females, the adrenal cortex secretes small amounts of weak androgens.

Adrenal Medulla

The inner region of the adrenal gland, the adrenal medulla functions as part of the autonomic nervous system. The hormone producing cells, called chromaffin cells, are innervated by sympathetic preganglionic neurons in the splanchnic nerve.

The two major hormones (catecholamines) synthesized by the adrenal medulla are epinephrine and norepinephrine, also called adrenaline and noradrenaline, respectively. In stressful situations, these hormones are secreted in the body.

TUMORS OF ADRENAL GLANDS

Tumors of the adrenal glands arise from the cortex or the medulla part of the adrenal gland. Adrenal tumors commonly present symptoms of excess secretion of hormones by the tumor.

Types of Adrenal Tumors

Tumors of the Adrenal Cortex
- Adrenocortical adenoma
- Adrenocortical carcinoma

Tumors of the Adrenal Medulla
- Neuroblastoma
- Pheochromocytoma

Adrenocortical Adenoma

Adrenocortical adenomas are tumors of the adrenal cortex. It is a benign tumor. When an adrenal adenoma produces hormones, it is called 'functioning', which produces excessive amounts of steroid hormones.

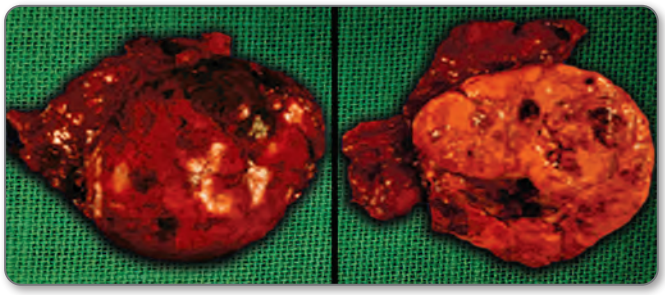


Fig. 8.56: Adrenocortical adenoma

If an adenoma does not produce a hormone it is termed 'nonfunctioning'. Adrenal adenomas are often found by chance during a scan of the body for an unrelated condition (Fig. 8.56).

Etiology

The cause of adrenal adenomas is unknown, but the current accepted theory is that they arise because of mutations (changes) in certain genes (which are not yet identified). Patients with genetic defects of the body systems that manufacture steroid hormones (e.g., congenital adrenal hyperplasia), especially those, whose condition is poorly controlled, may have a higher risk of adrenal adenomas.

Incidence

Adrenal adenoma occurs with increasing age. Benign adrenal adenomas are found in 1–32% of people at autopsy, with most studies showing a detection rate of about 5%.

About 6 % of patients over 60 years of age develop adrenal adenoma. No other predisposing factors have been defined, and prevention is not possible.

Clinical Manifestation

Usually adrenal adenomas are asymptomatic. However, even in asymptomatic proper investigations reveal that many adrenal adenomas produce abnormal amounts of steroid hormones. The most common abnormality is the production of too much cortisol, a steroid hormone involved in the response to stress and energy balance.

The glucocorticoid producing adrenal tumors shows symptoms and signs of Cushing syndrome such as obesity, hypertension, hyperglycemia, fatigue, depression, menstrual irregularities, proximal muscle weakness, acne, facial plethora, striae, fractures, and osteopenia.

Aldosterone-secreting tumors may present with resistant hypertension.

Evidence Based Practice

In a study, research group assessed 80 patients with Cushing's syndrome (CS) in a cross-sectional setting. Results indicate that, in a cross-sectional observational study, patients with CS were shown to have a particularly high susceptibility to pain and increased anxiety-associated personality traits. This study will expand on these previous findings.

Very rarely, bleeding can occur into adenomas and cause pain in the flanks or back.

Diagnostic Evaluation

Most adrenal adenomas are discovered by chance when an abdominal computed tomography (CT) or magnetic resonance imaging (MRI) scan is done for unrelated symptoms.

Adrenal masses require specialized scans and hormone investigations.

Tests will usually include:

- 24-hour urine collection for adrenaline and noradrenaline.
- Blood tests for: Potassium, renin and aldosterone activity, male sex hormones, female sex hormones.
- A "low-dose dexamethasone suppression" evaluates how well the body regulates cortisol production. It entails taking the synthetic steroid dexamethasone by mouth before having blood drawn to measure cortisol levels.

  In a normal condition, the body would decrease its own cortisol synthesis in response to the extra steroid of dexamethasone dose. Whereas, in the case of adrenal adenoma, despite the exogenous steroid, i.e., dexamethasone, there will be continued secretion of cortisol.

Nursing Alert

Patients taking an oral contraceptive pill should be advised to stop taking it at least 6 weeks before the blood tests. Because the hormonal composition of the pill may cause false interpretation, the blood levels of steroid hormones.

Management

The mainstay treatment for adenoma is the excision of affected adrenal gland.

When the left adrenal gland has to be removed, sometimes the spleen has to be removed also during the operation. Since the spleen is needed for fighting certain bacterial infections, In that case, patients should be routinely vaccinated against pneumococci and meningococci—the pneumococci (which cause lobar pneumonia) and meningococci (which cause bacterial meningitis).

Surgery

Adrenalectomy is the preferred surgery for the excision of tumor, also the surrounding healthy tissue until 1 cm margin may be excised. Laparoscopic surgery can be done if the tumor is noncancerous and is smaller than 5 cm. If the tumor is cancerous or larger than 5 cm, open surgery is recommended with one larger incision in the abdomen or back.

During surgery, the patient will need to be monitored and receive medication to treat high blood pressure. Patients with a tumor that produces very large amounts of hormones may require extensive medication need to stay in an intensive care unit for a short period after the surgery is performed.

Hormone Therapy

Because adrenal gland tumors produce excess hormones; therefore, various medications must be recommended to control the levels of hormones before, during, or after other treatments. These drugs include:

- Metyrapone (Metopirone)
- Metyrosine (Demser)
- Spironolactone (Aldactone)
- Streptozocin (Zanosar)

Chemotherapy

Systemic chemotherapy by various methods like an intravenous (IV) tube placed into a vein using a needle or in a pill or capsule that is swallowed (orally) gets into the bloodstream to reach tumor cells throughout the body. A chemotherapy treatment schedule consists of a specific number of cycles given over a set period of time. A patient may receive one drug or combinations of different drugs at a time.

Radiation Therapy

Radiation therapy is the use of high energy x-rays to destroy tumor cells and adrenal tumors require it in very rare cases. Similar Like chemotherapy, radiation therapy also have schedule of a specific number of treatments given over a set period of time.

The common side effects from radiation therapy are fatigue, mild skin reactions, upset stomach, and loose bowel movements.

Palliative Care

Along with the definitive treatment. It is also an important to relieve the patient's symptoms and side effects. The palliative or supportive care, gives support to the patient with his or her physical, emotional, and social needs. Palliative care includes the care which helps in reducing symptoms, improving quality of life, and supporting patients and their families through various treatments such as medication, nutritional changes, relaxation techniques, emotional support and other therapies.

Prognosis

The outlook of patients with an adrenal adenoma is excellent, provided that abnormal hormonal production is diagnosed and treated in a timely manner.

Adrenocortical Carcinoma

Adrenocortical carcinoma, also adrenal cortical carcinoma (ACC) and adrenal cortex cancer, is an aggressive cancer originating in the cortex of the adrenal gland.

Incidence

Adrenocortical carcinoma is a rare tumor, with incidence of 1–2 per million population annually. Adrenocortical carcinoma has a bimodal distribution by age, with cases clustering in children under 6, and in adults 30–40 years old.

Clinical Manifestation

Adrenocortical carcinoma may present differently in children and adults.

- Most tumors in children are functional, and virilization is by far the most common presenting symptom, followed by Cushing's syndrome.
- Among adults presenting with hormonal syndromes, Cushing's syndrome alone is most common, followed by mixed Cushing's and virilization. Feminization and Conn's syndrome occur in less than 10% of cases.
- For Cushing's syndrome (glucocorticoid excess), these include weight gain, muscle wasting, purple lines on the abdomen, a fatty "buffalo hump" on the neck, a "moonlike" face, and thinning, fragile skin (Fig. 8.57).
- Virilism (glucocorticoid and androgen overproduction) is most obvious in women, and may produce excess facial and body hair, acne, deepening of the voice, cessation of menstruation.
- Conn's syndrome (mineralocorticoid excess) is marked by high blood pressure, which can result in: headache hypokalemia (low serum potassium) which can produce muscle weakness, confusion, and palpitations (Fig. 8.58).
- Feminization (estrogen excess) is most readily noted in men, and includes breast enlargement, decreased libido and impotence.

Etiology

The exact etiology of adrenocortical cancer is unclear. It is thought to be associated with the mutation of tumor suppressor gene.

Management

- The only curative treatment is complete surgical excision of the tumor.

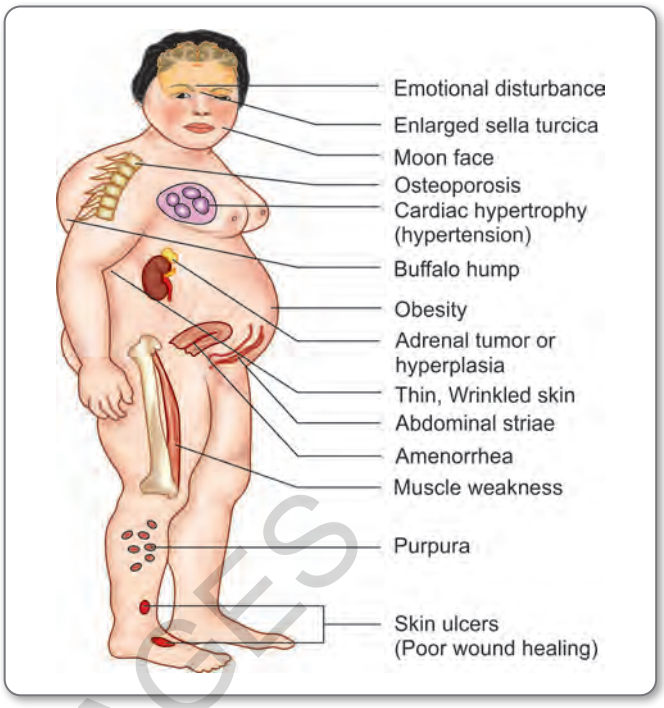


Fig. 8.57: Cushing's syndrome

Fig. 8.58: Conn's syndrome

- Radiation therapy and radio frequency ablation may be used for palliation in patients who are not surgical candidates.
- Chemotherapy includes the drug mitotane, an inhibitor of steroid synthesis which is toxic to cells of the adrenal cortex. Drugs consist of cisplatin, doxorubicin, etoposide, and mitotane.
- Hormonal therapy with steroid synthesis inhibitors such as aminoglutethimide may be used in a palliative manner to reduce the symptoms of hormonal syndromes.

Prognosis

Adrenocortical carcinoma generally, carries a poor prognosis and is unlike most tumors of the adrenal cortex, which are

benign (adenomas) and only occasionally cause Cushing's syndrome. The most important prognostic factors are age of the patient and stage of the tumor. Adrenocortical carcinoma has often invaded nearby tissues or metastasized to distant organs at the time of diagnosis, and the overall 5-year survival rate is only 20–35%.

Neuroblastoma

Definition: Neuroblastoma (NB) is the most prevalent extracranial childhood tumor. It is categorized as a neural crest progenitor cell-derived embryonal neuroendocrine tumor.

Incidence

According to some statistics, African-American children are more prone than Caucasian children to get the condition. Approximately 700 children are diagnosed with neuroblastoma in the United States each year (8.7 per million cases against 8.0 per million cases diagnosed).

Etiology

The exact causes of neuroblastoma are unknown.

According to current research, neuroblast cells—which the fetus produces—develop into neuroblastomas when they fail to mature into healthy nerve or adrenal cells and instead continue to grow and proliferate.

Clinical Manifestation

- The initial sign of a neuroblastoma is typically an unusual growth or lump, which is most frequently detected in the child's abdomen and causes discomfort or a feeling of fullness and agony.
- The pressure that the tumor exerts on the body also causes symptoms such as numbness and fatigue.
- If the cancer has progressed to the bone, it may lead to progressive bone pain.
- The cancer may produce bulging eyes and if it has advanced to the area behind the eye; in a rare instance, blindness may be the initial presenting symptom.
- Spinal cord compression can cause paralysis.
- Occasionally, people have high blood pressure, prolonged diarrhea, quick heartbeat, skin redness, and perspiration.
- Some children may also experience jerky or uncoordinated muscular movements or uncontrollable eye movements.

Diagnosis

- Blood and urine tests to examine the quantity of neurotransmitters are typically necessary for a neuroblastoma diagnosis.
- Neuroblastoma is confirmed as a diagnosis with an MRI and CT scan.
- Biopsy of the tumor or bone marrow tissue is used for making a definitive diagnosis of neuroblastoma.

Treatment

Children with neuroblastoma in all stages can receive treatment. Depending on the disease's stage, more than one of these therapies may be used. Therapeutic modalities are as follows:

- Treatment options include simple observation, surgical excision, chemotherapy, radiation therapy, stem cell transplantation, and immunotherapy due to the heterogeneity in tumor location, grade, and stage upon diagnosis.
- As a result, surgery can be completely avoided in infants with small tumors (less than 5 cm) by monitoring tumor progression with imaging every 6–12 weeks.
- The high-risk group can receive induction chemotherapy to lessen tumor burden at both the primary and metastatic locations, followed by myeloablative chemotherapy, stem-cell transplantation, and maximal surgical resection in order to treat widespread metastatic disease to the bone marrow, bone, lungs, and liver.

Prognosis

The prognosis depends upon:

- The cancer's stage, the child's age of diagnosis, the tumor's location, and an examination of the tumor cells under a microscope to determine their nature and status.
- Even when the disease has spread, infants have a higher cure rate than children over one year of age.
- The prognosis for a young child with neuroblastoma is generally positive; the anticipated 5 year survival rate for children whose disease started in infancy is about 85% and 35% for those whose disease started later.

Pheochromocytoma

Definition: Pheochromocytoma is a tumor of special cells (called chromaffin cells), most often found in the middle of the adrenal gland.

Incidence: This is a rare tumor, which occurs in young child of 5 days old to adults as old as 92 years old. Although they can be found at any time during life, they usually occur in adults between 30–40 years of age. Pheochromocytomas are somewhat more common in women than in men.

Survival rates for children with high-risk neuroblastoma show only modest improvement in recent years, compared to other childhood cancers.

Causes

The causes are unknown. A small minority (about 10–20%) of pheochromocytomas arise because a person has an inherited susceptibility to them.

Symptoms

Most common symptom of pheochromocytoma is hypertension. The other symptoms are extremely variable. These symptoms usually occur in episodes (or attacks) called paroxysms and include:

- Headaches
- Excess sweating
- Racing heart
- Rapid breathing
- Anxiety/nervousness
- Nervous shaking
- Pain in the lower chest or upper abdomen
- Nausea
- Heat intolerance

The frequency of episodes varies it can occur as often as 25 times a day or, as infrequently as once every few months and can last a few minutes, several hours or days. Usually, the attacks occur several times a week and last for about 15 minutes. After the episode is over, the person feels exhausted and fatigued.

Between the attacks, the following experience people with pheochromocytoma can have.

- Increased sweating
- Cold hands and feet
- Weight loss
- Constipation

Diagnosis

- **24-hour urinary catecholamine and metanephrine:** This test is designed to look for adrenaline and the breakdown products of adrenaline. Since the body gets rid of these hormones in the urine, those testing will need to collect their urine for 24 hours. The laboratory will determine whether or not the levels of hormones are too high. This test is very good at making the diagnosis of pheochromocytoma.
- **Serum catecholamine:** Measures the level of adrenaline compounds in the blood.

  A person needs to have an attack of symptoms either during the 24-hour urine collection time period or shortly before the blood is drawn for a serum test to ensure the test's accuracy.

  The imaging techniques CT scan and MRI are due to locate the tumor.

Treatment

- Treatment is surgical removal of the tumor. Before surgery, medications such as α-adrenergic blockers are given to block the effect of the hormones and normalize blood pressure. These medications are usually started 7–10 days prior to surgery. The surgery of choice is laparoscopic laparotomy.
- A small incision is made in the abdomen, the laparoscope is inserted and the tumor is removed.
- After surgery, blood and urine tests will be done to make sure hormone levels return to normal.
- If the hormone levels are still above normal, it may mean that some tumor tissue was not removed.
- Treatment also includes medication to control hypertension.
- If pheochromocytoma is malignant, radiation therapy and/or chemotherapy may be used (Fig. 8.59).

Prognosis

Untreated pheochromocytoma can be fatal due to complications of the high blood pressure. In the vast majority of cases, when the tumor is surgically removed, pheochromocytoma is cured. In the minority of cases (10%) where pheochromocytoma is malignant, prognosis depends on how far the cancer has spread, and the patient's age and general health. The overall median 5 year survival from the initial time of surgery and diagnosis is approximately 43%.

Prevention

Little is known about environmental and other causes of pheochromocytoma. Some of the tumors are due to inherited predisposition. Because of these factors, pheochromocytoma cannot be prevented.

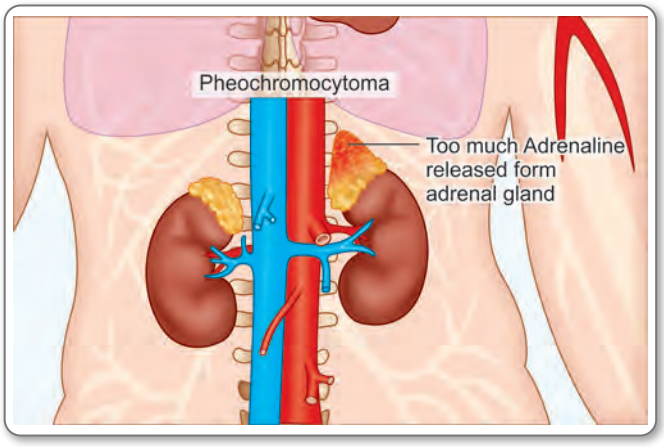


Fig. 8.59: Pheochromocytoma

Nursing Care Plan

Nursing Management of Patient with Pheochromocytoma

Assessment	Nursing Diagnosis	Goal	Implementation	Scientific Principles	Evaluation
Assess body temperature	Risk for infection related to altered immunologic response.	Prevention of infection.	• Assess patient for evidence of infection, check vital signs, monitor WBC, • Instruct all personnel in careful hand hygiene before and after entering room. • Change intravenous sites every other day. • Cleanse skin with povidone-iodine before arterial puncture or venipuncture. • Avoid intra muscular injections. • Encourage patient to ambulate in room.	To collect base line data	Decreases risk for infection.
Assess skin integrity	**Impaired skin integrity:** erythematous and wet desquamation reactions to radiation therapy.	Maintenance of skin integrity	• Assess the skin of the patient for erythema, wet desquamation. • Avoid the use of soaps, cosmetics, perfumes, powders, lotions and ointments, deodorants. • Use only lukewarm water to bathe the area. • Avoid rubbing or scratching the area. • Avoid shaving the area with the straight edged razor. • Avoid applying hot water bottles, ice and adhesive tape to the area. • Avoid exposing the area to sun light or cold weather. • Avoid tight clothing. Use cotton cloth. • Report any blistering.		Attains skin integrity.
Client look weak, loss weight	**Imbalanced nutrition:** Less than body requirements, related to anorexia, nausea and vomiting.	Maintenance of nutritional status.	• Avoid unpleasant sights, odors, sounds in the environment during meal time. • Suggest foods that are preferred and well tolerated by the patient, preferably high-calorie and high protein foods. • Encourage adequate fluid intake, but limit fluids at meal time. • Suggest smaller, more frequent meals. • Provide control of nausea and vomiting. • Encourage frequent oral hygiene. • Administer appetite stimulants as prescribed by physician.	• To improve appetite • Client will like the food • Fluid helps in digestion	Improved nutritional status
Not able to perform self-care activity.	Self-care deficit related to weakness, fatigue, muscle wasting, and altered sleep patterns.	Increased activity tolerance.	• Encourage several rest periods during the day. • Increase total hours of night time sleep. • Encourage adequate protein and calorie intake. • Encourage use of relaxation techniques. • Assess for fluid and electrolyte disturbances. • Administer blood products as prescribed.	It will preserve energy	Increased activity tolerance

Contd...

Assessment	Nursing Diagnosis	Goal	Implementation	Scientific Principles	Evaluation
Client is anxious, confused, not interested in the environment.	Anticipatory grieving related to loss; altered role functioning	Achieves improved body image.	• Encourage verbalization of fears and questions regarding disease, treatment and future implications. • Encourage active participation of patient and family in care and treatment decisions. • Encourage ventilation of negative feelings. • Allow for periods of crying and expression of sadness. • Advice professional counseling.	It will allow the client to ventilate anxiety. Counseling will help the client to face and solve the problems faced by him.	Exhibits decreased grieving.
Client shows withdrawal behavior. Non-participation in social activities.	Disturbed body image and situational low self-esteem related to change in appearance, function, and roles.	Improved body image and self-esteem.	• Assess patients feeling about body image and level of self-esteem • Encourage continued participation in activities and decision making. • Encourage patient to verbalize concerns • Assist patient in selecting and using scarves, clothing and hair pieces that increase his or her sense of attractiveness.	Help client to improve level of self-esteem.	Achieves improved body image.

Health Education

Health education to the patient with diagnosis of adrenal tumors:

- Regular follow-up care and blood tests for hormonal levels, WBCs.
- Yoga or other relaxation techniques may help manage pain.
- The presumed incidental adrenal mass should be re-imaged at 3 months, 6 months, and every 6–12 months, thereafter until it is evident that the mass is stable.
- Take prescribed medications as directed.
- Measure weight every day.
- Eat potassium rich foods.
- Learn and check blood pressure every day.
- Keep a record of all results.

Alternate Therapy

Acupuncture

Fine needles are inserted at specific points to stimulate, disperse, and regulate the flow of vital energy, and restore a healthy energy balance. In addition to pain relief, acupuncture is also used to improve well-being and treat acute, chronic, and degenerative conditions in children and adults.

Acupuncture can be used to restore hormonal balance, regulate energy levels, smooth emotions, help manage sleep and menstrual problems, restoring immune function and balancing the production and release of hormones. Acupuncture is so helpful in the treatment of endocrine disorders, as they focus on balancing all the glands.

Acupressure

Similar to acupuncture, but using finger pressure rather than fine needles on specific points along the body to treat ailments such as tension and stress, aches and pains, menstrual cramps, arthritis

Acupressure point is a very important one that is said to help controlling not only diabetes but also reduce headache and other ailments. Various points found on several different meridians are used to treat hypothyroidism. A point called Yintang, located between the eyebrows, activates the pituitary, which, in turn, controls the thyroid.

Aromatherapy

Using "essential oils" distilled from plants, aromatherapy treats emotional disorders such as stress and anxiety as well as a wide range of other ailments. Oils are massaged into the skin in diluted form, inhaled, or placed in baths. Aromatherapy is often used in conjunction with massage therapy, acupuncture, reflexology, herbology, chiropractic, and other holistic treatments.

In the endocrine system, oils like geranium, lavender, ginger, rosemary, etc., are used for over- and underactive glands.

Ayurvedic Medicine

Alternative medicine used from ancient times in the Indian subcontinent is known as Ayurveda. Over the course of more than two millennia, ayurvedic medicines have changed and evolved. Herbal remedies, specific diets, meditation, yoga, massage, laxatives, enemas, and medical oils are examples of therapies. Dosha balance is emphasized in Ayurvedic teach-

ings, and it is seen detrimental and likely to cause illness to repress one's natural inclinations.

Autogenic Training

It is a relaxing technique developed in Europe over a century ago that relies on bodily awareness of certain feelings and passive attention. Numerous stress-related problems, including anxiety, tension, sleeplessness, and exam stress, have been proven to be significantly improved by it. It has also been demonstrated that this therapy is beneficial for people with chronic medical disorders such thyroid disease, migraines, colitis, irritable bowel syndrome, diabetes, and high blood pressure.

Chinese (Oriental) Medicine

Oriental medical professionals are trained to treat a wide range of both acute and chronic disorders using a variety of both traditional and contemporary therapeutic techniques, such as acupuncture, herbal medication, massage, heat therapy, nutritional advice, and lifestyle counseling.

Herbalism

Herbalism is an age-old medicinal method that is still extensively used throughout most of the world. It makes use of organic plants or compounds derived from plants to treat a variety of diseases and improve the efficiency of the body's systems. Despite the fact that herbalism lacks a professional license in the United States, a variety of practitioners "prescribe" herbs.

Herbs can be used to treat conditions like Cushing syndrome, hyperthyroidism, hypothyroidism, and more.

Reflexology

This modality is based on the idea that specific points on the feet and hands correspond with organs and tissues throughout the body. With fingers and thumbs, the practitioner applies pressure to these points to treat a wide range of stress-related illnesses.

Foot reflexology is used for hyperthyroidism, hypothyroidism, hypoparathyroidism, hyperparathyroidism, hyperinsulinism, hypoinsulinism, hypogonadism, etc.

Yoga Therapy

The use of yoga is to address mental and physical problems while integrating body and mind. Sarvangasana is the most well recognized asana for thyroid gland. Other effective asanas include surya namaskar, pawanmuktasana, which are used for severe thyroid diseases. While medical science claims diabetes is incredible, many studies have proven that it responds very well to yogic management. It is important that diabetes undertake yogic therapy in conjunction with qualified medical supervision. The yoga sadhana and progress of every diabetes will vary according to individual conditions.

Summary

- Hormones are substances released by endocrine glands, and produce effect on specific organs and tissues.
- Endocrine glands are ductless glands which secretes hormones directly in the blood.
- Exocrine glands have ducts for releasing their secretions in the target organs.
- Hormones selectively act on their targets due to the specific receptor proteins present in these targets.
- The endocrine glands are the pineal gland (or pineal body), the hypophysis (or pituitary gland), the thyroid, the parathyroid, the endocrine part of the pancreas, the adrenal glands and the gonads (the testicles or ovaries).
- The pineal gland is located in the center of the head. It secretes the hormone melatonin, a hormone produced at night and related to the regulation of circadian rhythm (or the circadian cycle, the wakefulness-sleep cycle).
- The neurosecretory cells of the hypothalamus are stimulated by peripheral and central brain impulses.
- The feedback mechanism carries out the functions of a regulator, aiding and supporting the maintenance of the body's hormonal equilibrium.
- Positive feedback stimulates and boosts the hormone's release or synthesis. On the other side, the negative feedback prevents and suppresses the release of the hormone.
- Dwarfism is the medical terminology for short-stature. It is defined as height-vertex below two standard deviations (–2 SD) or in the third percentile for a given age and sex. Excessive secretion of GH in children may cause exaggerated bone growth and gigantism. In adults, excess GH may lead to acromegaly.
- Vasopressin, or ADH, regulates water in the body and therefore helps in the control of blood pressure.
- Diabetes insipidus (DI) is defined as the passage of large volumes (>3 L/24 h) of dilute urine (<300 mOsm/kg).
- Oxytocin acts on the uterus and the mammary glands. The target organs of ADH are the kidneys.
- The thyroid gland secretes the hormones thyroxine (T4), triiodothyronine (T3) and calcitonin.
- The term "goiter" refers to an enlarged thyroid gland. It may be multinodular, nodular, or diffuse.
- Hyperthyroidism is characterized by high levels of T3 and T4, which raises the basal metabolic rate.
- Hypothyroidism is a common endocrine condition caused by a lack of thyroid hormone. The most common cause of hypothyroidism is iodine insufficiency.
- The parathyroid glands are four small glands, embedded in posterior lobe of the thyroid. The hormone secreted by parathyroid glands called parathormone. It regulates calcium levels in the blood.
- Parathormone is also involved in increasing calcium absorption in the intestines via vitamin D activation. It also plays a role in the kidneys, promoting the tubular reabsorption of calcium.
- Pancreas is a mixed gland which secretes insulin and glucagon.
- Diabetes mellitus is a metabolic disorder caused by the deficient production or action of insulin. Polyuria, polydipsia and polyphagia are three main signs of diabetes mellitus.
- Diabetes mainly is two types—type 1 diabetes and type 2 diabetes. In type I diabetes, there is impaired production of insulin by the pancreas, and is caused by the destruction of the cells of the islets of Langerhans by autoantibodies (autoimmunity). Type II DM develops due to resistance of cells to the action of hormone insulin.
- Diabetes is treated with hypoglycemic agents such as insulin or oral medicines.

Assess Yourself

Long Answer Questions

1. Differentiate between hypothyroidism and hyperthyroidism on the basis of etiology, pathophysiology, clinical manifestations and treatment modalities.
2. Define diabetes mellitus. Elaborate medical and nursing management of diabetes mellitus.
3. Define Cushing syndrome. Explain its pathophysiology and clinical manifestations.
4. Differentiate between diabetes mellitus and diabetes insipidus.
5. Elaborate the etiology and management of adrenal tumor. Prepare a nursing care plan regarding care of patient who underwent adrenalectomy.

Write short notes on:

1. Syndrome of inappropriate antidiuretic hormone
2. Nursing management of diabetes mellitus
3. Hyperparathyroidism
4. Complications of diabetes mellitus
5. Insulin therapy for diabetes mellitus
6. Adrenal glands of mammals

Multiple Choice Questions

1. Which hormone is responsible for setting the basal metabolic rate and for maturation of the brain?
   a. Cortisol b. ACTH
   c. TSH d. Thyroxine
   e. None of the preceding
2. After thyroidectomy (removal of the thyroid gland), client is having:
   a. Decreased TSH secretion
   b. Increased T3 and thyroxine secretion
   c. Increased calcitonin secretion
   d. Increased TSH secretion
   e. Both b and c apply
3. Steroid hormones are secreted by:
   a. The adrenal cortex b. The gonads
   c. The thyroid d. Both a and b
   e. Both b and c
4. Oxytocin has the function that it:
   a. Allows milk secretion or "milk let-down"
   b. Is stored in the pars nervosa (posterior pituitary).
   c. Is produced by cells in the diencephalon (hypothalamus).
   d. Exerts important effects during childbirth
   e. All of the above
5. The action of glucocorticoids involves many functions, but only one of the following is a correct one:
   a. Increases inflammatory responses
   b. Decreases lipid hydrolysis (lipolysis)
   c. Increases glucose levels
   d. Retention of electrolytes by the kidneys
   e. Increases osteoclast activity
6. Which hormone causes increased calcium absorption from intestines?
   a. Calcitonin
   b. Calcitriol
   c. Thyroxine
   d. Pancreatic polypeptide
   e. Corticotropin releasing factor (CRF)
7. Insulin is secreted from which cells of pancreas?
   a. F-cells b. Principal cells
   c. α cells d. β cells
   e. Delta cells
8. If cortisol is secreted in less amount, it causes:
   a. Cretinism b. Diabetes mellitus
   c. Diabetes insipidus d. Addison's disease
   e. Graves disease
9. If there is decrease in insulin hormone receptors on cells, it will result in:
   a. Juvenile diabetes
   b. Type I diabetes mellitus
   c. Diabetes insipidus
   d. Insulin-dependent diabetes mellitus (IDDM)
   e. Type II diabetes mellitus
10. Vasopressin is also called:
    a. Cortisol b. hGH
    c. ADH d. Oxytocin
    e. Epinephrine
11. Out of the glands given below which gland does not belong to the endocrine system?
    a. Pituitary b. Thyroid
    c. Parathyroid d. Salivary
12. Which of the following is not a pancreatic hormone?
    a. Glucagon b. Aldosterone
    c. Insulin d. Somatostatin
13. Which of the following is not a hormone function?
    a. Increase cellular oxygen consumption
    b. Cause osteoblasts to divide
    c. Convert fibroblasts to osteoblasts
    d. Alter genetic expression
14. Releasing hormones are produced by the:
    a. Hypothalamus b. Pituitary
    c. Thyroid d. Adrenal cortex
15. Which is not the symptom of hyperthyroidism out of list given below?
    a. Loss of weight
    b. Increased mental awareness
    c. Increased heart rate
    d. Increased ability to sleep
16. Which hormone is deficient in diabetes insipidus?
    a. Insulin
    b. Vasopressin
    c. Aldosterone
    d. Atrial natriuretic peptide

17. If prolactin is secreted in excess, it may cause:
    a. Acromegaly
    b. Gynecomastia
    c. Dwarfism
    d. Early menopause

18. Action of ADH on kidney is at:
    a. Cortex
    b. Distal convoluted tubule
    c. Medulla
    d. Proximal convoluted tubule

19. TSH stimulation of the thyroid causes:
    a. Decreased blood flow
    b. Decreased in gland size
    c. Increased in follicular epithelium
    d. Increased in colloid

Fill in the Blanks

1. Pituitary GH secretion will _____ if client is having hyperglycemia.
2. When blood (somatomedin) increases, pituitary GH secretion will _____.
3. With the increase of GH secretion, glycogenolysis will _____.
4. As GH secretion decreases, fat catabolism will _____.
5. In hyperthyroidism, tissue O_2 consumption will _____.
6. As thyroid hormone levels increase, pituitary TSH secretion will _____.
7. As hypernatremia progresses, cortisol secretion will _____.
8. As hypoxia progresses, cortisol secretion will _____.
9. As cortisol secretion increases, blood (glucose) will _____.
10. As cortisol secretion increases, protein anabolism will _____.
11. As PTH levels increase, plasma (Ca^{2+}) will _____.
12. As plasma (Ca^{2+}) increases, calcitonin secretion will _____.

Answer Key

Multiple Choice questions

1.	d.	**2.**	a.	**3.**	a.	**4.**	d.	**5.**	a.	**6.**	a.	**7.**	d.	**8.**	d.	**9.**	e.	**10.**	e.
11.	d.	**12.**	b.	**13.**	a.	**14.**	a.	**15.**	d.	**16.**	b.	**17.**	b.	**18.**	b.	**19.**	c.		

Fill in the Blanks

1.	Increase	**2.**	Increase	**3.**	Increase	**4.**	Decrease	**5.**	Increase
6.	Decrease	**7.**	Decrease	**8.**	Decrease	**9.**	Increase	**10.**	Decrease
11.	Increase	**12.**	Increase						

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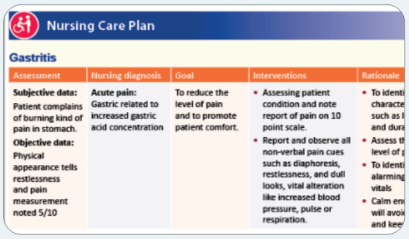
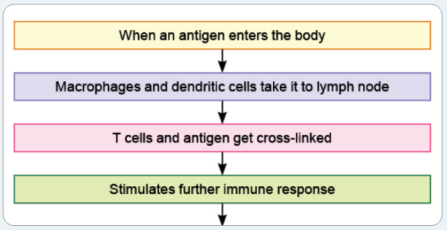
NEW! All the chapters include **NURSING CARE PLAN** in tabular form


Nursing Care Plan

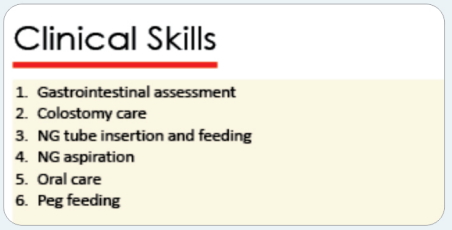
Gastritis

Assessment	Nursing diagnosis	Goal	Interventions	Rationale
Subjective data: Patient complains of burning kind of pain in stomach. **Objective data:** Physical appearance tells restlessness and pain measurement noted 5/10	**Acute pain:** Gastric related to increased gastric acid concentration	To reduce the level of pain and to promote patient comfort.	• Assessing patient condition and note report of pain on 10 point scale. • Report and observe all non-verbal pain cues such as diaphoresis, restlessness, and dull looks, vital alteration like increased blood pressure, pulse or respiration.	• To identi characte such as l and dura • Assess th level of p • To identi alarming vitals • Calm en will avoi and kee



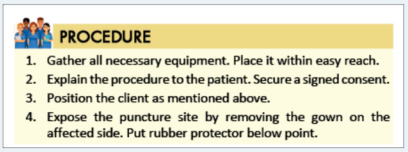
NEW! All disease conditions' pathophysiology have been explained through attractive **FLOWCHARTS**

NEW! Provides the list of **CLINICAL SKILLS** related to disease conditions

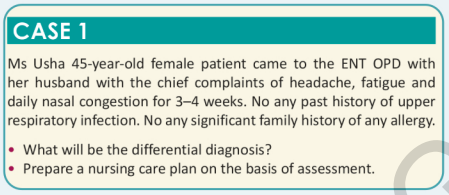

Clinical Skills

1. Gastrointestinal assessment
2. Colostomy care
3. NG tube insertion and feeding
4. NG aspiration
5. Oral care
6. Peg feeding

NEW! As per new INC syllabus with special emphasis on **CLINICAL PROCEDURES**


PROCEDURE

1. Gather all necessary equipment. Place it within easy reach.
2. Explain the procedure to the patient. Secure a signed consent.
3. Position the client as mentioned above.
4. Expose the puncture site by removing the gown on the affected side. Put rubber protector below point.

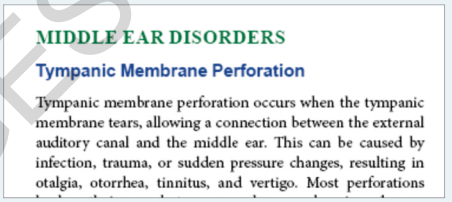

CASE 1

Ms Usha 45-year-old female patient came to the ENT OPD with her husband with the chief complaints of headache, fatigue and daily nasal congestion for 3–4 weeks. No any past history of upper respiratory infection. No any significant family history of any allergy.

- What will be the differential diagnosis?
- Prepare a nursing care plan on the basis of assessment.

NEW! Many clinical scenarios are included for critical thinking and clinical judgment **CASE STUDIES**

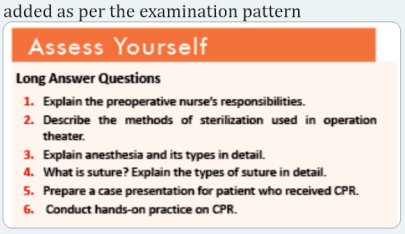
EXPANDED! Detailed health assessments of every disease have been incorporated


MIDDLE EAR DISORDERS

Tympanic Membrane Perforation

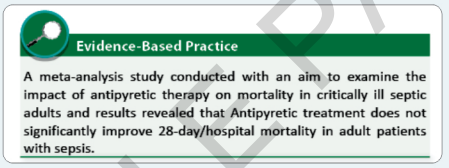
Tympanic membrane perforation occurs when the tympanic membrane tears, allowing a connection between the external auditory canal and the middle ear. This can be caused by infection, trauma, or sudden pressure changes, resulting in otalgia, otorrhea, tinnitus, and vertigo. Most perforations

NEW! **PRIORITIZATION QUESTIONS** have been added as per the examination pattern


Assess Yourself

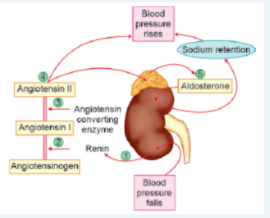
Long Answer Questions

1. Explain the preoperative nurse's responsibilities.
2. Describe the methods of sterilization used in operation theater.
3. Explain anesthesia and its types in detail.
4. What is suture? Explain the types of suture in detail.
5. Prepare a case presentation for patient who received CPR.
6. Conduct hands-on practice on CPR.


Evidence-Based Practice

A meta-analysis study conducted with an aim to examine the impact of antipyretic therapy on mortality in critically ill septic adults and results revealed that Antipyretic treatment does not significantly improve 28-day/hospital mortality in adult patients with sepsis.

NEW! **EVIDENCE-BASED** practices Alerts are added



UNDERSTAND! The content is presented in an easy-to-grasp manner using bullet points and diagrams

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