# SWARRNIM START UP AND INNOVATION UNIVERSITY

# AARIHANT INSTITUTE OF NURSING

COURSE NAME: ADULT HEALTH NURSING

**CROHN'S DISEASE**

## 1. DEFINITION

Crohn's Disease is a type of inflammatory bowel disease (IBD) characterized by chronic inflammation of the gastrointestinal (GI) tract. It can affect any part of the GI tract from the mouth to the anus but most commonly impacts the ileum (the last part of the small intestine) and the colon (the large intestine).

## 2. OBJECTIVE

The objectives of managing Crohn's disease aim to control inflammation, alleviate symptoms, and improve the patient's overall quality of life. Here's a comprehensive look at the primary goals:

1. Control Inflammation
- Objective: Reduce and manage inflammation in the gastrointestinal tract to minimize symptoms and prevent disease progression.
- Strategies: Use of anti-inflammatory medications such as corticosteroids, aminosalicylates, and immunosuppressants.

2. Alleviate Symptoms
- Objective: Relieve symptoms such as abdominal pain, diarrhea, and fatigue to improve the patient's comfort and daily functioning.
- Strategies: Implement symptomatic treatments, including antidiarrheal agents, pain relievers, and dietary modifications.

3. Promote Healing of the Gastrointestinal Tract
- Objective: Facilitate mucosal healing and prevent complications like strictures and fistulas.
- Strategies: Use of biologic agents and targeted therapies that help in mucosal healing and maintaining remission.

4. Prevent Complications

- Objective: Identify and address complications early, such as bowel obstructions, abscesses, and fistulas, to reduce the risk of severe outcomes.
- Strategies: Regular monitoring through imaging and endoscopic evaluations, along with surgical interventions if necessary.

5. Improve Nutritional Status

- Objective: Address and manage nutritional deficiencies and ensure adequate caloric and nutrient intake.
- Strategies: Use of dietary modifications, nutritional supplements, and in some cases, enteral or parenteral nutrition.

6. Enhance Quality of Life

- Objective: Improve overall well-being and daily functioning by managing disease symptoms effectively and providing support for psychological and social aspects.
- Strategies: Comprehensive care including counseling, support groups, and educational programs about disease management.

7. Educate and Support Patients

- Objective: Provide education about Crohn's disease, treatment options, and self-management techniques.
- Strategies: Educate patients and their families about medication adherence, symptom monitoring, and lifestyle adjustments.

8. Manage Exacerbations

- Objective: Prevent and manage flare-ups of the disease to avoid severe symptoms and complications.
- Strategies: Develop and follow action plans for flare-ups, including medication adjustments and prompt medical attention.

9. Monitor Disease Progression

- Objective: Regularly assess disease activity and progression to adjust treatment plans accordingly.

- Strategies: Routine follow-up appointments, lab tests, and imaging studies to monitor disease status and response to treatment.

10. Coordinate Comprehensive Care

- Objective: Ensure that all aspects of care are addressed through a multidisciplinary approach.
- Strategies: Collaborate with gastroenterologists, dietitians, surgeons, and other healthcare professionals to provide integrated care.

## 3. PRINCIPLE

regular follow-ups, continuous evaluation of treatment efficacy, and adjustments as needed.The principles of managing Crohn's disease focus on understanding the disease's chronic and inflammatory nature, addressing its complex manifestations, and applying a comprehensive treatment approach. Here are the core principles:

1. Chronic Inflammation Management

- Principle: Crohn's disease involves ongoing inflammation that affects various parts of the gastrointestinal (GI) tract. Effective management focuses on controlling this inflammation to minimize symptoms and prevent complications.
- Approach: Use anti-inflammatory and immunosuppressive medications to reduce inflammation and maintain remission.

2. Individualized Treatment

- Principle: Crohn's disease can present differently in each patient, requiring personalized treatment plans tailored to the severity and location of the disease.
- Approach: Develop and adjust treatment strategies based on individual patient needs, disease progression, and response to therapy.

3. Multidisciplinary Care

- Principle: The complexity of Crohn's disease necessitates a coordinated approach involving various healthcare professionals.
- Approach: Collaborate with gastroenterologists, surgeons, dietitians, psychologists, and other specialists to address all aspects of patient care, including medical, nutritional, and psychological needs.

4. Symptom Relief
- Principle: Addressing symptoms such as abdominal pain, diarrhea, and fatigue is crucial for improving quality of life.
- Approach: Implement symptomatic treatments, including medications to control diarrhea and pain, as well as dietary adjustments to manage symptoms effectively.

5. Nutritional Support
- Principle: Patients with Crohn's disease often experience nutritional deficiencies and malabsorption due to inflammation and disease-related factors.
- Approach: Provide nutritional counseling, manage deficiencies with supplements, and use enteral or parenteral nutrition if necessary to ensure adequate nutrient intake.

6. Monitoring and Preventing Complications
- Principle: Regular monitoring helps detect and manage potential complications, such as strictures, fistulas, and abscesses, early.
- Approach: Use routine imaging, endoscopic evaluations, and clinical assessments to monitor disease progression and prevent or address complications.

7. Patient Education and Self-Management
- Principle: Educating patients about their disease, treatment options, and self-management techniques is essential for effective disease control and adherence to treatment.
- Approach: Provide information on disease management, medication adherence, lifestyle modifications, and recognizing signs of flare-ups or complications.

8. Psychosocial Support
- Principle: Chronic illness like Crohn's disease can impact mental health and overall well-being, necessitating supportive care.

- Approach: Offer psychological support, counseling, and connect patients with support groups to address the emotional and social aspects of living with a chronic condition.

9. Long-Term Management

- Principle: Crohn's disease is a lifelong condition requiring ongoing management and adjustment of treatment strategies.
- Approach: Develop a long-term management plan that includes

10. Focus on Quality of Life

- Principle: The ultimate goal of treatment is to improve the patient's overall quality of life by managing symptoms, preventing complications, and supporting daily functioning.
- Approach: Integrate comprehensive care strategies to enhance physical health, emotional well-being, and overall life satisfaction.

## 4. ETOLOGY

The etiology of Crohn's disease, a chronic inflammatory bowel condition, is multifaceted and not yet fully understood. However, several factors are believed to contribute to its development:

1. Genetic Factors: There is a strong genetic component to Crohn's disease. Studies have identified various susceptibility genes associated with the disease, including those involved in immune system regulation and intestinal barrier function. For example, mutations in the NOD2/CARD15 gene are known to increase the risk of developing Crohn's disease.

2. Immune System Dysfunction: Crohn's disease is considered an autoimmune condition where the immune system erroneously attacks the gastrointestinal tract. This abnormal immune response leads to chronic inflammation and damage. The exact triggers for this immune dysfunction are not entirely clear but may involve an inappropriate reaction to intestinal microbes or other environmental factors.

3. Environmental Factors: Various environmental factors may play a role in the development of Crohn's disease. These include:

- Diet: Certain dietary patterns, such as high-fat or low-fiber diets, might influence the risk.
- Smoking: Tobacco use is a well-established risk factor for Crohn's disease and can also exacerbate the condition.
- Geography: The incidence of Crohn's disease varies geographically, with higher rates in industrialized countries, suggesting that environmental factors, including hygiene and sanitation, might be involved.

4. Microbiome: The gut microbiome, the community of microorganisms living in the intestines, may influence the onset and progression of Crohn's disease. Dysbiosis, an imbalance in the microbial community, might contribute to inflammation and disease development.

5. Impaired Intestinal Barrier: A compromised intestinal barrier, which allows harmful substances to penetrate the gut lining, can trigger an immune response leading to inflammation. Factors that impair the barrier function include genetic mutations, microbial imbalance, and environmental toxins.

6. Other Factors: Stress and psychological factors have also been explored as potential contributors, though they are typically considered secondary to the primary immunological and genetic factors.

In summary, Crohn's disease is likely caused by a complex interplay of genetic susceptibility, immune system dysfunction, environmental factors, and microbial influences. Ongoing research aims to better understand these interactions to improve prevention and treatment strategies.

## 5. PATHOPHYSIOLOGY

The pathophysiology of Crohn's disease involves a complex interaction between genetic, immune, and environmental factors, leading to chronic inflammation and damage to the gastrointestinal (GI) tract. Here's an overview of the key aspects:

1. Genetic Factors

- Genetic Predisposition: Genetic factors play a role in the susceptibility to Crohn's disease. Several genes, such as NOD2/CARD15, have been associated with an increased risk. These genes are involved in the immune response and recognition of bacterial pathogens.

2. Immune System Dysfunction

- Inflammatory Response: Crohn's disease is characterized by an inappropriate immune response to intestinal microbes or other environmental triggers. The immune system becomes activated and attacks the gut lining, leading to chronic inflammation.
- Inflammatory Cells: The disease involves an accumulation of immune cells such as macrophages, T lymphocytes (particularly Th1 and Th17 cells), and neutrophils. These cells release pro-inflammatory cytokines (e.g., TNF-alpha, IL-1, IL-6) and other mediators that contribute to inflammation and tissue damage.
- Disruption of Immune Tolerance: There is a failure in maintaining immune tolerance to the gut microbiota, which can exacerbate inflammation.

3. Mucosal Inflammation

- Transmural Inflammation: Crohn's disease affects all layers of the bowel wall (transmural inflammation), leading to damage beyond just the mucosa. This can cause thickening of the intestinal wall and fibrosis.
- Granuloma Formation: Granulomas, which are clusters of macrophages that transform into multinucleated giant cells, are a hallmark of Crohn's disease. They are found in about 30% of patients and are indicative of chronic inflammation.

4. Segmental Involvement

- Skip Lesions: Inflammation often occurs in discrete segments, or "skip lesions," where healthy areas of the intestine are interspersed between inflamed regions.
- Ileocolonic Involvement: The most common areas affected are the ileum (last part of the small intestine) and the colon (large intestine). However, Crohn's disease can affect any part of the GI tract from the mouth to the anus.

5. Structural Changes

- Strictures: Chronic inflammation leads to fibrosis and narrowing of the intestinal lumen, causing strictures (narrowed areas) that can obstruct the bowel.
- Fistulas: Inflammation and tissue damage can result in abnormal connections between different parts of the intestine (enteric fistulas) or between the intestine and other organs (e.g., bladder or skin).

- Abscesses: Pockets of pus can form in the inflamed tissue, leading to abscesses that may cause pain and fever.

6. Malabsorption and Nutritional Deficiencies

- Malabsorption: Inflammation and damage to the intestinal lining can impair nutrient absorption, leading to deficiencies in vitamins, minerals, and other nutrients.
- Weight Loss and Malnutrition: These deficiencies, along with increased metabolic demands from chronic inflammation, can result in weight loss and malnutrition.

7. Extraintestinal Manifestations

- Systemic Effects: Crohn's disease can also have manifestations outside the GI tract, including skin rashes, arthritis, and eye inflammation. These are thought to be related to the systemic inflammatory response.

8. Disease Course

- Relapsing and Remitting: The disease often follows a relapsing and remitting course, with periods of exacerbation (flare-ups) and remission (improvement or absence of symptoms).
- Segmental and Skip Lesions: Disease can affect different parts of the GI tract in a patchy manner.
- Complications: Includes strictures, fistulas, and abscesses, along with systemic effects and nutritional issues.

Understanding these mechanisms helps in developing targeted therapies and managing the disease effectively.

## 6. CLINICAL MANIFESTATION

Crohn's disease presents with a variety of signs and symptoms that can range from mild to severe and can affect different parts of the gastrointestinal (GI) tract. Here's a summary of the common signs and symptoms:

1. Abdominal Pain

- Characteristics: Often crampy and intermittent, located in the lower right abdomen if the ileum is involved, but can occur anywhere in the abdomen.

- Description: Pain is usually due to inflammation and can be severe during flare-ups.

2. Diarrhea

- Characteristics: Frequent, loose, and watery stools; can be persistent or occur intermittently.
- Description: May be accompanied by blood or mucus, especially if there is significant inflammation or ulceration.

3. Weight Loss

- Characteristics: Unintentional and significant weight loss due to reduced appetite, malabsorption of nutrients, and increased metabolic demands.
- Description: Can lead to malnutrition and overall physical deterioration.

4. Fatigue

- Characteristics: Persistent tiredness and lack of energy.
- Description: Often related to anemia (from blood loss or malabsorption), nutritional deficiencies, and the body's ongoing inflammatory response.

5. Fever

- Characteristics: Low-grade fever, especially during active disease or flare-ups.
- Description: Indicative of systemic inflammation or infection.

6. Nausea and Vomiting

- Characteristics: Can occur due to bowel obstruction, severe inflammation, or involvement of the upper GI tract.
- Description: May be intermittent or persistent, affecting overall comfort and nutritional intake.

7. Bloody Stools

- Characteristics: Presence of blood in the stool, which may be bright red or dark and tarry.
- Description: Indicative of inflammation or ulceration in the intestines.

8. Mucus in Stool

- Characteristics: Presence of mucus in the stool, often associated with inflammation.

- Description: Can be seen alongside other symptoms like diarrhea and abdominal pain.

9. Anorexia

- Characteristics: Loss of appetite and reduced food intake.
- Description: Often due to pain, nausea, or general malaise.

10. Extraintestinal Manifestations

- Skin Issues: Skin rashes, such as erythema nodosum (painful red nodules) or pyoderma gangrenosum (painful ulcers).
- Eye Inflammation: Conditions like uveitis or iritis.
- Joint Pain: Arthritis or arthralgia affecting various joints.
- Oral Lesions: Mouth sores or ulcers, also known as aphthous ulcers.

11. Abdominal Masses

- Characteristics: Palpable masses in the abdomen, often related to thickened bowel loops or abscesses.
- Description: Can be detected on physical examination or imaging studies.

12. Fistulas

- Characteristics: Abnormal connections between the intestine and other organs or the skin.
- Description: May cause discharge of stool or intestinal fluids through the skin or other areas, leading to discomfort and infection risk.

13. Constipation

- Characteristics: Less common, but can occur due to strictures or other obstructions.
- Description: Difficulty passing stool or infrequent bowel movements.

14. Bowel Obstruction

- Characteristics: Symptoms include severe abdominal pain, vomiting, and inability to pass gas or stool.
- Description: Can be due to strictures or adhesions in the intestines.

15. Night Sweats

- Characteristics: Episodes of sweating at night, which can be related to systemic inflammation or fever.
- Description: Can affect sleep quality and contribute to fatigue.

## 7. DIAGNOSTIC EVALUATION

Diagnosing Crohn's disease involves a combination of clinical assessment, laboratory tests, imaging studies, and endoscopic procedures. Here's an overview of the diagnostic evaluation process:

1. Clinical Assessment
   - Medical History: Detailed patient history, including symptoms (e.g., abdominal pain, diarrhea, weight loss), duration, and pattern of symptoms.
   - Physical Examination: Includes checking for signs such as abdominal tenderness, swelling, or masses.

2. Laboratory Tests
   - Blood Tests: To check for anemia, elevated white blood cell count, and inflammatory markers like C-reactive protein (CRP) or erythrocyte sedimentation rate (ESR).
   - Stool Tests: To rule out infections and check for markers of inflammation, such as fecal calprotectin or lactoferrin.

3. Imaging Studies
   - Abdominal Ultrasound: Useful for initial evaluation and to identify complications like bowel thickening or abscesses.
   - Computed Tomography (CT) Scan: Provides detailed images of the intestines and can help identify inflammation, strictures, fistulas, and other complications. A CT enterography, which is a specialized form of CT scan, is particularly useful for visualizing the small intestine.
   - Magnetic Resonance Imaging (MRI): Particularly useful for evaluating the small intestine and for assessing complications like fistulas and abscesses. MRI enterography is a specific type of MRI used for this purpose.

4. Endoscopic Procedures

- Colonoscopy: Direct visualization of the colon and the terminal ileum (the end of the small intestine) allows for diagnosis of inflammation, ulcerations, and other changes. Biopsies can be taken during this procedure to confirm the diagnosis.
- Upper Endoscopy (Esophagogastroduodenoscopy, EGD): If there are symptoms or signs of upper gastrointestinal involvement, this procedure allows visualization of the esophagus, stomach, and duodenum.

5. Histological Examination
- Biopsy: Tissue samples obtained during endoscopy are examined under a microscope to identify characteristic features of Crohn's disease, such as non-caseating granulomas, which can help confirm the diagnosis.

6. Capsule Endoscopy
- Capsule Endoscopy: Involves swallowing a small camera that takes pictures of the small intestine. This is particularly useful when traditional endoscopy cannot access certain parts of the small intestine.

7. Differential Diagnosis
- Ruling Out Other Conditions: It's essential to distinguish Crohn's disease from other conditions with similar symptoms, such as ulcerative colitis, infections, irritable bowel syndrome (IBS), and colorectal cancer. This often involves additional tests or imaging studies.

Each of these diagnostic tools provides different pieces of information and helps clinicians form a comprehensive view of the disease's presence and extent. The combination of clinical, laboratory, imaging, and endoscopic findings is used to confirm the diagnosis and determine the most appropriate treatment plan.

## 8. NURSING MANAGEMENT

Nursing management of Crohn's disease involves a comprehensive approach to address both the physical and emotional aspects of the condition. The goal is to control inflammation, alleviate symptoms, prevent complications, and support overall well-being. Here's a detailed overview of nursing management strategies:

1. Assessment and Monitoring

- Symptom Assessment: Regularly monitor and document symptoms such as abdominal pain, diarrhea, weight loss, and fever. Evaluate the frequency, severity, and impact on daily life.
- Vital Signs: Check and record vital signs, including temperature, pulse, blood pressure, and weight. Monitor for signs of dehydration, fever, or systemic inflammation.
- Nutritional Status: Assess dietary intake and monitor for signs of malnutrition or deficiencies. Track weight changes and assess growth in pediatric patients.

2. Medication Administration and Management

- Medications: Administer prescribed medications, including anti-inflammatory drugs (e.g., corticosteroids, aminosalicylates), immunosuppressants, and biologics. Ensure correct dosage and timing.
- Side Effects: Monitor for and report side effects of medications, such as infections, gastrointestinal disturbances, or skin reactions.
- Patient Education: Educate patients about the purpose, side effects, and proper use of their medications. Emphasize the importance of adherence to the treatment regimen.

3. Nutritional Support

- Dietary Modifications: Collaborate with dietitians to create individualized meal plans that accommodate the patient's symptoms and nutritional needs. This may involve low-residue diets or specific food avoidance.
- Supplementation: Provide or recommend nutritional supplements as needed to address deficiencies (e.g., vitamins, minerals, or enteral nutrition).
- Monitoring: Regularly monitor weight and nutritional status. Adjust dietary plans based on changes in symptoms or disease status.

4. Symptom Management

- Pain Relief: Use medications and non-pharmacological methods (e.g., heat application) to manage abdominal pain. Evaluate the effectiveness and adjust treatment as needed.
- Diarrhea Management: Administer antidiarrheal medications as prescribed and recommend dietary changes to reduce bowel movements and discomfort.

- Fever Management: Monitor body temperature and provide antipyretics if needed. Assess the cause of fever, whether due to infection or inflammation.

5. Preventing and Managing Complications

- Complications: Monitor for signs of complications such as strictures, fistulas, and abscesses. Perform regular assessments and follow-up imaging or endoscopic studies as indicated.
- Surgical Care: For patients undergoing surgery, provide pre-operative and post-operative care, including wound management, pain control, and monitoring for signs of infection or complications.

6. Psychological and Emotional Support

- Counseling: Offer emotional support and counseling to help patients cope with the stress of a chronic illness. Address issues related to anxiety, depression, or body image.
- Support Groups: Encourage participation in support groups or connect patients with resources for additional psychological and social support.

7. Patient Education and Self-Management

- Disease Education: Educate patients about Crohn's disease, its symptoms, and treatment options. Provide information on how to manage flare-ups and maintain remission.
- Self-Monitoring: Teach patients how to monitor their symptoms, recognize early signs of exacerbation, and when to seek medical attention.
- Lifestyle Modifications: Advise on lifestyle changes that may help manage symptoms, such as stress reduction techniques and smoking cessation.

8. Coordination of Care

- Multidisciplinary Team: Collaborate with other healthcare professionals, including gastroenterologists, dietitians, and psychologists, to provide comprehensive care.
- Care Planning: Develop and update individualized care plans based on ongoing assessments and patient needs.

9. Monitoring and Follow-Up

- Regular Follow-Up: Schedule and attend regular follow-up appointments to assess disease status, monitor treatment efficacy, and make necessary adjustments.
- Lab and Imaging Studies: Coordinate and interpret laboratory tests (e.g., blood counts, inflammatory markers) and imaging studies to monitor disease activity and treatment response.

10. Education on Lifestyle and Disease Management

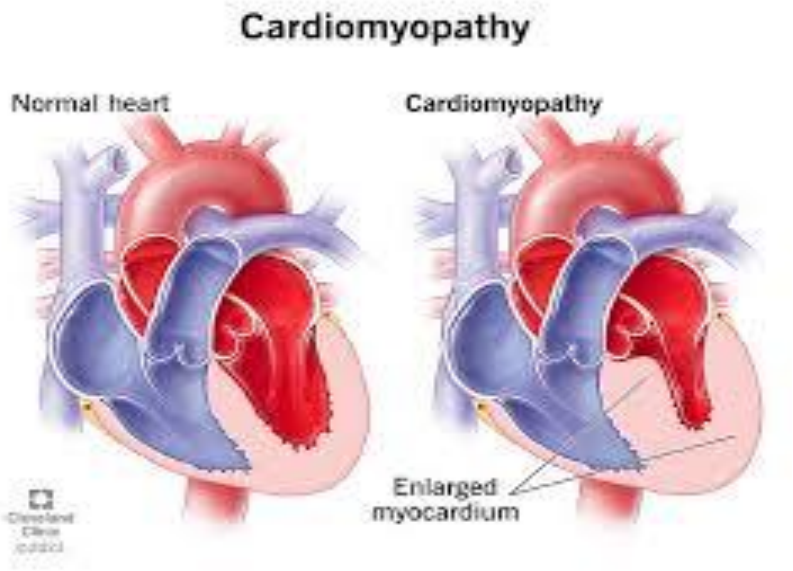
- Diet and Exercise: Advise on appropriate exercise routines and dietary habits that support overall health and may help manage Crohn's disease symptoms.
- Advance Care Planning: Discuss advance directives and end-of-life care options if appropriate, ensuring that patients' preferences are documented and respected.

By focusing on these aspects, nurses can play a critical role in managing Crohn's disease, improving patient outcomes, and enhancing the quality of life for individuals living with this chronic condition.

# CARDIOMYOPATHY

## 1. DEFINITION

Cardiomyopathy is a term used to describe a group of diseases that affect the heart muscle (myocardium). These diseases lead to changes in the heart's structure and function, which can impair its ability to pump blood effectively. Cardiomyopathy can result in heart failure, arrhythmias (irregular heartbeats), and other complications.




## 2. OBJECTIVE

The objectives of managing cardiomyopathy focus on improving heart function, alleviating symptoms, and preventing complications to enhance the patient's quality of life. Here's a detailed overview of the key objectives:

1. Improve Heart Function

- Objective: Enhance the heart's ability to pump blood effectively and manage the disease's impact on the heart muscle.
- Strategies: Use medications (e.g., beta-blockers, ACE inhibitors, diuretics) to support cardiac function, and consider interventions such as implantable devices or surgery if necessary.

2. Alleviate Symptoms

- Objective: Reduce symptoms such as shortness of breath, fatigue, chest pain, and swelling to improve daily functioning and comfort.
- Strategies: Implement symptom-specific treatments, such as diuretics for fluid retention, and provide education on lifestyle modifications and symptom management techniques.

**3.** Prevent Complication

- Objective: Minimize the risk of complications such as heart failure, arrhythmias, thromboembolic events, and sudden cardiac death.
- Strategies: Regular monitoring, use of anticoagulants or antiarrhythmic medications as needed, and prompt management of any emerging complications.

4. Manage Underlying Causes and Contributing Factors

- Objective: Address any underlying conditions or factors that may be contributing to cardiomyopathy, such as hypertension, diabetes, or thyroid disorders.
- Strategies: Implement treatment plans for associated conditions, including lifestyle changes, medications, and monitoring.

5. Improve Quality of Life

- Objective: Enhance the overall well-being of patients by managing disease symptoms, improving functional capacity, and supporting emotional health.
- Strategies: Provide supportive care, patient education, and access to resources such as counseling or support groups.

6. Educate and Empower Patients

- Objective: Ensure that patients understand their condition, treatment options, and self-management strategies.
- Strategies: Offer education on disease management, medication adherence, dietary modifications, and recognizing signs of worsening conditions.

7. Regular Monitoring and Follow-Up

- Objective: Track disease progression, treatment response, and potential side effects of therapies to adjust the management plan as needed.
- Strategies: Schedule regular follow-up visits, laboratory tests, and imaging studies to monitor heart function and overall health.

8. Optimize Medication Management

- Objective: Achieve optimal therapeutic effects while minimizing side effects and drug interactions.
- Strategies: Tailor medication regimens to the individual patient's needs, regularly review and adjust dosages, and monitor for side effects.

9. Support Lifestyle Modifications

- Objective: Implement lifestyle changes that support heart health and overall well-being.
- Strategies: Advise on dietary changes, regular physical activity (as appropriate), stress management, and smoking cessation.

10. Prepare for Advanced Care if Needed

- Objective: Prepare for potential advanced treatments, including heart transplantation or mechanical assist devices, if the condition progresses to end-stage heart failure.
- Strategies: Evaluate eligibility for advanced therapies, coordinate with specialists, and provide patient education on these options.

11. Coordinate Multidisciplinary Care

- Objective: Ensure comprehensive care by involving various healthcare professionals, including cardiologists, dietitians, psychologists, and other specialists.
- Strategies: Facilitate communication and coordination among healthcare providers to address all aspects of patient care.

4. By focusing on these objectives, healthcare providers can deliver effective and holistic management for individuals with cardiomyopathy, aiming to improve heart function, reduce symptoms, and enhance overall quality of life.

## 3. PRINCIPLE

The principles of managing cardiomyopathy involve understanding the underlying pathology of the disease, addressing its diverse effects on heart function, and applying a comprehensive, patient-centered approach to treatment. Here are the key principles:

1. Understanding the Disease

- Principle: Cardiomyopathy encompasses various types of heart muscle diseases, each with distinct pathophysiological mechanisms, including dilated, hypertrophic, restrictive, and arrhythmogenic cardiomyopathies.
- Approach: Accurate diagnosis and classification of the specific type of cardiomyopathy are crucial for tailoring effective treatment strategies.

2. Improving Heart Function

- Principle: The primary goal is to enhance the heart's ability to pump blood effectively, which may be compromised due to the disease.
- Approach: Utilize medications (e.g., beta-blockers, ACE inhibitors) and interventions (e.g., pacemakers, defibrillators) to support and improve cardiac function.

3. Alleviating Symptoms

- Principle: Management should focus on reducing symptoms such as dyspnea (shortness of breath), fatigue, and edema (swelling) to improve the patient’s quality of life.
- Approach: Employ symptomatic treatments, including diuretics for fluid retention, pain management, and lifestyle modifications.

4. Preventing Complications

- Principle: Cardiomyopathy can lead to serious complications like heart failure, arrhythmias, and thromboembolic events.
- Approach: Implement strategies to prevent and manage complications, including anticoagulant therapy for preventing blood clots, and antiarrhythmic medications or devices for managing arrhythmias.

5. Addressing Underlying Causes

- Principle: Identifying and managing any underlying conditions or contributing factors, such as hypertension or metabolic disorders, is essential.
- Approach: Treat contributing conditions aggressively and make lifestyle adjustments to manage risk factors.

6. Patient-Centered Care

- Principle: Treatment should be individualized based on the specific type of cardiomyopathy, patient's symptoms, and overall health status.
- Approach: Develop personalized care plans that address the patient's unique needs and preferences, and involve them in decision-making.

7. Ongoing Monitoring and Follow-Up

- Principle: Regular monitoring of the patient's condition and treatment response is critical for effective management.
- Approach: Schedule regular follow-up appointments, laboratory tests, and imaging studies to track disease progression and adjust treatment as necessary.

8. Multidisciplinary Collaboration

- Principle: Cardiomyopathy management often requires input from various healthcare professionals.

- Approach: Coordinate care among cardiologists, primary care providers, dietitians, psychologists, and other specialists to ensure comprehensive management.

9. Educating and Empowering Patients

- Principle: Providing education about the disease, treatment options, and self-management is crucial for effective care.
- Approach: Offer guidance on medication adherence, lifestyle changes, and recognizing symptoms that require medical attention.

10. Supporting Lifestyle Changes

- Principle: Lifestyle modifications can significantly impact the management of cardiomyopathy and overall heart health.
- Approach: Advise on dietary changes, physical activity, stress management, and smoking cessation to support overall cardiovascular health.

11. Preparing for Advanced Interventions

- Principle: In cases of advanced cardiomyopathy or end-stage heart failure, consideration for more intensive interventions may be necessary.
- Approach: Evaluate eligibility for heart transplantation or mechanical assist devices and provide appropriate referrals and support.

12. Psychosocial Support

- Principle: Chronic heart disease can impact mental health and overall well-being.
- Approach: Offer psychological support, counseling, and connect patients with support groups to address emotional and social aspects of living with cardiomyopathy.

By adhering to these principles, healthcare providers can offer effective, holistic management of cardiomyopathy, aiming to improve heart function, alleviate symptoms, prevent complications, and enhance the overall quality of life for affected individuals.

## 4. ETIOLOGY

Cardiomyopathy refers to a group of diseases affecting the heart muscle, impairing its ability to pump blood effectively. The etiology of cardiomyopathy is diverse, encompassing genetic, acquired, and environmental factors. Here's a detailed look at the various causes:

1. Genetic Factors

- Inherited Cardiomyopathies: Many forms of cardiomyopathy have a genetic basis. Genetic mutations can lead to structural and functional abnormalities in the heart muscle. Key types include:
  - Hypertrophic Cardiomyopathy (HCM): Often caused by mutations in genes encoding proteins of the cardiac sarcomere (the heart's contractile unit), such as MYH7 (beta-myosin heavy chain) and MYBPC3 (cardiac myosin-binding protein C).
  - Dilated Cardiomyopathy (DCM): Can be linked to mutations in genes such as TTN (titin) and LMNA (lamin A/C), affecting the heart's ability to contract and relax properly.
  - Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC): Associated with mutations in genes involved in cardiac cell adhesion and desmosome function, such as PKP2 (plakophilin-2).
  - Restrictive Cardiomyopathy (RCM): May be related to mutations in genes like TNNT2 (cardiac troponin T) or can be a manifestation of systemic diseases.

2. Acquired Factors

- Coronary Artery Disease (CAD): Chronic ischemia due to CAD can lead to cardiomyopathy, often manifesting as ischemic cardiomyopathy.
- Hypertension: Chronic high blood pressure can cause the heart muscle to thicken (hypertrophy) or become stiff (restrictive cardiomyopathy).
- Infections: Viral infections, such as those caused by viruses like myocarditis, can lead to inflammation and damage of the heart muscle.

- Toxins: Exposure to certain substances, including alcohol and drugs (e.g., chemotherapy agents like doxorubicin), can induce cardiomyopathy.
- Endocrine Disorders: Conditions such as thyroid disease (hyperthyroidism or hypothyroidism) or diabetes can contribute to cardiomyopathy through metabolic disturbances.
- Nutritional Deficiencies: Deficiencies in nutrients like thiamine (vitamin B1) can lead to dilated cardiomyopathy, as seen in conditions like beriberi.
- Autoimmune Diseases: Conditions like systemic lupus erythematosus (SLE) or rheumatoid arthritis can cause inflammatory cardiomyopathy.

3. Environmental and Lifestyle Factors
- Lifestyle Factors: Long-term substance abuse, including excessive alcohol consumption or recreational drug use, can cause cardiomyopathy.
- Pregnancy: Peripartum cardiomyopathy occurs towards the end of pregnancy or in the months following childbirth, with unclear exact causes but associated with factors like preeclampsia.

4. Idiopathic Cardiomyopathy
- In some cases, the cause of cardiomyopathy cannot be determined despite extensive investigation. These cases are termed idiopathic.

5. Systemic Diseases
- Systemic Diseases: Conditions such as hemochromatosis (iron overload) or amyloidosis (abnormal protein deposits) can lead to secondary cardiomyopathy.

6. Other Conditions
- Inflammatory Diseases: Diseases like sarcoidosis can involve the heart and cause cardiomyopathy.
- Genetic Syndromes: Certain genetic syndromes, like Noonan syndrome, can have cardiomyopathy as a feature.

Summary

The etiology of cardiomyopathy is complex, with various genetic, acquired, and environmental factors contributing to the development of the condition. Identifying the specific cause is crucial for determining the

most effective treatment and management strategies. Advances in genetics and diagnostic techniques are improving the ability to pinpoint the underlying causes and tailor interventions accordingly.

## 5. PATHOPHYSIOLOGY

The pathophysiology of cardiomyopathy involves various mechanisms depending on the specific type of cardiomyopathy. Each type affects the heart muscle differently, leading to impaired cardiac function. Here's an overview of the pathophysiological mechanisms underlying the major types of cardiomyopathy:

1. Dilated Cardiomyopathy (DCM)

Pathophysiology:

- Myocyte Dysfunction: In DCM, the heart's ventricles become dilated and the myocardium (heart muscle) becomes weakened. This leads to impaired contractility (the heart's ability to pump blood) and reduced ejection fraction (the fraction of blood ejected from the heart with each beat).
- Stretch and Fibrosis: The dilation of the ventricles causes stretching of the heart muscle fibers, which can lead to interstitial fibrosis (scarring of the heart tissue). Fibrosis disrupts normal electrical conduction and can lead to arrhythmias.
- Inflammatory and Genetic Factors: In some cases, inflammation (e.g., viral myocarditis) or genetic mutations contribute to the development of DCM. Chronic inflammation or genetic defects can damage and weaken the heart muscle.

2. Hypertrophic Cardiomyopathy (HCM)

Pathophysiology:

- Myocyte Hypertrophy: In HCM, there is abnormal thickening of the heart muscle, particularly in the ventricular walls. This hypertrophy can be asymmetric, affecting primarily the septum (the wall between the left and right ventricles).

- Impaired Diastolic Function: The thickened myocardium reduces the heart's ability to relax and fill properly with blood during diastole (the relaxation phase). This leads to increased filling pressures and symptoms of heart failure.
- Outflow Obstruction: In some cases, the thickened muscle obstructs blood flow from the left ventricle outflow tract (LVOT), particularly during systole (the contraction phase). This can cause dynamic left ventricular outflow tract obstruction.
- Genetic Mutations: HCM is often inherited and linked to mutations in genes encoding proteins of the cardiac sarcomere (the contractile unit of the heart muscle). These mutations disrupt normal muscle function and lead to hypertrophy.

3. Restrictive Cardiomyopathy (RCM)

Pathophysiology:

- Increased Stiffness: RCM is characterized by increased stiffness of the heart muscle, which impairs the heart’s ability to expand and fill with blood during diastole. This results in elevated filling pressures and symptoms of heart failure.
- Fibrosis or Infiltration: The increased stiffness can be due to fibrosis (excessive scarring) or infiltration of abnormal substances (such as amyloid deposits in amyloid cardiomyopathy). These changes decrease the elasticity of the heart muscle.
- Impaired Relaxation: The heart's ventricles cannot accommodate blood flow effectively, leading to elevated pressures in the heart and lungs, contributing to symptoms such as shortness of breath and fluid retention.

4. Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC)

Pathophysiology:

- Fibrofatty Replacement: ARVC is characterized by the replacement of the right ventricular myocardium with fibrous and fatty tissue. This replacement disrupts normal electrical conduction and can lead to arrhythmias.

- Myocyte Loss: There is loss of normal heart muscle cells (myocytes) and replacement with fibrous tissue, which leads to structural and electrical abnormalities in the right ventricle.
- Genetic Factors: ARVC often has a genetic basis, with mutations affecting proteins involved in cell-cell adhesion and structural integrity of the heart muscle. These mutations contribute to the loss of normal myocardial structure and function.

General Pathophysiological Mechanisms Across Types

- Impaired Cardiac Output: Regardless of the type, cardiomyopathy typically leads to a decrease in the heart's ability to pump blood effectively, resulting in reduced cardiac output and symptoms of heart failure.
- Altered Hemodynamics: Changes in heart muscle structure and function lead to alterations in hemodynamics, including increased filling pressures, impaired contractility, and altered blood flow dynamics.
- Arrhythmias: Many forms of cardiomyopathy are associated with increased risk of arrhythmias due to structural changes in the heart muscle, altered electrical conduction pathways, and fibrosis.
- Heart Failure: Progressive deterioration of heart function in cardiomyopathy often results in heart failure, characterized by symptoms like dyspnea (shortness of breath), edema (swelling), and fatigue.

Understanding the pathophysiology of cardiomyopathy helps in diagnosing the condition accurately and tailoring appropriate treatment strategies to manage the disease effectively and improve patient outcomes.

## 6. CLINICAL MANIFESATATION

Cardiomyopathy presents with a range of signs and symptoms that vary depending on the type and severity of the condition. Here's a summary of common signs and symptoms associated with different types of cardiomyopathy:

1. Dilated Cardiomyopathy (DCM)

Common Symptoms:

- Shortness of Breath (Dyspnea): Difficulty breathing, particularly during exertion or when lying flat, due to fluid accumulation in the lungs.
- Fatigue: Persistent tiredness and decreased exercise tolerance due to reduced cardiac output.
- Edema: Swelling in the legs, ankles, or abdomen from fluid retention.
- Palpitations: Sensation of irregular or rapid heartbeats due to arrhythmias.
- Chest Pain: Discomfort or pain in the chest, which may be related to heart strain or ischemia.
- Reduced Exercise Capacity: Difficulty performing physical activities that were previously manageable.
- Dizziness or Fainting (Syncope): Occasional dizziness or fainting episodes due to decreased blood flow or arrhythmias.

2. Hypertrophic Cardiomyopathy (HCM)

Common Symptoms:

- Shortness of Breath: Difficulty breathing during physical activity or at rest due to impaired diastolic filling and potential obstruction of blood flow.
- Chest Pain: Discomfort or pain in the chest, especially during exertion, due to increased cardiac workload or ischemia.
- Palpitations: Irregular heartbeats or rapid heart rates due to arrhythmias, including atrial fibrillation or ventricular tachycardia.
- Fatigue: Generalized weakness and reduced exercise capacity due to decreased cardiac function.
- Dizziness or Lightheadedness: Feeling faint or dizzy, which may be related to arrhythmias or reduced cardiac output.
- Fainting (Syncope): Sudden loss of consciousness, often related to arrhythmias or obstructive symptoms.

3. Restrictive Cardiomyopathy (RCM)

Common Symptoms:

- Shortness of Breath: Difficulty breathing, particularly when lying flat, due to elevated pressures in the heart and lungs.

- Fatigue: Persistent tiredness and reduced exercise tolerance due to impaired cardiac filling and reduced cardiac output.
- Edema: Swelling in the legs, ankles, or abdomen due to fluid accumulation.
- Abdominal Pain: Discomfort or pain in the abdomen due to liver congestion or fluid accumulation.
- Palpitations: Sensation of irregular or rapid heartbeats due to arrhythmias.
- Cough: Persistent cough, often due to pulmonary congestion.

4. Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC)

Common Symptoms:

- Palpitations: Sensation of irregular or rapid heartbeats, often due to arrhythmias originating in the right ventricle.
- Shortness of Breath: Difficulty breathing during exertion or at rest, potentially due to heart failure or reduced cardiac output.
- Fatigue: Generalized weakness and decreased exercise capacity due to impaired heart function.
- Dizziness or Lightheadedness: Feeling faint or lightheaded, which may be related to arrhythmias or reduced cardiac output.
- Fainting (Syncope): Sudden loss of consciousness, often related to arrhythmias or reduced blood flow to the brain.
- Chest Pain: Discomfort or pain in the chest, which may occur due to arrhythmias or strain on the heart.

General Symptoms Across All Types

- Heart Failure Symptoms: Symptoms like shortness of breath, fatigue, and edema are common across different types of cardiomyopathy due to the overall impact on heart function.
- Arrhythmias: Irregular heartbeats or palpitations are a frequent symptom across all types of cardiomyopathy due to structural and electrical changes in the heart muscle.
- Reduced Exercise Tolerance: Difficulty with physical activity due to impaired cardiac function is common in all types of cardiomyopathy.

When to Seek Medical Attention

- Severe Symptoms: Seek immediate medical attention if experiencing severe shortness of breath, chest pain, dizziness, or fainting.
- Worsening Symptoms: Contact a healthcare provider if symptoms such as fatigue, swelling, or palpitations worsen or become more frequent.

Recognizing these signs and symptoms early is crucial for diagnosing cardiomyopathy and initiating appropriate management to improve patient outcomes and quality of life.

## 7. DIAGNOSTIC EVALUATION

Diagnosing cardiomyopathy involves a comprehensive evaluation that combines clinical assessment, imaging studies, laboratory tests, and sometimes genetic testing. Here's an overview of the diagnostic approach:

1. Clinical Assessment

- Medical History: Detailed history of symptoms (e.g., shortness of breath, fatigue, palpitations, chest pain), family history of heart disease, and any relevant personal history (e.g., exposure to toxins, recent infections, or systemic illnesses).
- Physical Examination: Assessment of vital signs, presence of heart murmurs, signs of heart failure (e.g., edema, jugular venous distention), and other relevant findings.

2. Laboratory Tests

- Blood Tests: To identify biomarkers of heart damage and inflammation, such as:
  - B-type Natriuretic Peptide (BNP) or N-terminal proBNP: Elevated levels can indicate heart failure.
  - Cardiac Troponins: Elevated levels may suggest myocardial injury or stress.
  - Complete Blood Count (CBC), Electrolytes, Kidney Function Tests, and Thyroid Function Tests: To identify contributing factors like anemia, electrolyte imbalances, or thyroid dysfunction.
  - Genetic Testing: If a hereditary cardiomyopathy is suspected, testing for specific genetic mutations may be recommended.

3. Imaging Studies

- Electrocardiogram (ECG): Assesses heart rhythm, electrical activity, and can reveal signs of hypertrophy, arrhythmias, or ischemia.
- Echocardiogram: The primary tool for diagnosing cardiomyopathy. It provides detailed images of the heart's structure and function, including:
  - Chamber Sizes: To assess dilatation in dilated cardiomyopathy or hypertrophy in hypertrophic cardiomyopathy.
  - Wall Motion: To evaluate the heart's pumping ability and identify areas of dysfunction.
  - Ventricular Function: To assess ejection fraction (EF), an important measure of how well the heart pumps blood.
  - Diastolic Function: To evaluate how well the heart relaxes and fills with blood.
- Cardiac MRI: Provides detailed images of the heart muscle and can help assess:
  - Myocardial Structure and Function: To identify fibrosis, inflammation, or infiltrative processes.
  - Myocardial Scarring: Useful for diagnosing certain types of cardiomyopathy like arrhythmogenic right ventricular cardiomyopathy (ARVC) and differentiating it from other conditions.
- Cardiac CT Scan: Can be used to assess coronary artery anatomy and rule out coronary artery disease, which may mimic or contribute to cardiomyopathy.

4. Endomyocardial Biopsy

- Biopsy: A small sample of heart tissue is obtained via catheterization and examined microscopically. This is often used in cases where the diagnosis is uncertain and may help identify inflammatory or infiltrative conditions, such as myocarditis or amyloidosis.

5. Electrophysiological Studies

- Holter Monitor: Continuous ECG monitoring over 24-48 hours to identify arrhythmias that may not be detected during a standard ECG.
- Event Monitor: Similar to Holter monitoring but used for longer periods (weeks to months) to capture infrequent arrhythmias.

6. Stress Testing

- Exercise Stress Test: Assesses how the heart performs under physical stress and may help identify functional limitations and exercise-induced symptoms.

7. Specialized Testing

- Genetic Testing: For suspected hereditary cardiomyopathies, testing for specific genetic mutations can confirm a diagnosis and guide family screening and management.
- Sarcoidosis or Amyloidosis Evaluation: If these conditions are suspected, additional tests such as specific blood tests, imaging, or biopsy of affected organs may be required.

Summary

The diagnostic evaluation of cardiomyopathy is multi-faceted, combining clinical, laboratory, imaging, and sometimes genetic approaches to accurately diagnose the type and cause of cardiomyopathy. This comprehensive approach ensures appropriate management and treatment tailored to the specific type and underlying cause of the disease.

## 8. NURSING MANAGEMENT

Nursing management of cardiomyopathy involves a multifaceted approach to optimize heart function, manage symptoms, and improve the overall quality of life for patients. Here's a comprehensive guide to nursing management for patients with cardiomyopathy:

1. Assessment and Monitoring

- Vital Signs: Regularly monitor blood pressure, heart rate, respiratory rate, and temperature. Pay particular attention to signs of heart failure, such as elevated blood pressure or increased heart rate.
- Symptom Assessment: Evaluate symptoms such as shortness of breath, fatigue, chest pain, and edema. Document the severity, frequency, and triggers of symptoms.

- Fluid Status: Monitor for signs of fluid retention, including edema in the extremities or abdomen. Measure daily weights to detect weight gain indicative of fluid overload.
- Heart Sounds and Rhythm: Assess heart sounds for abnormalities such as murmurs or gallops and monitor for irregular heart rhythms through auscultation and ECG if needed.

2. Medication Management

- Administer Medications: Provide prescribed medications, which may include:
  - Diuretics: To manage fluid retention and reduce edema.
  - ACE Inhibitors: To decrease blood pressure and reduce the workload on the heart.
  - Beta-Blockers: To reduce heart rate and improve cardiac output.
  - Antiarrhythmics: To manage and prevent arrhythmias.
  - Anticoagulants: If prescribed, to reduce the risk of blood clots.
- Monitor for Side Effects: Watch for and report any adverse effects from medications, such as hypotension, electrolyte imbalances, or gastrointestinal issues.
- Patient Education: Educate patients about the purpose of their medications, proper dosage, potential side effects, and the importance of adherence to the prescribed regimen.

3. Symptom Management

- Shortness of Breath: Implement measures to alleviate dyspnea, such as positioning the patient in an upright position or using supplemental oxygen if indicated.
- Pain Management: Assess and manage chest pain or discomfort, which may involve administering analgesics and evaluating pain relief strategies.
- Edema Management: Elevate edematous limbs and use compression stockings if prescribed. Monitor fluid intake and output to manage fluid balance effectively.

4. Lifestyle and Dietary Modifications

- Dietary Management:

- Low-Sodium Diet: Educate patients on the importance of reducing sodium intake to manage fluid retention and blood pressure.
- Heart-Healthy Diet: Promote a diet rich in fruits, vegetables, lean proteins, and whole grains to support overall cardiovascular health.
- Exercise: Encourage appropriate levels of physical activity as tolerated, considering the patient's symptoms and limitations. Collaborate with physical therapists if needed.
- Weight Management: Advise patients on maintaining a healthy weight and monitor for any sudden weight changes that may indicate fluid retention.

5. Psychosocial Support

- Emotional Support: Provide psychological support to help patients cope with the stress and emotional impact of living with a chronic condition. Address any concerns or fears related to their diagnosis and treatment.
- Counseling and Resources: Offer information about support groups or counseling services to help patients manage the emotional and social aspects of their condition.

6. Patient Education

- Disease Education: Educate patients about cardiomyopathy, including its effects on heart function, potential complications, and the importance of treatment adherence.
- Self-Monitoring: Teach patients how to monitor their symptoms, recognize signs of worsening condition (e.g., increased shortness of breath, significant weight gain), and when to seek medical attention.
- Lifestyle Changes: Provide guidance on making necessary lifestyle changes, including dietary adjustments, smoking cessation, and stress management techniques.

7. Coordination of Care

- Multidisciplinary Approach: Collaborate with cardiologists, dietitians, physical therapists, and other healthcare professionals to ensure comprehensive care.
- Care Planning: Develop and update individualized care plans based on the patient’s needs, symptoms, and response to treatment.

8. Monitoring for Complications

- Heart Failure: Regularly assess for signs of worsening heart failure and manage accordingly. Ensure that patients are aware of symptoms that might indicate a worsening condition.
- Arrhythmias: Monitor for and manage arrhythmias that may arise from structural changes in the heart. Perform regular ECGs or Holter monitoring as required.

9. Advanced Care Planning

- End-of-Life Care: Discuss advanced care planning and end-of-life options if the patient's condition progresses to end-stage heart failure. Ensure that patients' wishes are documented and communicated with the care team.
- Heart Transplant Evaluation: For patients with severe cardiomyopathy, assess eligibility for heart transplantation or mechanical assist devices if applicable.

10. Follow-Up and Regular Visits

- Scheduled Appointments: Ensure regular follow-up visits to monitor the patient's condition, review treatment efficacy, and adjust care plans as needed.
- Patient Compliance: Evaluate and encourage adherence to treatment plans and follow-up schedules.

Effective nursing management of cardiomyopathy involves a holistic approach that integrates symptom management, patient education, lifestyle modifications, and coordination with a multidisciplinary team to improve patient outcomes and quality of life.

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# LIVER CIRRHOSIS

## 1. DEFINITION

Liver cirrhosis is a disease characterized by the presence of diffuse and chronic inflammation of the liver, followed by the proliferation connective tissue, degeneration and regeneration of liver cells, so that riot in the composition of the liver parenchyma.

## 2. OBJECTIVE

1. Prevent Disease Progression: Halt or slow down the progression of cirrhosis through medical treatments, lifestyle changes, and addressing underlying causes.
2. Manage Symptoms: Alleviate symptoms associated with cirrhosis, such as fatigue, itching, and jaundice, to improve quality of life.
3. Treat Complications: Address and manage complications that arise from cirrhosis, such as ascites (fluid buildup), hepatic encephalopathy (brain dysfunction),

and variceal bleeding (bleeding from dilated veins in the esophagus or stomach).

4. Improve Liver Function: Support and improve liver function where possible, through medications, diet modifications, and sometimes surgical interventions.

5. Prevent Liver Failure: Reduce the risk of liver failure by managing the disease effectively and addressing any contributing factors.

6. Evaluate for Liver Transplantation: For patients with advanced cirrhosis, assess eligibility for a liver transplant as a potential long-term solution.

7. Promote Healthy Lifestyle Choices: Encourage lifestyle changes such as

avoiding alcohol, maintaining a healthy diet, and managing other health conditions (e.g., diabetes, obesity).

## 3. PRINCIPLE

The principle of liver cirrhosis revolves around understanding the pathophysiology of the disease, its progression, and its impact on liver function. Here are the key principles:

1. Chronic Liver Injury: Liver cirrhosis begins with chronic injury to the liver cells. This injury can result from various factors such as chronic viral hepatitis, chronic alcohol consumption, non-alcoholic fatty liver disease, autoimmune hepatitis, or certain medications.

2. Inflammatory Response: Persistent liver injury leads to inflammation. The liver attempts to repair itself, but ongoing inflammation contributes to further damage.

3. Fibrosis Formation: In response to repeated injury and inflammation, the liver starts to produce excessive connective tissue, leading to fibrosis. This fibrous tissue replaces healthy liver tissue, forming scar tissue.

4. Distorted Liver Architecture: The accumulation of scar tissue disrupts the normal liver structure. This distortion impairs blood flow through the liver and affects its ability to perform essential functions.

5. Functional Impairment: As fibrosis progresses to cirrhosis, the liver's ability to perform its functions—such as detoxifying harmful substances, synthesizing proteins,

and producing bile—diminishes.

6. Complications: Advanced cirrhosis can lead to a variety of complications due to its impact on liver function and blood flow, including portal hypertension (increased blood pressure in the portal vein), ascites (fluid accumulation in the abdomen), hepatic encephalopathy (brain dysfunction), and increased risk of liver cancer.

7. Irreversibility: While some aspects of liver damage can be managed or improved, cirrhosis is generally considered irreversible. The focus is on halting progression and managing symptoms and complications.

8. Treatment Goals: Management aims to address the underlying cause (if possible), prevent further liver damage, manage complications, and improve quality of life. In advanced cases, liver transplantation may be considered.

## 4. ETIOLOGY

1. Malnutrition
2. Alcoholism
3. Viral hepatitis
4. Heart failure that causes hepatic vein dam
5. Wilson's disease (copper accumulation of excessive default)
6. Hemochromatosis (iron overload)
7. Toxic substances

There are 3 types of cirrhosis or scar formation in the heart:

1. Laennec cirrhosis (alcoholism, nutritional), where scarring typically surrounds the portal area. Often caused by chronic alcoholics.

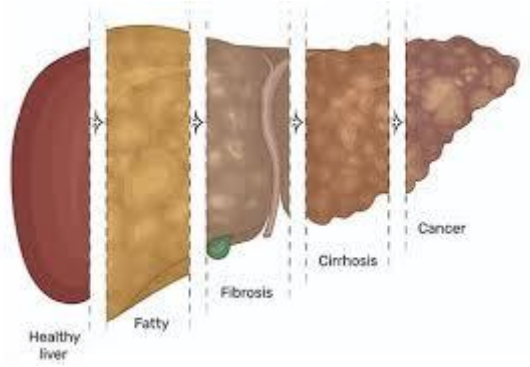
2. Cirrhosis pascanekrotik, where there is a band of scar tissue Further widening as a result of acute viral hepatitis that occurs previous.

3. Biliary cirrhosis, which occurs in the formation of scar tissue around the liver bile ducts.Which occurs due to biliary obstruction Chronic infections (cholangit).

## 5. PATHOPHYSIOLOGY

Although there are several factors involved in the etiology of cirrhosis, consumption of alcoholic beverages is considered as a causal factor main. Cirrhosis occurs with the highest frequency in the drink liquor. Despite the decrease in the intake of nutritional deficiency protein contributed to the liver damage in cirrhosis, but the intake Excessive alcohol is a major contributory factors fatty liver and the consequences thereof. however Thus, cirrhosis also have occurred in individuals who do not have drinking liquor and in individuals who diet normal but with high alcohol consumption (Smeltzer & Bare, 2001).

Most people seem to be more prone to this disease compared to other individuals without determined whether the individual is have the habit of drinking liquor or suffer malnutrition. Other factors may play a role, including Exposure to certain chemicals




(carbon tetrachloride, naphthalene chlorinated, asen or phosphorus) or schistosomiasis infections that contagious. The number of male patients with cirrhosis is twice more than women, and the majority of cirrhotic patients aged 40-60 year (Smeltzer and Bare, 2001).

Alcoholic cirrhosis or historically called cirrhosis Laennec characterized by diffuse scarring, loss of cell-liver cells were uniform, and a bit of regenerative nodules. so Now and sometimes called micronodular cirrhosis. Micronodular cirrhosis can also injuries caused by other liver. Three major lesions induced alcohol is an alcoholic fatty liver, alcoholic hepatitis, and alcoholic cirrhosis (Tarin, 2001)

## 5. CLINICAL MANIFESTATION

1. Enlarged Heart

At the beginning of the trip to liver cirrhosis, liver tend to enlarge and the cells filled with fat. The liver becomes hard and have sharp edges that can be detected through palpation. Abdominal pain can occur as a result of the enlargement of the liver fast and just happened resulting strain the liver fibrous sheath (kapsula Glissoni). on a trip more advanced disease, liver size will be reduced after scar tissue causes contraction of the heart tissue. If can be palpated, the surface of the heart will be felt bumpy (Nodular).

2. Obstruction Portal and Ascites

Manifestations in part caused by a failure Chronic liver function and partly by the obstruction of circulation portal. All blood from the digestive organs practically gathered in the portal vein and carried to the liver. Because heart cirrhotic not allow passage of free blood, then The blood flow will return to the spleen and tracts Gastrointestinal with the consequence that these organs a place of chronic passive congestion; in other words, the second The organ will be filled with blood and thus do not can work well. Patients with these circumstances tend to suffer from chronic dyspepsia and constipation or diarrhea. The patient's weight is gradually decreased. Protein-rich fluid and accumulate dirongga Peritoneal will cause ascites. This is demonstrated through perfusion of the existence of shifting dullness or a fluid wave. Splenomegaly also occur. Nets telangiectasia, or dilatation Superficial arteries causing reddish blue nets, which can often be seen through the inspection of the face and whole body.

3. varicose Gastrointestinal

Obstruction of blood flow through the liver caused by fibrotic changes also resulted in the formation of vessel collateral blood in the gastrointestinal system and pemintasan ( shunting) of blood vessels into the portal vein with a lower pressure. As a result, sufferers cirrhosis often exhibit abdominal distension of blood vessels A striking and visible on inspection of the abdomen (caput medusae), and distention of blood vessels throughout the tract gastrointestinal. Esophagus, stomach and lower rectum an area that is often experienced vessels formation collateral blood. Distension of the blood vessels will form varicose veins or hemorrhoids depending on the location.

Because its function is not to bear the blood volume and a high pressure due to cirrhosis, the blood vessels of this can rupture and cause bleeding. Therefore, assessment should include observation to determine bleeding real and hidden from tract gastrointestinal. Approximately 25% of patients hematemesis light; the rest will undergo massive hemorrhage from variceal rupture of the stomach and esophagus.

4. Edema

More advanced symptoms in liver cirrhosis caused by chronic liver failure. Plasma albumin concentration decline so that predisposes to edema. Production excessive aldosterone will cause sodium retention and water and potassium excretion.

5. Vitamin Deficiency and Anemia

Since the formation, use and storage Inadequate certain vitamins (especially vitamin A, C and K), then the signs of vitamin deficiency are often found, especially as hemorrhagic phenomena associated with the Chronic Gastritis vitamin K deficiency and disfunction Gastrointestinal together inadequate dietary intake and impaired liver function contributed to the anemia that often accompanies cirrhosis of the liver. Symptoms of anemia and nutritional status as well as the patient's poor health will lead to severe fatigue which impairs the ability to perform routine activities daily.

6. Mental setback

Other clinical manifestations are setback function mental with hepatic

encephalopathy and coma membakat. Therefore, a neurological examination needs to be done in cirrhosis hepatic and covers the general behavior of the patient, the ability cognitive, orientation to time and place, and speech patterns.

## 7. DIAGNOSTIC EVALUATION

Diagnosing liver cirrhosis involves a thorough evaluation that includes clinical assessment, laboratory tests, imaging studies, and sometimes biopsy. Here’s a detailed overview of the diagnostic approach:

1. Clinical Assessment

- Medical History: Includes symptoms (e.g., fatigue, jaundice, abdominal pain, ascites, and easy bruising), history of liver disease risk factors (e.g., alcohol use, viral hepatitis, medications), and family history of liver disease.
- Physical Examination: Focuses on signs of cirrhosis such as:
- Jaundice: Yellowing of the skin and eyes.
- Ascites: Fluid accumulation in the abdominal cavity.
- Edema: Swelling of the legs.
- Spider Angiomas: Small, spider-like blood vessels visible on the skin.
- Hepatomegaly or Splenomegaly: Enlarged liver or spleen.
- Palmar Erythema: Redness of the palms.

2. Laboratory Tests

- Liver Function Tests (LFTs): Assess liver enzyme levels and other markers to evaluate liver function:
- Alanine Aminotransferase (ALT) and Aspartate Aminotransferase (AST): Elevated levels can indicate liver injury.
- Alkaline Phosphatase (ALP) and Gamma-Glutamyl Transferase (GGT): Elevated levels may suggest cholestasis or bile duct obstruction.
- Bilirubin: Elevated levels can indicate jaundice and impaired liver function.
- Albumin: Low levels may suggest impaired liver function.
- Prothrombin Time (PT) and International Normalized Ratio (INR): Prolonged PT/INR can

indicate liver dysfunction and impaired synthesis of clotting factors.
- Hepatitis Panel: To determine if viral hepatitis (e.g., hepatitis B or C) is contributing to liver damage.
- Autoimmune Markers: Such as anti-nuclear antibodies (ANA) or anti-smooth muscle antibodies (ASMA) if autoimmune hepatitis is suspected.
- Iron Studies: To evaluate for conditions like hemochromatosis that can lead to cirrhosis.

3. Imaging Studies

- Abdominal Ultrasound: The primary imaging tool for diagnosing cirrhosis. It can identify:

- Liver Size and Texture: Detects hepatomegaly, nodular liver texture, and signs of portal hypertension.
- Ascites: Presence of fluid in the abdominal cavity.
- Splenomegaly: Enlarged spleen, which is a common sign of portal hypertension.
- Liver Masses: Detects potential liver tumors or nodules.
- CT Scan: Provides detailed cross-sectional images of the liver and can help assess liver size, texture, and complications like portal hypertension or liver tumors.
- MRI: Offers detailed images and can be useful for evaluating liver lesions or assessing liver iron content.

4. Liver Biopsy
- Percutaneous Liver Biopsy: Involves obtaining a small sample of liver tissue through a needle inserted into the liver. This is used to assess the degree of fibrosis and confirm the diagnosis of cirrhosis. It also helps to identify the underlying cause of cirrhosis.
- Transjugular Liver Biopsy: Performed through the jugular vein, this method is used in patients who are at higher risk for bleeding or have ascites.

5. Endoscopy
- Upper Gastrointestinal Endoscopy: Used to detect esophageal varices and gastric varices that are a complication of portal hypertension in cirrhosis. It helps assess the severity of portal hypertension and guide treatment decisions.

6. Elastography
- Transient Elastography (FibroScan): A non-invasive test that measures liver stiffness to estimate the degree of fibrosis. It is increasingly used to assess liver stiffness and monitor disease progression.

7. Other Specialized Tests
- Liver Fibrosis Scores: Such as the Child-Pugh score or MELD score, which help assess liver function and severity of cirrhosis. These scores are based on clinical and laboratory parameters.
- Genetic Testing: If hereditary conditions like hemochromatosis or Wilson’s disease are suspected.

Summary

Diagnosing liver cirrhosis involves a combination of clinical evaluation, laboratory tests, imaging studies, and sometimes biopsy to confirm the diagnosis, assess the severity, and identify the underlying cause. This comprehensive approach is essential for managing cirrhosis effectively and preventing or treating complications.

## 8. NURSING MANAGEMENT

Management according to Tarin (2001) are:

1. The patient is in good heart compensation is done Regular control, adequate rest, diet composition high high calorie protein, fat taste.
2. Cirrhotic patients with known causes such as:

a. Alcohol and drugs recommended stop use. Alcohol reduces protein intake into the body. With a high-calorie diet (300 calories), protein content of about 70- 90 grams of food a day for hinder the development of kolagenik can be tried with Award D penicilamine and Cochicine.

b. Hemokromatis Discontinued use of preparations containing iron / therapy flatfoot (desferioxamine). Sexy veins do 2x a week as much as 500cc for a year.

c. In chronic autoimmune hepatitis corticosteroids.

3. Treatment of complications arising

a. ascites Bed rest and begin a low-salt diet, salt intake 5.2 g/day. Low-salt diet combined with diuretic drugs. Initially the administration spironolactone at a dose of 100-200 mg once daily. responses diuretics can be monitored with a weight loss of 0.5 kg/day, without edema feet or 1 kg/day with their leg edema. When giving spironolactone inadequate could be combined with furosemide at a dose of 20-40 m /day. Provision of furosemide can be increased dosage if not no response, the maximum dose is 160 mg/day. paracentesis done when ascites is very large. Spending could ascites up to 4-6 liters and covered with albumin administration.

b. Esophageal variceal bleeding (haematemesis, hematemesis with melena or melena only)

1) Perform aspiration of gastric fluid containing blood to determine whether the

bleeding
has stopped or is still underway.
2) When bleeding a lot, the systolic pressure below 100 mmHg, pulse above 100 x/min
or Hb below 99% administration of IVFD by administering dextrose/copy and blood
transfusions to taste. 3) Given vasopressin 2 amps 0.1 g in 500cc D5% or
administration of normal saline for 4 hours can be repeated three times. 15
c. Encephalopathy
1) Correction of precipitating factors such as the provision of KCL in hypokalaemia.
2) Reducing protein intake of food to give appropriate diet.
3) Aspiration of gastric fluid for patients who have bleeding on varicose veins.
4) Antibiotics campisilin / cephalosporin in state systemic infection.
5) Liver transplantation.
d. Spontaneous bacterial peritonitis
Given the antibiotic of choice such as cefotaxime, amoxicillin, aminoglycosides.
e. Hepatorenal syndrome / nefropatik hepatic Regulate the balance of fluids and salts.

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# EMERGENCY NURSING

## 1. DEFINITION

Emergency nursing is a specialized field of nursing focused on providing immediate and comprehensive care to patients experiencing acute or life-threatening medical conditions.

## 2. OBJECTIVE

The primary objectives of emergency nursing are to ensure the immediate and effective care of patients in urgent and often high-stress situations. These objectives include:

1. Rapid Assessment and Triage: To quickly and accurately assess patients' conditions, prioritize care based on the severity of their symptoms, and triage patients efficiently to manage resources and ensure the most critical cases receive prompt attention.

2. Immediate Stabilization and Treatment: To provide urgent medical interventions and stabilize patients experiencing acute or life-threatening conditions, including administering medications, performing emergency procedures, and utilizing advanced life-support techniques.

3. Pain and Symptom Management: To alleviate pain and manage symptoms effectively, ensuring patient comfort and addressing any distress or discomfort during treatment.

4. Effective Communication: To communicate clearly and effectively with patients, their families, and other healthcare professionals, providing information about diagnoses, treatment plans, and expected outcomes.

5. Patient and Family Support: To offer emotional support and reassurance to patients and their families during crises, helping them navigate the healthcare system and cope with the stress of emergency situations.

6. Prevention of Complications: To minimize the risk of complications through careful monitoring, timely interventions, and the implementation of preventive measures to avoid further health deterioration.

7. Collaboration and Coordination: To work closely with other members of the healthcare team, including physicians, paramedics, and specialists, to ensure a coordinated and comprehensive approach to patient care.

8. Documentation and Continuity of Care: To accurately document all aspects of patient care and interventions, ensuring continuity of care and providing essential information for follow-up or referrals.

9. Education and Training: To stay current with emergency care protocols, advancements in medical practices, and ongoing professional development to deliver the highest standard of care.

10. Preparedness and Adaptability: To maintain readiness for a variety of emergency scenarios, adapt to evolving situations, and manage resources effectively to meet the needs of patients in a dynamic environment.

By achieving these objectives, emergency nurses play a crucial role in providing high-quality care, improving patient outcomes, and ensuring a positive impact on the overall emergency healthcare system.

## 3. PRINCIPLE

The principles of emergency nursing provide a foundation for delivering effective, efficient, and compassionate care in high-pressure situations. These principles guide emergency nurses in their practice and ensure that patient care is both timely and comprehensive. Key principles include:

1. Prioritization and Triage: Prioritizing patients based on the severity of their conditions is crucial. Triage systems are used to classify patients according to their immediate needs, ensuring that those with life-threatening conditions receive prompt attention while less urgent cases are managed appropriately.

2. Rapid Assessment: Conducting quick and accurate assessments of patients' conditions is essential. This involves identifying life-threatening issues, understanding the patient's medical history, and determining the most appropriate interventions.

3. Evidence-Based Practice: Utilizing the latest research and clinical guidelines to inform decision-making and interventions ensures that care is based on the most current and effective practices.

4. Holistic Care: Addressing not only the physical needs but also the emotional, psychological, and social aspects of patient care. This includes providing support and reassurance to patients and their families during crises.

5. Collaboration and Communication: Working effectively with other healthcare professionals, including physicians, paramedics, and support staff, to coordinate care and ensure comprehensive management of the patient's needs. Clear and accurate communication is vital for successful teamwork.

6. Patient Safety: Prioritizing patient safety by following established protocols, reducing the risk of errors, and implementing measures to protect patients from harm. This includes careful monitoring and prevention of complications.

7. Adaptability and Flexibility: Being able to adapt to rapidly changing situations and unexpected challenges is essential in emergency settings. Emergency nurses must be flexible and ready to handle a wide range of scenarios.

8. Ethical and Compassionate Care: Providing care that is both ethical and compassionate, respecting patients' rights, dignity, and preferences, and delivering care with empathy and respect, even in the most stressful situations.

9. Continuous Learning and Improvement: Committing to ongoing education and professional development to stay current with advancements in emergency care and continually improving practice through reflection and feedback.

10. Resource Management: Efficiently managing resources, including time, personnel, and equipment, to maximize the effectiveness of care and ensure that all patients receive the attention and treatment they need.

By adhering to these principles, emergency nurses can provide high-quality, patient-centered care and effectively manage the complexities and demands of emergency medical situations.

## 4. ETIOLOGY

In the context of emergency nursing, "etiology" refers to the underlying causes or origins of the acute medical conditions and situations that emergency nurses encounter. While the term "etiology" is more commonly associated with diagnosing the causes of diseases, in emergency nursing, it encompasses understanding and addressing the root causes of various emergency scenarios. Here’s how etiology applies to emergency nursing:

1. Traumatic Injuries: Understanding the cause of trauma is crucial for appropriate care. For instance, knowing whether an injury resulted from a fall, vehicle accident, or assault helps in guiding diagnostic tests, treatment plans, and follow-up care.

2. Acute Medical Conditions: Identifying the underlying cause of conditions such as chest pain, abdominal pain, or sudden loss of consciousness helps in providing targeted treatment. For example, distinguishing between a heart attack and a pulmonary embolism as the cause of chest pain influences treatment decisions.

3. Infections: Recognizing the source of an infection, such as a bacterial, viral, or fungal etiology, helps in choosing the right antibiotics or antiviral medications and implementing appropriate infection control measures.

4. Toxicological Emergencies: Identifying the substance involved in poisoning or overdose—whether it is a drug, chemical, or toxin—guides the administration of antidotes or supportive treatments.

5. Environmental and Lifestyle Factors: Understanding environmental factors like extreme temperatures or lifestyle choices (e.g., alcohol use) that contribute to emergencies allows for targeted interventions and preventive advice.

6. Chronic Disease Exacerbations: For patients with chronic conditions like diabetes or heart disease, identifying triggers or exacerbating factors helps in managing acute episodes and preventing future emergencies.

7. Psychiatric and Behavioral Issues: Determining the etiology of psychiatric crises, such as substance abuse or acute mental health disorders, helps in providing appropriate psychiatric and medical care.

8. Genetic and Congenital Conditions: Recognizing congenital or genetic conditions that lead to acute emergencies, such as metabolic crises or congenital heart defects, enables early intervention and specialized care.

Understanding the etiology of emergencies helps emergency nurses in several ways:

- Accurate Diagnosis: Helps in correctly diagnosing the patient’s condition, which is crucial for effective treatment.

- Targeted Treatment: Guides the selection of appropriate interventions, medications, and therapies based on the underlying cause.

- Prevention and Education: Aids in educating patients and families about risk factors and preventive measures, contributing to better long-term health management.

- Resource Allocation: Ensures that resources, including specialized treatments or consultations, are used appropriately based on the nature of the emergency.

Overall, a thorough understanding of the etiology of emergency situations allows emergency nurses to deliver precise, effective, and timely care, ultimately improving patient outcomes and enhancing the efficiency of emergency care.

## 5. PATHOPHYSIOLOGY

The pathophysiology of emergency nursing involves understanding the underlying mechanisms and processes of various acute medical conditions and emergencies that patients present with. This knowledge is crucial for emergency nurses to provide effective and timely care. Here's an overview of how pathophysiology applies to different types of emergencies:

1. Traumatic Injuries
   - Pathophysiology: Trauma can cause immediate tissue damage through physical forces. This may result in hemorrhage, inflammation, and subsequent complications like shock. For example, a fractured bone can cause bleeding, swelling, and pain, which can lead to compromised circulation and potential infection.
   - Emergency Care: Stabilization of the injury, pain management, and prevention of complications such as infection and shock.

2. Cardiovascular Emergencies
   - Pathophysiology: Conditions like myocardial infarction (heart attack) are caused by the obstruction of coronary arteries, leading to ischemia (lack of blood flow) and necrosis (tissue death) of the heart muscle. Arrhythmias or heart failure can disrupt normal heart function, affecting systemic circulation.
   - Emergency Care: Administering medications like thrombolytics, providing oxygen, and performing interventions like angioplasty or defibrillation to restore normal heart function.

3. Respiratory Emergencies

- Pathophysiology: Issues such as acute respiratory distress syndrome (ARDS) or chronic obstructive pulmonary disease (COPD) exacerbations involve inflammation and fluid accumulation in the lungs or airway obstruction, leading to impaired gas exchange.
- Emergency Care: Providing oxygen therapy, mechanical ventilation, or medications to manage airway inflammation and improve oxygenation.

4. Neurological Emergencies
- Pathophysiology: Conditions like stroke or seizures involve disruption in normal brain function due to factors such as hemorrhage, ischemia, or electrical disturbances. For instance, a stroke results from the interruption of blood flow to a part of the brain, causing cell death and neurological deficits.
- Emergency Care: Administering thrombolytics for ischemic stroke, controlling seizures with antiepileptic drugs, and providing supportive care to manage neurological symptoms.

5. Gastrointestinal Emergencies
- Pathophysiology: Acute conditions such as appendicitis or gastrointestinal bleeding involve inflammation, infection, or erosion of gastrointestinal tissues. For example, appendicitis results from the inflammation of the appendix, leading to abdominal pain and potential perforation.
- Emergency Care: Providing pain relief, managing fluid and electrolyte balance, and preparing for surgical intervention if necessary.

6. Endocrine Emergencies
- Pathophysiology: Conditions like diabetic ketoacidosis (DKA) result from severe insulin deficiency, leading to hyperglycemia and ketone production. This disrupts normal metabolic processes and can cause dehydration and electrolyte imbalances.
- Emergency Care: Administering insulin, fluids, and electrolytes to correct metabolic imbalances and stabilize the patient.

7. Infectious Emergencies
- Pathophysiology: Severe infections like sepsis result from the body's systemic inflammatory response to an infection, leading to widespread inflammation, organ dysfunction, and potential septic shock.
- Emergency Care: Administering broad-spectrum antibiotics, providing supportive care, and monitoring for signs of organ failure.

8. Toxicological Emergencies

- Pathophysiology: Poisoning or overdose affects normal biochemical processes, depending on the toxin. For example, opioid overdose suppresses the central nervous system, leading to respiratory depression and altered consciousness.
- Emergency Care: Providing antidotes (e.g., naloxone for opioid overdose), supportive care, and decontamination if applicable.

9. Psychiatric Emergencies

- Pathophysiology: Acute mental health crises, such as severe anxiety or psychosis, involve disruptions in normal brain function and neurotransmitter balance, affecting mood, perception, and behavior.
- Emergency Care: Providing psychiatric stabilization, medications, and safety measures to address immediate mental health needs.

10. Obstetric Emergencies

- Pathophysiology: Conditions like preeclampsia involve abnormal blood pressure and organ dysfunction during pregnancy. This can affect maternal and fetal well-being.
- Emergency Care: Monitoring and managing blood pressure, providing medications to prevent seizures, and preparing for possible delivery if necessary.

Understanding the pathophysiology of these conditions helps emergency nurses:

- Diagnose and Identify: Quickly recognize the underlying causes of symptoms and conditions.
- Implement Targeted Interventions: Choose appropriate treatments and interventions based on the pathophysiological processes involved.
- Monitor and Evaluate: Assess the effectiveness of treatments and make adjustments as needed based on the patient's response.

By integrating knowledge of pathophysiology with clinical skills, emergency nurses can provide effective, patient-centered care in critical situations.

## 6. CLINICAL MANIFESTATION

In emergency nursing, "clinical manifestations" refer to the signs and symptoms that patients present with during an acute medical situation. These manifestations help emergency nurses identify, assess, and prioritize care. The clinical manifestations can vary widely depending on the type of emergency. Here's an overview of clinical manifestations across different categories of emergencies:

1. Traumatic Injuries
   - Signs: Bruising, swelling, deformity, open wounds, or bleeding.
   - Symptoms: Pain, tenderness, immobility, or inability to use the affected limb.
   - Examples: A fractured bone might show visible deformity and severe pain, while a head injury could present with confusion or loss of consciousness.

2. Cardiovascular Emergencies
   - Signs: Cyanosis (bluish skin), elevated blood pressure, jugular vein distention, abnormal heart rhythms, or signs of heart failure (e.g., pulmonary edema).
   - Symptoms: Chest pain, shortness of breath, palpitations, dizziness, or sweating.
   - Examples: Myocardial infarction (heart attack) often presents with chest pain, radiating pain to the arm or jaw, nausea, and sweating.

3. Respiratory Emergencies
   - Signs: Abnormal breath sounds (e.g., wheezing, crackles), use of accessory muscles for breathing, cyanosis, or altered respiratory rate.
   - Symptoms: Shortness of breath, chest tightness, cough, or fatigue.
   - Examples: Asthma exacerbation might present with wheezing, difficulty breathing, and a cough, while acute respiratory distress syndrome (ARDS) can show severe hypoxia and rapid breathing.

4. Neurological Emergencies
   - Signs: Altered level of consciousness, unilateral weakness, facial droop, slurred speech, or seizures.
   - Symptoms: Severe headache, sudden confusion, visual disturbances, or loss of coordination.
   - Examples: Stroke may present with sudden onset of weakness on one side of the body, difficulty speaking, and severe headache. Seizures can manifest as convulsions, loss of consciousness, and postictal confusion.

5. Gastrointestinal Emergencies

- Signs: Abdominal tenderness, distension, abnormal bowel sounds, or signs of bleeding (e.g., hematemesis or melena).
- Symptoms: Severe abdominal pain, nausea, vomiting, or changes in bowel habits.
- Examples: Appendicitis typically presents with right lower quadrant pain, nausea, vomiting, and fever. Gastrointestinal bleeding might manifest as hematemesis (vomiting blood) or melena (black, tarry stools).

6. Endocrine Emergencies

- Signs: Dehydration, altered mental status, abnormal blood glucose levels, or signs of fluid and electrolyte imbalances.
- Symptoms: Confusion, excessive thirst, frequent urination, or fatigue.
- Examples: Diabetic ketoacidosis (DKA) can present with fruity-smelling breath, rapid breathing, and altered mental status, while hyperglycemic hyperosmolar syndrome (HHS) may show severe dehydration and confusion.

7. Infectious Emergencies

- Signs: Fever, rash, elevated white blood cell count, or signs of systemic infection (e.g., hypotension in sepsis).
- Symptoms: Chills, malaise, confusion, or localized pain depending on the infection site.
- Examples: Sepsis may present with fever, rapid heart rate, hypotension, and confusion. Meningitis could show symptoms like severe headache, neck stiffness, and sensitivity to light.

8. Toxicological Emergencies

- Signs: Altered mental status, abnormal vital signs, or signs of specific poisoning (e.g., pinpoint pupils in opioid overdose).
- Symptoms: Nausea, vomiting, dizziness, or drowsiness.
- Examples: Opioid overdose might present with respiratory depression, pinpoint pupils, and altered consciousness, while carbon monoxide poisoning could manifest with headache, dizziness, and confusion.

9. Psychiatric Emergencies

- Signs: Agitation, disorganized behavior, or self-harm.

- Symptoms: Severe anxiety, paranoia, hallucinations, or suicidal ideation.
- Examples: Acute psychosis may show disorganized speech, hallucinations, and paranoia, while a severe panic attack might present with chest pain, palpitations, and shortness of breath.

10. Obstetric Emergencies
- Signs: Abnormal fetal heart rate patterns, vaginal bleeding, or signs of preeclampsia (e.g., high blood pressure and swelling).
- Symptoms: Severe abdominal pain, contractions, or headaches.
- Examples: Preeclampsia can present with high blood pressure, edema, and proteinuria, while abruptio placentae might show vaginal bleeding, abdominal pain, and fetal distress.

In emergency nursing, recognizing these clinical manifestations allows for:

- Prompt Assessment: Quickly identifying the nature and severity of the emergency.
- Effective Intervention: Implementing appropriate treatments and interventions based on the symptoms and signs.
- Patient Monitoring: Ongoing assessment to track changes and responses to treatment.

Overall, clinical manifestations guide emergency nurses in delivering targeted, effective care, improving patient outcomes in urgent situations.

## 7. DIAGNOSTIC EVAULTION

Diagnostic evaluation in emergency nursing involves a systematic approach to identify and assess the underlying causes of a patient's acute condition. This evaluation is crucial for determining the appropriate treatment and ensuring effective patient care. Here’s a structured guide to diagnostic evaluation in emergency settings:

1. Initial Assessment

- Patient History: Gather relevant information quickly, including:
  - Chief Complaint: The primary reason for the visit or symptoms.

- Medical History: Past illnesses, surgeries, chronic conditions, and current medications.
- Allergies: Any known allergies, especially to medications or contrast agents.
- Recent Events: Details of the incident or symptoms onset (e.g., trauma, onset of pain).

- Physical Examination: Perform a thorough but rapid examination to assess:
- General Appearance: Look for signs of distress, cyanosis, pallor, or other immediate concerns.
- Vital Signs: Measure temperature, pulse, blood pressure, respiratory rate, and oxygen saturation.
- Focused Examination: Based on the presenting complaint, perform a focused examination of relevant systems (e.g., cardiovascular, respiratory, neurological).

2. Diagnostic Tests

- Laboratory Tests:
- Complete Blood Count (CBC): To assess for anemia, infection, or other hematological issues.
- Electrolytes and Renal Function: Serum electrolytes (e.g., sodium, potassium), blood urea nitrogen (BUN), and creatinine to evaluate kidney function and metabolic imbalances.
- Blood Glucose Levels: To check for hyperglycemia or hypoglycemia.
- Coagulation Profile: For patients with bleeding disorders or those on anticoagulants.

- Imaging Studies:
- X-rays: Useful for assessing fractures, pneumothorax, and other structural abnormalities.
- Ultrasound: Can be used for assessing internal bleeding, abdominal organs, and guiding certain procedures.
- CT Scan: Provides detailed images of internal structures, helpful in diagnosing trauma, hemorrhage, and certain infections.
- MRI: Less commonly used in the emergency setting but useful for detailed imaging of soft tissues and neurological issues.

- Electrocardiogram (ECG):
- Cardiac Assessment: Essential for evaluating arrhythmias, ischemia, and myocardial infarction.

3. Specialized Tests

- Toxicology Screen: If poisoning or overdose is suspected, tests can identify substances in the blood or urine.
- Culture and Sensitivity: If an infection is suspected, cultures (e.g., blood, urine, wound) are obtained to identify pathogens and guide antibiotic therapy.

4. Functional Assessments

- Neurological Assessment: Using tools like the Glasgow Coma Scale (GCS) to evaluate consciousness and neurological function.
- Cardiac Monitoring: Continuous ECG monitoring may be needed for patients with suspected cardiac issues.

5. Dynamic Monitoring

- Trend Analysis: Regularly monitor and review changes in vital signs, lab results, and clinical status to assess the response to treatment and adjust care plans as needed.

6. Clinical Decision-Making

- Synthesis of Findings: Combine results from history, physical examination, and diagnostic tests to form a comprehensive understanding of the patient's condition.
- Diagnostic Formulation: Develop a differential diagnosis list and prioritize investigations or treatments based on the most likely causes and severity of symptoms.

7. Documentation and Communication

- Accurate Records: Document all findings, test results, and clinical decisions thoroughly and promptly.
- Team Communication: Communicate findings and plans with the healthcare team to ensure coordinated and effective care.

8. Patient and Family Education

- Explanation of Findings: Explain diagnostic findings and next steps to the patient and their family in an understandable manner.
- Instructions: Provide clear instructions regarding follow-up care, medications, or any changes in lifestyle or activity.

Effective diagnostic evaluation in emergency nursing involves a combination of rapid assessment, targeted diagnostic testing, and continuous monitoring. This approach helps to identify critical conditions promptly, guide appropriate treatment, and ensure the best possible outcomes for patients in emergency situations.

## 8. NURSING MANAGEMENT

Nursing management in emergency settings requires a rapid, efficient, and systematic approach to ensure optimal patient outcomes. Here's a comprehensive guide to nursing management in emergency nursing:

1. Initial Assessment and Triage

- Primary Assessment (ABCDE):
  - Airway: Ensure the airway is open and unobstructed. Use maneuvers or devices as needed.
  - Breathing: Assess for adequate breathing. Check for respiratory rate, depth, and oxygen saturation. Administer oxygen if necessary.
  - Circulation: Evaluate pulse, blood pressure, and capillary refill. Manage any signs of shock or severe bleeding.
  - Disability: Perform a quick neurological assessment (e.g., Glasgow Coma Scale) to determine consciousness level and any potential neurological deficits.
  - Exposure: Fully expose the patient to identify all injuries or signs of illness while maintaining their dignity and warmth.

- Triage: Prioritize patients based on the severity of their condition using a standardized system (e.g., the Emergency Severity Index, or ESI). This helps in managing multiple patients efficiently.

2. Immediate Care and Interventions

- Stabilization: Administer necessary treatments to stabilize vital signs and manage life-threatening conditions. This may include:
  - Medications: Administer emergency medications as per protocols (e.g., epinephrine for anaphylaxis, naloxone for opioid overdose).
  - Intravenous Access: Establish IV access for fluid resuscitation, medication administration, or blood products.
  - Wound Care: Apply pressure to control bleeding, clean wounds, and dress injuries appropriately.

- Monitoring: Continuously monitor vital signs, oxygenation, and other relevant parameters. Use ECGs, blood glucose checks, and other diagnostic tools as needed.

3. Patient and Family Communication

- Communication: Provide clear and concise information to the patient and their family about the situation, procedures, and expected outcomes. Offer reassurance and emotional support.
- Consent: Obtain informed consent for emergency procedures or treatments, ensuring that the patient or their legal representative understands the risks and benefits.

4. Coordination and Documentation

- Team Coordination: Collaborate with other healthcare professionals (e.g., physicians, paramedics, social workers) to ensure cohesive care and address all aspects of the patient's needs.
- Documentation: Accurately document all assessments, interventions, and patient responses. Ensure that all records are completed in a timely manner and in accordance with legal and institutional requirements.

5. Post-Stabilization Care

- Reassessment: Continuously reassess the patient's condition and response to treatments. Be prepared to adjust care plans as needed.

- Preparation for Transfer: If the patient needs to be transferred to another facility or unit, ensure that all necessary information and documentation are prepared and communicated to receiving healthcare providers.

6. Psychosocial Support

- Emotional Support: Address the psychological needs of patients and families, offering support, empathy, and resources as necessary.
- Counseling: Provide or arrange for counseling services for those dealing with trauma or significant stress related to the emergency situation.

7. Infection Control

- Precautions: Follow strict infection control practices, including hand hygiene, use of personal protective equipment (PPE), and safe disposal of sharps and contaminated materials.

8. Education and Training

- Continuous Learning: Stay updated on the latest emergency care protocols, treatments, and technologies. Participate in regular training and simulation exercises to enhance skills.

Emergency nursing management requires a combination of rapid decision-making, technical skills, and compassionate care. Effective management ensures that patients receive timely and appropriate care, improving their chances of recovery and minimizing complications.

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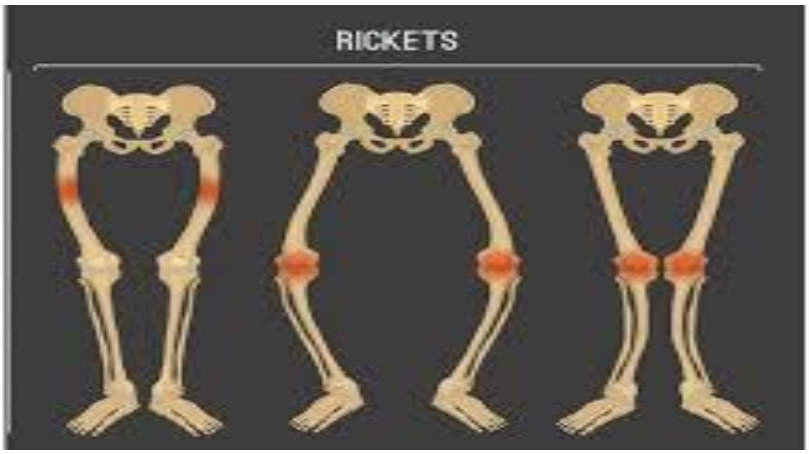
# RICKETS

## 1. DEFINITION

Rickets is a bone disorder that affects children, primarily due to a deficiency in vitamin D, calcium, or phosphate.




## 2. OBJECTIVE

The term "objective" in relation to rickets might be a bit unclear, as it usually refers to goals or purposes rather than a condition itself. However, if you're asking about the objective in managing or understanding rickets, here are some key goals:

1. Prevention: Ensure adequate intake of vitamin D, calcium, and phosphate through diet and supplementation to prevent the onset of rickets.

2. Diagnosis: Accurately identify rickets through clinical assessment, blood tests, and imaging to confirm the condition and determine its severity.

3. Treatment: Correct deficiencies in vitamin D, calcium, and phosphate through supplements and dietary adjustments to restore proper bone health and prevent complications.

4. Monitoring: Regularly monitor the child's bone health and growth to assess the effectiveness of treatment and make necessary adjustments.

5. Education: Inform caregivers and healthcare providers about the importance of nutrition, sunlight exposure, and early intervention to manage and prevent rickets effectively.

## 3. PRINCIPLE

The "principle" of rickets generally refers to the underlying cause and mechanism of the condition. Rickets is fundamentally a disorder of bone mineralization that occurs due to deficiencies in key nutrients, specifically vitamin D, calcium, or phosphate. Here are the main principles:

1. Bone Mineralization: Healthy bone development relies on adequate mineralization. Vitamin D, calcium, and phosphate are crucial for this process. Vitamin D facilitates the absorption of calcium and phosphate from the gut into the bloodstream.

2. Nutrient Deficiency: Rickets primarily arises when there is a deficiency of vitamin D, calcium, or phosphate. Without sufficient vitamin D, the body cannot effectively absorb calcium and phosphate, leading to weakened bones and impaired bone growth.

3. Impaired Calcium and Phosphate Balance: Inadequate vitamin D results in lower levels of calcium and phosphate in the blood, which disrupts the normal process of bone mineralization. This leads to the softening and weakening of bones.

4. Bone Deformation: The softening of bones due to poor mineralization results in deformities and other skeletal abnormalities. Common symptoms include bowed legs, a curved spine, and a protruding breastbone.

5. Diagnosis and Treatment: The principle of managing rickets involves addressing the underlying deficiencies. This is done through dietary changes, supplements, and sometimes medications to restore normal vitamin D, calcium, and phosphate levels, thereby promoting proper bone development and reducing symptoms.

In essence, the principle of rickets revolves around the critical role of vitamin D and minerals in maintaining bone health and the consequences that arise when these nutrients are deficient.

## 4. ETIOLOGY

The etiology of rickets primarily involves deficiencies or imbalances in key nutrients essential for bone health, particularly vitamin D, calcium, and phosphate. Understanding the causes of these deficiencies helps in diagnosing and treating rickets effectively. Here's a detailed look at the etiology of rickets:

1. Vitamin D Deficiency

- Inadequate Sunlight Exposure: Vitamin D is synthesized in the skin through exposure to ultraviolet (UV) rays from sunlight. Limited sunlight exposure, especially in regions with long winters or for individuals who stay indoors frequently, can lead to vitamin D deficiency.
- Dietary Deficiency: Inadequate intake of vitamin D-rich foods (such as fatty fish, fortified dairy products, and egg yolks) can contribute to deficiency, particularly in populations with limited access to these foods.
- Malabsorption Disorders: Conditions affecting the absorption of vitamin D in the gastrointestinal tract, such as celiac disease, Crohn's disease, or cystic fibrosis, can lead to deficiency.
- Dark Skin Pigmentation: Melanin in the skin reduces the skin's ability to produce vitamin D from sunlight. Individuals with darker skin may be at higher risk of deficiency, especially in areas with low UV exposure.

2. Calcium Deficiency

- Inadequate Dietary Intake: Insufficient calcium intake from dietary sources such as dairy products, green leafy vegetables, and fortified foods can lead to rickets. This is particularly important during periods of rapid growth in children.
- Impaired Absorption: Conditions that affect calcium absorption, including vitamin D deficiency and certain gastrointestinal disorders, can result in low calcium levels.
- High Phytate or Oxalate Intake: Foods high in phytates (e.g., whole grains) or oxalates (e.g., spinach) can interfere with calcium absorption, contributing to deficiency.

3. Phosphate Deficiency

- Inadequate Dietary Intake: Low intake of phosphate-rich foods (such as meats, nuts, and legumes) can contribute to phosphate deficiency.
- Renal Phosphate Loss: Certain kidney disorders can lead to excessive phosphate loss through urine, resulting in phosphate deficiency. Conditions like renal tubular acidosis or Fanconi syndrome can affect phosphate reabsorption in the kidneys.

4. Genetic Factors

- Inherited Disorders: Genetic conditions such as X-linked hypophosphatemic rickets, a form of rickets that results from a genetic defect affecting phosphate metabolism, can cause rickets. These conditions often require specific treatment approaches.

5. Other Contributing Factors

- Chronic Kidney Disease: Impaired kidney function can affect the conversion of vitamin D into its active form and disrupt phosphate and calcium balance, contributing to rickets.
- Medication Effects: Certain medications, such as anticonvulsants, can interfere with vitamin D metabolism and calcium absorption, increasing the risk of rickets.
- Malnutrition: General malnutrition, which may result from poverty, lack of access to adequate food, or underlying health conditions, can lead to deficiencies in vitamin D, calcium, and phosphate.

6. Environmental and Lifestyle Factors

- Cultural Practices: Practices such as covering the skin extensively for cultural or religious reasons can limit sunlight exposure, increasing the risk of vitamin D deficiency.
- Economic Factors: Limited access to vitamin D-rich foods or supplements due to financial constraints can also contribute to the development of rickets.

In summary, the etiology of rickets primarily revolves around deficiencies in vitamin D, calcium, and phosphate, either due to dietary insufficiencies, malabsorption issues, or other underlying health conditions. Identifying and addressing these factors is crucial for the prevention and treatment of rickets.

## 5. PATHOPHYSIOLOGY

The pathophysiology of rickets involves disruptions in bone mineralization primarily due to deficiencies in vitamin D, calcium, or phosphate. Here's a detailed look at how these deficiencies lead to the development of rickets:

1. Vitamin D Deficiency:

- Role of Vitamin D: Vitamin D is essential for the absorption of calcium and phosphate from the gastrointestinal tract. It also helps regulate calcium and phosphate levels in the blood and bone.
- Deficiency Consequences: When vitamin D levels are insufficient, the body's ability to absorb calcium and phosphate from the diet is impaired. This results in decreased levels of calcium and phosphate in the bloodstream.

2. Calcium and Phosphate Imbalance:
- Hypocalcemia: Low blood calcium levels stimulate the release of parathyroid hormone (PTH), which mobilizes calcium from bones and increases renal calcium reabsorption. Chronic hypocalcemia can lead to weakened bones.
- Hypophosphatemia: Low phosphate levels further disrupt bone mineralization, as phosphate is a crucial component of hydroxyapatite, the mineral matrix of bones.

3. Disruption of Bone Mineralization:
- Osteoid Formation: Bones are composed of an organic matrix (osteoid) and inorganic minerals. In rickets, the osteoid matrix is formed but is inadequately mineralized due to the lack of calcium and phosphate.
- Softening of Bones: The lack of adequate mineralization leads to the softening of bones, making them more susceptible to deformities under mechanical stress.

4. Bone Deformities:
- Growth Plate Abnormalities: In growing children, rickets affects the growth plates (epiphyseal plates) of long bones, leading to abnormal bone growth and deformities such as bowed legs or knock-knees.
- Skeletal Deformities: The weakened bone structure can result in deformities such as a protruding chest (pectus carinatum), a curved spine (kyphosis or scoliosis), and a larger head relative to the body size.

5. Secondary Effects:
- Compensatory Mechanisms: The body may attempt to compensate for low calcium by increasing bone resorption and releasing calcium from bones. This can lead to additional bone loss and structural changes.
- Metabolic Implications: Chronic rickets can affect overall metabolism and growth, leading to delayed development and other systemic health issues.

In summary, the pathophysiology of rickets centers around the inadequate mineralization of bones due to deficiencies in key nutrients, primarily vitamin D, calcium, and phosphate. This leads to soft, weak bones and various skeletal deformities. Addressing these deficiencies through appropriate supplementation and dietary changes is crucial for managing and treating rickets.

## 6. CLINICAL MANIFESTATION

The clinical manifestations of rickets can vary depending on the severity and duration of the condition. Common signs and symptoms include:

1. Bone Deformities:
   - Bowed Legs: One of the most recognizable signs, especially in weight-bearing areas like the legs.
   - Knock-Knees: A condition where the knees touch while the feet remain apart.
   - Curved Spine: This may present as scoliosis (side-to-side curvature) or kyphosis (forward curvature).

2. Growth Disturbances:
   - Delayed Growth: Children with rickets may experience slower growth compared to peers.
   - Delayed Dental Eruption: Teeth may come in later than expected.

3. Bone Pain and Tenderness:
   - Pain in the Bones: This can occur in various parts of the body, including the legs, arms, and ribs.
   - Tenderness Over Bones: Specific areas, particularly the ribs and long bones, may be tender to touch.

4. Soft Skulls:
   - Delayed Closure of Fontanelles: The soft spots on a baby's skull may remain open longer than usual.

- Frontal Bossing: An abnormal protrusion of the forehead due to softening of the skull bones.

5. Chest Deformities:

- Rachitic Rosary: Swelling at the junctions of the ribs and cartilage, giving a bead-like appearance.
- Pectus Carinatum: A protruding chest, also known as a "bird chest."

6. Muscle Weakness:

- Weakness and Fatigue: Reduced muscle strength can contribute to difficulty with physical activities and poor muscle tone.

7. Skeletal Abnormalities:

- Widened Growth Plates: Visible on X-rays, where the growth plates are widened and irregular.
- Abnormal Bone Density: X-rays may show areas of decreased bone density or thinning.

8. Other Symptoms:

- Delayed Motor Skills: Difficulty with crawling, walking, or other developmental milestones.
- Frequent Fractures: Due to weakened bones, children may experience more fractures than usual.

The severity and presentation of rickets can vary based on factors such as age, the underlying cause of the deficiency, and how long the condition has been present. Early diagnosis and treatment are crucial to managing symptoms and preventing long-term complications.

## 7. DIAGNOSTIC EVAULTION

Diagnosing rickets involves a combination of clinical assessment, laboratory tests, and imaging studies. The goal is to identify the underlying cause of the condition, assess the severity, and guide appropriate treatment. Here’s a comprehensive approach to the diagnostic evaluation of rickets:

1. Clinical Assessment

- Medical History:
  - Symptom Onset: Document the onset and progression of symptoms such as bone deformities, pain, or delayed growth.
  - Dietary History: Assess intake of vitamin D, calcium, and phosphate-rich foods.
  - Sunlight Exposure: Inquire about exposure to sunlight and any potential cultural or lifestyle factors affecting it.
  - Family History: Explore any family history of rickets or related disorders.

- Physical Examination:
  - Bone Deformities: Examine for signs of bone deformities such as bowed legs, knock-knees, and a protruding chest.
  - Growth and Development: Assess for delayed growth, developmental milestones, and changes in physical development.
  - Neurological Exam: Check for signs of muscle weakness or delayed motor skills.

2. Laboratory Tests

- Serum Calcium Levels:
  - Typically low in vitamin D deficiency-related rickets, though normal or even high in some forms of rickets.

- Serum Phosphate Levels:
  - Low in nutritional rickets or due to renal phosphate wasting.
  - Normal to low in cases of vitamin D deficiency or other forms of rickets.

- Serum 25-Hydroxyvitamin D:
  - Measure this to assess vitamin D status. Low levels are indicative of vitamin D deficiency.

- Parathyroid Hormone (PTH):
  - Elevated in response to low calcium levels, contributing to bone resorption and rickets.

- Alkaline Phosphatase:
  - Often elevated in rickets due to increased bone turnover and remodeling.

- Bone Biopsy (rarely used):
  - In cases where the diagnosis is uncertain, a bone biopsy can reveal histological changes characteristic of rickets.

3. Imaging Studies

- X-rays:
  - Long Bones: Look for characteristic changes such as widening of the growth plates, metaphyseal changes, and bone deformities.
  - Skeleton: Evaluate for signs of poor mineralization, such as bone softening or deformation.

- Ultrasound:
  - Can be used to assess soft tissue changes or guide the diagnosis in certain cases, although it is less commonly used for rickets specifically.

- Bone Densitometry:
  - In some cases, especially if osteoporosis is suspected, bone mineral density (BMD) measurements can be performed.

4. Genetic Testing

- Genetic Analysis (if indicated):
  - For suspected genetic forms of rickets, such as X-linked hypophosphatemic rickets, genetic testing can confirm specific genetic mutations.

5. Differential Diagnosis

- Assess for other conditions:

- Conditions such as osteomalacia, congenital bone disorders, or metabolic bone diseases may present similarly to rickets and need to be ruled out.

6. Follow-Up

- Monitor Response to Treatment: Regular follow-up with repeat laboratory tests and imaging studies to assess the effectiveness of treatment and make necessary adjustments.

In summary, diagnosing rickets involves a combination of thorough clinical evaluation, targeted laboratory tests to assess mineral and vitamin levels, and imaging studies to visualize bone changes. Accurate diagnosis is crucial for effective management and treatment of rickets.

## 8. NURSING MANAGEMENT

Nursing management of rickets involves a comprehensive approach to address the underlying causes, provide appropriate treatment, and support the patient's overall well-being. Here's a detailed guide to managing rickets in a nursing context:

1. Assessment and Monitoring

- Initial Assessment:
  - Vital Signs: Monitor vital signs to identify any abnormalities that may be related to rickets or its complications.
  - Physical Examination: Assess for signs of bone deformities, growth delays, and muscle weakness.
  - Growth and Development: Track growth patterns and developmental milestones to evaluate the impact of rickets on the child's progress.

- Ongoing Monitoring:
  - Laboratory Tests: Regularly monitor serum levels of calcium, phosphate, and vitamin D to evaluate the effectiveness of treatment and make necessary adjustments.

- Bone Health: Monitor for signs of worsening bone deformities or complications. Ensure regular follow-up appointments for imaging studies if indicated.

2. Nutritional Support

- Vitamin D Supplementation:
  - Dosage and Administration: Administer vitamin D supplements as prescribed, ensuring the correct dosage and monitoring for potential side effects.
  - Patient Education: Educate caregivers about the importance of vitamin D and adherence to supplementation.

- Calcium and Phosphate Intake:
  - Dietary Guidance: Provide guidance on dietary sources of calcium and phosphate, such as dairy products, green leafy vegetables, nuts, and meats.
  - Supplementation: If needed, administer calcium and phosphate supplements according to medical recommendations.

3. Medication Management

- Medication Administration:
  - Ensure Proper Dosing: Administer medications as prescribed, including vitamin D, calcium, and phosphate supplements.
  - Monitor for Side Effects: Watch for any adverse reactions or side effects related to medications and report them to the healthcare provider.

- Education and Compliance:
  - Instruction: Educate families on the importance of medication adherence and potential side effects.
  - Follow-Up: Schedule and encourage regular follow-up visits to monitor the patient's progress and adjust treatment as needed.

4. Physical Therapy and Rehabilitation

- Physical Therapy:
  - Exercise Programs: Collaborate with physical therapists to develop exercise programs that improve muscle strength and mobility.
  - Supportive Devices: Provide or assist with the use of supportive devices, such as braces or orthotics, to help correct or manage bone deformities.

- Mobility Assistance:
  - Assistive Devices: If needed, help with the provision of mobility aids like walkers or crutches to enhance the patient's ability to move safely.

5. Psychosocial Support

- Emotional Support:
  - Counseling: Provide emotional support to patients and families dealing with the stress and challenges of managing rickets.
  - Support Groups: Facilitate access to support groups or counseling services for additional support.

- Education:
  - Disease Understanding: Educate the patient and family about rickets, its causes, and the importance of treatment and lifestyle changes.
  - Self-Management: Teach families about monitoring symptoms and managing the condition at home.

6. Infection Prevention and General Care

- Infection Control:
  - Hand Hygiene: Follow strict hand hygiene practices to prevent infections.
  - Wound Care: If applicable, provide proper wound care and infection prevention measures.

- General Care:
  - Comfort Measures: Ensure the patient is comfortable, provide pain management as needed, and address any other symptoms or complications.

7. Coordination of Care

- Interdisciplinary Collaboration:
  - Team Meetings: Collaborate with physicians, dietitians, physical therapists, and other healthcare professionals to provide holistic care.
  - Care Plans: Develop and update individualized care plans based on the patient's needs and progress.

- Family Involvement:
  - Engagement: Involve family members in care planning and decision-making to ensure they understand and can support the patient's needs.

8. Follow-Up and Evaluation

- Regular Check-Ups:
  - Schedule Visits: Ensure regular follow-up visits to monitor the patient's condition and treatment effectiveness.
  - Adjustments: Adjust the care plan as needed based on follow-up evaluations and laboratory results.

In summary, nursing management of rickets requires a multifaceted approach that includes monitoring, nutritional and medication management, physical therapy, psychosocial support, and coordination of care. Effective management aims to address the underlying deficiencies, support physical and emotional well-being, and improve overall outcomes for patients with rickets.

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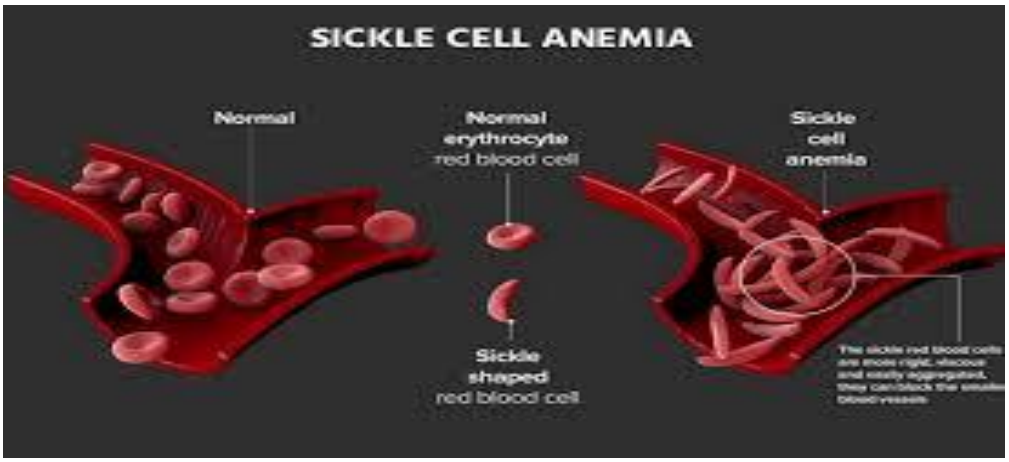
# SICKLE CELL ANEMIA

## 1. DEFINITION

Sickle cell anemia is a genetic disorder characterized by the production of abnormal hemoglobin, known as hemoglobin S, which causes red blood cells to become rigid, sticky, and shaped like a crescent or sickle. These abnormally shaped cells can cause blockages in small blood vessels, leading to various complications and symptoms.




## 2. OBJECTIVE

The objectives of managing sickle cell anemia are aimed at improving patient outcomes, minimizing complications, and enhancing quality of life. Here are the primary objectives:

1. Pain Management

- Reduce Pain Crises: Provide effective treatment to alleviate pain during vaso-occlusive crises, which are a hallmark of sickle cell anemia.
- Pain Relief Strategies: Use medications such as analgesics, opioids, and non-pharmacological methods (e.g., hydration, warmth) to manage pain.

2. Preventing and Managing Complications

- Infection Prevention: Implement measures to reduce the risk of infections, such as vaccinations, prophylactic antibiotics, and regular health screenings.
- Organ Damage Prevention: Monitor and manage potential damage to organs like the spleen, liver, kidneys, and lungs due to sickle cell disease.
- Stroke Prevention: Use blood transfusions or other therapies to reduce the risk of stroke, especially in children with a high risk of cerebrovascular events.

3. Anemia Management

- Address Anemia: Regularly monitor and manage anemia through treatments like blood transfusions, hydroxyurea, and iron supplementation if needed.
- Improve Hemoglobin Levels: Aim to maintain optimal hemoglobin levels to reduce symptoms of anemia and enhance overall well-being.

4. Enhancing Quality of Life

- Supportive Care: Provide comprehensive care that addresses physical, emotional, and psychological needs to improve the overall quality of life.
- Patient Education: Educate patients and their families about the disease, management strategies, and lifestyle modifications to empower them in self-care.

5. Promoting Healthy Growth and Development

- Monitor Growth: Regularly assess and support growth and developmental milestones in children with sickle cell anemia.
- Nutritional Support: Ensure adequate nutrition to support overall health and development, considering the specific needs related to sickle cell anemia.

6. Reducing Hospitalizations

- Prevent Crises: Implement strategies to reduce the frequency of painful crises and hospital admissions.
- Outpatient Management: Manage the disease effectively in outpatient settings to minimize the need for emergency interventions and hospital stays.

7. Utilizing Disease-Modifying Therapies

- Hydroxyurea: Use hydroxyurea to reduce the frequency of pain episodes and complications by increasing fetal hemoglobin (HbF) levels, which can reduce sickling.
- Bone Marrow Transplantation: Consider bone marrow transplantation as a potential curative treatment for eligible patients.

8. Providing Psychosocial Support

- Counseling and Support: Offer psychological and emotional support to help patients and families cope with the chronic nature of the disease and its impact on daily life.
- Support Groups: Facilitate access to support groups and resources to connect patients with others experiencing similar challenges.

9. Early Detection and Intervention

- Screening: Implement early screening and regular monitoring to identify and address complications before they become severe.
- Proactive Management: Use early interventions to manage emerging issues and prevent progression of the disease.

10. Research and Advancements

- Participate in Research: Encourage involvement in clinical trials and research studies aimed at improving treatments and understanding of sickle cell anemia.
- Stay Updated: Keep abreast of new developments in treatment options and care strategies to offer the most current and effective therapies.

In summary, the objectives of managing sickle cell anemia focus on alleviating pain, preventing and managing complications, improving quality of life, and providing comprehensive, supportive care. Effective management involves a multidisciplinary approach and aims to enhance both the physical and emotional well-being of patients.

## 3. PRINCIPLE

The principles of managing sickle cell anemia involve a multifaceted approach aimed at addressing the underlying pathophysiology of the disease, mitigating symptoms, and preventing complications. Here's a detailed overview of these principles:

1. Understanding the Pathophysiology

- Hemoglobin S Formation: Recognize that sickle cell anemia is caused by a genetic mutation leading to the production of abnormal hemoglobin S. This hemoglobin causes red blood cells to deform into a sickle shape under low oxygen conditions.
- Impaired Blood Flow: Understand that sickle-shaped cells can obstruct blood flow in small vessels, leading to pain, tissue damage, and organ dysfunction.

2. Pain Management

- Pain Relief: Prioritize effective pain management strategies for vaso-occlusive crises, including both pharmacological (analgesics, opioids) and non-pharmacological methods (hydration, heat application).
- Individualized Care: Tailor pain management plans to the patient's specific needs and response to treatment.

3. Preventing and Managing Complications

- Infection Prevention: Implement preventive measures such as vaccinations and prophylactic antibiotics to reduce the risk of infections, which can be exacerbated by spleen dysfunction.
- Organ Protection: Monitor and manage complications related to organ damage, such as stroke, acute chest syndrome, and priapism, to minimize long-term damage and improve quality of life.

4. Anemia Management

- Transfusion Therapy: Use blood transfusions to manage severe anemia and prevent complications like stroke, especially in high-risk patients.
- Hydroxyurea Therapy: Administer hydroxyurea to increase fetal hemoglobin (HbF) levels, which reduces sickling and decreases the frequency of pain crises and other complications.

5. Promoting Healthy Growth and Development

- Growth Monitoring: Regularly monitor children with sickle cell anemia to ensure proper growth and development.
- Nutritional Support: Provide appropriate nutritional advice and supplementation to support overall health and address specific needs related to anemia and disease management.

6. Psychosocial Support

- Emotional Well-being: Address the psychological and emotional aspects of living with a chronic condition through counseling and support services.
- Education: Educate patients and families about the disease, treatment options, and strategies for managing daily life with sickle cell anemia.

7. Disease Modifications and Advanced Therapies

- Bone Marrow Transplant: Consider bone marrow or stem cell transplantation as a potential curative option for eligible patients, especially in children.
- Clinical Trials: Encourage participation in research and clinical trials to access new treatments and contribute to advancing knowledge and therapies.

8. Preventive Care and Monitoring

- Routine Screenings: Implement routine screenings to detect and manage complications early, such as regular eye exams, renal function tests, and pulmonary evaluations.
- Preventive Measures: Employ strategies to prevent crises, such as maintaining adequate hydration, managing stress, and avoiding extreme temperatures.

9. Patient-Centered Care

- Personalized Approach: Provide individualized care plans based on the patient's specific needs, preferences, and response to treatment.
- Family Involvement: Engage family members in the care process to ensure they understand the disease, treatment, and how they can support the patient.

10. Education and Awareness

- Patient Education: Educate patients about the importance of adherence to treatment plans, recognizing symptoms of complications, and making lifestyle adjustments.
- Public Awareness: Increase awareness about sickle cell anemia in the community to promote early diagnosis and support research and advocacy efforts.

In summary, the principles of managing sickle cell anemia focus on understanding the disease's pathophysiology, effective pain management, preventing and managing complications, and providing comprehensive, patient-centered care. These principles aim to improve patient outcomes, enhance quality of life, and support overall health and well-being.

## 4. ETIOLOGY

Sickle cell anemia is a genetic disorder caused by a specific mutation in the hemoglobin gene. Understanding its etiology involves examining the genetic basis of the disease, its inheritance pattern, and the resultant pathological effects on red blood cells. Here's a detailed breakdown:

1. Genetic Mutation

- Hemoglobin Gene Mutation: Sickle cell anemia is caused by a mutation in the HBB gene, which provides instructions for making the beta-globin subunit of hemoglobin. The mutation involves a single nucleotide change in the DNA sequence.

- Point Mutation: The specific mutation is a point mutation in the sixth codon of the beta-globin gene, where adenine (A) is replaced by thymine (T). This results in the substitution of glutamic acid with valine in the beta-globin chain, creating abnormal hemoglobin S (HbS).

2. Inheritance Pattern

- Autosomal Recessive: Sickle cell anemia is inherited in an autosomal recessive manner. This means that an individual must inherit two copies of the sickle cell gene (one from each parent) to manifest the disease.

- Carrier State (Sickle Cell Trait): If an individual inherits only one copy of the sickle cell gene and one normal beta-globin gene, they are a carrier, known as having sickle cell trait. Carriers generally do not exhibit the symptoms of sickle cell anemia but can pass the gene to their offspring.

3. Pathophysiology

- Abnormal Hemoglobin S Formation: The mutation causes the production of hemoglobin S, which differs from normal hemoglobin (hemoglobin A) in its structure and function. Hemoglobin S molecules tend to aggregate under low oxygen conditions, forming long, rigid structures.

- Red Blood Cell Deformation: When hemoglobin S aggregates, it causes red blood cells to deform into a sickle shape. These sickle-shaped cells are less flexible and more prone to breaking apart (hemolysis) than normal disc-shaped red blood cells.

4. Consequences of Sickle Cell Formation

- Vaso-Occlusive Crises: Sickle-shaped cells can block small blood vessels, impeding blood flow and oxygen delivery to tissues. This blockage leads to painful episodes known as vaso-occlusive crises and can cause tissue damage.

- Chronic Hemolysis: The sickle cells are fragile and break down more rapidly than normal red blood cells, leading to chronic hemolytic anemia. This results in a lower-than-normal number of red blood cells and associated symptoms like fatigue and pallor.

- Organ Damage: Repeated vaso-occlusive events and chronic anemia can lead to damage in various organs, including the spleen (functional asplenia), liver, kidneys, and lungs.

5. Genetic Variants and Population Distribution

- Geographic Distribution: The sickle cell gene is prevalent in regions where malaria is or was common, such as parts of Africa, the Middle East, India, and the Mediterranean. The sickle cell trait provides some protection against malaria, which is believed to be a selective advantage in these regions.

- Genetic Variants: There are different genetic variants of hemoglobin S, but the most common form associated with sickle cell anemia is HbS. Other variants, such as HbSC (sickle cell-hemoglobin C disease) and HbSβ-thalassemia, also affect the severity and clinical presentation of the disease.

6. Environmental and Non-Genetic Factors

- Oxygen Levels and Stress: Environmental factors such as high altitudes, dehydration, and infections can exacerbate sickling and contribute to crises.
- Body Temperature: Extreme temperatures can also influence the sickling process and trigger symptoms.

In summary, sickle cell anemia is caused by a genetic mutation in the HBB gene that leads to the production of abnormal hemoglobin S. This mutation results in sickle-shaped red blood cells that can cause various complications, including vaso-occlusive crises and chronic anemia. The disease is inherited in an autosomal recessive manner, and its prevalence is notably higher in populations historically exposed to malaria.

## 5. PATHOPHYSIOLOGY

The pathophysiology of sickle cell anemia involves several interconnected processes that lead to the characteristic symptoms and complications of the disease. Here's a detailed breakdown:

1. Abnormal Hemoglobin Formation

- Hemoglobin S Production: In sickle cell anemia, a mutation in the HBB gene results in the production of abnormal hemoglobin known as hemoglobin S (HbS) instead of normal hemoglobin A (HbA). This mutation involves a substitution of glutamic acid with valine in the beta-globin chain of hemoglobin.

- Hemoglobin Aggregation: Under low oxygen conditions, hemoglobin S molecules aggregate and form long, rigid polymers. This aggregation causes the red blood cells to deform into a sickle shape.

2. Red Blood Cell Deformation and Hemolysis

- Sickle-Shaped Cells: The sickle-shaped red blood cells are less flexible and more rigid compared to normal, disc-shaped red blood cells. These abnormally shaped cells can cause several issues:

- Impaired Flexibility: Sickle cells are less able to navigate through small blood vessels, which can lead to blockages and reduced blood flow.
- Increased Fragility: Sickle cells are more prone to breaking apart (hemolysis) than normal red blood cells, leading to chronic hemolytic anemia.

3. Vaso-Occlusive Crises

- Blockage of Blood Vessels: Sickle-shaped cells can obstruct blood flow in capillaries and small arteries. This blockage causes reduced oxygen delivery to tissues and organs, leading to pain and tissue damage.

- Painful Episodes: The obstruction of blood flow results in vaso-occlusive crises, characterized by sudden, severe pain in various parts of the body, including the bones, joints, and abdomen.

4. Chronic Hemolytic Anemia

- Increased Red Cell Turnover: The rapid destruction of sickle-shaped red blood cells leads to a shortage of red blood cells (anemia). This anemia causes symptoms such as fatigue, pallor, and shortness of breath.

- Splenic Dysfunction: The spleen, which is responsible for filtering out damaged red blood cells, can become overwhelmed or damaged by the increased hemolysis, leading to functional asplenia (reduced spleen function).

5. Organ Damage

- Repeated Ischemia: The recurrent episodes of vaso-occlusive crises can cause damage to various organs. For example:
  - Kidney Damage: Reduced blood flow can impair kidney function, leading to complications like hematuria (blood in urine) and nephropathy.
  - Lung Damage: Repeated pulmonary emboli and decreased oxygen delivery can lead to acute chest syndrome and chronic lung damage.
  - Bone Damage: Osteonecrosis or avascular necrosis of the bones can occur due to reduced blood flow.

6. Inflammatory Response

- Endothelial Activation: The sickle cells can trigger inflammation and activation of the endothelial cells lining the blood vessels. This inflammatory response contributes to further vaso-occlusive events and increases the risk of infection.

- Adhesion of Sickle Cells: Sickle cells can adhere to the blood vessel walls, contributing to the obstruction of blood flow and the inflammatory process.

7. Complications

- Stroke: Sickle cell disease increases the risk of cerebrovascular accidents (strokes) due to blockages in the cerebral blood vessels.

- Infections: The spleen's reduced function increases susceptibility to infections, particularly by encapsulated bacteria like Streptococcus pneumoniae.

- Priapism: Prolonged, painful erections (priapism) can occur due to trapped blood in the penile vessels.

8. Triggers and Exacerbating Factors

- Environmental Factors: Extreme temperatures, high altitudes, dehydration, and infections can exacerbate sickling and increase the risk of vaso-occlusive crises.

- Physiological Stress: Physical or emotional stress can also trigger or worsen symptoms.

In summary, the pathophysiology of sickle cell anemia involves the production of abnormal hemoglobin S, which causes red blood cells to deform into a sickle shape. These sickle-shaped cells lead to blocked blood flow, chronic hemolytic anemia, and organ damage. The disease is characterized by vaso-occlusive crises, increased hemolysis, and a range of complications resulting from reduced blood flow and tissue ischemia.

## 6. CLINICAL MANIFESTATION

The clinical manifestations of sickle cell anemia result from the complex interactions between abnormal hemoglobin, red blood cell sickling, and the resultant effects on various organs and systems. Here are the primary clinical manifestations:

1. Painful Episodes (Vaso-Occlusive Crises)

- Symptoms: Acute and severe pain episodes, often occurring in the bones, joints, chest, and abdomen. These crises are caused by the obstruction of blood flow in small vessels, leading to tissue ischemia.
- Frequency: The frequency and intensity of pain episodes can vary widely among individuals.

2. Chronic Anemia

- Fatigue: Due to the decreased number of functional red blood cells, patients often experience chronic fatigue, weakness, and pallor.
- Shortness of Breath: Reduced oxygen-carrying capacity of the blood can lead to dyspnea (shortness of breath), especially during exertion.

3. Splenic Complications

- Functional Asplenia: Repeated vaso-occlusive crises and hemolysis can lead to damage of the spleen, resulting in reduced or absent spleen function. This increases the risk of infections, particularly by encapsulated bacteria (e.g., Streptococcus pneumoniae).
- Splenomegaly: In children, the spleen may be enlarged (splenomegaly) due to sequestration of sickle cells, although it may become atrophied (autosplenectomy) in adults.

4. Acute Chest Syndrome

- Symptoms: A serious complication characterized by chest pain, fever, cough, and difficulty breathing. It is caused by pulmonary infection, infarction, or embolism.
- Risk: It is a leading cause of hospitalization and can be life-threatening.

5. Stroke

- Symptoms: Neurological deficits such as sudden weakness or numbness in the limbs, difficulty speaking, and loss of vision, which are signs of cerebrovascular accidents (strokes).
- Prevalence: Children with sickle cell anemia are at higher risk of stroke, which can result in long-term neurological impairment.

6. Hand-Foot Syndrome (Dactylitis)

- Symptoms: Painful swelling of the hands and feet, often seen in infants and young children. This condition results from vaso-occlusive episodes in the small blood vessels of the extremities.

7. Priapism

- Symptoms: Painful, prolonged erections without sexual stimulation. Priapism occurs due to trapped blood in the penile vessels and can lead to erectile dysfunction if not treated promptly.

8. Leg Ulcers

- Symptoms: Chronic, non-healing ulcers on the legs, typically seen in older patients. These ulcers result from poor blood flow and repeated episodes of vaso-occlusive crises.

9. Bone and Joint Complications

- Symptoms: Chronic pain, deformities, or osteonecrosis (death of bone tissue) due to impaired blood supply. Commonly affected areas include the hips and knees.

10. Renal Complications

- Symptoms: Hematuria (blood in urine), proteinuria (protein in urine), and potential renal failure due to sickle cell-induced damage to the kidneys.

11. Eye Complications

- Symptoms: Retinopathy (damage to the retina) which can lead to vision problems. Sickle cell anemia can cause changes in the blood vessels of the eye, potentially leading to vision loss.

12. Growth and Development Issues

- Symptoms: Delayed growth and puberty in children due to chronic anemia and frequent illness. Growth may be stunted due to the systemic impact of the disease.

13. Psychosocial Impact

- Symptoms: Emotional and psychological challenges including stress, depression, and anxiety due to the chronic nature of the disease and frequent hospitalizations.

14. Delayed Wound Healing

- Symptoms: Poor wound healing due to impaired blood flow and chronic anemia. This can affect both minor and major injuries or surgical wounds.

In summary, the clinical manifestations of sickle cell anemia are diverse and can impact multiple organ systems, leading to a range of symptoms including severe pain, chronic anemia, increased susceptibility to infections, and complications affecting the spleen, lungs, brain, and other areas. Management of these manifestations requires a comprehensive, multidisciplinary approach to address both acute crises and long-term health needs.

## 7. DIAGNOSTIC EVALUATION

The diagnostic evaluation of sickle cell anemia involves several key tests and procedures to confirm the diagnosis, assess the severity of the disease, and monitor for complications. Here's a comprehensive overview:

1. Newborn Screening

- Purpose: Many countries have routine newborn screening programs to identify sickle cell anemia early, even before symptoms appear.
- Method: A blood sample is taken from the heel (heel prick test) shortly after birth and tested for the presence of hemoglobin S.

2. Hemoglobin Electrophoresis

- Purpose: This test confirms the diagnosis of sickle cell anemia by identifying the type of hemoglobin present in the blood.

- Method: A blood sample is analyzed to separate different types of hemoglobin. In sickle cell anemia, hemoglobin S (HbS) will be present in higher amounts.

3. Complete Blood Count (CBC)

- Purpose: To assess overall blood health and detect anemia.
- Method: Measures levels of hemoglobin, hematocrit, and red blood cells. In sickle cell anemia, the hemoglobin level is typically low, and there may be an increased number of reticulocytes (immature red blood cells).

4. Peripheral Blood Smear

- Purpose: To examine the shape and size of red blood cells.
- Method: A blood sample is spread on a slide and stained. The presence of sickle-shaped red blood cells can be observed under a microscope, confirming sickling.

5. Sickle Cell Solubility Test

- Purpose: To screen for the presence of hemoglobin S.
- Method: Detects the presence of sickle hemoglobin in the blood by assessing the solubility of hemoglobin in a solution. A positive result suggests sickle cell anemia or sickle cell trait.

6. Genetic Testing

- Purpose: To identify the specific mutations in the HBB gene that cause sickle cell anemia and to confirm the diagnosis if hemoglobin electrophoresis is inconclusive.
- Method: Analyzes DNA from a blood sample to detect the sickle cell mutation.

7. Additional Tests for Complications

- Renal Function Tests: To assess kidney function, including blood urea nitrogen (BUN) and serum creatinine levels, as kidney damage can occur in sickle cell anemia.

- Liver Function Tests: To monitor liver health, as the liver can be affected by sickle cell disease.

- Chest X-Ray: To evaluate for acute chest syndrome, pneumonia, or other pulmonary complications associated with sickle cell anemia.

- Ultrasound: Used to assess for splenomegaly, assess the spleen's size and function, and to monitor for gallstones and liver conditions.

- Transcranial Doppler (TCD) Ultrasound: Specifically used in children to assess blood flow in the brain and predict the risk of stroke. Regular TCD screenings help guide the need for blood transfusion therapy to prevent stroke.

- Eye Exam: A comprehensive eye examination to detect sickle cell retinopathy or other ocular complications.

8. Bone Marrow Biopsy (Less Common)

- Purpose: To evaluate the bone marrow's production of red blood cells, especially if there is an unusual presentation or if other conditions need to be ruled out.
- Method: A sample of bone marrow is obtained, usually from the pelvis, and examined for abnormalities.

9. Hydroxyurea Monitoring

- Purpose: If hydroxyurea therapy is being used, regular monitoring of blood counts and potential side effects is necessary.
- Method: Includes routine blood tests to monitor hemoglobin levels, white blood cell counts, and potential effects on the bone marrow.

Summary

In summary, the diagnostic evaluation of sickle cell anemia involves a combination of newborn screening, hemoglobin electrophoresis, complete blood count, peripheral blood smear, and additional tests as needed. These evaluations help confirm the diagnosis, assess the severity of the disease, and monitor for complications, guiding appropriate management and treatment strategies. Regular monitoring and follow-up are crucial for managing the disease and improving patient outcomes.

## 8. NURSING MANAGEMENT

Nursing management of sickle cell anemia involves a comprehensive approach to address both the immediate needs during crises and the long-term management of the disease. The goal is to alleviate symptoms, prevent complications, and enhance the patient's overall quality of life. Here's a detailed overview:

1. Pain Management

- Assessment: Regularly assess pain using appropriate scales (e.g., numerical rating scale, Wong-Baker FACES scale) and evaluate the characteristics, location, and intensity of pain.
- Pharmacological Interventions: Administer pain medications as prescribed, which may include acetaminophen, nonsteroidal anti-inflammatory drugs (NSAIDs), or opioids for severe pain.
- Non-Pharmacological Interventions: Use complementary methods such as hydration, warmth (heating pads), relaxation techniques, and distraction therapies to help manage pain.
- Education: Teach patients and families about pain management strategies, including when and how to use prescribed medications and alternative methods.

2. Hydration and Nutrition

- Hydration: Encourage adequate fluid intake to prevent dehydration, which can trigger or worsen vaso-occlusive crises. Monitor fluid balance and adjust intake based on patient needs.
- Nutrition: Provide a balanced diet rich in essential nutrients to support overall health and manage anemia. Address any dietary restrictions or preferences and ensure adequate caloric intake.

3. Preventing and Managing Complications

- Infection Prevention: Educate on the importance of vaccinations (e.g., pneumococcal, influenza) and prophylactic antibiotics. Monitor for signs of infection and promptly address any symptoms.
- Organ Function Monitoring: Regularly assess organ function, including renal and hepatic functions, and perform routine screenings for potential complications such as stroke or acute chest syndrome.
- Hydroxyurea Therapy: Monitor for side effects and efficacy if the patient is on hydroxyurea. Regular blood tests are needed to monitor blood counts and adjust dosage as necessary.

4. Emergency Management

- Crisis Management: Be prepared to provide prompt intervention during vaso-occlusive crises or other acute complications, such as acute chest syndrome or priapism. Follow protocols for managing these emergencies.
- Transfusion Therapy: Administer blood transfusions as prescribed and monitor for transfusion reactions. Educate patients and families about the purpose and potential side effects of transfusions.

5. Patient and Family Education

- Disease Education: Provide education on the nature of sickle cell anemia, its symptoms, and the importance of adherence to treatment plans. Include information on recognizing and responding to early signs of complications.
- Self-Care: Teach patients and families self-care techniques, including pain management strategies, hydration, and infection prevention.
- Lifestyle Modifications: Advise on lifestyle changes to minimize triggers for sickling, such as avoiding extreme temperatures, managing stress, and maintaining a healthy lifestyle.

6. Psychosocial Support

- Emotional Support: Offer psychological support and counseling to help patients and families cope with the chronic nature of the disease and the impact on daily life.

- Support Groups: Facilitate access to support groups and resources for connecting with others who have sickle cell anemia.
- Stress Management: Provide resources and strategies for managing stress and improving coping mechanisms.

7. Monitoring and Follow-Up

- Regular Check-Ups: Schedule and coordinate regular follow-up appointments to monitor disease progression, assess treatment effectiveness, and address any new or ongoing issues.
- Health Maintenance: Ensure that patients receive regular health screenings, including eye exams, dental check-ups, and bone health evaluations.

8. Coordination of Care

- Multidisciplinary Approach: Work with a multidisciplinary team, including hematologists, pain specialists, social workers, and dietitians, to provide comprehensive care.
- Care Coordination: Ensure continuity of care and effective communication between various healthcare providers involved in the patient's care.

9. Education on Long-Term Management

- Genetic Counseling: Provide information and resources about genetic counseling for patients and families to understand inheritance patterns and implications for future generations.
- Advanced Therapies: Discuss options for advanced treatments, such as bone marrow transplantation, if applicable, and provide information about clinical trials or research opportunities.

10. Documentation and Reporting

- Accurate Records: Maintain accurate and detailed records of symptoms, treatments, and responses to interventions. Document patient education and any changes in condition.
- Communication: Communicate effectively with the healthcare team and ensure that all relevant information is shared to coordinate care and improve outcomes.

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# CEREBRAL PALSY

## 1. DEFINITION

Cerebral palsy is a neurological disorder that results from damage to one or more areas of the brain that control muscle movements. This damage disrupts the brain's ability to coordinate and regulate muscle movements, leading to various motor impairments and physical disabilities. The damage to the brain is typically non-progressive, meaning that it does not worsen over time, though the symptoms and their impact can change as the individual grows.

## 2. OBJECTIVE

The objectives of managing cerebral palsy (CP) focus on improving the quality of life for individuals with the condition by addressing their unique needs and challenges. These objectives aim to enhance physical function, maximize independence, and support overall well-being. Here's a detailed outline of the objectives:

1. Enhance Motor Function and Mobility

- Goal: Improve motor skills and mobility to help individuals perform daily activities and move more effectively.
- Strategies:
  - Implement physical therapy to enhance muscle strength, coordination, and balance.
  - Utilize assistive devices (e.g., walkers, wheelchairs) as needed to support mobility and independence.
  - Provide orthotic devices (e.g., braces) to improve posture and movement.

2. Maximize Functional Independence

- Goal: Increase the individual's ability to perform daily living activities independently.
- Strategies:
  - Engage in occupational therapy to develop skills for self-care activities (e.g., dressing, grooming, eating).

- Modify the home environment to make it more accessible and supportive of independence.
- Encourage adaptive techniques and tools to assist with daily tasks.

3. Improve Communication and Social Interaction

- Goal: Facilitate effective communication and social interaction to enhance quality of life and social inclusion.
- Strategies:
  - Provide speech and language therapy to improve verbal communication and alternative communication methods if needed (e.g., communication devices).
  - Support social skills development and encourage participation in social activities and peer interactions.

4. Manage Associated Health Conditions

- Goal: Address and manage any co-occurring health conditions that may affect the individual's well-being.
- Strategies:
  - Monitor and treat associated conditions such as seizures, intellectual disabilities, vision or hearing impairments.
  - Coordinate care with specialists to manage and address specific health issues related to cerebral palsy.

5. Promote Physical and Emotional Well-Being

- Goal: Support overall health and emotional well-being to improve the individual's quality of life.
- Strategies:
  - Encourage participation in recreational and physical activities to promote physical health and reduce stress.
  - Provide psychological support and counseling to address emotional and behavioral challenges.
  - Educate families and caregivers about the importance of a healthy lifestyle and mental health support.

6. Facilitate Educational and Vocational Opportunities

- Goal: Ensure that individuals with cerebral palsy have access to educational and vocational opportunities suited to their abilities.
- Strategies:
  - Collaborate with educational professionals to develop an Individualized Education Plan (IEP) or equivalent, tailored to the individual's needs.
  - Support vocational training and job placement services to help individuals achieve their career goals and gainful employment.

7. Support Family and Caregiver Needs

- Goal: Provide support and resources for families and caregivers to enhance their ability to care for and support the individual with cerebral palsy.
- Strategies:
  - Offer family counseling, support groups, and respite care to help manage caregiving responsibilities and reduce stress.
  - Provide information and training on how to effectively care for the individual and navigate available resources and services.

8. Advocate for Accessibility and Inclusion

- Goal: Promote societal awareness and advocate for accessibility and inclusion in various settings.
- Strategies:
  - Work with community organizations and policymakers to improve accessibility in public spaces, transportation, and educational institutions.
  - Raise awareness about cerebral palsy and advocate for the rights and inclusion of individuals with disabilities.

9. Monitor and Adjust Treatment Plans

- Goal: Continuously assess and adjust treatment plans to meet evolving needs and optimize outcomes.
- Strategies:

- Regularly review and update treatment and therapy plans based on progress, changes in condition, and feedback from the individual and family.
- Ensure ongoing communication between healthcare providers, therapists, and caregivers to adapt interventions as needed.

In summary, the objectives of managing cerebral palsy include enhancing motor function and mobility, maximizing functional independence, improving communication and social interaction, managing associated health conditions, promoting overall well-being, and supporting educational, vocational, and family needs. These goals aim to improve the individual's quality of life and ensure they can achieve their fullest potential.

## 3. PRINCIPLE

The principles of managing cerebral palsy (CP) are grounded in a comprehensive, multidisciplinary approach that addresses the complex needs of individuals with the condition. These principles guide the treatment and care strategies to optimize functional outcomes and improve the overall quality of life. Here's an overview of the key principles:

1. Individualized Care

- Principle: Each person with cerebral palsy has unique needs, abilities, and challenges. Management plans should be tailored to the individual's specific condition and goals.
- Application: Develop personalized treatment plans based on comprehensive assessments of the individual's motor abilities, cognitive function, communication skills, and social needs. Regularly update these plans as the individual's needs and circumstances change.

2. Multidisciplinary Approach

- Principle: Effective management of cerebral palsy requires collaboration among various healthcare professionals, including neurologists, physical therapists, occupational therapists, speech therapists, and social workers.

- Application: Coordinate care among specialists to provide a holistic approach to treatment. This may involve regular team meetings to review progress, share insights, and adjust interventions as needed.

3. Functional Improvement

- Principle: The primary goal of treatment is to enhance functional abilities and maximize independence in daily activities.

- Application: Focus on therapies and interventions that improve motor skills, coordination, and functional independence. Prioritize activities and exercises that help the individual achieve their personal goals and participate in daily life.

4. Early Intervention

- Principle: Early diagnosis and intervention are crucial for improving outcomes and preventing or minimizing secondary complications.
- Application: Implement therapeutic and supportive interventions as soon as cerebral palsy is diagnosed. Early intervention can address developmental delays and promote optimal growth and development.

5. Holistic Care

- Principle: Address the physical, emotional, and social aspects of the individual's life to support overall well-being.
- Application: Provide a comprehensive care plan that includes physical therapy, occupational therapy, speech therapy, psychological support, and social services. Consider the individual's emotional needs, family dynamics, and social environment.

6. Family-Centered Care

- Principle: Involve families in the care process, recognizing their role in supporting and advocating for the individual with cerebral palsy.

- Application: Engage families in treatment planning and decision-making. Provide education and support to help them understand the condition, manage care, and navigate available resources.

7. Goal-Oriented Therapy

- Principle: Set specific, measurable, achievable, relevant, and time-bound (SMART) goals for therapy and intervention.
- Application: Develop clear objectives for each therapeutic intervention based on the individual's needs and progress. Regularly evaluate and adjust goals to reflect changes in the individual's abilities and priorities.

8. Evidence-Based Practice

- Principle: Utilize interventions and treatments that are supported by scientific research and clinical evidence.
- Application: Stay informed about the latest research and best practices in managing cerebral palsy. Implement therapies and strategies that have been proven effective through clinical studies and evidence-based guidelines.

9. Preventative Care

- Principle: Implement strategies to prevent secondary complications and optimize long-term health.
- Application: Monitor for and address potential complications such as musculoskeletal deformities, contractures, and pressure sores. Provide preventive care measures, including regular check-ups, vaccinations, and health screenings.

10. Empowerment and Advocacy

- Principle: Empower individuals with cerebral palsy and their families to advocate for their needs and rights.
- Application: Support self-advocacy and self-management skills. Provide resources and information about rights, services, and community support. Encourage participation in advocacy efforts to improve access and quality of care.

11. Continuous Evaluation and Adaptation

- Principle: Regularly assess the effectiveness of interventions and make necessary adjustments to the care plan.
- Application: Conduct ongoing evaluations of the individual's progress and the impact of interventions. Be flexible and responsive to changes in the individual's condition and needs.

In summary, the principles of managing cerebral palsy emphasize individualized, multidisciplinary, and holistic care. Early intervention, goal-oriented therapy, and evidence-based practices are central to optimizing functional outcomes and improving quality of life. Engaging families, preventing complications, and empowering individuals are key components of effective management.

## 4. ETIOLOGY

The etiology of cerebral palsy (CP) is diverse and involves a range of prenatal, perinatal, and postnatal factors that disrupt normal brain development and function. The condition results from brain injury or abnormal brain development, which affects motor control and coordination. Here is an overview of the primary causes and contributing factors:

1. Prenatal Causes

- Genetic Factors:
  - Genetic Mutations: Some cases of cerebral palsy are associated with genetic mutations or abnormalities that affect brain development or function.
  - Family History: A family history of neurological disorders or congenital conditions may increase the risk.

- Maternal Infections:
  - Infections During Pregnancy: Infections such as rubella, cytomegalovirus, toxoplasmosis, and herpes simplex virus can interfere with fetal brain development.
  - Inflammatory Responses: Maternal inflammation due to infections can impact fetal brain development and lead to cerebral palsy.

- Maternal Health Conditions:
  - Chronic Diseases: Conditions like diabetes, hypertension, or thyroid disorders in the mother can increase the risk of cerebral palsy.
  - Nutritional Deficiencies: Inadequate maternal nutrition or deficiencies in essential vitamins and minerals (e.g., folic acid) can impact fetal brain development.

- Exposure to Toxins:
  - Substance Abuse: Maternal use of drugs, alcohol, or tobacco during pregnancy can affect brain development and increase the risk of cerebral palsy.
  - Environmental Toxins: Exposure to certain environmental toxins or chemicals during pregnancy may contribute to the risk.

2. Perinatal Causes

- Birth Complications:
  - Oxygen Deprivation (Hypoxia): Lack of adequate oxygen during labor and delivery, often due to complications like umbilical cord compression or placental abruption, can cause brain damage.
  - Birth Trauma: Physical injury during delivery, such as head trauma or prolonged labor, can contribute to the development of cerebral palsy.

- Premature Birth:
  - Prematurity: Infants born prematurely (before 37 weeks of gestation) are at higher risk due to the underdevelopment of the brain and other organs. Premature infants may suffer from conditions like intraventricular hemorrhage (bleeding in the brain), which can lead to cerebral palsy.

- Low Birth Weight:
  - Intrauterine Growth Restriction (IUGR): Infants with low birth weight or those who experience restricted growth in the womb are at increased risk for cerebral palsy.

3. Postnatal Causes

- Brain Infections:
  - Infections After Birth: Infections such as meningitis or encephalitis occurring in the postnatal period can damage the brain and result in cerebral palsy.

- Severe Jaundice:
  - Kernicterus: Uncontrolled severe jaundice (high bilirubin levels) in newborns can lead to kernicterus, a type of brain damage that can cause cerebral palsy.

- Head Trauma:
  - Injury: Traumatic brain injury due to accidents or falls after birth can contribute to the development of cerebral palsy.

4. Unknown or Multifactorial Causes

- Idiopathic Cases:
  - Unclear Causes: In some cases, the exact cause of cerebral palsy is unknown. It may result from a combination of genetic, environmental, and developmental factors that are not fully understood.

- Complex Interactions:
  - Multifactorial Etiology: The development of cerebral palsy can result from a complex interplay of multiple factors, including genetic predispositions and environmental influences, which can make it challenging to pinpoint a single cause.

Summary

Cerebral palsy arises from various factors that affect brain development or cause brain injury before, during, or shortly after birth. These include genetic factors, maternal infections and health conditions, birth complications, premature birth, and postnatal injuries or infections. In many cases, the exact cause may not be identifiable, and the condition may result from a combination of multiple factors. Early identification and management of risk factors, along with comprehensive care, are crucial in addressing the needs of individuals with cerebral palsy.

# 5. PATHOPHYSIOLOGY

The pathophysiology of cerebral palsy (CP) involves a complex interplay of brain injury and developmental abnormalities that disrupt the normal functioning of the brain's motor control systems. The condition affects motor control, muscle tone, and coordination, leading to a range of physical disabilities. Here's a detailed overview of the pathophysiology:

1. Brain Injury and Developmental Abnormalities

- Brain Injury:
  - Lesions: Cerebral palsy often results from damage to specific areas of the brain responsible for motor control, including the motor cortex, basal ganglia, and cerebellum. The type and location of the brain injury influence the specific motor impairments and symptoms experienced.
  - Types of Lesions: Lesions can include infarcts (areas of dead tissue due to lack of blood flow), hemorrhages (bleeding), or malformations (abnormal brain development).

- Developmental Abnormalities:
  - Malformations: Abnormal brain development can occur due to genetic factors, maternal health conditions, or environmental influences. These malformations may affect brain structures involved in motor control and coordination.

2. Disruption of Motor Pathways

- Motor Cortex:
  - Damage to Motor Cortex: The motor cortex, located in the frontal lobe of the brain, plays a crucial role in planning and executing voluntary movements. Injury or abnormal development in this area can impair motor function, leading to difficulties with movement control.

- Basal Ganglia:
  - Dysfunction of Basal Ganglia: The basal ganglia, a group of structures involved in regulating movement and coordination, can be affected in certain types of cerebral palsy (e.g., dyskinetic CP). Damage to the

basal ganglia results in abnormal movement patterns, such as involuntary movements and muscle tone abnormalities.

- Cerebellum:
  - Impairment of Cerebellum: The cerebellum is responsible for coordinating and fine-tuning movements. Damage or abnormal development in the cerebellum can lead to issues with balance, coordination, and motor control, as seen in ataxic CP.

3. Abnormal Muscle Tone and Motor Control

- Spasticity:
  - Increased Muscle Tone: In spastic cerebral palsy, damage to the motor pathways results in increased muscle tone or stiffness. This leads to exaggerated reflexes and resistance to movement, causing difficulty with voluntary movements and coordination.

- Dystonia:
  - Involuntary Movements: In dyskinetic cerebral palsy, abnormal motor control due to basal ganglia dysfunction results in involuntary, irregular movements and muscle contractions. This can cause writhing or jerky movements and affect posture and coordination.

- Ataxia:
  - Impaired Coordination: In ataxic cerebral palsy, damage to the cerebellum disrupts the coordination and precision of movements. This can result in unsteady gait, difficulty with fine motor tasks, and problems with balance.

4. Secondary Musculoskeletal Changes

- Contractures:
  - Joint Stiffness: Abnormal muscle tone and spasticity can lead to joint contractures, where muscles and tendons become shortened and stiff. This restricts the range of motion and affects functional mobility.

- Deformities:

- Postural Changes: Over time, imbalances in muscle tone and joint contractures can lead to skeletal deformities, such as scoliosis (curvature of the spine) or hip dislocations.

5. Neuroplasticity and Compensation

- Adaptation and Compensation:
  - Neuroplasticity: The brain's ability to adapt and reorganize itself (neuroplasticity) plays a role in the development of motor skills and functional abilities. In individuals with cerebral palsy, neuroplasticity can lead to the development of compensatory mechanisms to improve function despite brain injury.
  - Compensatory Strategies: Therapies often focus on enhancing these compensatory strategies to improve functional outcomes and quality of life.

6. Impact on Other Systems

- Cognitive and Sensory Systems:
  - Cognitive Function: Although cerebral palsy primarily affects motor control, some individuals may also experience cognitive impairments or developmental delays. The extent of cognitive involvement varies depending on the type and severity of cerebral palsy.
  - Sensory Function: Sensory processing issues, such as difficulties with vision or hearing, may also be present, impacting overall function and quality of life.

Summary

The pathophysiology of cerebral palsy involves a range of brain injuries and developmental abnormalities that disrupt motor pathways and brain structures responsible for movement control. This leads to abnormal muscle tone, motor control issues, and secondary musculoskeletal changes. While the condition primarily affects motor function, it can also impact cognitive and sensory systems. The management of cerebral palsy aims to address these disruptions and improve functional abilities through various therapeutic approaches.

## 6. CLINICAL MANIFESTATION

Cerebral palsy (CP) presents with a diverse range of clinical manifestations, which vary based on the type and severity of the condition. The manifestations can affect motor control, muscle tone, posture, and coordination, and may also include other associated conditions. Here's a detailed overview of the clinical manifestations of cerebral palsy:

1. Motor Impairments

- Spasticity:
  - Characteristics: Increased muscle tone or stiffness that leads to difficulty with voluntary movements. This can result in exaggerated reflexes and resistance to movement.
  - Types: Spastic hemiplegia (one side of the body), spastic diplegia (primarily the legs), or spastic quadriplegia (all four limbs).

- Dyskinesia:
  - Characteristics: Involuntary, irregular movements due to damage to the basal ganglia. This can include writhing (athetosis) or jerky movements (chorea).
  - Types: Dyskinetic CP can involve a mix of movements, including dystonia (sustained muscle contractions) and athetosis (slow, writhing movements).

- Ataxia:
  - Characteristics: Difficulty with balance, coordination, and precise movements due to damage to the cerebellum.
  - Manifestations: Unsteady gait, poor coordination, and difficulty with fine motor tasks such as writing or using utensils.

2. Muscle Tone Abnormalities

- Hypertonia:
  - Characteristics: Increased muscle tone that causes stiffness and rigidity. Movements may be restricted and require more effort.
  - Impact: Can lead to contractures and joint deformities over time.

- Hypotonia:
  - Characteristics: Reduced muscle tone that results in weakness and poor posture control. Often seen in some types of cerebral palsy, such as hypotonic CP.
  - Impact: May lead to difficulties with holding the head up, sitting, or walking.

3. Postural and Gait Abnormalities

- Postural Changes:
  - Characteristics: Abnormalities in posture, such as an increased or decreased arch in the back, or asymmetrical posture.
  - Manifestations: May include scoliosis (curved spine) or kyphosis (hunchback).

- Gait Disturbances:
  - Characteristics: Abnormal walking patterns such as scissoring (legs crossing each other) or a wide-based gait.
  - Impact: Can affect the ability to walk independently and may require the use of assistive devices.

4. Fine and Gross Motor Skill Impairments

- Gross Motor Skills:
  - Characteristics: Difficulty with large movements such as sitting, standing, and walking.
  - Manifestations: Delayed milestones in crawling, walking, or running.

- Fine Motor Skills:
  - Characteristics: Challenges with small, precise movements involving the hands and fingers.
  - Manifestations: Difficulty with tasks like buttoning shirts, writing, or using utensils.

5. Speech and Communication Difficulties

- Speech Impairments:

- Characteristics: Difficulty with articulation, volume, or clarity of speech. Some individuals may have slurred speech or be non-verbal.
- Impact: May require speech therapy and the use of augmentative and alternative communication (AAC) devices.

- Language Delays:
- Characteristics: Challenges in understanding and using language effectively.
- Impact: May affect the ability to follow instructions, engage in conversations, or express needs.

6. Cognitive and Behavioral Issues

- Cognitive Impairments:
- Characteristics: Some individuals with cerebral palsy may experience intellectual disabilities or developmental delays.
- Impact: Varies from mild learning difficulties to more significant cognitive impairments.

- Behavioral and Emotional Challenges:
- Characteristics: May include difficulties with social interactions, attention, or behavioral regulation.
- Impact: Can affect social relationships and overall emotional well-being.

7. Associated Conditions

- Seizures:
- Characteristics: Epileptic seizures are common in some individuals with cerebral palsy.
- Impact: May require ongoing management with antiepileptic medications.

- Vision and Hearing Impairments:
- Characteristics: Problems with vision (e.g., strabismus, nystagmus) or hearing (e.g., hearing loss).
- Impact: Can affect sensory perception and overall development.

- Feeding and Swallowing Difficulties:
- Characteristics: Issues with chewing, swallowing, or feeding may be present.

- Impact: May require specialized feeding techniques or dietary modifications.

- Growth and Developmental Delays:
  - Characteristics: Delays in physical growth or developmental milestones.
  - Impact: May need intervention to support healthy growth and development.

Summary

Cerebral palsy presents with a range of clinical manifestations, including motor impairments (spasticity, dyskinesia, ataxia), muscle tone abnormalities, postural and gait changes, fine and gross motor skill difficulties, speech and communication challenges, and associated conditions like seizures and sensory impairments. The severity and specific manifestations vary widely among individuals, and a multidisciplinary approach is often required to address the diverse needs and improve the quality of life for those affected by cerebral palsy.

## 7. DIAGNOSTIC EVALUATION

Diagnosing cerebral palsy (CP) involves a comprehensive evaluation to confirm the presence of the condition, determine its type, and identify associated conditions. The diagnostic process typically includes a combination of medical history, physical examination, and various diagnostic tests. Here's a detailed overview of the diagnostic evaluation for cerebral palsy:

1. Medical History

- Prenatal and Birth History:
  - Maternal Health: Review the mother's health during pregnancy, including any infections, chronic conditions, or complications.
  - Labor and Delivery: Investigate any issues during labor and delivery, such as oxygen deprivation, trauma, or premature birth.

- Developmental History:

- Milestone Achievement: Assess the child's developmental milestones (e.g., sitting, crawling, walking) to identify delays or abnormalities.

- Family History:
  - Genetic Factors: Explore any family history of neurological disorders or developmental conditions.

2. Physical Examination

- Neurological Examination:
  - Motor Function: Evaluate muscle tone, coordination, and motor skills. Look for signs of spasticity, dyskinesia, or ataxia.
  - Reflexes: Check for abnormal reflexes, such as exaggerated or diminished reflexes.
  - Posture and Gait: Assess posture, balance, and walking patterns.

- Assessment of Associated Conditions:
  - Cognitive and Behavioral: Evaluate cognitive development and any potential behavioral issues.
  - Sensory Abilities: Check for vision and hearing impairments.

3. Diagnostic Imaging

- Magnetic Resonance Imaging (MRI):
  - Purpose: Provides detailed images of the brain to identify areas of damage, malformations, or structural abnormalities.
  - Use: Helps confirm the diagnosis and determine the type of cerebral palsy based on the location and extent of brain injury.

- Computed Tomography (CT) Scan:
  - Purpose: Offers detailed images of the brain, though less detailed than MRI. Useful for identifying major brain injuries or abnormalities.
  - Use: Often used if MRI is not available or suitable.

- Ultrasound:

- Purpose: Used in premature infants to assess for intraventricular hemorrhage (bleeding in the brain).
- Use: Helps identify early signs of brain injury or abnormalities.

4. Neurophysiological Tests

- Electroencephalogram (EEG):
  - Purpose: Measures electrical activity in the brain to detect seizure activity or other abnormal brain functions.
  - Use: Particularly important if seizures are present or suspected.

- Evoked Potentials:
  - Purpose: Assess the brain's response to visual, auditory, or sensory stimuli.
  - Use: Helps evaluate sensory pathways and brain function.

5. Genetic and Metabolic Testing

- Genetic Testing:
  - Purpose: Identifies any genetic mutations or abnormalities that may be associated with cerebral palsy.
  - Use: Useful if there is suspicion of a genetic cause or in cases with a family history of genetic disorders.

- Metabolic Screening:
  - Purpose: Tests for metabolic disorders that can mimic or contribute to symptoms similar to cerebral palsy.
  - Use: Includes screening for conditions such as metabolic syndromes or inborn errors of metabolism.

6. Developmental and Psychological Evaluation

- Developmental Assessment:
  - Purpose: Evaluates the child's cognitive, motor, language, and social development.
  - Use: Identifies developmental delays and helps in creating a comprehensive management plan.

- Psychological Evaluation:
  - Purpose: Assesses behavioral, emotional, and social aspects.

- Use: Helps identify any psychological or behavioral issues that may require intervention.

7. Additional Evaluations

- Speech and Language Assessment:
  - Purpose: Evaluates speech, language, and communication skills.
  - Use: Identifies any speech or language difficulties and guides therapy needs.

- Occupational Therapy Assessment:
  - Purpose: Assesses fine motor skills, daily living activities, and sensory processing.
  - Use: Helps in planning interventions to improve functional independence.

- Physical Therapy Assessment:
  - Purpose: Evaluates gross motor skills, muscle strength, and coordination.
  - Use: Guides physical therapy interventions to improve motor function and mobility.

Summary

The diagnostic evaluation of cerebral palsy involves a thorough medical history, detailed physical examination, and a range of diagnostic tests, including imaging studies (MRI, CT scan), neurophysiological tests (EEG, evoked potentials), genetic and metabolic testing, and developmental assessments. The goal is to confirm the diagnosis, determine the type of cerebral palsy, assess the extent of brain injury, and identify any associated conditions. This comprehensive evaluation helps in developing an effective, individualized management plan to address the child's needs and improve their quality of life.

## 8. NURSING MANAGEMENT

Nursing management of cerebral palsy (CP) involves a multidisciplinary approach focused on optimizing the individual's functional abilities, promoting independence, and improving quality of life. Nurses play a crucial role in coordinating care, implementing therapeutic interventions, and providing education and support to patients and their families. Here's a detailed overview of nursing management strategies for cerebral palsy:

1. Assessment and Monitoring

- Regular Monitoring:
  - Neurological Status: Assess motor function, muscle tone, reflexes, and any changes in neurological status.
  - Growth and Development: Monitor developmental milestones and physical growth to identify any delays or needs for intervention.

- Complication Surveillance:
  - Secondary Conditions: Watch for complications such as contractures, pressure sores, respiratory infections, or gastrointestinal issues.
  - Seizure Monitoring: If seizures are present, monitor frequency, duration, and triggers, and ensure appropriate seizure management.

2. Therapeutic Interventions

- Medication Administration:
  - Antispasticity Agents: Administer medications such as baclofen or dantrolene as prescribed to manage spasticity and muscle tone.
  - Antiepileptics: If seizures are present, ensure adherence to antiepileptic medications and monitor for side effects.
  - Pain Management: Assess and manage pain, using medications and non-pharmacological methods as needed.

- Physical Therapy:
  - Exercise Programs: Collaborate with physical therapists to implement individualized exercise programs that improve strength, coordination, and mobility.
  - Mobility Aids: Assist with the use of mobility aids such as wheelchairs, walkers, or braces.

- Occupational Therapy:
  - Daily Living Skills: Support occupational therapy interventions that enhance fine motor skills and promote independence in daily activities.

- Adaptive Equipment: Facilitate the use of adaptive devices and modifications to assist with tasks such as feeding, dressing, and personal hygiene.

- Speech Therapy:
  - Communication Aids: Work with speech therapists to support communication development and use of augmentative and alternative communication (AAC) devices if necessary.
  - Swallowing Support: Assist with techniques and strategies for safe feeding and swallowing, and monitor for signs of aspiration or choking.

3. Education and Support

- Patient and Family Education:
  - Condition Understanding: Provide information about cerebral palsy, its impact, and the available treatments and therapies.
  - Home Care: Educate families on how to manage care at home, including the use of assistive devices, medication administration, and recognizing signs of complications.

- Emotional and Psychological Support:
  - Counseling: Offer support to address the emotional and psychological challenges faced by individuals with cerebral palsy and their families.
  - Support Groups: Connect families with support groups or community resources for additional support and information.

4. Care Coordination

- Multidisciplinary Team Collaboration:
  - Care Plan Coordination: Work with a team of healthcare professionals, including physicians, therapists, and social workers, to develop and implement a comprehensive care plan.
  - Regular Meetings: Participate in team meetings to discuss progress, adjust interventions, and address any emerging needs.

- Transition Planning:

- School and Community Integration: Support transitions to school and community settings by coordinating with educators, therapists, and community resources.
- Adolescent and Adult Care: Plan for transitions from pediatric to adult care, including addressing changes in healthcare needs and services.

5. Preventive Care

- Infection Prevention:
  - Hygiene: Emphasize good hygiene practices to prevent infections, especially if the individual has compromised mobility or skin integrity.
  - Vaccinations: Ensure that routine vaccinations are up-to-date to prevent preventable diseases.

- Skin Care:
  - Pressure Ulcer Prevention: Implement strategies to prevent pressure sores, including regular repositioning and use of pressure-relieving devices.
  - Skin Inspection: Regularly inspect the skin for signs of breakdown or infection, especially in areas prone to pressure ulcers.

- Nutrition and Hydration:
  - Balanced Diet: Support nutritional needs by promoting a balanced diet and addressing any feeding difficulties or special dietary requirements.
  - Hydration: Ensure adequate fluid intake to prevent dehydration and support overall health.

6. Safety and Advocacy

- Safety Measures:
  - Home Safety: Provide recommendations for modifying the home environment to ensure safety, such as installing grab bars and removing tripping hazards.
  - Emergency Preparedness: Educate families on emergency procedures and how to respond to medical emergencies, such as seizures or respiratory issues.

- Advocacy:

- Access to Services: Advocate for access to necessary services and resources, including therapy, educational support, and community programs.
- Rights and Resources: Inform families about their rights and available resources, including financial assistance, legal rights, and support networks.

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# SWARRNIM START UP AND INNOVATION UNIVERSITY

# AARIHANT INSTITUTE OF NURSING

COURSE NAME : ANATOMY AND PHYSIOLOGY

# THE RESPIRATORY SYSTEM

## THE RESPIRATORY SYSTEM:

The respiratory system consists of the set of organs and tissues involved in the uptake of oxygen from the atmosphere and the release of carbon dioxide generated during aerobic respiration. This gas exchange is also called breathing or external respiration.

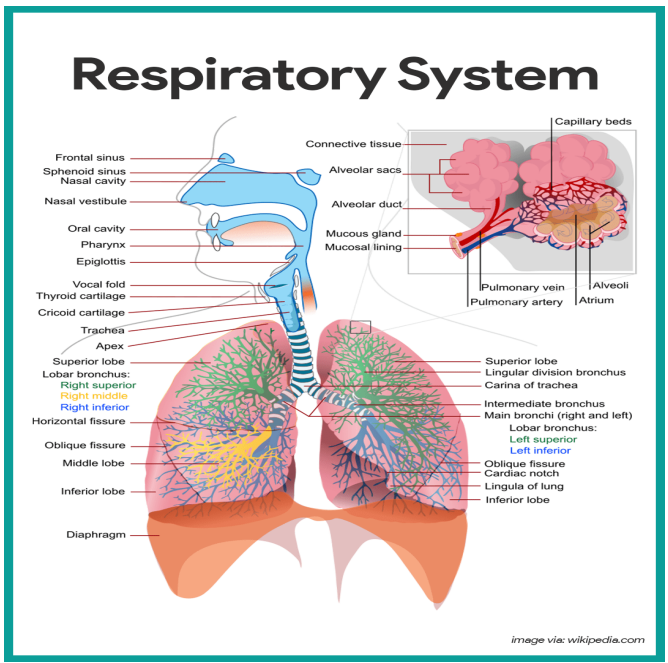
Organs specialized for breathing usually **contain moist structures with large surface areas to allow the diffusion of gases.** They are also adapted to protect the organism from the invasion of pathogens along those surfaces.

In fish, this gas exchange occurs through gills. Some invertebrates, like cockroaches, have simple respiratory systems made of interconnecting tubules directly delivering oxygen to tissues. In humans and other mammals, there is an extensive, highly vascularized organ system specialized for gas exchange.

**The respiratory system begins in the nose, continues into the pharynx and larynx, leads to the trachea which branch to create bronchi, and finally down the bronchioles into the lungs.** This respiratory tree ends in puffy structures called alveoli that are made of a single layer of squamous cells, surrounded by a network of capillaries. Gas exchange occurs within alveoli. Since external respiration in many vertebrates involves lungs, it is also called pulmonary ventilation. Changes to the volume and pressure in the lungs are the primary driving forces for breathing.

The functions of the respiratory system are:

1. **Oxygen supplier.** The job of the respiratory system is to keep the body constantly supplied with oxygen.
2. **Elimination.** Elimination of carbon dioxide.
3. **Gas exchange.** The respiratory system organs oversee the gas exchanges that occur between the blood and the external environment.
4. **Passageway.** Passageways that allow air to reach the lungs.
5. **Humidifier.** Purify, humidify, and warm incoming air.



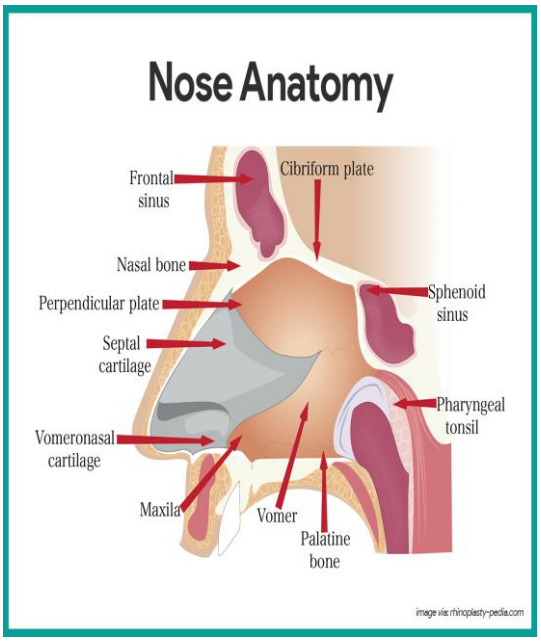

**Anatomy of the Respiratory System**

The organs of the respiratory system include the nose, pharynx, larynx, trachea, bronchi, and their smaller branches, and the lungs, which contain the alveoli.

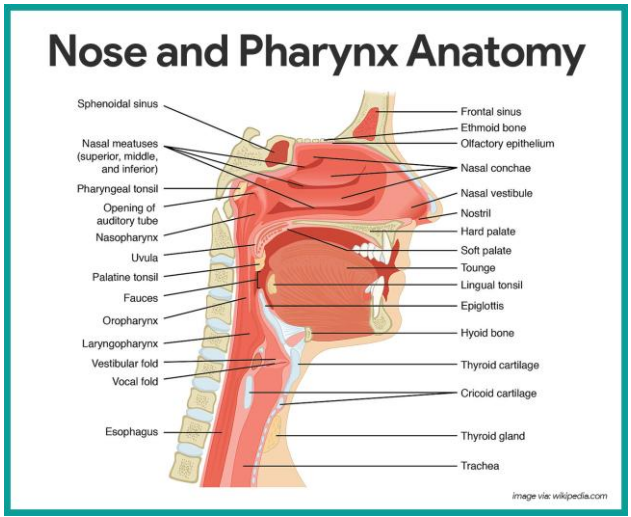
The Nose

The nose is the only externally visible part of the respiratory system.

- **Nostrils.** During breathing, air enters the nose by passing through the nostrils, or nares.
- **Nasal cavity.** The interior of the nose consists of the nasal cavity, divided by a midline **nasal septum**.
- **Olfactory receptors.** The olfactory receptors for the sense of smell are located in the mucosa in the slitlike superior part of the nasal cavity, just beneath the ethmoid bone.
- **Respiratory mucosa.** The rest of the mucosal lining, the nasal cavity called the respiratory mucosa, rests on a rich network of thin-walled veins that warms the air as it flows past.
- **Mucus.** In addition, the sticky mucus produced by the mucosa's glands moistens the air and traps incoming bacteria and other foreign debris, and **lysozyme enzymes** in the mucus destroy bacteria chemically.




- **Ciliated cells.** The ciliated cells of the nasal mucosa create a gentle current that moves the sheet of contaminated mucus posteriorly toward the throat, where it is swallowed and digested by stomach juices.
- **Conchae.** The lateral walls of the nasal cavity are uneven owing to three mucosa-covered projections, or lobes called conchae, which greatly increase the surface area of the mucosa exposed to the air, and also increase the air turbulence in the nasal cavity.
- **Palate.** The nasal cavity is separated from the oral cavity below by a partition, the palate; anteriorly, where the palate is supported by bone, is the **hard palate;** the unsupported posterior part is the **soft palate**.
- **Paranasal sinuses.** The nasal cavity is surrounded by a ring of paranasal sinuses located in the frontal, sphenoid, ethmoid, and maxillary bones; theses sinuses lighten the skull, and they act as a resonance chamber for speech.




**PHARYNX**

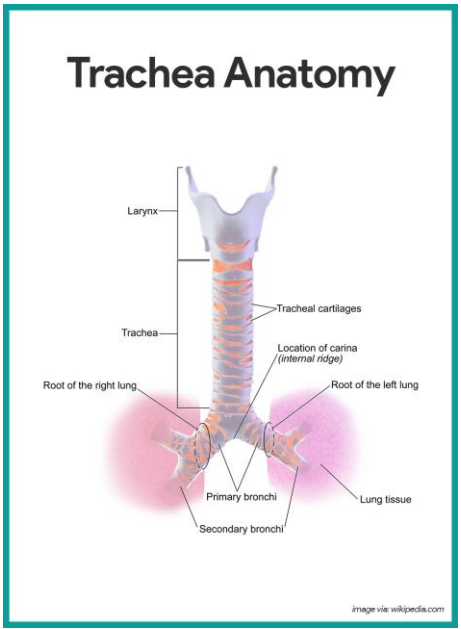
- **Size.** The pharynx is a muscular passageway about **13 cm (5 inches)** long that vaguely resembles a short length of red garden hose.
- **Function.** Commonly called the **throat**, the pharynx serves as a common passageway for food and air.
- **Portions of the pharynx.** Air enters the superior portion, the **nasopharynx**, from the nasal cavity and then descends through the **oropharynx** and **laryngopharynx** to enter the larynx below.
- **Pharyngotympanic tube.** The pharyngotympanic tubes, which drain the middle ear open into the nasopharynx.
- **Pharyngeal tonsil.** The pharyngeal tonsil, often called **adenoid** is located high in the nasopharynx.
- **Palatine tonsils.** The palatine tonsils are in the oropharynx at the end of the soft palate.
- **Lingual tonsils.** The lingual tonsils lie at the base of the tongue.

**LARYNX**

The larynx or **voice box** routes air and food into the proper channels and plays a role in speech.

- **Structure.** Located inferior to the pharynx, it is formed by eight rigid hyaline cartilages and a spoon-shaped flap of elastic cartilage, the **epiglottis**.
- **Thyroid cartilage.** The largest of the hyaline cartilages is the shield-shaped thyroid cartilage, which protrudes anteriorly and is commonly called **Adam's apple**.
- **Epiglottis.** Sometimes referred to as the **"guardian of the airways"**, the epiglottis protects the superior opening of the larynx.
- **Vocal folds.** Part of the mucous membrane of the larynx forms a pair of folds, called the vocal folds, or **true vocal cords**, which vibrate with expelled air and allows us to speak.
- **Glottis.** The slitlike passageway between the vocal folds is the glottis.

- **Length.** Air entering the trachea or **windpipe** from the larynx travels down its length (10 to 12 cm or about 4 inches) to the level of the **fifth thoracic vertebra**, which is approximately midchest.



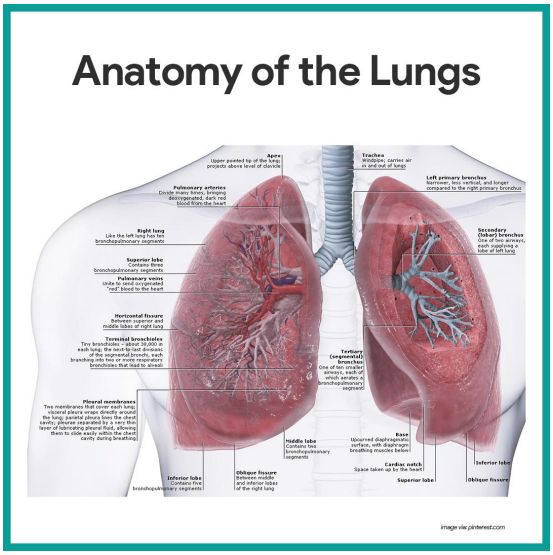

- **Structure.** The trachea is fairly rigid because its walls are reinforced with **C-shaped rings** of hyaline cartilage; the open parts of the rings abut the esophagus and allow it to expand anteriorly when we swallow a large piece of food, while the solid portions support the trachea walls and keep it patent, or open, in spite of the pressure changes that occur during breathing.
- **Cilia.** The trachea is lined with ciliated mucosa that beat continuously and in a direction opposite to that of the

incoming air as they propel mucus, loaded with dust particles and other debris away from the lungs to the throat, where it can be swallowed or spat out.

Main Bronchi

- **Structure.** The right and left main (primary) bronchi are formed by the division of the trachea.
- **Location.** Each main bronchus runs obliquely before it plunges into the medial depression of the lung on its own side.
- **Size.** The right main bronchus is wider, shorter, and straighter than the left.

**LUNGS**

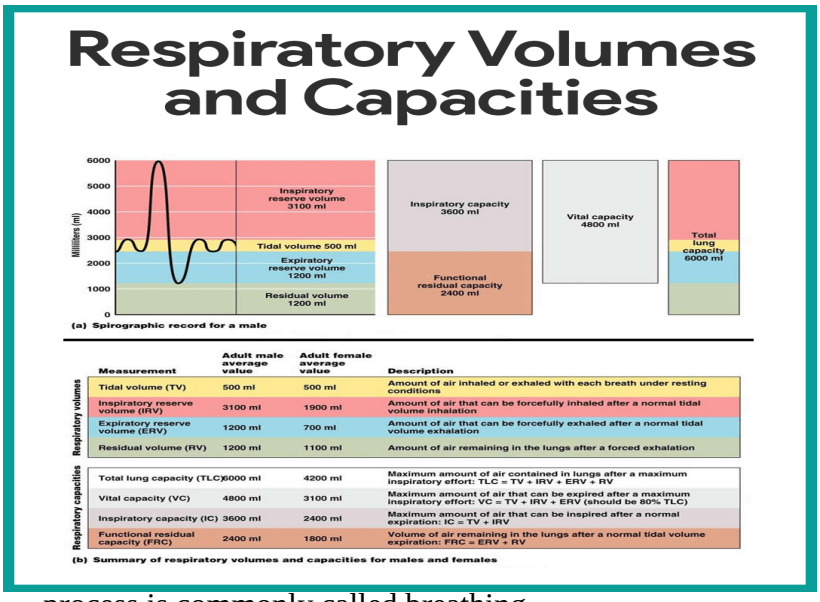



- **Location.** The lungs occupy the entire thoracic cavity except for the most central area, the **mediastinum**, which houses the heart, the great blood vessels, bronchi, esophagus, and other organs.
- **Apex.** The narrow, superior portion of each lung, the apex, is just deep into the clavicle.
- **Base.** The broad lung area resting on the diaphragm is the base.
- **Division.** Each lung is divided into lobes by fissures; the left lung has **two lobes**, and the right lung has **three**.
- **Pleura.** The surface of each lung is covered with a visceral serosa called the **pulmonary**, or **visceral pleura,** and the walls of the thoracic cavity are lined by the **parietal pleura**.
- **Pleural fluid.** The pleural membranes produce pleural fluid, a slippery serous secretion that allows the lungs to glide easily over the thorax wall during breathing movements and causes the two pleural layers to cling together.
- **Pleural space.** The lungs are held tightly to the thorax wall, and the pleural space is more of a potential space than an actual one.
- **Bronchioles.** The smallest of the conducting passageways are the bronchioles.
- **Alveoli.** The terminal bronchioles lead to the respiratory zone structures, even smaller conduits that eventually terminate in alveoli or air sacs.
- **Respiratory zone.** The respiratory zone, which includes the respiratory bronchioles, alveolar ducts, alveolar sacs, and alveoli, is the only site of gas exchange.
- **Conducting zone structures.** All other respiratory passages are conducting zone structures that serve as conduits to and from the respiratory zone.

- **Stroma.** The balance of the lung tissue, its stroma, is mainly elastic connective tissue that allows the lungs to recoil passively as we exhale.

The Respiratory Membrane

- **Wall structure.** The walls of the alveoli are composed largely of a single, thin layer of squamous epithelial cells.
- **Alveolar pores.** Alveolar pores connect neighboring air sacs and provide alternative routes for air to reach alveoli whose feeder bronchioles have been clogged by mucus or otherwise blocked.
- **Respiratory membrane.** Together, the alveolar and capillary walls, their fused basement membranes, and occasional elastic fibers construct the respiratory membrane (air-blood barrier), which has gas (air) flowing past on one side and blood flowing past on the other.
- **Alveolar macrophages.** Remarkably efficient alveolar macrophages sometimes called **"dust cells"**, wander in and out of the alveoli picking up bacteria, carbon particles, and other debris.
- **Cuboidal cells.** Also scattered amid the epithelial cells that form most of the alveolar walls are chunky cuboidal cells, which produce a lipid (fat) molecule called **surfactant**, which coats the gas-exposed alveolar surfaces and is very important in lung function.




**Physiology of the Respiratory System**

The major function of the respiratory system is to supply the body with oxygen and to dispose of carbon dioxide. To do this, at least four distinct events, collectively called respiration, must occur.

Respiration

- **Pulmonary ventilation.** Air must move into and out of the lungs so that gasses in the air sacs are continuously refreshed, and this process is commonly called breathing.
- **External respiration.** Gas exchange between the pulmonary blood and alveoli must take place.
- **Respiratory gas transport.** Oxygen and carbon dioxide must be transported to and from the lungs and tissue cells of the body via the bloodstream.

- **Internal respiration.** At systemic capillaries, gas exchanges must be made between the blood and tissue cells.

Mechanics of Breathing

- **Rule.** Volume changes lead to pressure changes, which lead to the flow of gasses to equalize pressure.
- **Inspiration.** Air is flowing into the lungs; the chest is expanded laterally, the rib cage is elevated, and the diaphragm is depressed and flattened; lungs are stretched to the larger thoracic volume, causing the intrapulmonary pressure to fall and air to flow into the lungs.
- **Expiration.** Air is leaving the lungs; the chest is depressed and the lateral dimension is reduced, the rib cage is descended, and the diaphragm is elevated and dome-shaped; lungs recoil to a smaller volume, intrapulmonary pressure rises, and air flows out of the lung.
- **Intrapulmonary volume.** Intrapulmonary volume is the volume within the lungs.
- **Intrapleural pressure.** The normal pressure within the pleural space, the intrapleural pressure, is always negative, and this is the major factor preventing the collapse of the lungs.
- **Nonrespiratory air movements.** Nonrespiratory movements are a result of reflex activity, but some may be produced voluntarily such as coughing, sneezing, crying, laughing, hiccups, and yawning.

**Respiratory Volumes and Capacities**

- **Tidal volume.** Normal quiet breathing moves approximately 500 ml of air into and out of the lungs with each breath.
- **Inspiratory reserve volume.** The amount of air that can be taken in forcibly over the tidal volume is the inspiratory reserve volume, which is normally between 2100 ml to 3200 ml.
- **Expiratory reserve volume.** The amount of air that can be forcibly exhaled after a tidal expiration, the expiratory reserve volume, is approximately 1200 ml.
- **Residual volume.** Even after the most strenuous expiration, about 1200 ml of air still remains in the lungs and it cannot be voluntarily expelled; this is called residual volume, and it is important because it allows gas exchange to go on continuously even between breaths and helps to keep the alveoli inflated.
- **Vital capacity.** The total amount of exchangeable air is typically around 4800 ml in healthy young men, and this respiratory capacity is the vital capacity, which is the sum of the tidal volume, inspiratory reserve volume, and expiratory reserve volume.
- **Dead space volume.** Much of the air that enters the respiratory tract remains in the conducting zone passageways and never reaches the alveoli; this is called the dead space volume and during a normal tidal breath, it amounts to about 150 ml.
- **Functional volume.** The functional volume, which is the air that actually reaches the respiratory zone and contributes to gas exchange, is about 350 ml.

- **Spirometer.** Respiratory capacities are measured with a spirometer, wherein as a person breathes, the volumes of air exhaled can be read on an indicator, which shows the changes in air volume inside the apparatus.

**Respiratory Sounds**

- **Bronchial sounds.** Bronchial sounds are produced by air rushing through the large respiratory passageways (trachea and bronchi).
- **Vesicular breathing sounds.** Vesicular breathing sounds occur as air fills the alveoli, and they are soft and resemble a muffled breeze.

**External Respiration, Gas Transport, and Internal Respiration**

- **External respiration.** External respiration or pulmonary gas exchange involves oxygen being loaded and carbon dioxide being unloaded from the blood.
- **Internal respiration.** In internal respiration or systemic capillary gas exchange, oxygen is unloaded and carbon dioxide is loaded into the blood.
- **Gas transport.** Oxygen is transported in the blood in two ways: most attaches to hemoglobin molecules inside the RBCs to form oxyhemoglobin, or a very small amount of oxygen is carried dissolved in the plasma; while carbon dioxide is transported in plasma as bicarbonate ion, or a smaller amount (between 20 to 30 percent of the transported carbon dioxide) is carried inside the RBCs bound to hemoglobin.

**CONTROL OF RESPIRATION**

***Neural Regulation***

- **Phrenic and intercostal nerves.** These two nerves regulate the activity of the respiratory muscles, the diaphragm, and external intercostals.
- **Medulla and pons.** Neural centers that control respiratory rhythm and depth are located mainly in the medulla and pons; the medulla, which sets the basic rhythm of breathing, contains a pacemaker, or self-exciting inspiratory center, and an expiratory center that inhibits the pacemaker in a rhythmic way; pons centers appear to smooth out the basic rhythm of inspiration and expiration set by the medulla.
- **Eupnea.** The normal respiratory rate is referred to as eupnea, and it is maintained at a rate of **12 to 15 respirations/minute**.
- **Hyperpnea.** During exercise, we breathe more vigorously and deeply because the brain centers send more impulses to the respiratory muscles, and this respiratory pattern is called hyperpnea.

***Non-neural Factors Influencing Respiratory Rate and Depth***

- **Physical factors.** Although the medulla's respiratory centers set the basic rhythm of breathing, there is no question that physical factors such as talking, coughing, and exercising can modify both the rate and depth of breathing, as well as an increased body temperature, which increases the rate of breathing.

- **Volition (conscious control).** Voluntary control of breathing is limited, and the respiratory centers will simply ignore messages from the cortex (our wishes) when the oxygen supply in the blood is getting low or blood pH is falling.
- **Emotional factors.** Emotional factors also modify the rate and depth of breathing through reflexes initiated by emotional stimuli acting through centers in the hypothalamus.
- **Chemical factors.** The most important factors that modify respiratory rate and depth are chemical- the levels of carbon dioxide and oxygen in the blood; increased levels of carbon dioxide and decreased blood pH are the most important stimuli leading to an increase in the rate and depth of breathing, while a decrease in oxygen levels become important stimuli when the levels are dangerously low.
- **Hyperventilation.** Hyperventilation blows off more carbon dioxide and decreases the amount of carbonic acid, which returns blood pH to the normal range when carbon dioxide or other sources of acids begin to accumulate in the blood.
- **Hypoventilation.** Hypoventilation or extremely slow or shallow breathing allows carbon dioxide to accumulate in the blood and brings blood pH back into normal range when blood starts to become slightly alkaline.

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# ANATOMY AND PHYSIOLOGY OF THE SKIN

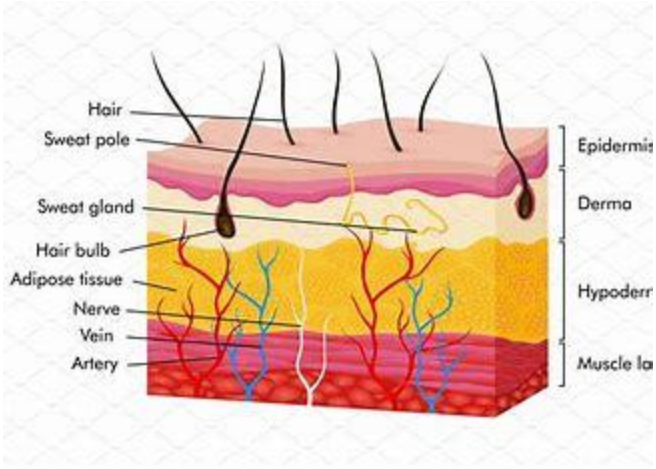
## INTRODUCTION

Skin diseases affect 20-33% of the UK population at any one time (All Parliamentary Group on Skin, 1997) and surveys suggest around 54% of the UK population will experience a skin condition in a given year (Schofield et al, 2009). Nurses will observe the skin daily while caring for patients and it is important they understand it so they can recognise problems when they arise. The skin and its appendages (nails, hair and certain glands) form the largest organ in the human body, with a surface area of 2m2 (Hughes, 2001). The skin comprises 15% of the total adult body weight; its thickness ranges from

### Structure of the Skin

The skin is divided into several layers, as shown in Fig 1. The epidermis is composed mainly of keratinocytes. Beneath the epidermis is the basement membrane (also known as the dermo-epidermal junction); this narrow, multilayered structure anchors the epidermis to the dermis. The layer below the dermis, the hypodermis, consists largely of fat. These structures are described below.

**Epidermis**




The epidermis is the outer layer of the skin, defined as a stratified squamous epithelium, primarily comprising keratinocytes in progressive stages of differentiation (Amirlak and Shahabi, 2017). Keratinocytes produce the protein keratin and are the major building blocks (cells) of the epidermis.

As the epidermis is avascular (contains no blood vessels), it is entirely dependent on the underlying dermis for nutrient delivery and waste disposal through the basement membrane. The prime function of the epidermis is to act as a physical and biological barrier to the external environment, preventing penetration by irritants and allergens.

At the same time, it prevents the loss of water and maintains internal homeostasis (Gawkrodger, 2007; Cork, 1997). The epidermis is composed of layers; most body parts have four layers, but those with the thickest skin have five.

**The layers are:**

- Stratum corneum (horny layer);
- Stratum lucidum (only found in thick skin – that is, the palms of the hands, the soles of the feet and the digits);
- Stratum granulosum (granular layer);
- Stratum spinosum (prickle cell layer);
- Stratum basale (germinative layer).

The epidermis also contains other cell structures. Keratinocytes make up around 95% of the epidermal cell population – the others being melanocytes, Langerhans cells and Merkel cells (White and Butcher, 2005). Keratinocytes. Keratinocytes are formed by division in the stratum basale. As they move up through the stratum spinosum and stratum granulosum, they differentiate to form a rigid internal structure.

The skin is the largest organ of the human body, serving as a protective barrier between the internal body and the external environment. It plays a crucial role in various physiological processes. Here's an overview of the anatomy and physiology of the skin:

**Anatomy of the Skin**

1. **Epidermis**:
    - **Stratum Corneum**: Outermost layer, composed of dead, flattened keratinocytes that form a tough, protective layer.
    - **Stratum Lucidum**: A thin, clear layer found only in the thick skin of the palms and soles.

- **Stratum Granulosum**: Contains keratinocytes that are undergoing a process called keratinization, where they lose their nuclei and organelles and become more rigid.
- **Stratum Spinosum**: A layer where keratinocytes are connected by desmosomes, giving the cells a spiny appearance.
- **Stratum Basale**: The deepest layer, where new keratinocytes are produced through mitosis. It also contains melanocytes, which produce melanin, the pigment responsible for skin color.

2. **Dermis**:
   - **Papillary Layer**: The upper portion of the dermis that contains capillaries, lymph vessels, and sensory neurons. It forms the dermal papillae, which interlock with the epidermis.
   - **Reticular Layer**: The thicker, deeper layer of the dermis that contains dense irregular connective tissue, collagen, and elastin fibers. It houses hair follicles, sweat glands, sebaceous glands, blood vessels, and nerves.
3. **Hypodermis (Subcutaneous Layer)**:
   - Consists of loose connective tissue and fat (adipose tissue). It provides insulation, energy storage, and cushioning to protect underlying muscles and bones. The hypodermis also connects the skin to underlying tissues.

**Physiology of the Skin**

1. **Protection**:
   - The skin acts as a physical barrier against mechanical injuries, pathogens, and harmful substances. The stratum corneum, with its layer of dead keratinized cells, provides a tough shield, while the acidic pH of the skin surface inhibits microbial growth.
2. **Regulation of Body Temperature**:
   - The skin helps regulate body temperature through the process of sweating and the dilation or constriction of blood vessels. Sweat glands release sweat, which cools the body as it evaporates, while blood vessels in the dermis can constrict to reduce heat loss or dilate to increase heat dissipation.
3. **Sensation**:
   - The skin contains a variety of sensory receptors that detect touch, pressure, pain, temperature, and vibration. These receptors allow the skin to communicate information about the external environment to the brain.
4. **Excretion**:
   - The skin plays a minor role in excretion by eliminating waste products such as urea, salts, and water through sweat.

5. **Synthesis of Vitamin D**:
    - When exposed to UV radiation from sunlight, the skin synthesizes vitamin D, which is essential for calcium absorption and bone health.
6. **Immune Defense**:
    - The skin contains specialized cells like Langerhans cells in the epidermis, which play a role in immune responses by recognizing and processing foreign antigens.
7. **Storage of Lipids and Water**:
    - The hypodermis stores fat that can be used as an energy reserve and provides insulation. The skin also serves as a reservoir for water, helping to maintain fluid balance.

This comprehensive understanding of the skin's anatomy and physiology highlights its critical role in maintaining overall health and homeostasis.

**REFERENCE:**

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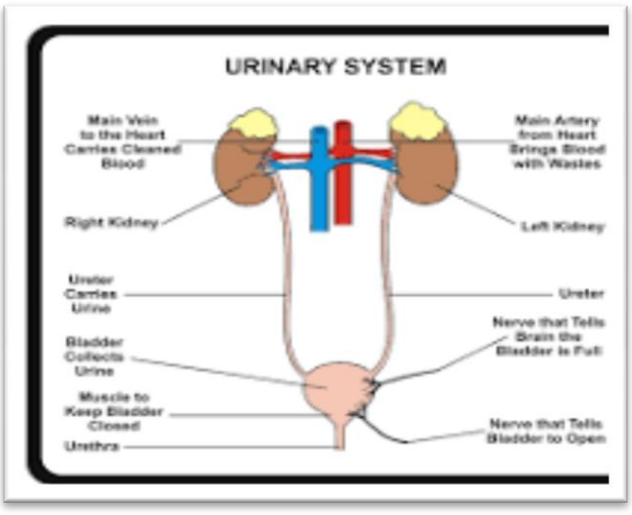
# THE RENAL SYSTEM

The renal system, also known as the urinary system, plays a critical role in maintaining the body's internal environment by regulating the balance of water, electrolytes, and the elimination of waste products. It consists of several key structures:

**1. Kidneys:**

- **Location**: Two bean-shaped organs located on either side of the spine, just below the ribcage.
- **Function**: The kidneys filter blood to remove waste products and excess substances, forming urine. They also regulate blood pressure, electrolyte balance, and red blood cell production through the hormone erythropoietin.




**2. Ureters:**

- **Description**: Thin, muscular tubes that transport urine from each kidney to the bladder.

- **Function**: Ureters propel urine from the kidneys to the bladder using peristaltic movements.

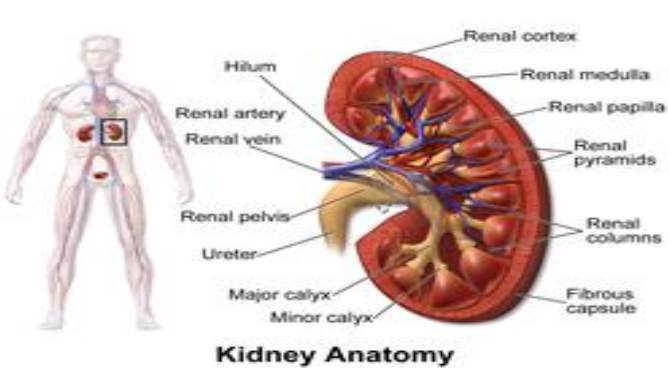
**3. Bladder:**

- **Description**: A hollow, muscular organ located in the pelvis.
- **Function**: The bladder stores urine until it is ready to be excreted. The bladder can expand and contract, holding urine until voluntary urination occurs.

**4. Urethra:**

- **Description**: A tube that leads from the bladder to the outside of the body.
- **Function**: The urethra is responsible for expelling urine from the body during urination. In males, the urethra also carries semen during ejaculation.

**Functions of the Renal System:**



Kidney Anatomy

- **Filtration**: The kidneys filter blood, removing waste products like urea, creatinine, and excess ions, which are then excreted as urine.
- **Reabsorption**:Essential substances like glucose, certain ions, and water are reabsorbed back into the bloodstream, ensuring the body retains necessary nutrients and fluids.
- **Secretion**: The renal system actively secretes certain substances, such as hydrogen ions and drugs, into the urine, helping maintain pH balance and detoxify the body.
- **Excretion**: The final urine, containing waste products and excess substances, is excreted from the body.

**Regulation:**

- The renal system helps regulate blood pressure through the renin-angiotensin-aldosterone system (RAAS), and it plays a role in maintaining acid-base balance, electrolyte levels, and fluid volume.

In summary, the renal system is essential for filtering blood, maintaining homeostasis, and eliminating waste, making it vital for overall health.

The kidneys are highly specialized organs responsible for filtering blood and maintaining the body's internal balance. Here's how they work:

**1. Blood Filtration:**

- **Glomerulus**: Each kidney contains about a million tiny filtering units called nephrons. Each nephron has a glomerulus, a small bundle of capillaries where blood is filtered. The glomerulus acts as a sieve, allowing water, salts, glucose, and waste products to pass through while retaining larger molecules like proteins and blood cells.
- **Bowman's Capsule**: Surrounding the glomerulus is the Bowman's capsule, which collects the filtered fluid, known as filtrate. This filtrate contains waste products along with useful substances like water, glucose, and electrolytes.

**2. Reabsorption:**

- After filtration, the filtrate moves into the **renal tubule**, where most of the water, glucose, and essential ions (like sodium, potassium, and calcium) are reabsorbed back into the bloodstream. This occurs mainly in the **proximal convoluted tubule** and the **loop of Henle**.
- The kidneys reabsorb nearly all of the water and nutrients your body needs, allowing waste products and excess substances to be concentrated in the urine.

**3. Secretion:**

- The renal tubule also actively secretes certain substances, like hydrogen ions, potassium, and certain drugs, into the filtrate. This process helps maintain the body's acid-base balance and ensures the elimination of unwanted substances.

**4. Concentration of Urine:**

- As the filtrate passes through the **distal convoluted tubule** and the **collecting duct**, the kidneys adjust the concentration of urine based on the body's needs. If the body needs to conserve water, more water is reabsorbed, and the urine becomes more concentrated. Conversely, if there is excess water in the body, less water is reabsorbed, resulting in more dilute urine.
- The hormone **antidiuretic hormone (ADH)** plays a key role in this process by increasing water reabsorption in the collecting ducts.

**5. Excretion:**

- The final product, now called urine, is collected in the **renal pelvis** of each kidney and transported through the **ureters** to the **bladder**. Urine is stored in the bladder until it is excreted from the body through the **urethra**.

**Regulation:**

- **Blood Pressure**: The kidneys help regulate blood pressure through the renin-angiotensin-aldosterone system (RAAS). When blood pressure is low, the kidneys release renin, which triggers a cascade of events leading to the retention of sodium and water, increasing blood volume and pressure.
- **Red Blood Cell Production**: The kidneys produce erythropoietin, a hormone that stimulates the production of red blood cells in the bone marrow, in response to low oxygen levels in the blood.

**Waste Products Removed by the Kidneys:**

- **Urea**: Produced from the breakdown of proteins.
- **Creatinine**: A waste product from muscle metabolism.
- **Uric Acid**: Produced from the breakdown of nucleotides (building blocks of DNA and RNA).
- **Excess Ions**: Such as sodium, potassium, and calcium, depending on the body's needs.

**Key Points:**

- The kidneys filter around 120-150 liters of blood daily, producing about 1-2 liters of urine.
- They are vital in maintaining the body's fluid balance, electrolyte levels, and overall homeostasis.

By filtering blood, reabsorbing necessary substances, and excreting waste, the kidneys play a crucial role in keeping the body's internal environment stable and healthy.

**REFERENCE:**

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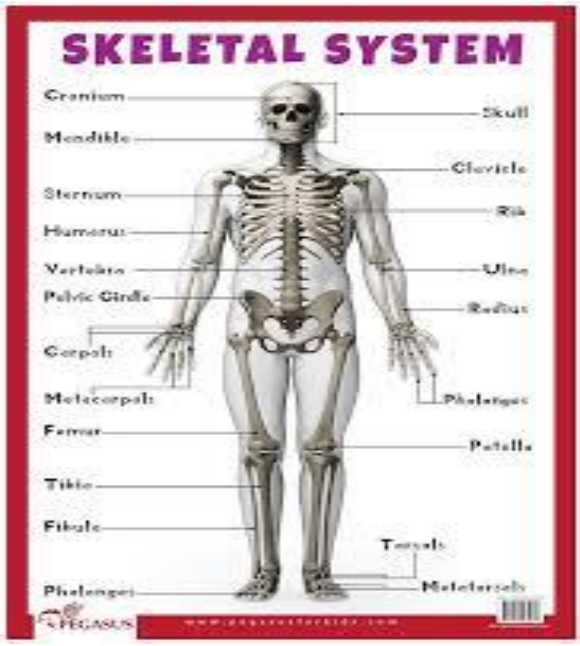
# SKELETAL SYSTEM

### Skeletal System Anatomy and Physiology

### 1. Overview of the Skeletal System

The skeletal system consists of bones, cartilage, and ligaments. It forms the structural framework of the body, supports and protects vital organs, enables movement, and houses bone marrow for blood cell production.

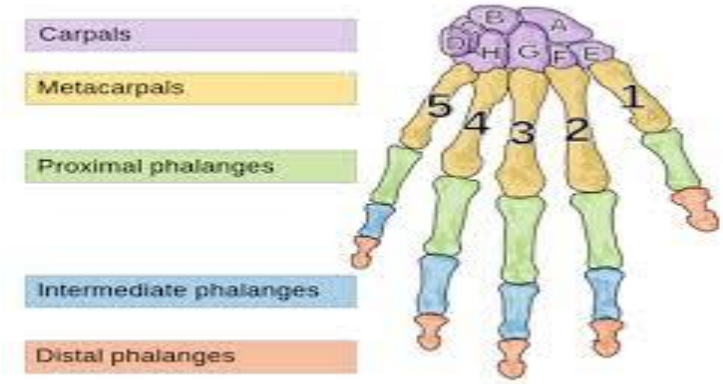
### 2. Anatomy of the Skeletal System



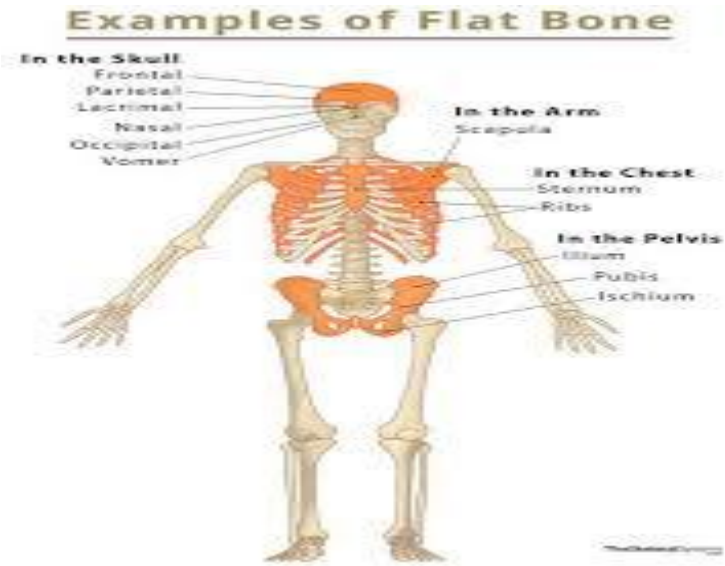

## A. Bone Types and Structure

1. **Bone Types**
   - **Long Bones:** Longer than they are wide, e.g., femur, tibia, humerus. They have a central shaft (diaphysis) and two ends (epiphyses).


   - **Short Bones:** Approximately equal in length and width, e.g., carpals (wrist bones), tarsals (ankle bones).




- **Flat Bones:** Thin and flat, e.g., skull bones, ribs, sternum. They provide broad surfaces for muscle attachment and protection.




- **Irregular Bones:** Complex shapes that don't fit into other categories, e.g., vertebrae, certain facial bones.

- **Sesamoid Bones:** Embedded within tendons, e.g., patella (kneecap). They protect tendons from stress and wear.


2. **Bone Structure**
   - **Compact Bone:** Dense and forms the outer layer of bones. It is composed of osteons (Haversian systems) that provide strength.
   - **Spongy Bone (Cancellous Bone):** Found inside bones, especially at the ends of long bones. It has a porous, lattice-like structure that is lightweight and provides space for bone marrow.
   - **Bone Marrow:** Soft tissue inside the bone. **Red marrow** produces blood cells, while **yellow marrow** stores fat.
3. **Bone Cells**
   - **Osteoblasts:** Cells that build new bone by producing the bone matrix.
   - **Osteocytes:** Mature bone cells that maintain bone tissue.
   - **Osteoclasts:** Cells that break down bone tissue, involved in bone resorption.
4. **Bone Tissue Composition**
   - **Organic Matrix:** Contains collagen fibers and ground substance, providing flexibility and tensile strength.
   - **Inorganic Matrix:** Consists of mineral salts, mainly calcium phosphate, providing rigidity and hardness.

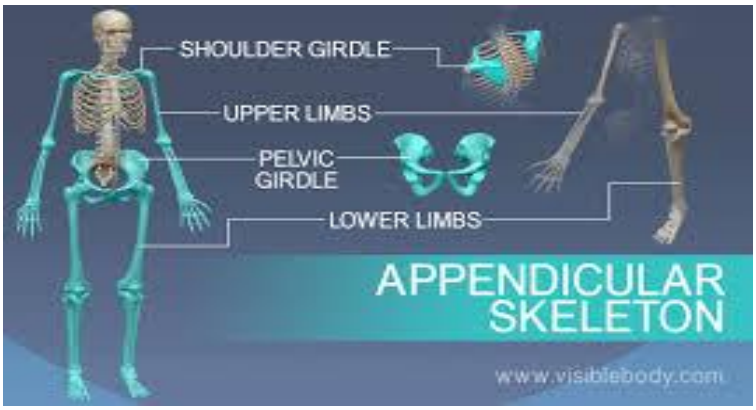
**B. Major Bones and Bone Groups**

1. **Axial Skeleton**
   - **Skull:**
     - **Cranium:** Protects the brain. Composed of the frontal, parietal, temporal, occipital, sphenoid, and ethmoid bones.
     - **Facial Bones:** Includes the maxilla, mandible, nasal bones, zygomatic bones, and others.
   - **Vertebral Column (Spine):**
     - **Cervical Vertebrae:** 7 vertebrae in the neck region.
     - **Thoracic Vertebrae:** 12 vertebrae in the chest region, each articulating with a rib.
     - **Lumbar Vertebrae:** 5 vertebrae in the lower back.
     - **Sacrum:** 5 fused vertebrae forming the back of the pelvis.

- **Coccyx:** 4 fused vertebrae (tailbone).

- **Thoracic Cage:**
  - **Ribs:** 12 pairs of ribs that protect the chest cavity.
  - **Sternum:** Breastbone in the center of the chest.


2. **Appendicular Skeleton**
   - **Shoulder Girdle:**
     - **Clavicle:** Collarbone.
     - **Scapula:** Shoulder blade.
   - **Upper Limbs:**
     - **Humerus:** Upper arm bone.
     - **Radius and Ulna:** Forearm bones.
     - **Carpals:** Wrist bones.
     - **Metacarpals:** Bones of the hand.
     - **Phalanges:** Finger bones.
   - **Pelvic Girdle:**
     - **Ilium, Ischium, Pubis:** Hip bones.
   - **Lower Limbs:**
     - **Femur:** Thigh bone.
     - **Patella:** Kneecap.
     - **Tibia and Fibula:** Lower leg bones.
     - **Tarsals:** Ankle bones.
     - **Metatarsals:** Bones of the foot.
     - **Phalanges:** Toe bones.




## 3. Physiology of the Skeletal System

### A. Bone Development and Growth

1. **Ossification:** The process by which bone tissue forms. It occurs in two ways:
   - **Intramembranous Ossification:** Formation of bone directly from mesenchymal tissue, primarily in flat bones of the skull.
   - **Endochondral Ossification:** Formation of bone from hyaline cartilage, typical of long bones.
2. **Bone Growth:** Occurs at the epiphyseal plates (growth plates) during childhood and adolescence, where new bone is added. This process stops when the plates ossify and the bones reach their full length.
3. **Bone Remodeling:** Ongoing process where old bone is replaced by new bone. It is essential for maintaining bone strength and mineral homeostasis. This involves the balanced activity of osteoblasts and osteoclasts.

### B. Bone Functions

1. **Support:** Provides a rigid framework that supports the body's weight and posture.
2. **Protection:** Shields internal organs from injury (e.g., the skull protects the brain, the rib cage protects the heart and lungs).
3. **Movement:** Acts as levers to which muscles attach. Muscle contractions pull on bones to produce movement.
4. **Mineral Storage:** Bones store calcium and phosphorus, which can be released into the bloodstream to maintain mineral balance.
5. **Blood Cell Production:** The red marrow within bones produces red blood cells, white blood cells, and platelets.
6. **Energy Storage:** Yellow marrow stores lipids which can be used as an energy reserve.

### C. Joint Function and Types

1. **Joints (Articulations):** Connections between bones that allow movement. They are classified based on their structure and function:
   - **Fibrous Joints:** Bones are connected by fibrous tissue, allowing little to no movement (e.g., sutures of the skull).

- **Cartilaginous Joints:** Bones are connected by cartilage, allowing limited movement (e.g., intervertebral discs).
- **Synovial Joints:** Most common and movable type of joint, characterized by a fluid-filled joint capsule (e.g., knee, shoulder). They allow for a range of movements:
  - **Hinge Joints:** Allow movement in one plane (e.g., elbow, knee).
  - **Ball-and-Socket Joints:** Allow for rotational movement and multiple axes (e.g., shoulder, hip).
  - **Pivot Joints:** Allow rotation around a single axis (e.g., atlantoaxial joint in the neck).
  - **Saddle Joints:** Allow movement in two planes (e.g., thumb joint).
  - **Gliding Joints:** Allow bones to glide past each other (e.g., wrist joints).

**D. Bone Health and Repair**

1. **Bone Healing:** Involves several stages:
   - **Inflammatory Phase:** Blood clot forms at the fracture site, followed by inflammation.
   - **Repair Phase:** Formation of a soft callus followed by a hard callus as new bone is laid down.
   - **Remodeling Phase:** The hard callus is remodeled to form new bone, restoring normal bone structure.
2. **Bone Diseases and Disorders:**
   - **Osteoporosis:** A condition characterized by weakened bones due to decreased bone density.
   - **Osteoarthritis:** Degenerative joint disease affecting cartilage.
   - **Rickets/Osteomalacia:** Softening of bones due to vitamin D deficiency.

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# ANATOMY OF THE ENDOCRINE SYSTEM

**1. Hypothalamus**

- **Location**: Situated in the brain, just below the thalamus and above the pituitary gland.
- **Anatomy**: A small region of the brain involved in maintaining homeostasis.
- **Function**: Controls the pituitary gland through the release of regulatory hormones (releasing and inhibiting hormones).

**2. Pituitary Gland**

- **Location**: Found at the base of the brain, within the sella turcica of the sphenoid bone.

- **Anatomy**: Divided into two lobes:
    - **Anterior Pituitary**: Larger, glandular tissue. Produces hormones that regulate other endocrine glands.
    - **Posterior Pituitary**: Composed of nerve endings from the hypothalamus. Stores and releases hormones.

**3. Thyroid Gland**

- **Location**: Located in the anterior neck, just below the larynx.
- **Anatomy**: Butterfly-shaped with two lobes connected by an isthmus.
- **Function**: Secretes thyroid hormones (T4 and T3) that influence metabolic rate, and calcitonin, which helps control calcium levels.

**4. Parathyroid Glands**

- **Location**: Four small glands located on the posterior surface of the thyroid gland.
- **Anatomy**: Small, oval-shaped glands.
- **Function**: Produce parathyroid hormone (PTH), which regulates calcium and phosphate levels in the blood.

**5. Adrenal Glands**

- **Location**: Situated on top of each kidney.
- **Anatomy**: Composed of two parts:
    - **Adrenal Cortex**: The outer layer, which produces corticosteroids, including cortisol (regulates metabolism and stress response), aldosterone (regulates sodium and potassium balance), and sex hormones.
    - **Adrenal Medulla**: The inner part, which produces catecholamines such as adrenaline (epinephrine) and noradrenaline (norepinephrine), involved in the fight-or-flight response.

**6. Pancreas**

- **Location**: Located in the abdomen, behind the stomach.
- **Anatomy**: Has both endocrine and exocrine functions. The endocrine part consists of the islets of Langerhans.
- **Function**: Produces insulin and glucagon (regulate blood glucose levels) and digestive enzymes.

**7. Gonads (Ovaries and Testes)**

- **Location**: Ovaries are located in the pelvic cavity, while testes are located in the scrotum.
- **Anatomy**:
  - **Ovaries**: Produce estrogen and progesterone.
  - **Testes**: Produce testosterone.
- **Function**: Regulate reproductive functions and secondary sexual characteristics.

**8. Pineal Gland**

- **Location**: Located in the brain, near the center, between the two hemispheres.
- **Anatomy**: Small, pea-shaped gland.
- **Function**: Produces melatonin, which regulates sleep-wake cycles and seasonal biological rhythms.

**9. Thymus**

- **Location**: Located in the upper chest, behind the sternum.
- **Anatomy**: A gland that shrinks with age.
- **Function**: Produces thymosin, which is crucial for the development of T lymphocytes (T cells) in the immune system.

**Physiology of the Endocrine System**

**Hormone Function**

- **Hormones**: Chemical messengers secreted into the bloodstream by endocrine glands.
- **Regulation**: Hormones regulate processes such as metabolism, growth, development, mood, and reproductive functions.
- **Types of Hormones**:
  - **Peptide Hormones**: Water-soluble, include insulin and growth hormone.

- **Steroid Hormones**: Lipid-soluble, derived from cholesterol, include cortisol and sex hormones.
- **Amino Acid Derivatives**: Include thyroid hormones and catecholamines like adrenaline.

**Hormone Secretion**

- **Feedback Mechanisms**: Hormone levels are regulated by feedback loops, which can be:
  - **Negative Feedback**: Most common; reduces the output of the endocrine gland to maintain homeostasis (e.g., thyroid hormones regulating thyroid-stimulating hormone [TSH] release).
  - **Positive Feedback**: Amplifies a process, such as oxytocin during childbirth.

**Hormone Transport and Action**

- **Transport**: Hormones travel through the bloodstream to target organs or tissues.
- **Action**: Hormones bind to specific receptors on target cells, triggering a response. This can involve changes in gene expression, enzyme activity, or cell function.

**Integration with Nervous System**

- **Hypothalamus-Pituitary Axis**: The hypothalamus controls the pituitary gland, which in turn regulates other endocrine glands.
- **Stress Response**: The adrenal medulla and cortex release hormones like adrenaline and cortisol to manage stress.

**Endocrine Disorders**

- **Examples**:
  - **Diabetes Mellitus**: Caused by issues with insulin production or action.
  - **Thyroid Disorders**: Such as hypothyroidism or hyperthyroidism.
  - **Adrenal Insufficiency**: Conditions like Addison's disease affect adrenal hormone production.

Understanding the endocrine system's anatomy and physiology helps in diagnosing and treating various hormonal imbalances and disorders, ensuring proper bodily function and health.

Reference:

Ross and Wilson: anatomy and Physiology

www.google.com

# SWARRNIM START UP AND INNOVATION UNIVERSITY

# AARIHANT INSTITUTE OF NURSING

COURSE NAME : CHILD HEALTH NURSING

# INTRA UTERINE GROWTH RESTRICTION (IUGR)

## INTRODUCTION

Intrauterine growth restriction (IUGR) is a common diagnosis in obstetrics and carries an increased risk of perinatal mortality and morbidity.

**DEFINITION:** Intrauterine growth restriction refers to a fetus whose weight is below the 10th percentile of the average for its gestational age.

IUGR is defined as failure of the fetus to achieve its genetic growth potential.

**INCIDENCE:** It comprises of 1/3rd of low birth weight babies. Among term babies it is 5% and preterm babies 15%.

**ETIOLOGY:**

**Maternal:**

- **Constitutiona**l: Small women, maternal genetic and racial background are associated with small babies.
- **Poor maternal nutrition** before and during the pregnancy
- **Maternal diseases**: Heart disease, preeclampsia or eclampsia, anemia, chronic renal disease etc.
- **Toxins:** Alcohol abuse, drug addiction, smoking

**Fetal:** there is enough substrate in maternal blood and also crosses the placenta but is not utilized by the fetus.The failure of non utilization may be due to**:**

- **Structural anomalies**: cardiovascular, renal or others
- **Chromosomal abnormality:** Turner's syndrome ,trisomies (13.18,21)
- **Infection:** TORCH agents, malaria
- **Multiple pregnancies:** there is mechanical hindrance to growth and excessive fetal demand.

**Placental:**

- **Poor uterine blood flow** to the placenta for a long time.
- **Placental pathology**: Placenta praevia , Abruption, Infarction, Circumvallete and Mosaichism.

**TYPES**: based on the clinical evaluation and ultrasound examination the small fetuses are divided into:

1. Fetuses that are small and healthy, normal pondrel index and subcutaneous fat and usually have an uneventful neonatal course.
2. Fetuses where growth is restricted by pathological process (true IUGR). Depending upon the relative size of their head, abdomen and femur, the fetuses are subdivided into:

**Symmetrical (20%)**: The fetus is affected from the noxious effect very early in the phase of cellular hyperplasia. The pathologic process is intrinsic to the fetus and involves all the organs including the head.

**Asymmetrical (80%)**: the fetus is affected in the later months during the phase of cellular hypertrophy. The total cell number remains the same but size is smaller than normal.

**Features of symmetrical and asymmetrical IUGR**

| Symmetrical | Asymmetrical |
|---|---|
| Uniformally small | Head larger than abdomen |
| Pondrel index (birth weight/crown-heel3) | low |
| HC:AC and FL:AC ratios normal | elevated |
| Total cell number- less, cell size normal | Normal cell size smaller |
| Neonatal course complicated with poor prognosis | Usually un complicated with good prognosis. |

**PATHOPHYSIOLOGY**

Due to reduced availability of the nutrients in the mother or its reduced transfer by the placenta to the fetus and also due to reduced utilization by the fetus brain cell size (asymmetric) as well as cell numbers (symmetric) are reduced. Liver glycogen is reduced. There is oligohydramnios as the renal and pulmonary contributions to amniotic fluid are diminished due to reduced blood flow to these organs. The fetus is at risk for intrauterine hypoxia and acidosis leading to intrauterine death.

**DIAGNOSIS**

**Clinical**

- Symphysis fundal height less than expected for gestational age after 24 weeks. A lag of 4 cm or more suggest growth restriction.
- Maternal weight gain remains stationary or at times falling during the second half of pregnancy.

**Ultrasound Biometry**

- Ultrasound biometry of the fetus is now the gold standard for assessing fetal growth. The measurements most commonly used are the **biparietal diameter, head circumference, abdominal circumference** and **femur length**. Percentiles have been established for each of these parameters, and fetal weight can be calculated. The most sensitive indicator of symmetric and asymmetric IUGR is the abdominal circumference, which has a sensitivity of over 95 percent if the measurement is

below the 2.5th percentile. Accurate dating of the pregnancy is essential in the use of any parameter. In the absence of reliable dating, serial scans at two- or three-week intervals must be performed to identify IUGR. It should always be remembered that each parameter measured has an error potential of about one week up to 20 gestational weeks, about two weeks from 20 to 36 weeks of gestation, and about three weeks thereafter.

- **Head circumference to the abdominal circumference (HC/AC) ratio**. Between 20 and 36 weeks of gestation, the HC/AC ratio normally drops almost linearly from 1.2 to 1.0. The ratio is normal in the fetus with symmetric growth restriction and elevated in the infant with asymmetric growth restriction.
- **Amniotic fluid volume:** a vertical pocket of amniotic fluid <2cm suggest IUGR
- **Femur length(FL):** it is not affected in asymmetric IUGR. The FL/AC ratio is 22 at all gestational ages from 21 weeks to term. FL/AC ratio > 23.5 suggest IUGR.

**Doppler velocimetry:** elevated uterine artery systolic/ diastolic(S/D) ratio(>2.6) and or presence of diastolic notch are associated with IUGR. The presence of diastolic notch in uterine artery suggests incomplete invasion of placental trophoblasts to the uterine spiral arteries.

**Three-dimensionalUltrasonography**

**Pondrel index PI**: it is determined by dividing the estimated fetal weight by the third power of crown- heel length( weight/length$^3$. PI below 10$^{th}$ percentile is taken as IUGR

**Middle cerebral artery Doppler**

**Biochemical markers**: erythropoietin level in cord blood is high in fetuses with IUGR.

**PHYSICAL FEATURES of IUGR BABY AT BIRTH**

- Weight deficit at birth is about 600gm below the minimum in percentile standard. Length is unaffected.
- HC is relatively larger than body in asymmetric variety.
- Physical features show dry and wrinkled skin because of less subcutaneous fat, scaphoid abdomen, thin meconium stained vernix caseosaand thin umbilical cord.**Old Man Look** for the baby.
- Baby is alert active and has normal cry. Reflexes are normal.

**COMPLICATIONS**

**Fetal**

a. **Antenatal :**chronic fetal distress, fetal death
b. **Intranatal :**hypoxia and acidosis
c. **After birth**
   **Immediate:**asphyxia and RDS
   Hypoglycemia ,Meconium Aspiration Syndrome, Hyperviscosity Syndrome, Necrotizing Enterocolitis
   Microcoagulation leading to DIC during 1$^{st}$ day of life.
   Hypothermia, Pulmonary Hemorrhage, Polycythemia, Intraventricular Hemorrhage.

**Late:**symmetrical IUGR baby is likely to grow slowly after birth. Asymmetrical IUGR baby tend to catch up growth in early infancy. The fetuses having retardation of growth evidenced before third trimester are likely to have retarded neurological and intellectual development in infancy.

**MANAGEMENT**

IUGR increases the risk for intrauterine death. If this condition is suspected, the pregnant woman will be closely monitored with several pregnancy ultrasounds to measure the baby's growth, movements, blood flow, and fluid around the baby. Non-stress testing will also be done. Depending on the results of these tests, delivery may be necessary.

**Management protocol of IUGR**

- To confirm IUGR and the type
- To exclude congenital malformation and genetic disorder
- To treat the specific cause if found
- Fetal surveillance
    - DFMC
    - NST, Cardiotopography
    - Biophysical profile
    - Umbilical artery Doppler Flow velocimetry

Pregnancy < 37 weeks

Pregnancyequalling or more than 37 weeks

Termination

Severe IUGR

Mild IUGR

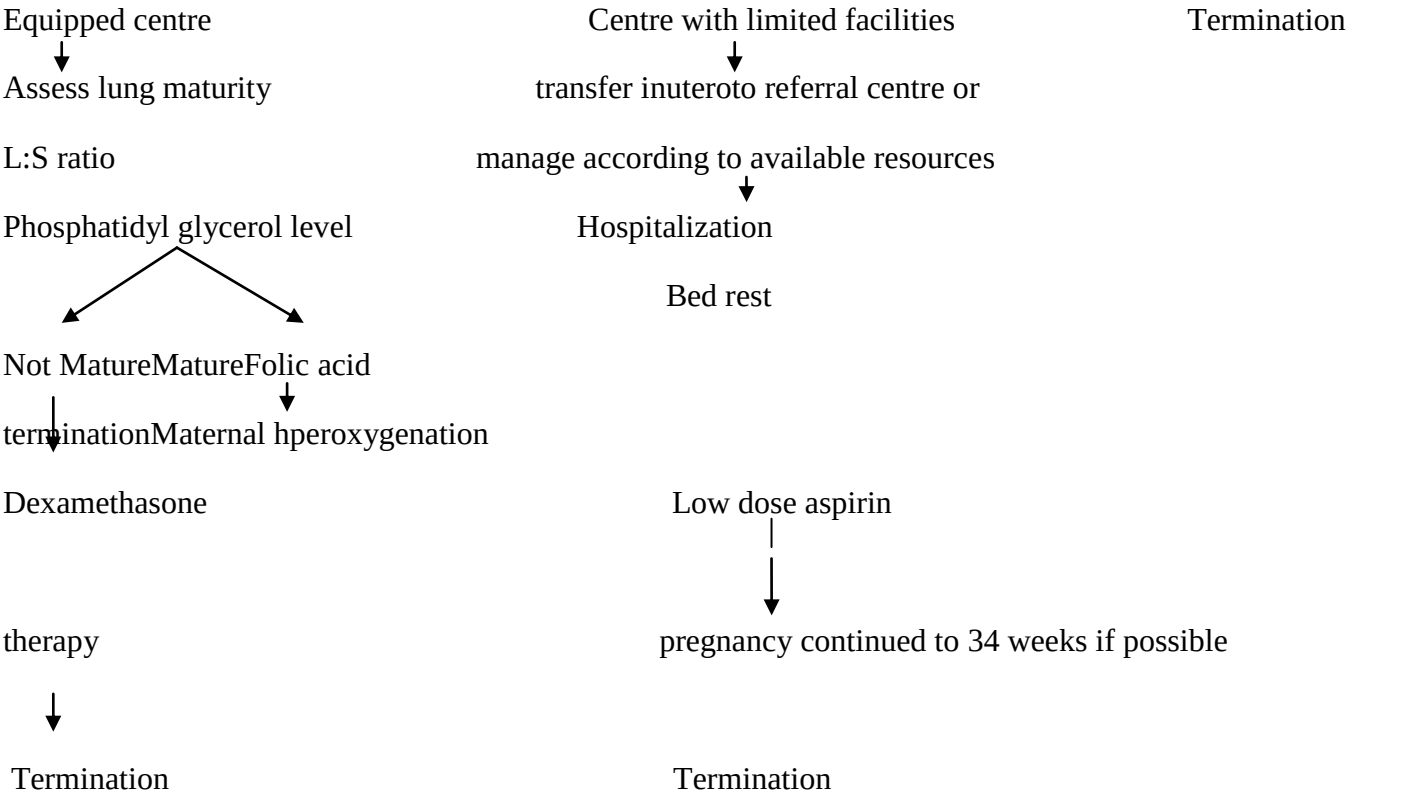
Dual problem

Prematurity & Dysmaturity Increased rest

Folic acid

weeks

Fetal monitoring till 37




**Methods of termination**

- Low rupture of membranes followed by oxytocin if pregnancy beyond 34 weeks with favourable cervix and head deep in the pelvis.$PGE_2$ gel can be used if cervix is unfavourable.
- Intrapartum monitoring by clinical, continuos electronic and scalp blood sampling may be needed as risk for intrapartum asphyxia is high.
- Caesarean section

**CONCLUSION**

IUGR remains a challenging problem for clinicians. Most cases of IUGR occur in pregnancies in which no risk factors are present; therefore, the clinician must be alert to the possibility of a growth disturbance in all pregnancies. No single measurement helps secure the diagnosis; thus, a complex strategy for diagnosis and assessment is necessary. The ability to diagnose the disorder and understand its pathophysiology still outpaces the ability to prevent or treat its complications. The current therapeutic goals are to optimize the timing of delivery to minimize hypoxemia and maximize gestational age and maternal outcome.

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# CHAPTER NAME: - LOW BIRTH WEIGHT BABIES (LBW)

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The World Health Organization (WHO) defines low birth weight (LBW) as a birth weight of less than 2,500 grams (5.5 pounds). LBW can be caused by premature birth, fetal growth restriction, or both. Risk factors for LBW include preterm labor, chronic health conditions, infections, problems with the placenta, and not gaining enough weight during pregnancy.

## ➢ Objective of low birth weight baby.

- In modern times, improvement in knowledge and technology has greatly influenced the health of children. However, past decade was marked by limited progress in reducing infant mortality largely due to a failure in reduction of neonatal mortality.
- There is widely shared but mistaken idea that improvement in newborn health requires advanced technologies and highly specialized staff. The reality is that many conditions that result in perinatal death can be prevented or treated without sophisticated and expensive technology. What is required is essential care during child birth and immediate postpartum period and a few critical interventions for the newborn during the first days of life.
- In this era of evidence based medicine the criteria of diagnosis and management of illness changes frequently, hence, objective of this training to specialists working at referral/ district level is to make them acquaint about the recent trends in the management of common newborn problems. The module for training of pediatricians has been developed in three sections "Module - A" contains care of newborn babies with common problems like Birth asphyxia, Sepsis, Jaundice, Birth injuries and Convulsions, "Module -B" contains Care of Low Birth Weight (LBW) babies and "Module - C" contains Intensive Care of a Sick Newborn.
- We hope that these modules will serve as a useful guide during training and afterwards in practice for the specialist in Pediatrics working at referral/district hospitals.

## ➢ Causes of low birth weight baby :-

| | |
|---|---|
| **1.** | **Maternal factors** |
| • | Medical disease of the mother during pregnancy. |
| • | Complications of pregnancy e.g., placenta previa and antepartum hemorrhage |
| • | Incompetence of cervix |
| • | Maternal infections |
| • | Previous premature delivery |
| **2** | **Fetal factors** |
| • | Multiple pregnancy |
| • | Congenital malformation |
| **3** | **Medical factors** |
| • | The delivery of the fetus may have to be induced before full term on medical grounds under the following circumstances: |
| • | Uncontrolled diabetes mellitus in the mother. |
| • | Severe cardiac illness. |
| • | Hypertension, toxemia |
| • | Fetal hypoxia and fetal distress |
| • | Severe rhesus iso-immunization in the mother or hydrops fetalis. |
| • | Severe intrauterine growth retardation |
| **4** | **iatrogenic** |
| • | Improper diagnosis of maturity in elective deliveries |

## ➢ Principles of Management of LBW Infants

| | |
|---|---|
| **1** | **Care at birth** |
| • | Suitable place of delivery 'in-utero' transfer to a place it optimum facilities if a LBW delivery is anticipated. |

| | |
|---|---|
| • | Prevention of hypothermia |
| • | Efficient resuscitation. |
| **2** | **Appropriate place of care** |
| • | Birth weight > 1800 eg: Home care, if the baby is otherwise well |
| • | Birth weight 1500-1800 eg : Secondary level newborn unit |
| • | Birth weight < 1500 eg: Tertiary level newborn care (or intensive care) |
| **3** | **Thermal protection** |
| • | Delay bathing. |
| • | Maternal contact. |
| • | Kangaroo mother care. |
| • | Warm room. |
| • | External heat source (incubator, radiant warmer) |
| **4** | **Fluids and feeds** |
| • | Intravenous fluids for very small babies and those who are sick. |
| • | Expressed breast milk with gavage or katori spoon. |
| • | Direct breastfeeding. |
| **5** | **Monitoring and early detection of complications** |
| • | Weight and other clinical signs. |
| • | Electronic monitoring |
| • | Biochemical monitoring |
| **6** | **Appropriate management of specific complications** |

## ➢ Arrest of premature labour :-

Advances in perinatal care including fabrication of a variety of electronic gadgets cannot compare with unique security and optimal care provided to the fetus by the uteroplacental unit. Efforts should always be made to arrest the progress of premature labor.

## ➢ Antenatal corticosteroids:-

Antenatal administration of corticosteroids is one of the most cost-effective perinatal strategies which must be universally exploited. It is associated with 50 percent reduction in the incidence of RDS due to surfactant deficiency. It provides additional benefits by reducing the incidence of intraventricular hemorrhage and necrotizing enter colitis. The overall neonatal mortality is reduced

by 40 percent by this simple and cheap intervention. Injection betamethasone 12 mg IM every 24 hours for 2 doses or dexamethasone 6 mg IM every 12 hours for 4 doses should be administered to the mother if labor starts or is induced before 34 weeks of gestation. Betamethasone is more potent and is associated with reduced risk of side effects.

## ➢ CARE OF PRETERM BABIES:-

1. **Optimal management at birth:** - When a preterm baby is anticipated, the delivery should be attended by a senior pediatrician, fully prepared to resuscitate the baby. The delayed 13 clamping of cord helps in improving the iron stores of the baby. It may also reduce the incidence and severity of hyaline membrane disease.
   - Elective intubation of extremely LBW babies (< 1000g) is practiced in some centers to support breathing and for prophy lactic administration of exogenous surfactant.
   - The baby should be promptly dried, kept effectively covered and warm. Vitamin K 0.5 mg should be given intramuscularly.
   - The baby should be transferred by the doctor or nurse (not a nursing orderly!) to the NICU as soon as breathing is established.
2. **Monitoring**: - The following clinical parameters should be monitored by specially trained nurses. The frequency of monitoring depends upon the gestational maturity and clinical status of the baby.
   - Vital signs with the help of multi-channel vital sign monitor (noninvasive with alarms).
   - Activity and behavior.
   - Color; Pink, pale, grey, blue, yellow.
   - Tissue perfusion Adequate tissue perfusion is suggested by pink color, capillary refill over upper chest of < 3 sec, warm and pink extremities, normal blood pressure, urine output of >1.5 ml/kg/hr, absence of metabolic acidosis and lack of any disparity between paO2 and SaO2. Fluids, electrolytes and ABG's.
   - Tolerance of feeds; Vomiting, gastric residuals, abdominal girth.
   - Look for development of RDS, apnea attacks, sepsis, PDA, NEC, IVH etc.
   - Weight gain velocity.
3. **Provide in-utero milieu: -** Uterus provides ideal ambient conditions to the baby. All attempts should be made to create uterus like baby-friendly ecology in the nursery.
   - Create a soft, comfortable, "nestled" and cushioned bed.
   - Avoid excessive light, excessive sound, rough handling and painful procedures. Use effective analgesia and sedation for procedures.

- Provide warmth.
- Ensure asepsis.
- Prevent evaporative skin loses by effectively covering the baby, application of oil or liquid paraffin to the skin and increasing humidity to near 100 percent.
- Provide effective and safe oxygenation.
- Uterus is able to provide unique parenteral nutrition. Efforts should be made to provide at least partial parental nutrition and give atrophic feeds with expressed breast milk (EBM).
- Provide rhythmic gentle tactile and kinesthetic stimulation like skin-to skin contact, interaction, music, caressing and cuddling.

4. **Position of the baby:** - Most babies love to lie in a prone position; they cry less and feel more comfortable. It relieves abdominal discomfort by passage of flatus and reduces risk of aspiration. Prone posture improves ventilation, increases dynamic lung compliance and enhances arterial oxygenation. Unsupervised prone positioning, beyond neonatal period, has been recognized as a risk factor for SIDS.
5. **Thermal comfort A:-** pre-warmed open care system or incubator should be available at all times to receive any baby with hypothermia or with a birth weight of less than 2000g. The baby should be nursed in a thermo neutral environment with a servo sensor geared to maintain skin temperature of mid-epigastria region at 36.5 0C so that there is virtually no or minimal metabolic thermo genesis.
   - Application of oil or liquid paraffin on the skin reduces convective heat loss and evaporative water losses.
   - The extremely LBW baby should be covered with a cellophane or thin transparent plastic sheet to prevent convective heat loss and evaporative losses of water from skin.
   - As soon as baby's condition stabilizes he should be covered with a Perspex shield or effectively clothed with a frock, cap, socks and mittens.
   - After one week or so, stable babies with a birth weight of < 1200 g should preferably be nursed in an intensive care incubator. It is associated with reduced chances of handling, better temperature control, reduced evaporative losses from skin and better weight gain velocity.
   - The mother should be encouraged to provide Kangaroo-Mother.
   - The mother should be encouraged to provide Kangaroo-MotherCare (KMC) to prevent hypothermia, to promote bonding and breast feeding and to transmit healing electromagnetic vibrations of love and compassion to her baby.
6. **DIETARY PRINCIPLES:-**

- Proteins:- 10% of daily calories should be derived from proteins.
- Recommended allowance for LBW neonates is 3-4 gms/kg/day.
- Carbohydrates: - Should provide 40% energy.
- Recommended allowance is 10-15 gms/kg/day.
- Fats :- Should provide 50% of total energy.
- Recommended allowance is 5.4- 7.2 gms/kg/day.
- Electrolytes
- Calcium & Phosphorus
- Iron
- Vitamins
- OXYGEN THERAPY
- PHOTOTHERAPY
- PREVENTION OF NOSOCOMIAL INFECTIONS

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# SPINA BIFIDA

## 1. DEFINITION

**Spina bifida** is a type of neural tube defect (NTD) that occurs during early fetal development. It involves a defect in the formation of the spinal cord and its protective covering, leading to a range of physical and neurological impairments. The condition arises when the neural tube, which forms the spine and surrounding structures, does not close completely during early pregnancy.

## 2. OBJECTIVES

The objectives of managing spina bifida are focused on optimizing outcomes for affected individuals through a combination of medical, surgical, and supportive interventions. These objectives aim to address the condition's various aspects, including its physical, neurological, and psychosocial impacts. Here's a detailed overview:

1. Early Detection and Diagnosis

- Objective: Ensure early diagnosis of spina bifida through prenatal screening and diagnostic testing.

- Approach: Utilize maternal blood tests (e.g., alpha-fetoprotein levels), detailed ultrasounds, and amniocentesis to identify the condition as early as possible.

2. Surgical Intervention

- Objective: Correct or minimize the physical defect and associated complications through surgical repair.

- Approach: Perform surgery to close the defect in the spinal column and protect the spinal cord and nerves. This is typically done shortly after birth or, in some cases, prenatally.

3. Neurological Preservation and Management

- Objective: Preserve and enhance neurological function and minimize damage to the spinal cord and nerves.

- Approach: Provide early and ongoing assessment of neurological function, including motor skills and sensory abilities, and implement rehabilitation strategies such as physical therapy and occupational therapy.

4. Management of Associated Conditions

- Objective: Address and manage complications often associated with spina bifida, such as hydrocephalus, bladder and bowel dysfunction, and orthopedic issues.

- Approach:

  - Hydrocephalus: Monitor and manage with interventions such as shunt placement.

  - Bladder and Bowel Dysfunction: Implement strategies like catheterization programs, bowel management plans, and medications as needed.

  - Orthopedic Issues: Provide orthopedic evaluations and treatments for issues like scoliosis or joint deformities.

5. Supportive Care and Rehabilitation

- Objective: Enhance the quality of life and functional independence through supportive care and rehabilitation.

- Approach:

  - Physical Therapy: To improve mobility, strength, and coordination.

  - Occupational Therapy: To support daily living activities and adaptive skills.

  - Speech and Language Therapy: If needed, for communication or swallowing difficulties.

6. Psychosocial Support

- Objective: Provide emotional and psychological support to individuals with spina bifida and their families.

- Approach:

  - Counseling: Offer psychological support to address the emotional impact of living with spina bifida.

  - Support Groups: Facilitate access to support groups and resources for both patients and families.

7. Education and Self-Management

- Objective: Educate patients and families about the condition, treatment options, and self-care strategies.

- Approach:

  - Patient Education: Provide information on managing the condition, recognizing signs of complications, and using assistive devices.

  - Family Education: Support families in understanding the needs of their child and managing care effectively.

8. Long-Term Monitoring and Follow-Up

- Objective: Ensure ongoing care and monitoring to address evolving needs and complications over time.

- Approach: Schedule regular follow-up appointments for comprehensive evaluations, including neurosurgical assessments, orthopedic evaluations, and routine screenings.

9. Prevention of Secondary Complications

- Objective: Prevent or manage secondary complications associated with spina bifida, such as infections or pressure sores.

- Approach: Implement preventive measures, such as regular skin care, infection control practices, and routine medical check-ups.

10. Promote Quality of Life

- Objective: Enhance overall quality of life by supporting physical, emotional, and social well-being.

- Approach: Address individual needs, provide appropriate therapies, and encourage participation in educational and social activities.

These objectives are aimed at providing a comprehensive and multidisciplinary approach to managing spina bifida, ensuring that individuals receive the care they need to achieve the best possible outcomes and lead fulfilling lives.

**TYPES**

Spina bifida is classified into several types based on the severity of the spinal defect and associated complications. Here's a detailed overview of the main types:

1. Spina Bifida Occulta

- Description: The mildest and most common form of spina bifida. In this type, there is a small defect in the bony encasement of the spinal cord (the vertebrae), but the spinal cord and nerves are not exposed or damaged.

- Characteristics:

  - No Visible Defect: The defect is covered by skin and muscles, so it may not be apparent without imaging studies.

  - Symptoms: Often asymptomatic, but can sometimes cause minor issues such as localized back pain or changes in skin sensations. In some cases, symptoms may include neurological signs like muscle weakness or sensory changes.

  - Diagnosis: Typically identified incidentally through X-rays or MRI conducted for other reasons.

2. Meningocele

- Description: In this type, the protective coverings of the spinal cord (the meninges) protrude through the defect in the vertebrae, forming a sac. The sac contains cerebrospinal fluid (CSF) but does not typically include spinal cord tissue.

- Characteristics:

  - Visible Sac: A noticeable sac filled with CSF may be visible on the back.

  - Neurological Impact: Generally less severe than myelomeningocele, but some neurological impairment may still occur depending on the location and extent of the defect.

  - Symptoms: May include mild to moderate motor or sensory deficits, depending on the location of the defect.

  - Diagnosis: Confirmed through imaging studies such as ultrasound, MRI, or CT scans.

3. Myelomeningocele

- Description: The most severe form of spina bifida, where both the meninges and the spinal cord (or nerve roots) protrude through the defect in the vertebrae. This results in a significant neurological impairment.

- Characteristics:

- Visible Sac: A sac containing both spinal cord tissue and CSF protrudes through the defect. This sac is usually covered by a thin membrane.

- Neurological Impact: Significant, often including paralysis or weakness in the lower limbs, loss of sensation, and other neurological deficits.

- Associated Conditions: Commonly associated with hydrocephalus (accumulation of cerebrospinal fluid in the brain), bladder and bowel dysfunction, and orthopedic problems like scoliosis or clubfoot.

- Symptoms: Severe motor and sensory deficits below the level of the defect, bowel and bladder incontinence, and other related complications.

- Diagnosis: Typically diagnosed through prenatal ultrasound, and confirmed with further imaging and clinical evaluation after birth.

Summary

The severity of spina bifida varies from mild cases (spina bifida occulta) that may be asymptomatic or have minor symptoms, to severe cases (myelomeningocele) that involve significant neurological impairment and require comprehensive medical and supportive care. Early diagnosis and intervention are crucial for managing the condition and improving outcomes.

## ETIOLOGY

The etiology of spina bifida involves a combination of genetic, environmental, and developmental factors that disrupt the normal closure of the neural tube during early fetal development. Here's a detailed look at these contributing factors:

1. Genetic Factors

- Genetic Mutations: Certain genetic mutations and variations can increase the risk of spina bifida. These mutations may affect the development of the neural tube or its closure.

- Family History: A family history of spina bifida or other neural tube defects increases the risk for future pregnancies. Genetic predispositions can be passed down through generations.

- Genetic Syndromes: Some genetic syndromes or chromosomal abnormalities may be associated with a higher risk of spina bifida.

2. Nutritional Factors

- Folic Acid Deficiency: Insufficient levels of folic acid (vitamin B9) during pregnancy are one of the most significant risk factors. Folic acid is crucial for the proper development of the neural tube. Deficiency can lead to incomplete closure of the neural tube.

- Maternal Nutrition: Overall maternal nutrition and health can impact the risk of spina bifida. Adequate intake of essential vitamins and minerals is vital during pregnancy.

3. Environmental Factors

- Maternal Diabetes: Poorly controlled diabetes during pregnancy is associated with an increased risk of neural tube defects, including spina bifida.

- Obesity: Maternal obesity is a risk factor for spina bifida and other congenital anomalies. It can affect nutrient absorption and overall fetal development.

- Exposure to Certain Medications: Use of certain anti-seizure medications (e.g., valproic acid) during pregnancy can increase the risk of spina bifida. These medications may interfere with the normal development of the neural tube.

4. Preconception and Maternal Health

- Uncontrolled Chronic Conditions: Conditions such as chronic hypertension or poorly managed pre-existing health issues can increase the risk of spina bifida.

- Infections: Certain infections during early pregnancy might affect neural tube development and increase the risk of spina bifida.

5. Developmental Factors

- Neural Tube Closure: Spina bifida occurs due to the failure of the neural tube to close properly during early fetal development. This process typically occurs within the first few weeks of pregnancy, and any disruption during this critical period can lead to spina bifida.

6. Socioeconomic and Lifestyle Factors

- Socioeconomic Status: Lower socioeconomic status can be associated with higher risk due to factors like limited access to prenatal care and lower rates of folic acid supplementation.

- Lifestyle Factors: Certain lifestyle factors, such as smoking or excessive alcohol consumption, might impact fetal development and increase the risk of neural tube defects.

Summary

The etiology of spina bifida is complex and multifactorial, involving an interplay of genetic, nutritional, environmental, and developmental factors. While the exact cause of spina bifida is not fully understood, addressing modifiable risk factors, such as ensuring adequate folic acid intake before conception and during early pregnancy, can significantly reduce the risk of spina bifida and other neural tube defects.

**PATHOPHYSIOLOGY**

The pathophysiology of spina bifida involves a failure in the closure of the neural tube during early fetal development, leading to defects in the spinal cord and surrounding structures. Here's a detailed look at the underlying mechanisms and consequences:

1. Neural Tube Development

- Neural Tube Formation: The neural tube forms from the ectodermal layer of the embryo and is the precursor to the central nervous system, including the brain and spinal cord. It typically closes around the fourth week of gestation.

- Neural Tube Closure: The closure of the neural tube involves the fusion of the neural folds along the midline. Failure of this closure leads to various forms of spina bifida.

2. Types of Spina Bifida and Their Pathophysiology

1. Spina Bifida Occulta

- Pathophysiology: This is the mildest form where there is a defect in the bony encasement (vertebral arch) but the spinal cord and nerves remain within the spinal canal. The defect is covered by skin and muscles, and often goes unnoticed.

- Consequences: It usually does not cause significant neurological impairment but may be associated with minor symptoms like localized back pain or skin changes over the defect.

2. Meningocele

- Pathophysiology: In meningocele, the meninges (the protective coverings of the spinal cord) herniate through the defect in the vertebral arch. The sac contains cerebrospinal fluid (CSF) but does not include spinal cord tissue.

- Consequences: The protruding sac may cause some neurological impairment depending on the defect's location and size, though it is generally less severe compared to myelomeningocele.

3. Myelomeningocele

- Pathophysiology: This is the most severe form where both the meninges and the spinal cord (or nerve roots) protrude through the defect in the vertebrae. The protruding sac contains both CSF and neural tissue.

- Consequences:

- Neurological Impairments: The exposure of spinal cord tissue leads to significant motor and sensory deficits below the level of the defect. This can include paralysis, loss of sensation, and problems with autonomic functions such as bladder and bowel control.

- Hydrocephalus: Often associated with an accumulation of CSF in the brain, leading to increased intracranial pressure and the need for surgical intervention, such as shunt placement.

- Infection Risk: The open spinal cord can be susceptible to infections, which can exacerbate neurological damage and complicate management.

3. Secondary Pathophysiological Effects

- Hydrocephalus: Often seen in conjunction with myelomeningocele, it results from impaired CSF flow or absorption. The resulting increased intracranial pressure can damage brain tissue and affect cognitive and developmental functions.

- Orthopedic Issues: Secondary skeletal deformities, such as scoliosis, hip dislocations, and contractures, can arise due to muscle imbalances and abnormal gait patterns.

- Bladder and Bowel Dysfunction: Damage to the nerves controlling the bladder and bowel can lead to incontinence, urinary tract infections, and constipation or fecal incontinence.

- Skin Problems: Due to limited mobility and potential loss of sensation, patients may be at risk for pressure sores or skin infections.

4. Genetic and Environmental Interactions

- Genetic Factors: Certain genetic mutations or predispositions can affect neural tube closure. For instance, mutations in genes involved in folate metabolism or other critical pathways may contribute to the risk.

- Environmental Influences: Factors such as maternal folic acid deficiency, diabetes, and exposure to certain drugs or toxins can disrupt the normal development of the neural tube and increase the risk of spina bifida.

Summary

The pathophysiology of spina bifida revolves around the failure of the neural tube to close properly during embryonic development. This defect results in varying degrees of spinal cord exposure, leading to a range of neurological and physical complications. The severity of these complications depends on the type of spina bifida and the level of spinal involvement. Understanding the underlying mechanisms helps guide management and intervention strategies to improve outcomes and quality of life for individuals affected by spina bifida.

**CLINICAL MANIFESATION**

The clinical manifestations of spina bifida vary widely depending on the type of spina bifida and the location and severity of the defect. Here's an overview of the common clinical manifestations for each type:

1. Spina Bifida Occulta

- Asymptomatic Presentation: Often asymptomatic and may not be detected unless discovered incidentally through imaging studies or X-rays.

- Possible Symptoms:

  - Localized Back Pain: Mild discomfort or pain in the lower back.

  - Skin Changes: Birthmarks, hair tufts, or pigmented lesions over the defect site.

  - Neurological Symptoms: Rarely, it may cause minor neurological symptoms such as numbness or weakness in the lower limbs.

2. Meningocele

- Visible Defect: A sac-like protrusion covered by skin, containing cerebrospinal fluid and the meninges. The sac is usually located on the back and may vary in size.

- Possible Symptoms:

  - Neurological Impact: Varies depending on the location and extent of the defect. Neurological impairments are typically less severe than those seen in myelomeningocele but may include mild motor or sensory deficits.

  - Bladder and Bowel Function: May have some degree of dysfunction, but often less severe compared to myelomeningocele.

  - No or Mild Hydrocephalus: Hydrocephalus is less common but may occur in some cases.

3. Myelomeningocele

- Visible Defect: A large sac on the back containing both cerebrospinal fluid and spinal cord tissue. This sac is often covered by a thin membrane or skin.

- Neurological Manifestations:

  - Motor Function: Significant motor impairment below the level of the defect, which can range from weakness to complete paralysis of the lower limbs.

  - Sensory Function: Loss of sensation below the level of the defect, leading to reduced ability to feel touch, pain, or temperature.

  - Bladder and Bowel Dysfunction: Severe problems with bladder and bowel control, leading to incontinence and potential urinary tract infections or constipation.

- Hydrocephalus: Frequently associated with increased cerebrospinal fluid in the brain, which can lead to increased intracranial pressure, head enlargement, and developmental delays. It often requires surgical intervention, such as shunt placement.

- Orthopedic Issues:

- Scoliosis: Abnormal curvature of the spine.

- Clubfoot: Malformation of the feet.

- Hip Dislocation: Dislocated or malformed hips.

- Skin Problems: Risk of pressure sores and skin infections due to limited mobility and sensation.

- Chiari Malformation: Some individuals may develop a type of brain malformation where the cerebellum extends into the spinal canal.

4. General Manifestations Across Types

- Infections: Risk of infection in areas where the skin is compromised or in the area of the defect.

- Psychosocial Impact: Emotional and psychological challenges due to the physical limitations and social implications of the condition.

Summary

The clinical manifestations of spina bifida are influenced by the type and severity of the defect. While spina bifida occulta may be asymptomatic or have minor symptoms, more severe forms like meningocele and myelomeningocele are associated with significant neurological deficits, physical deformities, and functional impairments. Early diagnosis and management are crucial for addressing these manifestations and improving outcomes for individuals with spina bifida.

**DIAGNOSTIC EVALUTION**

The diagnostic evaluation of spina bifida involves a combination of prenatal screening, imaging studies, and clinical assessments to confirm the diagnosis, determine the severity, and plan appropriate management. Here’s an overview of the diagnostic evaluation process:

1. Prenatal Screening

- Maternal Serum Alpha-Fetoprotein (AFP) Test:

  - Purpose: Measures the level of alpha-fetoprotein, a protein produced by the fetal liver, in the mother's blood.

  - Findings: Elevated AFP levels can indicate an increased risk of neural tube defects, including spina bifida. However, elevated AFP is not specific and can be influenced by other factors.

- Ultrasound:

  - Purpose: Provides detailed images of the fetus and can help detect physical abnormalities.

  - Findings: Can visualize the presence of a defect in the spinal column and assess the size and location of any protruding sac. It also helps in detecting associated conditions like hydrocephalus.

- Amniocentesis:

  - Purpose: Involves taking a sample of amniotic fluid to analyze fetal DNA and protein levels.

  - Findings: Elevated levels of AFP in the amniotic fluid can confirm the suspicion of neural tube defects. Amniocentesis can also provide genetic information about the fetus.

2. Postnatal Diagnostic Evaluation

- Physical Examination:

  - Purpose: To assess the newborn for visible signs of spina bifida, such as a sac on the back and any associated physical or neurological abnormalities.

  - Findings: Includes examination for skin changes, motor and sensory deficits, and signs of hydrocephalus.

- Ultrasound:

  - Purpose: To further evaluate the defect and associated complications, including hydrocephalus.

  - Findings: Provides detailed imaging of the spinal cord and surrounding structures.

- Magnetic Resonance Imaging (MRI):

  - Purpose: Offers detailed images of the spinal cord, spinal nerves, and associated structures.

  - Findings: Helps in assessing the extent of the defect, the relationship between the spinal cord and the surrounding tissues, and any associated abnormalities such as Chiari malformation or tethered cord syndrome.

- Computed Tomography (CT) Scan:

  - Purpose: Provides cross-sectional images of the spine and can be used if MRI is not available or feasible.

  - Findings: Helps in visualizing the bony structures of the spine and detecting any associated anomalies.

- Neuroimaging for Hydrocephalus:

  - Purpose: To diagnose and monitor hydrocephalus.

  - Findings: Includes assessing ventricular size and intracranial pressure, typically through ultrasound or MRI.

3. Genetic and Metabolic Testing

- Genetic Testing:

- Purpose: To identify genetic mutations that may be associated with spina bifida and other related conditions.

- Findings: Can provide information about genetic predispositions and guide further management and counseling.

- Metabolic Testing:

- Purpose: To assess for any metabolic or nutritional factors that might be contributing to the defect.

- Findings: Helps in identifying deficiencies or imbalances that could affect fetal development.

4. Functional and Developmental Assessments

- Neurological Examination:

- Purpose: To assess motor function, sensory perception, and reflexes.

- Findings: Evaluates the extent of neurological impairment and helps in planning rehabilitation and therapy.

- Orthopedic Evaluation:

- Purpose: To identify and assess any skeletal deformities or orthopedic issues.

- Findings: Includes evaluating for scoliosis, hip dislocation, and clubfoot.

Summary

Diagnostic evaluation of spina bifida involves a multidisciplinary approach, including prenatal screening, imaging studies, genetic testing, and functional assessments. Early and accurate diagnosis is crucial for planning appropriate interventions and management strategies, which can significantly impact outcomes and quality of life for individuals with spina bifida.

**NURSING MANAGEMENT**

Nursing management of spina bifida is multifaceted, involving the coordination of care to address both immediate and long-term needs of the patient. It encompasses medical, developmental, and psychosocial aspects to optimize outcomes and improve quality of life. Here's a comprehensive overview of nursing management strategies:

1. Preoperative Care

- Wound Care:

  - Objective: Prevent infection and protect the defect site.

  - Approach: Use sterile techniques to cover and protect the defect with a moist, sterile dressing. Avoid direct contact with the sac to prevent infection and injury.

- Monitoring:

  - Objective: Observe for signs of infection, leakage of cerebrospinal fluid (CSF), and changes in neurological status.

  - Approach: Monitor for redness, swelling, or discharge at the defect site. Check vital signs regularly and assess neurological function.

- Patient and Family Education:

  - Objective: Prepare the family for the surgical repair and post-operative care.

  - Approach: Provide information about the surgical procedure, post-operative care, and potential complications. Discuss the importance of timely medical attention for any changes in condition.

2. Postoperative Care

- Pain Management:

  - Objective: Manage and alleviate pain post-surgery.

  - Approach: Administer prescribed pain medications and use non-pharmacological methods, such as positioning and relaxation techniques.

- Wound Care and Monitoring:

  - Objective: Ensure proper healing of the surgical site and prevent infection.

  - Approach: Inspect the surgical site regularly for signs of infection or complications. Maintain a clean and dry environment, and follow protocols for dressing changes.

- Hydrocephalus Management:

  - Objective: Monitor for signs of hydrocephalus and manage as needed.

  - Approach: Observe for symptoms such as an enlarged head circumference, bulging fontanelles, or changes in neurological status. Coordinate with the healthcare team for shunt placement and management if required.

3. Neurological and Developmental Care

- Neurological Monitoring:

  - Objective: Assess and address neurological deficits.

  - Approach: Perform regular neurological assessments to evaluate motor and sensory function. Document changes and collaborate with physical and occupational therapists.

- Developmental Support:

  - Objective: Support developmental milestones and function.

- Approach: Work with developmental specialists to provide appropriate therapy and support. Encourage activities that promote motor skills, cognitive development, and socialization.

4. Bladder and Bowel Management

- Bladder Care:

- Objective: Manage bladder dysfunction and prevent complications.

- Approach: Implement a catheterization program if necessary, and educate families on techniques for intermittent catheterization. Monitor for urinary tract infections and provide guidance on hydration and hygiene.

- Bowel Management:

- Objective: Address bowel incontinence and promote regular bowel function.

- Approach: Develop and implement a bowel management plan that may include dietary modifications, medications, and scheduled bowel routines. Educate families on techniques to manage bowel function and recognize signs of constipation.

5. Orthopedic and Musculoskeletal Care

- Mobility and Physical Therapy:

- Objective: Enhance mobility and prevent complications from immobility.

- Approach: Collaborate with physical therapists to develop and implement a mobility plan, including exercises to strengthen muscles and improve range of motion. Assist with the use of mobility aids as needed.

- Orthopedic Assessments:

  - Objective: Identify and address skeletal deformities.

  - Approach: Monitor for signs of scoliosis, clubfoot, or hip dislocation. Coordinate with orthopedic specialists for evaluations and interventions.

6. Psychosocial Support

- Emotional and Psychological Support:

  - Objective: Address the emotional impact of spina bifida on the patient and family.

  - Approach: Provide counseling and support for coping with the diagnosis and its implications. Facilitate access to support groups and resources.

- Family Education and Support:

  - Objective: Empower families to manage care effectively and understand the condition.

  - Approach: Offer education on spina bifida, care techniques, and resources. Support families in navigating healthcare systems and accessing community services.

7. Long-Term Follow-Up and Care Coordination

- Routine Monitoring:

  - Objective: Ensure ongoing care and address any emerging issues.

- Approach: Schedule regular follow-up appointments with healthcare providers, including neurosurgeons, urologists, orthopedists, and developmental specialists. Monitor for complications and coordinate care.

- Transition Planning:

- Objective: Prepare for transitions in care as the patient grows.

- Approach: Develop a transition plan for moving from pediatric to adult care, and provide guidance on self-management and independent living skills.

Summary

Nursing management of spina bifida requires a comprehensive approach that addresses immediate postoperative needs, ongoing medical care, developmental support, and psychosocial well-being. By providing holistic care, education, and support, nurses play a crucial role in optimizing outcomes and improving the quality of life for individuals with spina bifida.

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# THALASSEMIA

## 3. DEFINITION

Thalassemia is a group of inherited blood disorders characterized by the production of abnormal hemoglobin, which impairs the blood's ability to carry oxygen effectively. Hemoglobin is the protein in red blood cells that binds oxygen and delivers it to tissues throughout the body.

## 4. OBJECTIVE

The primary objectives related to thalassemia are:

1. Early Diagnosis and Screening: Identify individuals with thalassemia or at risk of carrying the trait early, often through genetic testing or newborn screening programs. Early diagnosis helps in managing the condition more effectively and preventing complications.

2. Management and Treatment: Provide appropriate treatment to manage symptoms and complications. This may include regular blood transfusions, iron chelation therapy to prevent iron overload, and folic acid supplementation. For severe cases, bone marrow or stem cell transplants may be considered.

3. Genetic Counseling: Offer genetic counseling to individuals and families to understand the inheritance patterns of thalassemia, assess the risk for future children, and make informed reproductive decisions.

4. Preventive Measures: Implement community-based screening programs to identify carriers of thalassemia and educate them about the condition to reduce the incidence of the disease.

5. Research and Development: Support research to improve treatment options, explore potential cures, and understand the genetic and biological mechanisms underlying thalassemia.

6. Patient Support and Education: Provide support services and education to help individuals with thalassemia and their families manage the condition, cope with its impact on daily life, and access resources and support networks.

## 3. PRINCIPLES

The principles of managing and understanding thalassemia involve several key aspects:

1. Genetic Basis: Thalassemia is a genetic disorder caused by mutations in the genes responsible for producing hemoglobin. Understanding these genetic mutations is fundamental to diagnosing and managing the condition. It involves recognizing the inheritance patterns (autosomal recessive) and the specific genes affected (alpha or beta-globin).

2. Diagnosis: Accurate and early diagnosis is crucial. This typically involves blood tests to check for anemia and abnormal hemoglobin patterns, and genetic tests to identify specific mutations. Newborn screening programs can help detect thalassemia early, allowing for timely intervention.

3. Management and Treatment:

- Regular Monitoring: Patients require regular blood tests and check-ups to monitor their hemoglobin levels and overall health.

- Blood Transfusions: Many patients need regular blood transfusions to maintain adequate hemoglobin levels.

- Iron Chelation Therapy: Chronic blood transfusions can lead to iron overload. Iron chelation therapy is used to remove excess iron from the body and prevent damage to organs.

- Supportive Care: This includes managing symptoms like fatigue and ensuring adequate nutrition.

4. Genetic Counseling: For families affected by thalassemia, genetic counseling is essential to understand the risk of passing the condition to future generations. This includes discussing reproductive options and potential genetic testing for family members.

5. Prevention:

- Carrier Screening: Screening for carriers of thalassemia, especially in populations at higher risk, helps identify individuals who may pass the condition to their children.

- Education: Educating at-risk populations about thalassemia, its inheritance, and the importance of screening can help prevent the birth of children with severe forms of the disease.

6. Research and Advancements: Ongoing research aims to improve treatment options, explore potential cures, and enhance understanding of the disease. This includes gene therapy, new drug developments, and advances in stem cell research.

7. Patient and Family Support: Providing comprehensive support services, including psychological support, educational resources, and community networks, is vital for helping patients and their families manage the emotional and practical aspects of living with thalassemia.

By adhering to these principles, healthcare providers can offer effective care and support to individuals affected by thalassemia, improving their quality of life and health outcomes.

## 5. ETIOLOGY

The etiology of thalassemia involves genetic mutations that affect the production of hemoglobin, the oxygen-carrying protein in red blood cells. Here's a detailed look at the underlying causes:

Genetic Mutations

1. Hemoglobin Structure and Function: Hemoglobin is composed of four globin chains: two alpha (α) and two beta (β) chains. Thalassemia results from mutations in the genes responsible for producing these chains:

- Alpha Thalassemia: Caused by mutations or deletions in one or more of the four alpha-globin genes (HBA1 and HBA2). Each parent contributes two alpha-globin genes, and the severity of the disease depends on the number of affected genes.

- Beta Thalassemia: Caused by mutations in one or both of the two beta-globin genes (HBB). Each parent contributes one beta-globin gene, and the severity depends on whether one or both genes are affected.

2. Types of Mutations:

- Point Mutations: Single nucleotide changes in the DNA sequence can lead to dysfunctional hemoglobin production.

- Deletions: Large segments of DNA are missing, which can lead to the complete absence of certain globin chains.

- Insertions: Extra DNA segments are inserted into the gene sequence, disrupting normal function.

- Frame-Shift Mutations: Insertions or deletions that shift the reading frame of the gene, leading to abnormal protein production.

Inheritance Patterns

1. Autosomal Recessive Inheritance: Both parents must carry a mutated gene for thalassemia for a child to be affected. Carriers of a single mutated gene are typically asymptomatic but can pass the gene to their offspring.

2. Homozygous vs. Heterozygous:

- Homozygous: An individual has two copies of the mutated gene (one from each parent), leading to a more severe form of the disease.

- Heterozygous: An individual has one normal and one mutated gene, often resulting in a milder form or being a carrier without significant symptoms.

Ethnic and Geographic Variability

Thalassemia is more common in certain populations, particularly those from Mediterranean regions, parts of Africa, the Middle East, South Asia, and Southeast Asia. This geographic distribution is related to historical selection pressures, such as malaria, which may have influenced the frequency of thalassemia mutations in these populations.

Genetic Diversity

Different mutations can cause varying degrees of severity within thalassemia. For instance, specific mutations in the beta-globin gene can lead to beta-thalassemia major (severe form) or beta-thalassemia minor (mild or carrier state). Similarly, the number of affected alpha-globin genes determines the severity of alpha-thalassemia.

Understanding these genetic and environmental factors is crucial for diagnosing, managing, and providing genetic counseling for individuals and families affected by thalassemia

**PATHOPHYSIOLOGY**

The pathophysiology of thalassemia revolves around the disruption in the production of hemoglobin, which leads to anemia and related complications. Here's a detailed explanation of the underlying mechanisms:

1. Hemoglobin Production Disruption

- Normal Hemoglobin Structure: Hemoglobin is composed of four globin chains—two alpha (α) and two beta (β) chains—each bound to an iron-containing heme group. The normal production of hemoglobin requires a balanced synthesis of these chains.

- Thalassemia Impact: In thalassemia, genetic mutations impair the production of either alpha or beta globin chains:

- Alpha Thalassemia: Caused by reduced or absent production of alpha-globin chains due to mutations in the alpha-globin genes. This imbalance leads to excess beta-globin chains, forming abnormal hemoglobin (such as Hemoglobin H).

- Beta Thalassemia: Caused by reduced or absent production of beta-globin chains due to mutations in the beta-globin genes. This results in excess alpha-globin chains, which are unstable and can aggregate, forming inclusions.

2. Ineffective Erythropoiesis

- Ineffective Erythropoiesis: In thalassemia, the bone marrow attempts to produce more red blood cells (erythropoiesis) to compensate for the ineffective hemoglobin. However, the red blood cells produced are often malformed, have a shorter lifespan, and are less functional.

- Destruction of Red Blood Cells: The abnormal red blood cells are prematurely destroyed in the spleen (extravascular hemolysis) or in the bone marrow. This contributes to chronic anemia.

3. Anemia and Hemolysis

- Anemia: The reduced production of functional red blood cells and the increased destruction of abnormal cells lead to a decreased number of circulating red blood cells and lower hemoglobin levels, causing anemia.

- Hemolysis: Abnormal red blood cells are broken down more rapidly than normal, which contributes to further anemia and can lead to increased bilirubin levels, resulting in jaundice.

4. Compensatory Mechanisms

- Expansion of Bone Marrow: To meet the increased demand for red blood cells, the bone marrow expands and becomes hyperplastic. This can cause skeletal deformities, especially in the facial bones.

- Splenomegaly: The spleen enlarges due to its increased role in filtering and removing abnormal red blood cells. This can contribute to further anemia and discomfort.

5. Iron Overload

- Iron Overload: Frequent blood transfusions, a common treatment for thalassemia, can lead to iron overload in the body, as each transfusion introduces additional iron. This excess iron deposits in various organs, including the heart, liver, and pancreas, causing potential damage (secondary hemochromatosis).

- Iron Chelation Therapy: To manage iron overload, patients often require chelation therapy, which helps remove excess iron from the body.

6. Chronic Complications

- Organ Damage: Prolonged anemia and iron overload can lead to chronic damage to organs such as the liver (cirrhosis), heart (cardiomyopathy), and endocrine glands (diabetes, hypogonadism).

- Growth and Development Issues: Children with severe thalassemia may experience delayed growth and development due to anemia and related complications.

Understanding the pathophysiology of thalassemia is crucial for effective management and treatment. It helps in tailoring interventions such as blood transfusions, iron chelation, and supportive care to address the specific needs and complications associated with the disease.

**CLINICAL MANIFESATION**

The clinical manifestations of thalassemia vary depending on the type and severity of the disease. Symptoms often result from anemia, ineffective erythropoiesis, and related complications. Here's an overview of the clinical features associated with thalassemia:

1. General Symptoms

- Fatigue: Due to anemia, patients commonly experience tiredness and weakness.

- Paleness: Pale skin (pallor) is a common sign of anemia.

- Shortness of Breath: Reduced hemoglobin levels impair oxygen delivery to tissues, leading to breathlessness.

2. Alpha Thalassemia

- Mild Forms: In alpha thalassemia trait or minor forms, symptoms may be minimal or absent. Individuals may experience mild anemia or no symptoms at all.

- Moderate to Severe Forms: In more severe cases, such as Hemoglobin H disease or Hemoglobin Bart's hydrops fetalis, symptoms can be more pronounced:

  - Hemoglobin H Disease: Symptoms include moderate to severe anemia, jaundice, splenomegaly, and growth retardation.

  - Hemoglobin Bart's Hydrops Fetalis: A severe form that often leads to stillbirth or death shortly after birth due to extreme anemia and fluid accumulation in the fetus.

3. Beta Thalassemia

- Beta Thalassemia Minor (Trait): Individuals typically have mild anemia and may be asymptomatic or experience only mild symptoms like fatigue.

- Beta Thalassemia Intermedia: Patients may have moderate anemia and may exhibit symptoms such as:

  - Anemia: Moderate to severe, with symptoms like pallor, fatigue, and weakness.

  - Splenomegaly: Enlarged spleen due to increased work in filtering abnormal red blood cells.

  - Bone Changes: Expansion of the bone marrow can lead to skeletal deformities, particularly in the facial bones and skull.

- Beta Thalassemia Major (Cooley’s Anemia): This severe form presents with:

  - Severe Anemia: Requires regular blood transfusions.

  - Jaundice: Due to hemolysis (breakdown of red blood cells) and elevated bilirubin levels.

  - Growth Retardation: Delayed growth and development in children.

- Bone Deformities: Due to excessive bone marrow expansion, including changes in facial bones and a "chipmunk" face appearance.

- Splenomegaly and Hepatomegaly: Enlargement of the spleen and liver due to the increased workload in filtering abnormal cells.

- Iron Overload: From frequent blood transfusions, leading to potential damage to organs like the heart, liver, and pancreas.

4. Complications

- Iron Overload: Chronic transfusions can lead to excess iron accumulation, causing:

- Cardiac Complications: Such as cardiomyopathy and heart failure.

- Liver Damage: Including cirrhosis and potential liver failure.

- Endocrine Issues: Including diabetes (secondary to pancreatic damage) and delayed puberty.

- Infections: Increased risk of infections due to splenomegaly and possible spleen dysfunction.

- Delayed Puberty: In children, delayed sexual development and growth retardation can occur.

5. Additional Manifestation

- Leg Ulcers: Especially in individuals with beta thalassemia major, which can be related to microvascular damage.

- Bone Pain: Due to increased hematopoiesis (blood cell production) in the marrow and bone deformities.

The severity and specific manifestations of thalassemia can vary widely, and regular monitoring and personalized treatment are essential to managing the condition and improving the quality of life for affected individuals.

**DIAGNOSTIC EVAULATION**

Diagnostic evaluation for thalassemia involves a combination of blood tests, genetic analysis, and clinical assessments. Here's a comprehensive overview of the diagnostic approach:

1. Clinical History and Physical Examination

- Family History: Assessing family history can help identify individuals at risk, especially in populations where thalassemia is more common.

- Symptoms Review: Evaluating symptoms such as fatigue, pallor, jaundice, and growth delays.

- Physical Examination: Checking for signs like splenomegaly, hepatomegaly, and bone deformities.

2. Laboratory Tests

- Complete Blood Count (CBC):

  - Anemia: Low hemoglobin and hematocrit levels.

  - Red Blood Cell Indices: Low mean corpuscular volume (MCV) and mean corpuscular hemoglobin (MCH) are typical in thalassemia.

- Peripheral Blood Smear:

  - Microcytic Hypochromic Anemia: Small, pale red blood cells are often observed.

  - Target Cells: Cells with a central area of hemoglobin surrounded by a ring of pallor.

- Hemoglobin Electrophoresis:

  - Alpha Thalassemia: May show increased levels of Hemoglobin H or Hemoglobin Bart's, depending on the severity.

  - Beta Thalassemia: Elevated levels of Hemoglobin A2 and/or Hemoglobin F. Hemoglobin F (fetal hemoglobin) is increased in beta-thalassemia major and intermedia.

- Iron Studies:

  - Serum Iron, Ferritin, and Total Iron-Binding Capacity (TIBC): These tests help differentiate thalassemia from iron deficiency anemia. In thalassemia, iron levels are typically normal or elevated, while in iron deficiency anemia, iron levels and ferritin are low.

3. Genetic Testing

- Molecular Genetic Testing: Identifies specific mutations in the alpha or beta-globin genes. This is crucial for:

  - Confirming Diagnosis: Especially in cases where the phenotype is not clear from blood tests alone.

  - Carrier Screening: To determine if an individual is a carrier of thalassemia mutations.

- Prenatal Genetic Testing: For expecting parents at risk of having a child with thalassemia, methods such as chorionic villus sampling (CVS) or amniocentesis can be used to detect thalassemia genes in the fetus.

4. Additional Diagnostic Tools

- Bone Marrow Examination: Sometimes performed to evaluate ineffective erythropoiesis and to rule out other causes of anemia.

- Imaging Studies: X-rays or MRI may be used to assess bone deformities or organ enlargement (e.g., splenomegaly and hepatomegaly).

5. Differential Diagnosis

- Iron Deficiency Anemia: Often differentiated based on iron studies and response to iron supplementation.

- Anemia of Chronic Disease: Distinguished by clinical context and additional laboratory findings.

Summary

The diagnostic evaluation for thalassemia involves a combination of clinical evaluation, laboratory tests, genetic analysis, and sometimes imaging studies. Accurate diagnosis is essential for effective management and treatment, including potential genetic counseling and planning for appropriate therapies.

**NURSING MANAGEMENT**

Nursing management for thalassemia involves a multifaceted approach aimed at addressing the needs of patients through direct care, education, and support. Here's a comprehensive overview of nursing management strategies:

1. Monitoring and Assessment

- Regular Vital Signs: Monitor vital signs, including heart rate, blood pressure, and temperature, to detect signs of anemia or complications.

- Hemoglobin Levels: Regularly assess hemoglobin and hematocrit levels to determine the need for blood transfusions and to monitor treatment efficacy.

- Growth and Development: For pediatric patients, monitor growth parameters and developmental milestones to identify any delays or complications early.

- Iron Overload Monitoring: Regularly check serum ferritin levels and other indicators of iron overload if the patient is receiving blood transfusions.

2. Blood Transfusion Management

- Preparation: Ensure appropriate blood type matching and cross-matching.

- Administration: Administer blood transfusions as prescribed, monitoring for adverse reactions such as fever, allergic reactions, or transfusion-related acute lung injury (TRALI).

- Post-Transfusion Care: Monitor for signs of transfusion reactions and assess for any immediate post-transfusion symptoms.

3. Iron Chelation Therapy

- Medication Management: Administer iron chelation medications as prescribed to prevent iron overload. Common medications include deferoxamine, deferasirox, and deferiprone.

- Side Effects Management: Educate patients about potential side effects and monitor for adverse reactions, such as gastrointestinal issues or renal complications.

- Compliance Monitoring: Ensure adherence to chelation therapy regimens and assess for barriers to compliance.

4. Symptom Management

- Pain Management: Address any pain associated with anemia, bone deformities, or other complications. Use appropriate pain relief measures and interventions.

- Supportive Care: Provide supportive care for symptoms such as fatigue and weakness, which may include energy conservation techniques and assistance with daily activities.

5. Education and Counseling

- Disease Education: Educate patients and families about thalassemia, its progression, and management strategies.

- Medication Adherence: Teach about the importance of adhering to prescribed treatments, including transfusions and chelation therapy.

- Complication Awareness: Inform about potential complications, such as iron overload and its effects, and how to recognize and address them.

6. Genetic Counseling and Family Planning

- Genetic Counseling: Provide or facilitate access to genetic counseling for families to discuss inheritance patterns, risks, and reproductive options.

- Family Support: Offer support and information to help families cope with the emotional and practical aspects of managing thalassemia.

7. Coordination of Care

- Multidisciplinary Approach: Collaborate with hematologists, dietitians, social workers, and other healthcare professionals to provide comprehensive care.

- Referral Services: Refer patients to specialists as needed, such as endocrinologists for endocrine issues or cardiologists for heart-related complications.

8. Psychosocial Support

- Emotional Support: Provide emotional support to patients and families, addressing concerns, fears, and stress related to living with thalassemia.

- Support Groups: Encourage participation in support groups where patients and families can share experiences and gain additional support.

9. Preventive Care

- Vaccinations: Ensure that patients receive appropriate vaccinations, especially if they are at increased risk for infections due to splenomegaly or other factors.

- Regular Screenings: Facilitate regular screenings for complications, such as liver function tests for iron overload and routine cardiac evaluations.

10. Emergency Care

- Emergency Preparedness: Be prepared to manage acute complications, such as severe anemia or transfusion reactions, and ensure that emergency protocols are in place.

Effective nursing management of thalassemia requires a holistic approach that integrates clinical care, patient education, emotional support, and coordination with other healthcare providers. Regular monitoring and proactive management strategies help improve patient outcomes and quality of life.

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# NURSING CARE OF NEONATE

## INTRODUCTION: -

Majority of newborn babies do not develop any serious disorders and they need routine newborn care which can be provided by the mothers under nurse's supervision. High risk mothers are likely to give birth to preterm or low birth weight babies who are prone to suffer from a number of disorders. Common causes of morbidity in newborn babies include hypoglycemia, shock, RDS, jaundice, neonatal sepsis, and neonatal seizures. Most neonatal disorders are limited to preterm and low birth weight babies. In this unit, common neonatal disorders such as hypoglycemia, RDS, shock, jaundice, neonatal sepsis, neonatal seizures and monitoring of sick neonate are being discussed. Hence you will learn about the definition, causes, pathogenesis, types, signs and symptoms, diagnosis and management of these disorders. You will also learn about how to monitor a sick neonate.

## HYPOGLYCEMIA:-

**Definition:**-Hypoglycemia is defined as a blood glucose level of less than 45 mg/dl in all newborns. Low birth weight and sick neonates are prone to develop low blood sugar which increases morbidity and mortality. It is important to monitor, diagnose and treat hypoglycemia early for a favorable outcome.

Neonates at Risk:-

• Premature and LBW neonates especially those weighing less than 2.0 kg

. • Infants of diabetic mother.

• Sick neonate (perinatal asphyxia, hypothermia, poor and/or delayed feeding, sepsis, shock, respiratory distress and polycythemia).

## Signs and Symptoms:-

The signs and symptoms of hypoglycemia are very nonspecific and can mimic any illness. The common signs and symptoms are:

• Lethargy, weak cry and poor sucking

• Temperature instability

• Poor respiratory effort, apnea or cyanosis

• Excessive jitteriness, convulsions or hypotonic

## Management of Hypoglycemia:-

Management of hypoglycemia includes the following: -

• Establish an IV line if one is not already in place. Give a bolus of 2 ml/kg body weight of 10% glucose IV slowly over 5 minutes. Hypoglycemic babies with convulsions may be given 4 – 5 ml/kg of 10% glucose as the initial bolus.

• If an IV line cannot be established quickly, give 2 ml/kg body weight of 10% glucose by gastric tube.

• Start infusion of dextrose at the daily maintenance volume according to the baby's age so as to provide a Glucose Infusion Rates (GIR) of 6 mg/kg/min

• Measure blood glucose 30 minutes after starting the infusion of glucose and then every four to six hours.

• If the blood glucose is less than 25 mg/dl, repeat the bolus of glucose (as given above) and increase concentration of glucose to 8 mg/kg/min in the infusion.

If the blood glucose is less than 45 mg/dl but is at least 25 mg/dl at any measurement, increase the glucose infusion rate by 2 mg/kg/min and measure blood glucose after 30 min.

• Continue the infusion at this rate until 2 consecutive values 6 hrs apart are above 45 mg/dl.

• Allow the baby to begin breastfeeding. If the baby cannot be breastfed, give expressed breast milk using katori spoon and/or paladin.

• As the baby's ability to feed improves, slowly decrease (over a two to three-day period) the volume of IV glucose while increasing the volume of oral feeds.

## RESPIRATORY DISTRESS SYNDROME (RDS):-

Respiratory distress accounts for significant morbidity and mortality in neonates. It occurs in 4 to 6 percent of neonates. Many of the conditions causing respiratory distress are preventable. Early recognition and prompt management are required.

## Definition and Types:-

Respiratory distress is defined as a condition characterized by the presence of fast breathing with respiratory rate > 60/minute in a quiet resting baby, inspiratory recessions, expiratory grunting, flaring of nostrils with or without cyanosis. Based on the assessment by using respiratory distress score, it can be categorized into three types i.e. mild, moderate and severe or impending respiratory failure. For scoring and evaluation of the severity of respiratory distress.

## Etio-pathogenesis:-

A number of causes are responsible for respiratory distress but the most common are obstruction of the baby's airway by mucus, blood, liquor or meconium, infection etc. The common causes are listed below:

Preterm baby:-

• Respiratory distress syndrome

• Congenital Pneumonia

• Miscellaneous causes: hypothermia, hypoglycemia Term baby

• Transient tachypnea of newborn (TTNB)

• Meconium aspiration

• Pneumonia

• Asphyxia Surgical causes

• Diaphragmatic hernia

• Tracheo-esophageal fistula

• Bilateral choanal artesian other causes

• Cardiac (congenital cardiac defects)

• Metabolic (acidosis, alkalosis, hypoglycemia, hypothermia)

Common Signs, Symptoms and Diagnosis:-

Whatever may be the cause of respiratory distress, the signs and symptoms will be common besides the signs and symptoms specific to the cause itself. We shall learn about the common signs and symptoms of RDS. We will also learn about the diagnosis. Diagnosis is made by history, signs and symptoms and diagnostic tests.

**History:** A detailed relevant antenatal and peri-natal history should be taken based on the common causes:

• Gestation

• Onset of distress

• Previous preterm babies with respiratory distress

• Antenatal steroid prophylaxis if preterm delivery

• Rupture of Membranes > 24 hours, Intra-partum fever, chorio-amnionitis

• Meconium stained amniotic fluid

• Asphyxia

• Maternal diabetes mellitus Signs and symptoms Common signs and symptoms of respiratory distress syndrome are fast breathing, respiratory rate > 60 per minute, recession of intercostals and sub costal muscles, flaring of nostrils, pallor or cyanosis of skin and mucus membrane.

Examination of the baby :-

Examination of the baby is done to assess for the following:

• Severity of respiratory distress

• Neurological status • Blood Pressure, Capillary filling Time (CFT)

• Hepatomegaly

• Cyanosis

• Features of sepsis

• Malformations

## Diagnostic modalities:-

The diagnosis is based on the X-ray findings and the sepsis screen. Blood culture is also done.

1) Chest X-ray

To look for

• Respiratory Distress Syndrome (RDS) - Air bronchogram, decreased lung volume and hazy lungs

• Meconium Aspiration Syndrome (MAS) - Fluffy shadows involving both lungs with hyperinflation

• Pneumonia - Infiltrates

• Pulmonary hemorrhage, RDS - White out (Opaque lung) 2) Sepsis screen: TLC, DLC, CRP, Micro ESR, IT Ratio, ANC (Antenatal check up) 3) Blood Culture: This may give a clue to the infectious etiology of the respiratory distress.

Management of RDS:-

Management of RDS includes prevention of RDS, general management for mild and moderate to severe RDS and specific management.

Prevention of RDS can be done by the following:-

• Antenatal corticosteroid therapy is a simple and effective therapy that prevents RDS.

• Optimal effect of antenatal steroids is seen if delivery occurs after 24 hrs of starting therapy.

• Recommended dose is Inj Betamethasone 12 mg IM every 24 hrs × 2 doses or Inj. Dexamethasone 6 mg IM every 12 hrs × 4 doses, given to mothers with preterm labour or APH before 34 wks of gestation.

**Mild respiratory distress is managed as per the following:-**

• Monitor for respiratory distress and oxygen saturation. Give oxygen if needed.

• Give expressed breast milk by gastric tube.

• When oxygen is no longer needed, allow the baby to begin breastfeeding. If the baby cannot be breastfed, continue giving expressed breast milk using an alternative feeding method. • If the breathing difficulty worsens at any time during the observation period: treat for moderate breathing difficulty.

• All babies with mild and transient respiratory distress, do not need antibiotics. However, if the respiratory distress persists for more than 6 hours or there are risk factors, start antibiotics after taking a sepsis screen. Once respiratory distress settles and the sepsis screen is negative – STOPANTIBIOTICS.

**General supportive management for moderate to severe respiratory distress is as follows:**

• Give oxygen with oxygen hood or nasal cannula to achieve appropriate oxygen saturation (oxygen administration has been discussed in Block 1, Practical 6).

• Maintain normal body temperature (see section on hypothermia).

• Give IV fluids if the baby does not accept feeds or has severe respiratory distress.

• Maintain blood glucose, if it is low treat hypoglycemia.

• If baby has apnea

a) Stimulate breathing by rubbing the back or flicking the sole.

b) If the baby does not begin to breathe immediately provide positivepressure ventilation with bag and mask.

c) Aminophylline if baby is preterm

d) If recurrent apnea spells, treat for sepsis and organize transfer to a specialized centre for assisted ventilation.

**Specific management for moderate to severe respiratory distress is as follows:-**

• Monitor and record the baby's respiratory rate, presence of chest in drawing or grunting on expiration, and episodes of apnea every hour until the baby no longer requires oxygen and then for an additional 24 hours.

• Monitor the baby's response to oxygen by monitoring the level of oxygen saturation

. • Insert an oro-gastric tube to empty the stomach of air and secretions.

• After taking a sepsis screen including blood culture, start antibiotics.

• When the baby begins to show signs of improvement do the following: a) Give expressed breast milk by oro-gastric tube. b) Allow the baby to begin breastfeeding as the respiratory distress settles. Baby can be put on to breast while on oxygen by nasal cannula with continuous monitoring. c) If the baby cannot be breastfed, give expressed breast milk using a cup and spoon or paladai.

## NEONATAL SEPSIS:-

**Definition and Types:-**

Neonatal sepsis is the most important cause of neonatal deaths in the community, accounting for over half of them. It refers to the presence of bacterial blood stream infection (BSI) in neonate. If diagnosed early and treated with good supportive care and antibiotics, it is possible to save most cases of neonatal sepsis.

Etio-pathogenesis:-

Most cases of neonatal sepsis in the community are caused by Escherichia coli and Staphylococcus aureus. In hospitals, Klebsiella pneumonia is also a common organism. Early-onset (< 72 hrs) neonatal sepsis is caused by organisms prevalent in the maternal genital tract or in the delivery area. The risk factors for early-onset sepsis include the following: -

• Low birth weight

• Prolonged rupture of membranes >24 hrs;

• Foul smelling liquor,

• Multiple per vaginum examinations,

• Intrapartum maternal fever,

• Difficult or prolonged labour early onset sepsis manifests frequently as pneumonia and less commonly as septicemia or meningitis.

Congenital deformities, also known as congenital anomalies or birth defects, are conditions present at birth that can affect various parts of the body. Here are some common congenital deformities

1. Cleft Lip and/or Cleft Palate: These occur when the tissues of the lip and/or the roof of the mouth (palate) do not fully come together during fetal development.

2. Down Syndrome: Caused by an extra chromosome 21, this genetic disorder results in developmental and intellectual disabilities, as well as characteristic physical features.

3. Spina Bifida: This neural tube defect happens when the spine and surrounding tissues do not fully close during early fetal development, leading to varying degrees of disability.

4. Clubfoot: A condition where a baby's foot or feet are turned inward and downward, making it difficult to place the foot flat on the ground.

5. Congenital Heart Defects: These are structural problems with the heart that are present at birth. Examples include septal defects (holes in the heart's walls), tetralogy of Fallot, and patent ductus arteriosus.

6. Hip Dysplasia: A condition where the hip joint doesn't properly form, which can lead to dislocation and difficulty with movement.

7. Hydrocephalus: This involves an accumulation of cerebrospinal fluid in the brain, leading to increased pressure inside the skull.

8. Polydactyly: The presence of extra fingers or toes.

9. Syndactyly: A condition where two or more fingers or toes are fused together.

10. Achondroplasia: A form of dwarfism caused by a genetic mutation that affects bone growth.

These conditions can vary widely in severity and impact on an individual's health and development. Early diagnosis and intervention often help manage and treat many congenital deformities effectively.

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# SWARRNIM START UP AND INNOVATION UNIVERSITY

# AARIHANT INSTITUTE OF NURSING

COURSE NAME : NUTRITION AND DIETETICS

# LIPID COMPOSITION

Lipids are a diverse group of organic compounds that are primarily composed of carbon, hydrogen, and oxygen. They play crucial roles in energy storage, cell membrane structure, and signaling. Here's an overview of the composition and metabolism of lipids:

**Composition of Lipids**

Lipids are broadly classified into several categories based on their structure and function:

1. **Fatty Acids**:
    - **Structure**: Fatty acids are long chains of carbon atoms with a carboxyl group (—COOH) at one end and a methyl group (—$CH_3$) at the other.
    - **Saturation**:
        - **Saturated Fatty Acids**: Have no double bonds between carbon atoms. They are typically solid at room temperature (e.g., butter).
        - **Unsaturated Fatty Acids**: Have one or more double bonds. They can be:
            - **Monounsaturated**: One double bond (e.g., oleic acid).
            - **Polyunsaturated**: Two or more double bonds (e.g., linoleic acid, omega-3 fatty acids).
2. **Triglycerides (Triacylglycerols)**:
    - **Structure**: Composed of three fatty acids esterified to a glycerol backbone. Triglycerides are the main form of stored energy in adipose tissue.
    - **Function**: Serve as the body's primary energy reservoir and provide insulation and protection to organs.
3. **Phospholipids**:
    - **Structure**: Composed of two fatty acids, a glycerol backbone, and a phosphate group. The phosphate group is often linked to additional molecules like choline, serine, or inositol.
    - **Function**: Phospholipids are key components of cell membranes, forming a bilayer that separates the internal and external environments of cells.
4. **Steroids**:

- **Structure**: Composed of four fused carbon rings with various functional groups attached. Cholesterol is the most well-known steroid.
- **Function**: Steroids play roles in cell membrane structure, serve as precursors for steroid hormones (e.g., testosterone, estrogen), and are involved in the synthesis of vitamin D and bile acids.

5. **Glycolipids**:
   - **Structure**: Composed of a carbohydrate attached to a lipid molecule, usually a fatty acid.
   - **Function**: Glycolipids are involved in cell recognition, signaling, and adhesion, particularly in the nervous system.

**Metabolism of Lipids**

Lipid metabolism encompasses the processes by which lipids are synthesized, broken down, and utilized in the body for energy and structural purposes.

**1. Lipid Digestion and Absorption:**

- **Digestion**:
  - Lipid digestion begins in the small intestine, where bile salts emulsify fats, breaking them into smaller droplets.
  - **Pancreatic Lipase**: An enzyme secreted by the pancreas, breaks down triglycerides into free fatty acids and monoglycerides.
- **Absorption**:
  - The resulting fatty acids and monoglycerides are absorbed by the intestinal cells (enterocytes) and reassembled into triglycerides.
  - These triglycerides are packaged into lipoprotein particles called chylomicrons, which enter the lymphatic system and eventually the bloodstream.

**2. Lipid Transport:**

- **Lipoproteins**: Lipids are transported in the blood as part of lipoprotein complexes, which include:
  - **Chylomicrons**: Transport dietary triglycerides from the intestine to peripheral tissues.
  - **Very Low-Density Lipoproteins (VLDL)**: Transport triglycerides synthesized in the liver to peripheral tissues.
  - **Low-Density Lipoproteins (LDL)**: Deliver cholesterol to cells throughout the body.
  - **High-Density Lipoproteins (HDL)**: Collect excess cholesterol from cells and transport it back to the liver for excretion.

**3. Lipid Catabolism (Beta-Oxidation):**

- **Mobilization**:

- In response to energy needs, stored triglycerides in adipose tissue are broken down into free fatty acids and glycerol through the action of hormone-sensitive lipase.

- **Transport**:
  - Free fatty acids are transported to tissues like muscle and liver via the bloodstream, bound to albumin.
- **Beta-Oxidation**:
  - Fatty acids are taken up by cells and transported into the mitochondria.
  - Inside the mitochondria, fatty acids undergo beta-oxidation, a process that breaks down the fatty acid chain into two-carbon units called acetyl-CoA.
  - Each cycle of beta-oxidation produces one molecule of acetyl-CoA, NADH, and $FADH_2$.
- **Acetyl-CoA Utilization**:
  - Acetyl-CoA enters the citric acid cycle (Krebs cycle) where it is further oxidized to produce ATP, carbon dioxide, and water.
  - During prolonged fasting or low-carbohydrate diets, acetyl-CoA can also be converted into ketone bodies (ketogenesis), which serve as an alternative energy source for the brain and other tissues.

**4. Lipid Synthesis (Lipogenesis):**

- **De Novo Fatty Acid Synthesis**:
  - Occurs primarily in the liver and adipose tissue when excess glucose and carbohydrates are converted into fatty acids.
  - Acetyl-CoA is carboxylated to form malonyl-CoA, which is then used to elongate the fatty acid chain in a process involving the enzyme fatty acid synthase
  - **Triglyceride Synthesis**:
  - Newly synthesized fatty acids are esterified with glycerol to form triglycerides, which are then stored in adipose tissue or secreted as VLDL by the liver.
  - **Cholesterol Synthesis**:
  - Cholesterol is synthesized from acetyl-CoA through a multi-step process, with HMG-CoA reductase being the rate-limiting enzyme. Cholesterol is essential for membrane structure, hormone synthesis, and bile acid production.
  - **5. Lipid Storage and Mobilization:**
  - **Storage**:
  - Excess lipids, particularly triglycerides, are stored in adipose tissue, where they can be mobilized during periods of fasting or increased energy demand.

- **Mobilization**:
- During fasting or exercise, hormones like glucagon and epinephrine activate hormone-sensitive lipase, leading to the breakdown of triglycerides into free fatty acids and glycerol, which are released into the bloodstream.

## PROCESS OF LIPID COMPOSITION

The composition of lipids involves the synthesis and modification of lipid molecules, which include fatty acids, triglycerides, phospholipids, and cholesterol. Here's a detailed look at the processes involved in lipid composition:

### 1. Fatty Acid Synthesis (Lipogenesis)

Fatty acids are synthesized primarily in the liver and adipose tissue from excess glucose and carbohydrates. This process is known as de novo lipogenesis.

**Steps in Fatty Acid Synthesis:**

1. **Acetyl-CoA Formation**:
   - Acetyl-CoA is derived from the breakdown of glucose through glycolysis and the conversion of pyruvate into acetyl-CoA in the mitochondria.
   - In the cytoplasm, acetyl-CoA is converted into malonyl-CoA by the enzyme acetyl-CoA carboxylase, which is the first step in fatty acid synthesis.
2. **Fatty Acid Chain Elongation**:
   - Fatty acid synthesis involves the enzyme complex known as **fatty acid synthase**.
   - Malonyl-CoA provides the carbon units for elongation.
   - The process includes several cycles of addition, reduction, and dehydration, leading to the formation of a long-chain fatty acid. For example, palmitate (16 carbons) is a common product.
3. **Termination**:
   - The newly synthesized fatty acid is released from the fatty acid synthase complex.
4. **Modification**:
   - Fatty acids can be further modified by elongation and desaturation (adding double bonds), resulting in various types of fatty acids like stearic acid, oleic acid, and linoleic acid.

### 2. Triglyceride Synthesis

Triglycerides (triacylglycerols) are the main form of energy storage in the body and are composed of three fatty acids esterified to a glycerol backbone.

**Steps in Triglyceride Synthesis:**

1. **Glycerol-3-Phosphate Formation**:
    - Glycerol-3-phosphate is derived from glucose through glycolysis or directly from glycerol via glycerol kinase.
2. **Fatty Acid Activation**:
    - Fatty acids are activated by converting them into fatty acyl-CoA, a reaction catalyzed by acyl-CoA synthetase.
3. **Esterification**:
    - Fatty acyl-CoA molecules are esterified to glycerol-3-phosphate in a stepwise fashion by the enzyme **acyltransferase**:
        - **1st Step**: One fatty acyl-CoA is attached to glycerol-3-phosphate to form lysophosphatidic acid.
        - **2nd Step**: A second fatty acyl-CoA is added to form phosphatidic acid.
        - **3rd Step**: Phosphatidic acid is dephosphorylated to form diacylglycerol, which is then esterified with a third fatty acyl-CoA to form triglyceride.
4. **Storage**:
    - Triglycerides are stored in adipose tissue for later use or transported to other tissues as part of lipoproteins.

**3. Phospholipid Synthesis**

Phospholipids are essential components of cell membranes and consist of two fatty acids, a glycerol backbone, and a phosphate group.

**Steps in Phospholipid Synthesis:**

1. **Glycerol-3-Phosphate Formation**:
    - Similar to triglyceride synthesis, phospholipids start with glycerol-3-phosphate.
2. **Fatty Acid Attachment**:
    - Two fatty acids are added to glycerol-3-phosphate, forming phosphatidic acid.
3. **Formation of Phosphatidylcholine or Phosphatidylethanolamine**:
    - Phosphatidic acid is converted to various phospholipids by adding different head groups. For example:

- **Phosphatidylcholine**: Synthesized from phosphatidic acid and choline.
- **Phosphatidylethanolamine**: Synthesized from phosphatidic acid and ethanolamine.

4. **Incorporation into Membranes**:
    - Phospholipids are integrated into cellular membranes, contributing to their fluidity and functionality.

**4. Cholesterol Synthesis**

Cholesterol is a sterol that plays a critical role in cell membrane structure and is a precursor for steroid hormones, vitamin D, and bile acids.

**Steps in Cholesterol Synthesis:**

1. **Acetyl-CoA to HMG-CoA**:
    - Acetyl-CoA is converted into HMG-CoA (3-hydroxy-3-methylglutaryl-CoA) by HMG-CoA synthase.
2. **Mevalonate Formation**:
    - HMG-CoA is reduced to mevalonate by HMG-CoA reductase, the rate-limiting enzyme in cholesterol synthesis.
3. **Isoprenoid Units**:
    - Mevalonate is converted into isoprenoid units, which are five-carbon building blocks.
4. **Squalene Formation**:
    - Isoprenoid units are assembled into squalene, a 30-carbon compound.
5. **Cholesterol Formation**:
    - Squalene undergoes cyclization and further modifications to form cholesterol.

**5. Lipid Modification**

Lipids can undergo various modifications to become functional or to adjust their roles within the body:

1. **Hydroxylation**: Adding hydroxyl groups to fatty acids or cholesterol.
2. **Phosphorylation**: Adding phosphate groups to phospholipids.
3. **Desaturation**: Introducing double bonds into fatty acids.

**Conclusion**

The composition of lipids involves the synthesis and modification of various lipid classes, including fatty acids, triglycerides, phospholipids, and cholesterol. These processes ensure the proper formation of lipid molecules that are crucial for energy storage, cell membrane structure, and various physiological functions. Proper regulation of lipid metabolism is essential for maintaining health and preventing metabolic disorders.

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# MINERAL DEFICIENCIES

## DEFINTION

Mineral deficiencies can have a range of effects on health, depending on which mineral is lacking. Here's a quick overview of some common minerals and the potential issues that arise from their deficiencies:

1. **Iron**: An iron deficiency often leads to anemia, which can cause fatigue, weakness, and pallor. It's particularly common in women of childbearing age, pregnant women, and vegetarians.
2. **Calcium**: Lack of calcium can lead to bone-related issues such as osteoporosis or rickets in children. It can also cause muscle cramps and spasms.
3. **Magnesium**: Magnesium deficiency might result in muscle cramps, mental disorders, or irregular heart rhythms. It's also linked to fatigue and weakness.
4. **Potassium**: Low potassium levels can cause weakness, fatigue, and muscle cramps. Severe deficiency can lead to more serious issues like irregular heartbeats.
5. **Zinc**: Zinc deficiency can impair immune function, cause hair loss, and lead to skin issues like rashes. It may also affect taste and smell.
6. **Iodine**: Lack of iodine can cause goiter (enlarged thyroid gland) and hypothyroidism, which can lead to weight gain, fatigue, and cold intolerance.
7. **Selenium**: A deficiency in selenium might lead to cardiovascular problems and weakened immune function.
8. **Copper**: Deficiency in copper can cause anemia, bone abnormalities, and issues with blood vessels.

If you suspect you have a mineral deficiency, it's a good idea to consult with a healthcare provider for proper diagnosis and treatment. They can recommend dietary changes or supplements to help correct the deficiency.

**Iron deficiency:** is a common nutritional problem that can lead to iron deficiency anemia, where the body doesn't have enough iron to produce hemoglobin, the protein in red blood cells that carries oxygen. Here's a more detailed look at iron deficiency:

## Symptoms

Iron deficiency symptoms can vary but may include:

- **Fatigue and Weakness**: Reduced oxygen delivery to tissues can make you feel tired and weak.
- **Paleness**: Your skin may look paler than usual.
- **Shortness of Breath**: You might feel out of breath with even mild exertion.
- **Dizziness or Lightheadedness**: Low iron can affect blood flow to the brain.
- **Cold Hands and Feet**: Poor circulation due to low hemoglobin can lead to feeling cold.
- **Brittle Nails**: Nails may become thin, brittle, or spoon-shaped.
- **Hair Loss**: Increased hair shedding may occur.
- **Cravings for Non-Food Items**: Some people develop cravings for non-nutritive substances like ice or dirt, a condition known as pica.

## Causes

Iron deficiency can be caused by several factors:

- **Inadequate Dietary Intake**: Not consuming enough iron-rich foods.
- **Increased Iron Needs**: During pregnancy, menstruation, or growth spurts in children.
- **Absorption Issues**: Conditions like celiac disease or inflammatory bowel disease can affect iron absorption.
- **Blood Loss**: Heavy menstrual periods, gastrointestinal bleeding, or frequent blood donations can deplete iron levels.
- **Poor Iron Utilization**: Certain chronic diseases can interfere with how the body uses iron.

## Diagnosis

Iron deficiency is typically diagnosed through blood tests, including:

- **Complete Blood Count (CBC)**: To check hemoglobin and hematocrit levels.
- **Serum Ferritin**: Measures stored iron in the body.
- **Serum Iron and Total Iron-Binding Capacity (TIBC)**: Helps evaluate iron levels and how well iron is transported in the blood.

## Treatment

- **Dietary Changes**: Increase intake of iron-rich foods such as red meat, poultry, fish, beans, lentils, tofu, and fortified cereals. Pairing iron-rich foods with vitamin C-rich foods (like oranges or bell peppers) can enhance iron absorption.
- **Iron Supplements**: Your doctor may recommend iron supplements if dietary changes aren't sufficient. It's important to follow the dosage instructions, as excessive iron can be harmful.
- **Treating Underlying Causes**: If there's an underlying condition causing the deficiency, addressing that condition is crucial.

## Prevention

Maintaining a balanced diet with adequate iron and being mindful of conditions that can affect iron absorption can help prevent deficiency. If you're at higher risk or have symptoms of deficiency, regular check-ups with your healthcare provider can help manage and prevent issues.

If you suspect you have iron deficiency or are experiencing symptoms, it’s important to consult a healthcare professional for proper evaluation and treatment.

**Copper deficiency :** is relatively rare but can lead to a range of health issues. Copper is an essential trace mineral involved in various physiological processes, including iron metabolism, formation of red blood cells, and maintenance of nerve and immune system health. Here’s a detailed overview of copper deficiency:

## Symptoms

Copper deficiency can manifest in several ways, including:

- **Anemia**: Similar to iron deficiency anemia, copper deficiency can lead to anemia due to impaired iron metabolism.
- **Fatigue and Weakness**: Due to anemia or decreased energy production.
- **Neurological Issues**: Symptoms can include numbness, tingling, or a lack of coordination due to its role in nerve health.
- **Bone Abnormalities**: Potential for brittle bones or osteoporosis.
- **Skin and Hair Changes**: Changes such as thinning hair, premature graying, or skin depigmentation.
- **Immune System Problems**: Increased susceptibility to infections due to impaired immune function.
- **Heart Problems**: Rarely, severe deficiency can lead to cardiovascular issues.

## Causes

Copper deficiency can arise from various factors:

- **Inadequate Dietary Intake**: Although rare, insufficient dietary copper can occur, particularly in diets very low in copper-rich foods.
- **Malabsorption**: Conditions like celiac disease, Crohn's disease, or other gastrointestinal disorders can impair copper absorption.
- **Genetic Disorders**: Diseases like Menkes syndrome, which affects copper absorption, or Wilson's disease, which affects copper metabolism.
- **Excessive Zinc**: High levels of zinc can interfere with copper absorption and lead to deficiency.
- **Certain Medications**: Long-term use of certain medications may affect copper levels.

## Diagnosis

Diagnosis typically involves:

- **Blood Tests**: Measuring copper levels in the blood and serum ceruloplasmin (a protein that carries copper in the bloodstream).
- **Urine Tests**: Checking copper levels in urine can also provide diagnostic information.
- **Genetic Testing**: If a genetic disorder is suspected.

## Treatment

Treatment depends on the underlying cause of the deficiency:

- **Dietary Adjustments**: Increase intake of copper-rich foods such as shellfish, nuts, seeds, whole grains, and leafy green vegetables.
- **Copper Supplements**: May be prescribed if dietary changes are insufficient, but should be taken under medical supervision to avoid excessive copper levels.
- **Addressing Underlying Conditions**: Treating any gastrointestinal disorders or other conditions contributing to the deficiency.
- **Managing Zinc Intake**: Reducing excessive zinc intake if it's contributing to the copper deficiency.

## Prevention

A balanced diet that includes a variety of foods can usually prevent copper deficiency. If you have conditions that affect nutrient absorption or are at risk for deficiencies, regular check-ups and consultations with a healthcare provider can help manage and prevent issues.

If you suspect you have a copper deficiency or are experiencing related symptoms, it's important to consult with a healthcare provider for a proper diagnosis and treatment plan.

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# VITAMIN DEFICIENCIES

Vitamin deficiencies can lead to a range of health problems, depending on which vitamin is lacking. Here's an overview of some common vitamin deficiencies, their symptoms, causes, and treatments:

### Vitamin A Deficiency

### Symptoms:

- Night blindness
- Dry, scaly skin
- Frequent infections
- Bitot's spots (white patches on the eyes)
- Poor wound healing

### Causes:

- Inadequate dietary intake
- Malabsorption disorders (e.g., celiac disease, Crohn's disease)
- Liver disorders

### Treatment:

- Increase intake of vitamin A-rich foods (e.g., liver, fish, dairy products, carrots, sweet potatoes)

- Vitamin A supplements if necessary

## Vitamin B1 (Thiamine) Deficiency

### Symptoms:

- Fatigue
- Weakness
- Nerve damage (neuropathy)
- Beriberi (can cause swelling, pain, and difficulty walking)
- Wernicke-Korsakoff syndrome (a serious condition affecting the brain, often seen in alcoholics)

### Causes:

- Poor diet (e.g., in chronic alcoholics)
- Malabsorption
- Certain medical conditions (e.g., chronic illnesses)

### Treatment:

- Increase intake of thiamine-rich foods (e.g., whole grains, pork, legumes)
- Thiamine supplements

## Vitamin B12 (Cobalamin) Deficiency

### Symptoms:

- Fatigue
- Weakness
- Anemia
- Numbness or tingling in the hands and feet
- Difficulty walking
- Cognitive disturbances (memory problems, confusion)

### Causes:

- Poor dietary intake (especially in vegans)
- Malabsorption (e.g., pernicious anemia, gastrointestinal conditions)
- Certain medications

## Treatment:

- Increase intake of vitamin B12-rich foods (e.g., meat, fish, dairy products)
- Vitamin B12 supplements or injections if necessary

## Vitamin C Deficiency

## Symptoms:

- Scurvy (characterized by bleeding gums, joint pain, and anemia)
- Dry skin
- Bruising
- Weakness and fatigue

## Causes:

- Inadequate dietary intake
- Smoking or excessive alcohol consumption
- Certain medical conditions

## Treatment:

- Increase intake of vitamin C-rich foods (e.g., citrus fruits, strawberries, bell peppers)
- Vitamin C supplements if needed

# Vitamin D Deficiency

## Symptoms:

- Bone pain and tenderness
- Muscle weakness
- Increased risk of fractures
- Rickets (in children) or osteomalacia (in adults)

## Causes:

- Insufficient sun exposure
- Poor dietary intake
- Malabsorption disorders

## Treatment:

- Increase exposure to sunlight
- Consume vitamin D-rich foods (e.g., fatty fish, fortified dairy products)
- Vitamin D supplements

## Vitamin E Deficiency

### Symptoms:

- Muscle weakness
- Coordination problems
- Vision problems
- Immune system issues

**Causes:**

- Rare genetic disorders
- Malabsorption syndromes
- Premature birth in infants

## Treatment:

- Increase intake of vitamin E-rich foods (e.g., nuts, seeds, vegetable oils)
- Vitamin E supplements if necessary

## Vitamin K Deficiency

## Symptoms:

- Easy bruising
- Excessive bleeding from wounds
- Bleeding gums
- Bone health issues

## Causes:

- Inadequate dietary intake
- Malabsorption disorders
- Certain medications (e.g., anticoagulants)

## Treatment:

- Increase intake of vitamin K-rich foods (e.g., green leafy vegetables, broccoli)
- Vitamin K supplements if needed

## General Approach to Addressing Vitamin Deficiencies:

1. **Diagnosis**: Blood tests and clinical evaluations can diagnose vitamin deficiencies.
2. **Dietary Changes**: Incorporating foods rich in the deficient vitamin.
3. **Supplements**: Taking vitamin supplements as advised by a healthcare provider.
4. **Addressing Underlying Causes**: Managing any health conditions that may be contributing to the deficiency.

If you suspect a vitamin deficiency or have symptoms related to it, consult with a healthcare provider for appropriate testing and treatment.

**Vitamin K :** deficiency, while relatively uncommon, can lead to significant health issues due to its essential role in blood clotting and bone health. Here's a detailed overview:

## Symptoms

Vitamin K deficiency can cause a range of symptoms, primarily related to bleeding and bone health:

- **Easy Bruising**: Skin may bruise easily, even with minor injuries.
- **Excessive Bleeding**: Prolonged bleeding from cuts, gum bleeding, or nosebleeds.
- **Bleeding Gums**: Swelling and bleeding of the gums, especially noticeable after brushing or flossing.
- **Unusual Bleeding**: Blood in urine or stool, or heavy menstrual bleeding.
- **Bone Health Issues**: Increased risk of fractures due to weakened bones (though this is more commonly seen in long-term deficiencies).

## Causes

Vitamin K deficiency can arise from several factors:

- **Inadequate Dietary Intake**: Diets low in vitamin K-rich foods can contribute to deficiency.
- **Malabsorption**: Conditions affecting the digestive system, such as celiac disease, Crohn's disease, or cystic fibrosis, can impair absorption of vitamin K.
- **Medication Interference**: Certain medications, such as anticoagulants (e.g., warfarin), can interfere with vitamin K metabolism and utilization.

- **Liver Disease**: Since vitamin K is processed in the liver, liver diseases can impact its metabolism and absorption.
- **Antibiotics**: Long-term use of broad-spectrum antibiotics can affect the gut bacteria responsible for synthesizing vitamin K2.

## Diagnosis

Diagnosis of vitamin K deficiency typically involves:

- **Blood Tests**: Measurement of prothrombin time (PT) or international normalized ratio (INR) can indicate bleeding problems related to vitamin K. Specific tests for vitamin K levels are also available but less commonly used.
- **Medical History**: Reviewing dietary intake, medications, and any gastrointestinal conditions.
- **Clinical Evaluation**: Assessing symptoms and physical signs of bleeding.

## Treatment

Treatment for vitamin K deficiency includes:

- **Dietary Changes**: Increasing intake of vitamin K-rich foods, such as:
  - **Vitamin K1**: Found in green leafy vegetables (e.g., spinach, kale, broccoli), Brussels sprouts, and certain vegetable oils.
  - **Vitamin K2**: Found in fermented foods (e.g., natto), dairy products, and animal liver.
- **Vitamin K Supplements**: Oral supplements of vitamin K1 (phylloquinone) or vitamin K2 (menaquinone) may be prescribed, depending on the specific needs and severity of the deficiency.
- **Addressing Underlying Conditions**: Managing any health issues or medication interactions contributing to the deficiency.
- **Adjustment of Medications**: For those on anticoagulants, adjusting the medication dose or balancing with vitamin K intake may be necessary, under medical supervision.

## Prevention

To prevent vitamin K deficiency:

- **Balanced Diet**: Ensure a diet that includes sufficient vitamin K-rich foods.

- **Regular Check-Ups**: For individuals on medications affecting vitamin K or with conditions impacting absorption, regular monitoring and adjustments may be needed.
- **Supplements as Needed**: In cases where dietary intake alone is insufficient, supplements may be recommended.

If you suspect a vitamin K deficiency or are experiencing related symptoms, it's important to consult a healthcare provider for proper evaluation and treatment.

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# COMMON MINOR ALIMENT

## common minor aliment

1. **Common Cold**: Caused by a viral infection, symptoms include a runny nose, sore throat, coughing, and sneezing.
2. **Headache**: Can be tension-type, migraine, or sinus-related. Symptoms include pain in the head, often accompanied by nausea or light sensitivity.
3. **Indigestion**: Often caused by overeating or consuming spicy foods. Symptoms **include bloating, discomfort, and nausea.**

4. **Muscle Strain**: Caused by overuse or sudden movement, resulting in pain and stiffness in the affected muscle.
5. **Minor Cuts and Scrapes**: Typically result from minor accidents, causing surface wounds that may bleed a little and can be treated with basic first aid.
6. **Allergic Reactions**: Minor reactions to allergens like pollen or certain foods, causing symptoms like itching, sneezing, or mild rashes.
7. **Sore Throat**: Often due to a viral infection or irritation from dry air, leading to pain or scratchiness in the throat.

These are generally manageable at home with rest, hydration, and over-the-counter remedies. However, if symptoms persist or worsen, it's always a good idea to consult a healthcare professional.

The common cold is a viral infection of the upper respiratory tract. It's highly prevalent and typically caused by rhinoviruses, though other viruses can also be involved. Here's a quick overview:

## Symptoms:

- **Runny or stuffy nose**: Often one of the first symptoms, with mucus that may start out clear and then become thicker or yellowish.
- **Sore throat**: Can be mild and may improve within a day or two.
- **Cough**: Usually a dry or productive cough that can persist after other symptoms have improved.
- **Sneezing**: Frequent sneezing is common.
- **Mild fever**: More common in children than adults.
- **Headache**: Can accompany other symptoms.
- **Fatigue**: Feeling generally tired or unwell.

## Causes:

- **Viruses**: Rhinoviruses are the most common cause, but other viruses such as coronaviruses, adenoviruses, and respiratory syncytial virus (RSV) can also cause colds.

## Transmission:

- **Direct Contact**: Touching surfaces contaminated with the virus and then touching your face.
- **Airborne Droplets**: When an infected person coughs or sneezes, releasing droplets that can be inhaled by others.

## Prevention:

- **Hand Hygiene**: Wash hands frequently with soap and water.
- **Avoid Close Contact**: Stay away from people who are sick.
- **Cover Coughs and Sneezes**: Use tissues or your elbow, and dispose of tissues properly.
- **Boost Immune System**: Eat a balanced diet, stay hydrated, and get regular exercise.

## Treatment:

- **Rest**: Give your body time to fight off the virus.
- **Hydration**: Drink plenty of fluids to stay hydrated and help loosen mucus.
- **Over-the-Counter Medications**: Decongestants, antihistamines, or pain relievers can help alleviate symptoms.
- **Home Remedies**: Warm teas, honey, and steam inhalation can provide relief.

## When to See a Doctor:

- **Persistent Symptoms**: If symptoms last more than 10-14 days or worsen.
- **Severe Symptoms**: High fever, difficulty breathing, or chest pain.

The common cold is usually mild and resolves on its own, but managing symptoms and taking preventive measures can help you feel better faster and reduce the risk of spreading the virus.

cough

A cough is a common reflex action to clear the throat and airways of mucus, irritants, or foreign particles. It can be caused by a variety of conditions, ranging from minor irritants to more serious health issues. Here's a breakdown of what you need to know:

## Types of Coughs:

1. **Dry Cough**: No mucus is produced. It can be caused by irritation or inflammation in the throat, often due to viral infections like the common cold or allergies.
2. **Productive (Wet) Cough**: Produces mucus or phlegm. This type of cough is often associated with infections like bronchitis or pneumonia.

## Common Causes:

- **Viral Infections**: Such as colds, flu, or COVID-19.
- **Allergies**: To pollen, dust, pet dander, or other allergens.
- **Sinusitis**: Post-nasal drip can cause coughing.
- **Gastroesophageal Reflux Disease (GERD)**: Acid reflux can irritate the throat.
- **Irritants**: Smoke, pollution, or strong odors.
- **Chronic Conditions**: Asthma, chronic bronchitis, or chronic obstructive pulmonary disease (COPD).

## Symptoms:

- **Dry Cough**: Often feels scratchy or ticklish in the throat.
- **Productive Cough**: Mucus may be clear, yellow, green, or sometimes blood-tinged.
- **Associated Symptoms**: Fever, sore throat, shortness of breath, or wheezing.

## Treatment:

- **For Dry Cough**:
    - **Hydration**: Drink plenty of fluids to soothe the throat.
    - **Humidifier**: Adding moisture to the air can ease irritation.
    - **Cough Suppressants**: Over-the-counter medications like dextromethorphan.
- **For Productive Cough**:
    - **Expectorants**: Medications like guaifenesin can help loosen mucus.

- **Steam Inhalation**: Helps to loosen mucus and soothe airways.
- **Honey**: A spoonful of honey can soothe the throat and reduce coughing.

## Home Remedies:

- **Warm Teas**: Herbal teas, especially those with honey or ginger, can provide relief.
- **Saltwater Gargle**: Helps to soothe a sore throat.
- **Steam Inhalation**: Inhaling steam from a bowl of hot water or a warm shower can help.

## When to See a Doctor:

- **Persistent Cough**: Lasts more than three weeks or worsens.
- **Severe Symptoms**: Includes high fever, difficulty breathing, or chest pain.
- **Blood**: If you cough up blood or have blood-tinged mucus.

## Prevention:

- **Good Hygiene**: Wash hands frequently to avoid infections.
- **Avoid Irritants**: Stay away from smoke and other pollutants.
- **Manage Allergies**: Use antihistamines or other allergy treatments if needed.

Coughing is usually a symptom rather than a condition on its own, so identifying and treating the underlying cause is key to effective relief.

## ➢ REFERENCE

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**SWARRNIM START UP AND INNOVATION UNIVERSITY**
**AARIHANT INSTITUTE OF NURSING**

# SWARRNIM START UP AND INNOVATION UNIVERSITY

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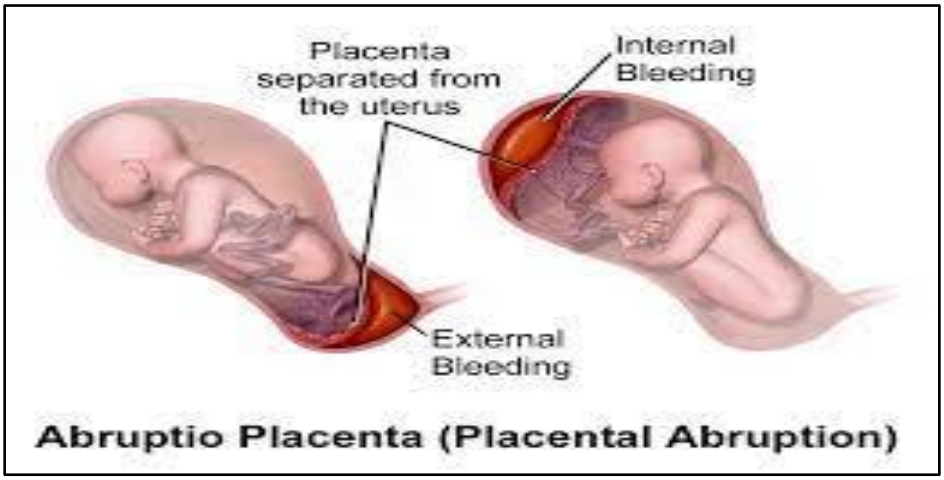
# COURSE NAME : OBSTRETICS AND GYNECOLOGY

# ANTEPARTUM HEMORRAGE

INTRODUCTION:

In obstetrics, ante partum hemorrhage is a bleeding from the vagina during pregnancy from twenty weeks gestational age to term. It should be considered a medical emergency (regardless of whether there is pain) and medical attention should be sought immediately, as if it is left untreated it can lead to death of the mother and/or fetus. Bleeding without pain is most frequently bloody show, which is benign; however, it may also be placenta previa (in which both the mother and fetus are in danger). Painful APH is most frequently placental abruption.




MEANING:

Ante partum hemorrhage (APH) is a bleeding from or in to the genital tract, occurring from 24+0 weeks of pregnancy and prior to the birth of the baby. The most important causes of APH are placenta praevia and placental abruption, although these are not the most common.

DEFINITION:

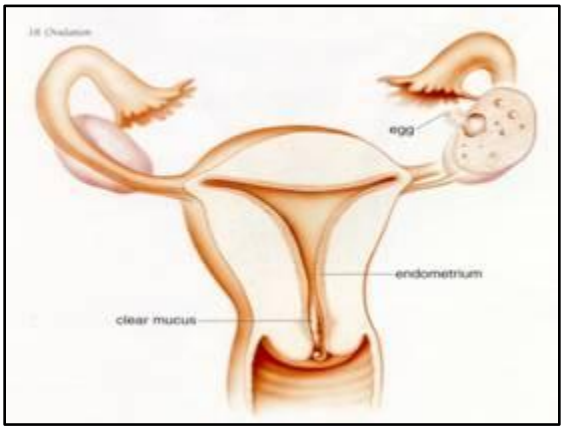
- Ante partum hemorrhage (APH) is defined as a bleeding from or in to the genital tract, occurring after 28 weeks of pregnancy till the birth of the baby.

  - NIMA BHASKAR

- It is defined as bleeding from or into the genital tract after 28$^{th}$ week of pregnancy but before the birth of the baby.

  - DC DUTTA

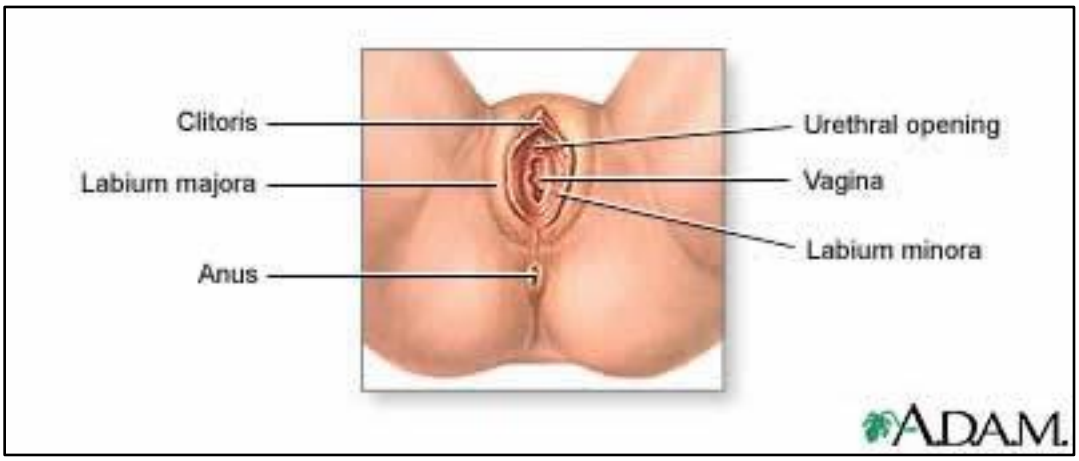
ANATOMY AND PHYSIOLOGY OF REPRODUCTIVE SYSTEM:



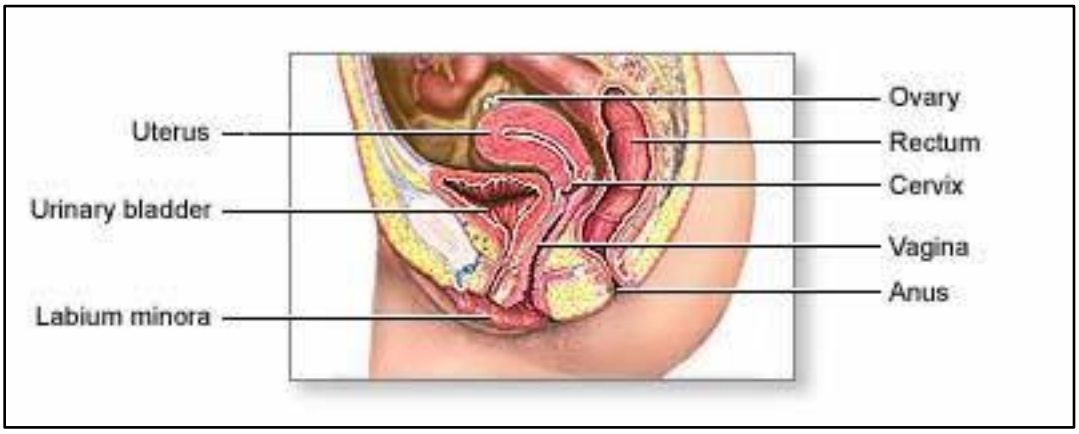

The female reproductive anatomy includes the study of the **external** and **internal** structures**.**

**EXTERNAL FEMALE REPRODUCTIVE SYSTEM:**

The **external genitalia,** also called the **vulva,** includes the **Mons pubis** (a fatty mound which covers the **pubic bone)**, the **labia majora** (outer lips of the vagina), the **labia minora** (the inner lips of the vagina), the **vaginal opening,** the **urethral opening** (opening of the **urethra**, a tube which carries urine from the bladder outside of the body), the **clitoris** (a small structure with sensitive nerve endings located within the labia minora, the sole purpose of which is for sexual arousal and pleasure), and the **perineum** (the space between the **anus** (the rectal opening), and the vaginal opening).




**INTERNAL FEMALE REPRODUCTIVE SYSTEM:**




The **internal reproductive anatomy** includes the **uterus**, two **ovaries**, two **fallopian tubes**, the **urethra,** the **pubic bone**, and the **rectum**. The uterus contains an inner lining called the **endometrium** (which builds ups and sheds monthly in response to hormonal stimulation). The lower portion of the uterus is called the **cervix,** which contains a small opening called the **os**. Menstrual blood flows through the os into the vagina during menstruation. **Semen** travels through the os into the uterus and the fallopian tubes following ejaculation during sexual intercourse. The cervical os **dilates** (opens) during childbirth.

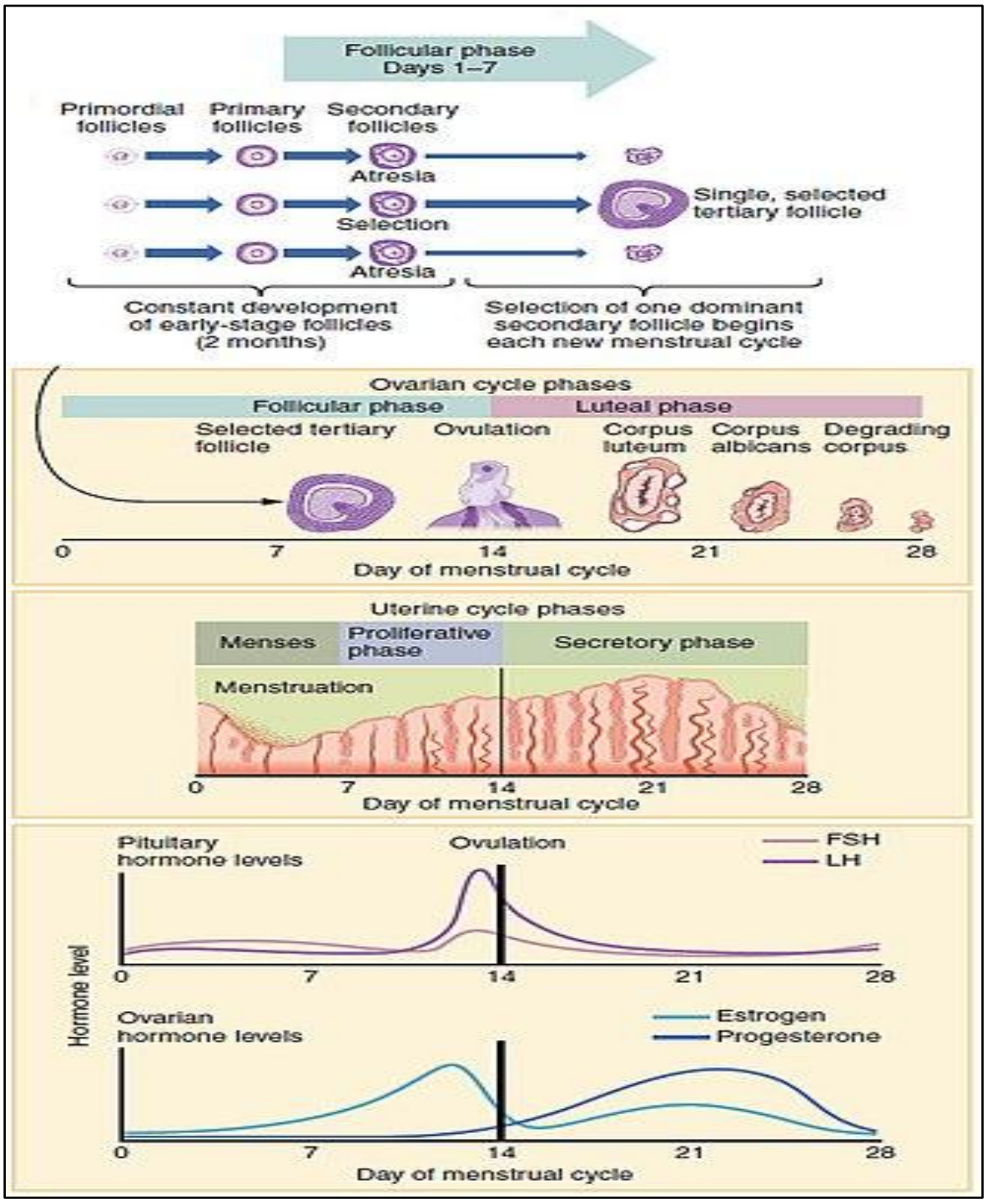
The ovaries, two small almond-shaped structures located on each side of the uterus, are the female gonads (reproductive glands). Female babies are born with over 400,000 ova (the gametes, also referred

to as egg cells or oocytes), which are stored in the ovaries. The female body does not produce any additional ova. The ovaries produce estrogen and progesterone. The ovaries are close to, but not actually connected to the fallopian tubes, thin tube-like structures that are the site of fertilization, the fusion of the male and female gametes.

MENSTRUAL /HORMONAL CYCLE:

The hormonal cycle facilitates maturation and rupture of the ovarian follicle resulting in the release of an ovum (the female reproductive or germ cell). Each month a series of changes take place which prepares the uterus for pregnancy. This cycle (menstrual cycle) is described below:

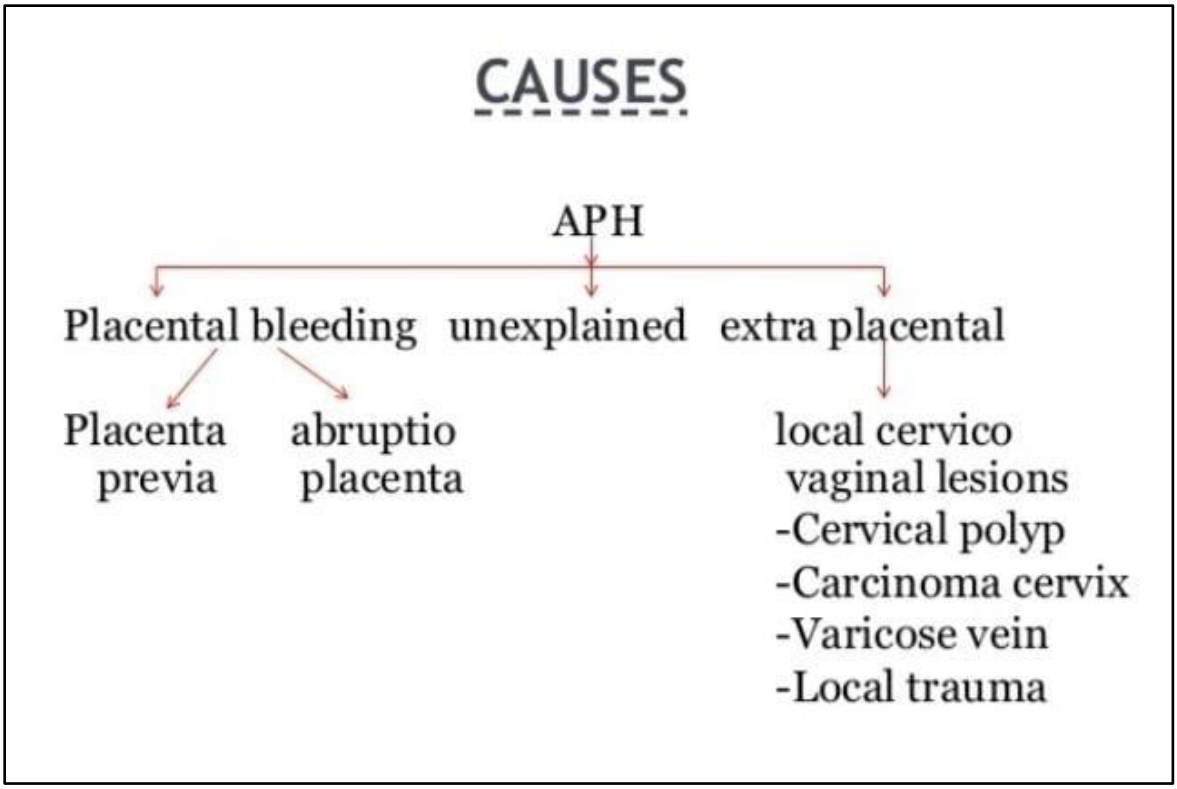
- The first day of menstruation (referred to as Day 1) occurs when levels of estrogen and progesterone are low. In response to these low levels, the hypothalamus secretes gonadotrophin releasing hormone (GnRH) which triggers the anterior pituitary gland to release two hormones: follicle stimulating hormone (FSH), and luteinizing hormone (LH).
- FSH stimulates the development of many follicles within the ovary. One dominant follicle takes over. As it continues to grow, it produces increasing amounts of estrogen, which stimulates the release of LH, and inhibits FSH, which suppresses further follicular development.
- When LH levels are highest (LH surge), the ovarian follicle “ruptures” and releases one ovum, which is “swept” into the fallopian tube by hair-like projections called cilia that line the fimbriae (the fringe-like end of the fallopian tube that is closest to the ovary). This process is called ovulation. Increasing estrogen levels causes the cervical mucus (vaginal secretions) to become clear and profuse and the os to dilate. These two actions may facilitate the transport of semen (containing sperm) from the vagina, through the uterus, and into the fallopian tube.
- Following ovulation, the ruptured follicle is transformed into the corpus luteum, a glandular mass that continues to produce estrogen and high levels of progesterone. The progesterone causes the endometrium to thicken, preparing it for implantation of a fertilized egg. If fertilization takes place during ovulation, hormonal levels remain high, essential for the maintenance of the pregnancy.




- If fertilization does not occur, the corpus luteum shrinks and levels of both estrogen and progesterone decrease. The withdrawal of estrogen and progesterone cause the blood vessels of the endometrial (uterine) lining to “break” resulting in vaginal bleeding (menstruation). The average menstrual cycle is 28-35 days, and menstrual flow usually continues for three to seven days, although there are variations among women.

- Following menstruation, estrogen and progesterone levels are low, triggering the hypothalamus to once again release GnRH, starting the entire cycle again. If fertilization does take place, menstruation will not reoccur for the duration of the pregnancy

**CAUSES OF ANTE PARTUM HEMORRHAGE:**

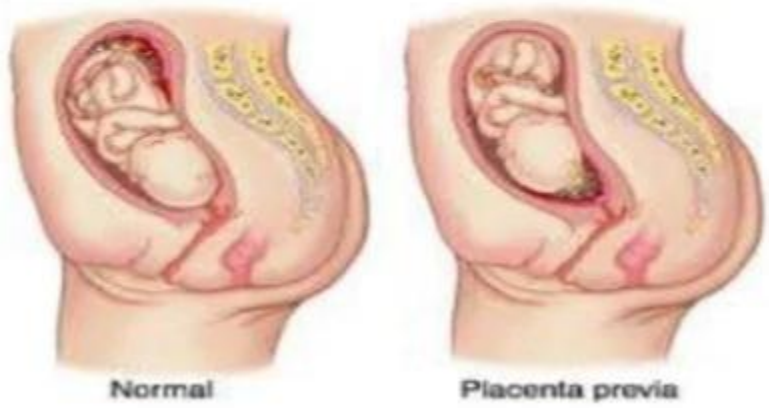



Placenta praevia:

Placenta praevia refers to when the placenta of a growing fetus is attached abnormally low within the uterus. Intermittent ante partum hemorrhaging occurs in 72% of women living with placenta praevia.

# PLACENTA PRAEVIA

- When the placenta is implanted partially or completely over the lower uterine segment it is called placenta praevia.




ETIOLOGY:

➔ Dropping theory:
The fertilized ovum drops down and implanted in the lower uterine segment. Poor decidual reaction in the upper uterine segment may be the cause. Failure of zona pellucid to disappear at time can be hypothetical possibility. This explains the formation of central placenta previa.

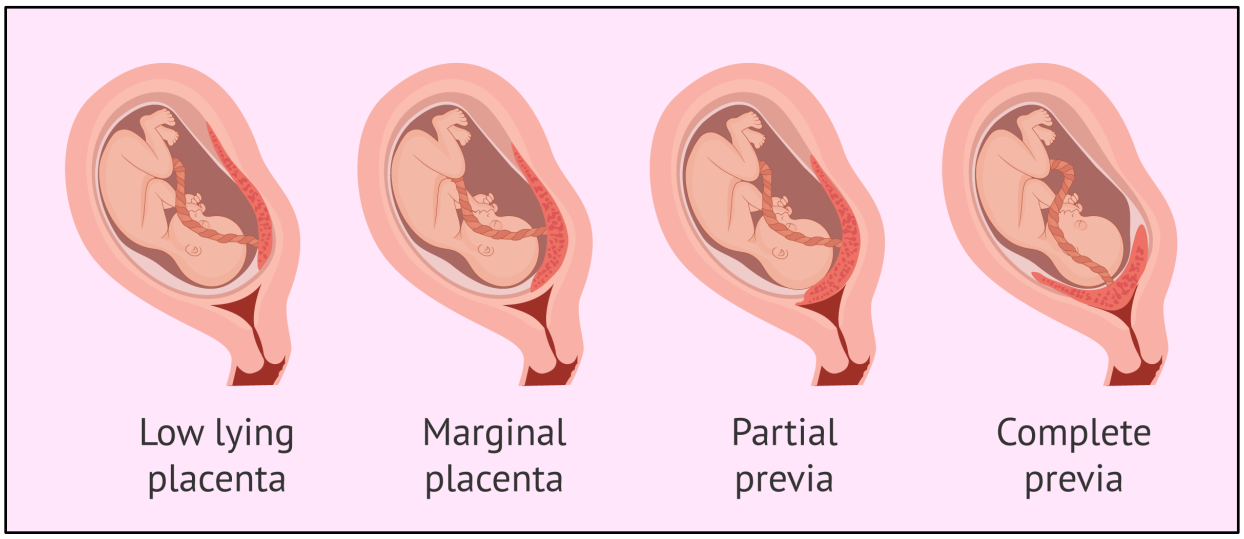
➔ Persistence of chorionic activity:
Chorionic activity in the deciduas capsular is and its subsequent development into capsular placenta which comes in contact with deciduas Vera of the lower segment can explain the formation of lesser degrees of placenta previa.

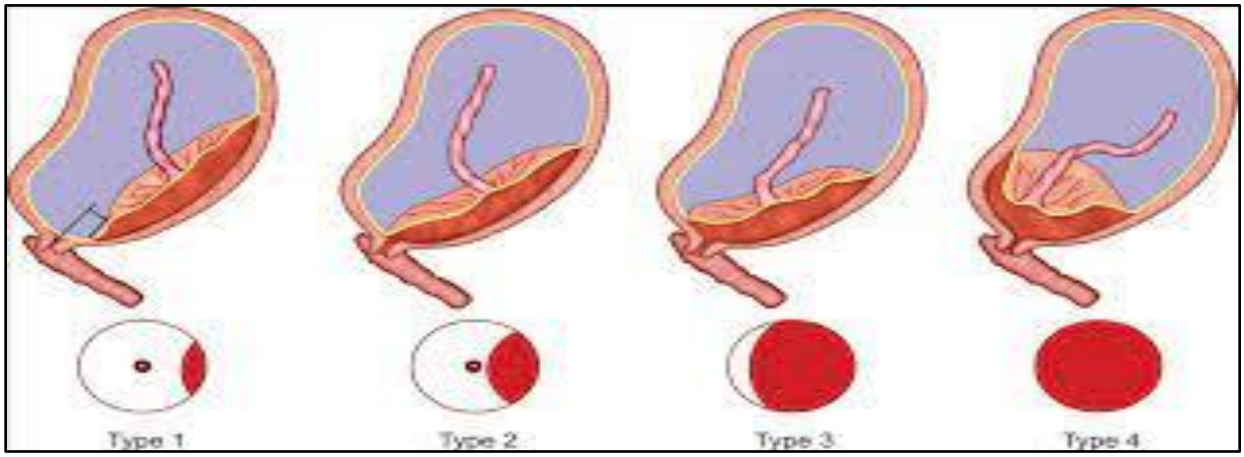
➔ Defective deciduas:
It results in spreading of the chorionic villi over a wide area in the uterine wall to get nourishment. During this process, not only the placenta becomes membranous but encroaches onto the lower segment, such a placenta praevia may invade the underlying deciduas or myometrium to cause placenta accreta, increta or percreta.

➔ Big surface area of the placenta:
As twins may encroach onto the lower segment.

TYPES OR DEGREES OF PLACENTA PRAEVIA:

There are four types of placentapraevia depending upon the degree of extension of placenta to the lower segment.




The severity of a patient's placenta praevia depends on the location of placental attachment;

| Type | Location of Placental Attachment |
|---|---|
| Type 1 | Lower segment of uterus, no attachment to the cervix |

| | |
|---|---|
| Type 2 | Touching but not covering the internal orifice of the cervix |
| Type 3 | Partially covering the internal orifice of the cervix |
| Type 4 | Completely covering the internal orifice of the cervix |

- Types 1 and 2 are classified as minor placental praevia as these typically result in minor ante partum hemorrhaging.
- Types 3 and 4 are referred to as major placental praevia due to the risk of heavy hemorrhaging in the case of a rupture due to the location of placental attachment.

CLINICAL FEATURES OF PLACENTA PREVIA:

SYMPTOMS:

- The only symptom of placenta previa is vaginal bleeding.
- Bleeding- sudden, painless, apparently causeless and recurrent.
- The bleeding is unassociated with pain unless labor starts simultaneously.

SIGNS:

- General condition and anemia are proportionate to the visible blood loss.

ON EXAMINATION:

- The uterus is not tender on palpation.
- The height of uterus corresponds to the gestational age as calculated from the LMP.
- FHR is usually normal.
- The presenting part of the fetus is high up and can be easily palpated through the abdomen.
- There may be abnormal presentations like breech or face presentation.

VAGINAL EXAMINATION:

P/V is indicated only if active treatment is initiated. This may provoke a severe attack of bleeding so it should be done with the following precautions:

In the operating room,

- Under general anesthesia
- Cross- matched blood is in hand,
- Operating room is ready for immediate caesarean section.
- If index finger is introduced gently through the dilated cervix, the placenta can be felt as a tough fibrous mass.

TEST FOR PLACENTA PREVIA:

Ultrasonography is the most valuable aid in the diagnosis of placenta previa.

TREATMENT:

AT HOME:

- Arrange for immediate transfer to the hospital.
- No vaginal examination or pack, only a sterile vulval pad is applied.
- Anti-shock measures as Pethidine IM, fluids and blood transfusion may be given in the way to the hospital if bleeding is severe.

AT HOSPITAL:

- Assessment of the patient's condition, general and abdominal examination and resuscitation if needed.
- At least 2 units of cross matched blood should be available.
- Ultrasonography for differentiation between abruption placentae, marginal bleeding and placenta praevia.
- Assessment of fetal viability age, position and presentation.

MANAGEMENT:

I. IF THE MOTHER IS NOT IN LABOR:

- LOOK FOR AMOUNT OF BLEEDING:
  - If the bleeding is severe, continue anti-shock measures and do immediate caesarean section.
  - If completed 37 weeks or more, pregnancy is terminated by induction of labor or caesarean section.
  - If less than 37 weeks, conservative treatment is indicated till the end of 37 weeks but not more.
- CONSERVATIVE TREATMENT:
  - The patient is kept hospitalized with bed rest and observation till delivery.
  - Observation of fetal wellbeing.
  - Anti- D immunoglobulin is given for the Rh-negative mother.

II. IF THE MOTHER IS IN LABOR:

Vaginal delivery is allowed if the following findings are fulfilled:

- Placenta praevia is lateralis or marginalis anterior.
- Bleeding is slight.
- Vertex presentation.
- Partially dilated cervix to allow amniotomy. As it allows descent of head so it compresses the placental site preventing further bleeding.

III. CAESAREAN SECTION IS INDICATED IN:

- Placenta praevia centralis whether complete or incomplete or incomplete even if the fetus is dead.
- Placenta praeviamarginalis posterior.
- Severe bleeding.

- Presentation other than vertex.
- Other obstetric indications as contracted pelvis, cord prolapsed and elderly primigravida.
- It allows better control of bleeding from the placental site.

- COMPLICATIONS:

MATERNAL:

a. During pregnancy:
   - Abortion.
   - Premature labor.
   - Ante partum hemorrhage.
   - Malpresentation and non-engagement.

b. During labor:
   - Premature rupture of membranes.
   - Cord prolapsed
   - Inertia.
   - Postpartum hemorrhage.
   - Retained placenta.

FETAL:

- Fetal mortality is 20%.
- Prematurity.
- Asphyxia.
- Malformations (2%).

PLACENTA ABRUPTIO

- DEFINITION:

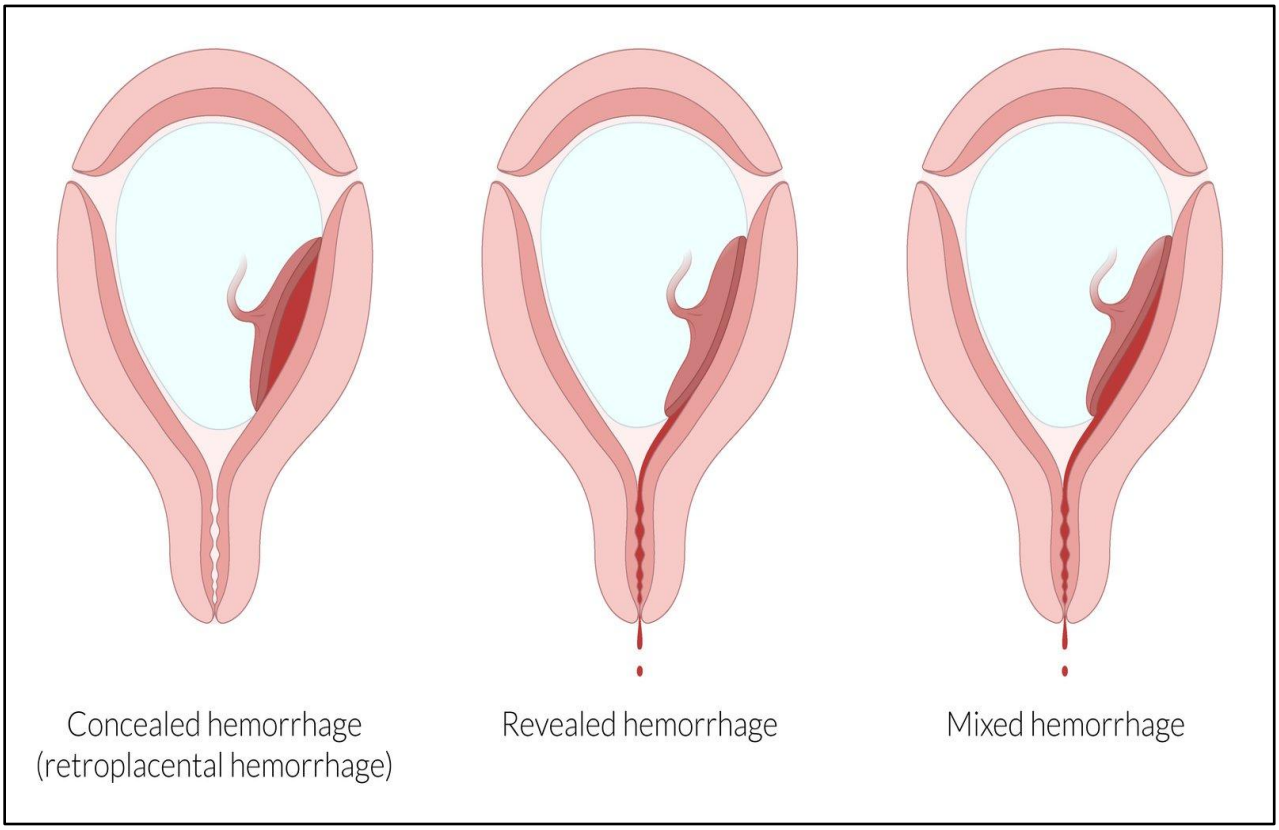
Placental abruption is also called “abruption”. It is type of ante partum hemorrhage where there is premature separation of a normally situated placenta in the upper part of the uterus before delivery of the baby, or sometimes even before labor begins.

Bleeding occurs between the placenta and the uterine wall and can either trickle out between the amniotic membranes or collect as a blood clot that gradually increases in size.

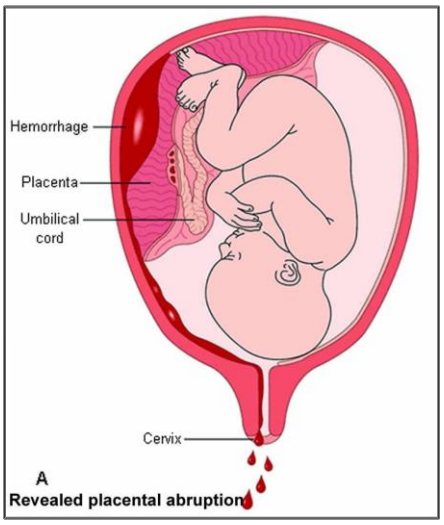
- CLINICAL TYPES OF PLACENTAL ABRUPTION:

There are three clinical types of placental abruption:

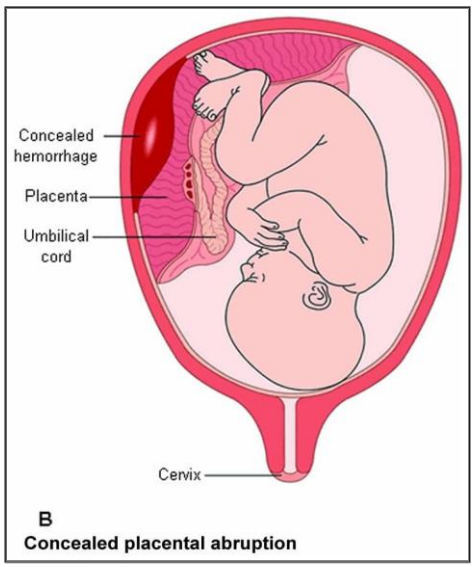
i. Revealed type.
ii. Concealed type.
iii. Mixed type.




i. REVEALED TYPE: This is a mild type of placental abruption. In this type of placental abruption, the bleeding that occurs behind the placenta trickles down between the membranes and the uterine walls to be revealed at the vaginal opening. Since there is no collection of blood behind the placenta, separation of the placenta from the uterus is usually less than in the other types.




ii. CONCEALED TYPE: The blood fails to trickle down and collects between the placenta and the uterine wall. The enlarging blood clot further dissects out the placenta from its bed and placental separation can occur over a large area.

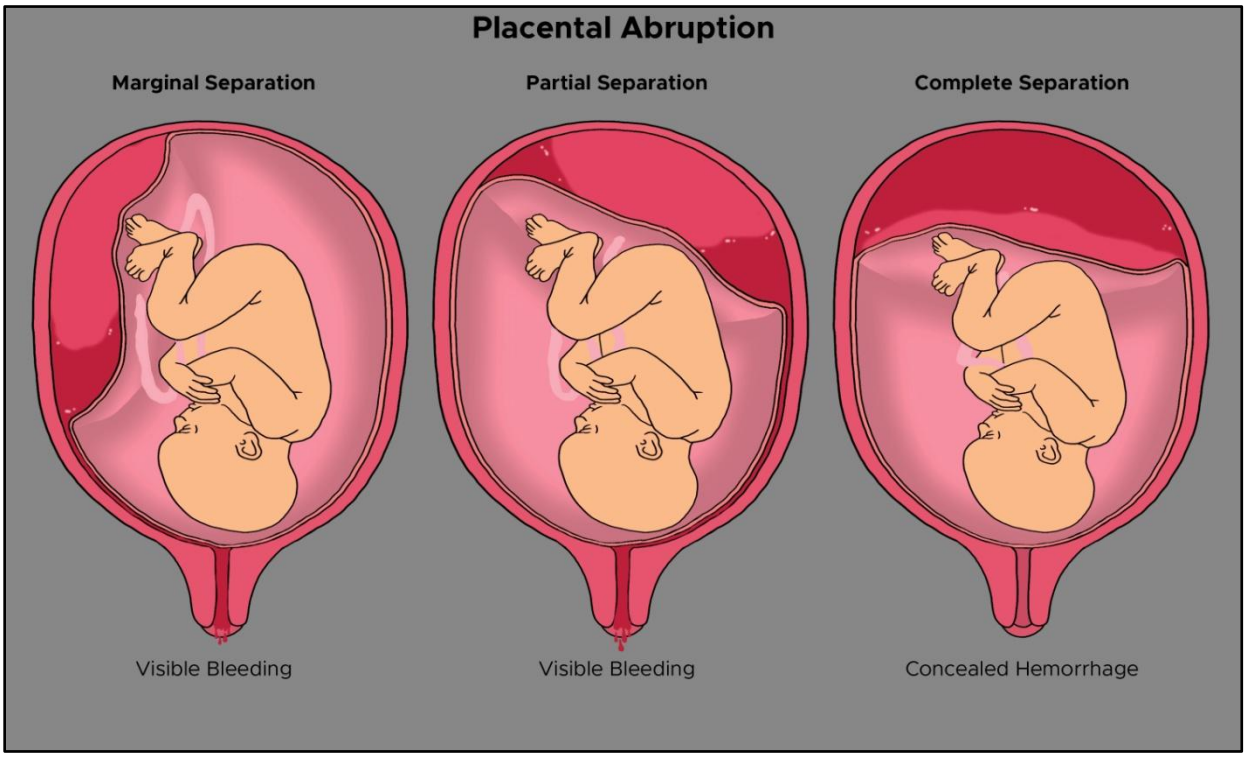



iii. MIXED TYPE: in this type, part of the blood trickles down and part collects behind the placenta. Like the concealed type, this is also a dangerous type of placental abruption as the blood clot continues to dissect out the placenta from the placental bed.

- DEGREES OF PLACENTAL ABRUPTION:

Abruption placenta may be classified in three types of separation:

1. Marginal/ low separation.
2. Moderate/ high separation.
3. Severe/ complete separation.




1. MARGINAL/ LOW SEPARATION:
   This occurs when the separation is low and is not complete and is not complete; vaginal hemorrhage is evident.

2. MODERATE / HIGH SEPARATION:
   This occurs when the separation is high in the uterine segment; causing the fundus of the uterus to rise. The fetus is in grave danger because of lack of oxygen. External hemorrhage will probably not be present here, whereas the amniotic fluid will be a port-wine color.

3. SEVERE/ COMPLETE SEPARATION:

This occurs when the fetus head is present in the cervical os that prevents external hemorrhage. The fetus is in grave danger, and an immediate cesarean section will probably be needed in order to save the baby's and mother's lives.

- CAUSES OF PLACENTAL ABRUPTION:

  - PREMATURE RU PTURE OF MEMBRANES: It can lead to acute infection inside the uterus. This infection is believed to be a leading cause of placental abruption.

  - TOXEMIA OF PREGNANCY: The high blood pressure associated with pre-eclamptic toxemia (PET) or toxemia of pregnancy is frequently associated with placental abruption.

  - CHRONIC HYPERTENSION: High blood pressure present even before the start of pregnancy can also cause placental abruption.

  - TRAUMATIC: Mechanical traumas such as forceful cephalic version, a fall on the abdomen, a short cord that pulls on the placenta during labor pains or overstimulation of the uterus during induction of labor.

  - UNKNOWN CAUSE: sometimes no cause can be identified.

- SIGNS AND SYMPTOMS:

  The signs and symptoms vary depending on whether the placental abruption is of the revealed or concealed type.

Revealed placental abruption:

- Vaginal bleeding : The bleeding is mild to moderate. The blood is blackish red in color and trickles continuously from the vagina.
- Pain: Pain may be mild or absent. But most patients complain of a general discomfort over the abdomen.
- Symptoms of other diseases: symptoms of other disease processes like PET, diabetes or essential hypertension may be present.
- On examination: Localized pain may be present over the uterus at the site of implantation of the placenta.

CONCEALED PLAECNTAL ABRUPTION:

- Vaginal bleeding: There may be no bleeding in the concealed type but in the mixed type, a little trickle of blood may be seen.
- Pain :Pain is acute, agonizing and occurs suddenly ,may be severe .
- Symptoms of other diseases: other diseases like PET, diabetes or essential hypertension may be present.
- Shock: the patient may be unconscious when brought to the hospital and show all the signs and symptoms of acute blood loss like a thin thread pulse, low blood pressure, cold , clammy arms and legs, etc.
- On examination: the patient appears pale and anemic. The uterus is tense, tender and hard. The fetal parts are felt easily.

TREATMENT OF PLACENTAL ABRUPTION:

REVEALED PLACENTAL ABRUPTION:

- If bleeding is slight:
  - If the patient is stable and USG shows minimal retro placental bleeding with a healthy immature fetus- conservative treatment with hospital admission, bed rest and careful monitoring is done.
  - A caesarean section is done once the fetus reaches maturity.

- If bleeding is considerable:
  - If it is believed that the bleeding is enough to compromise the life of the mother, a caesarean section is done, regardless of whether fetus is mature or not.

CONCEALED PLACENTAL ABRUPTION:

- If the patient has come in shock, she is promptly resuscitated with IV fluids, blood transfusion, etc.
- An emergency caesarean section is done as early as possible to cut down blood loss.
- If the patient is in labor, she is allowed to proceed, keeping her ready for a caesarean section.
- In most cases, the fetus is dead at the time of treatment.

CESSARIAN HYSETERECTOMY:

- There may be even bleeding into the muscle and blood vessels of the uterus, causing injury and damage.
- A caesarian hysterectomy (removal of uterus) becomes necessary to control the hemorrhage.

Journal: Ante partum hemorrhage and its feto-maternal outcome- retrospective study.

*Saloni K. Gandhi, Ayushi P. Vamja, Kishor P. Chauhan*

DOI: https://dx.doi.org/10.18203/2320-1770.ijrcog20204811

Published: 2020-10-27

Abstract

**Background:** Ante partum hemorrhage (APH) is defined as any bleeding from or into the genital tract after the period of viability and before the delivery of the baby. Aim of the research was to study the feto-maternal outcome in patients with APH.

**Methods:** The present study was a retrospective observational study undertaken in Obstetrics and Gynecology department of Dhiraj General Hospital, during a period of 1.5 years from November 2018 to May 2020 in 84 cases of ante partum hemorrhage. Only patients with APH >28 weeks gestational age and willing to participate in study were included. Open STAT statistical software has been used to analyze the data in this study.

**Results:** The incidence of ante partum hemorrhage was 2.86%. Maximum patients of APH lie between the age group of 26-34 years. In abruptio placenta (AP) 65% and in placenta previa (PP) 77.2% of the patients were

multiparous. APH presents mostly between 34-36 weeks. Around 90% patients of APH required blood transfusion. APH overall shows increased rate of cesarean sections up to 62%. Around 9.5% patients went into shock, 4.7% had disseminated intravascular coagulation (DIC), 3.5% postpartum hemorrhage (PPH) and 8.3% had wound gap and puerperal pyrexia. 23.8% babies had asphyxia of which 60% were contributed to PP and 40% were in AP group. Respiratory distress syndrome was in 7.1% babies of which both groups equally contributed. Septicemia was seen in 13% and jaundice in 29.8%.

**Conclusions:** Higher rates of neonatal intensive care unit (NICU) admission and stay were seen with these complications. This study showed 20.2% perinatal deaths as outcome of APH and 14.2% still births.

SUMMARY:

The knowledge about ante partum hemorrhage is very important for nurses to know the cause, signs and symptoms in order to render effective nursing care and management of ante partum hemoorhage.

CONCLUSION:

Till now we discussed about ante partum hemorrhage. Hence, this knowledge will help the nurses to apply practical skills in clinical areas, nursing education and in research programmes.

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# OBSTETRIC EMERGENCIES

## DEFINITION

Obstetrical emergencies are life threatening medical conditions that occur in pregnancy or during labor or after delivery.

## VASA PREVIA INCIDENCE

The actual incidence is extremely difficult to estimate, it appears that vasa previa complicates approximately 1 in 2,500 births.

## DEFINITION

It is an abnormality of the cord that occurs when one or more blood vessels from the umbilical cord or placenta cross the cervix but it is not covered by Wharton's jelly.

This condition can cause hypoxia to the baby due to pressure on the blood vessels.

It is a life threatening condition.

## ETIOLOGY

These vessels may be from either

- Velamentous insertion of umbilical cord

- placental lobe joined to the main disk of the placenta.
- Low-lying placenta
- Previous delivery by C-section.

**SYMPTOMS**

- The baby's blood is a darker red color due to lower oxygen levels of a fetus
- Sudden onset of painless vaginal bleeding, especially in their second and third trimesters
- If very dark burgundy blood is seen when the water breaks, this may be an indication of vasa previa

**DIAGNOSIS**

- Classical triad Painless vaginal bleeding
- Colour doppler- vessel crossing the membranes over the internal cervical os
- Membrane rupture
- Fetal bradycardia.

**MANAGEMENT**

1. Antepartum

- The patient should be monitored closely for preterm labor, bleeding or rupture of membranes.
- Steroids should be administered at about 32 weeks.
- Hospitalization at 32 weeks is reasonable.
- Take patient for emergency cesarean section if membranes are ruptured.
- Fetal growth ultrasounds should be performed at least every 4 weeks.
- Cervical length evaluations may help in assessing the patient's risk for preterm delivery or rupture of the membranes

2. Intrapartum

- The patient should not be allowed to labor. She should be delivered by elective cesarean at about 35 weeks
- Delaying delivery until after 36 weeks increases the risk of membrane rupture.
- Care should be taken to avoid incising the fetal vessels at the time of cesarean delivery.

- If vasa previa is recognized during labor in an undiagnosed patient, she should be delivered by urgent cesarean. The placenta should be examined to confirm the diagnosis

3. Postpartum

- Routine postpartum management as for cesarean delivery.
- If the fetus is born after blood loss, transfusion of blood without delay may be life-saving.
- It is important to have O negative blood or type-specific blood available immediately for neonatal transfusion.

**NURSING MANAGEMENT**

- Assess bleeding, color, amount
- Administer iv fluids.
- Administer oxygen.
- Strict vitals and FHS monitoring.
- Prepare patient for caesarean section.
- Reserve blood if (Hct >30%)

**AMNIOTIC FLUID EMBOLISM INCIDENCE**

Amniotic fluid embolism syndrome is rare. Most studies indicate that the incidence rate is between 1 and 12 cases per 100,000 deliveries

**DEFINATION**

An amniotic fluid embolism is rare but serious condition that occur when amniotic fluid, fetal material, such as hair, enters the maternal bloodstream.

The body respond in 2 phases

- ❖ The initial phase is one of pulmonary vasospasm causing hypoxia, hypotension, pulmonary edema and cardiovascular collapse.
- ❖ The second phase sees the development of left ventricular failure, with hemorrhage and coagulation disorders and further uncontrollable hemorrhage

**ETIOLOGY**

- A maternal age of 35 years
- older Caesarean or instrumental vaginal delivery
- Polyhydramnios Cervical laceration or uterine rupture
- Placenta previa or abruption
- Amniocentesis
- Eclampsia
- Abdominal trauma
- Ruptured uterine or cervical veins
- Ruptured membranes

## SIGNS AND SYMPTOMS

- Sudden shortness of breath
- Excess fluid in the lungs
- Sudden low blood pressure
- Sudden circulatory failure Life- threatening problems with blood clotting (disseminated intravascular coagulopathy)
- Altered mental status
- Nausea or vomiting
- Chills
- Rapid heart rate
- Fetal distress
- Seizures
- Coma

## DIAGNOSIS

- Chest X-ray: May show an enlarged right atrium and ventricle and prominent proximal pulmonary artery and pulmonary edema.
- Lung scan: May demonstrate some areas of reduced radioactivity in the lung field.
- Central venous pressure (CVP) with an initial rise due to pulmonary hypertension and eventually a profound drop due to severe hemorrhage.

- Coagulation profile: decreased platelet count, decreased fibrinogen and a fibrinogenemia, prolonged PT and PTT, and presence of fibrin degradation products.
- Cardiac enzymes levels may be elevated
- Echocardiography may demonstrate acute left heart failure, acute right heart failure or severe pulmonary hypertension

**MANAGEMENT**

- Maintain systolic blood pressure > 90 mm Hg.
- Urine output > 25 ml/hr
- Re-establishing uterine tone
- Correct coagulation abnormalities
- Administer oxygen to maintain normal saturation.
- Intubate if necessary.
- Initiate cardiopulmonary resuscitation (CPR) if the patient arrests. If she does not respond to resuscitation, perform a cesarean delivery.
- Treat hypotension with crystalloid and blood products.
- Consider pulmonary artery catheterization in patients who are haemodynamically unstable. Continuously monitor the fetus.
- trauma to the uterus must be avoided during maneuvers such as insertion of a pressure catheter or rupture of membranes.
- Incision of the placenta during caesarean delivery should also be avoided

**NURSING MANAGEMENT**

- ✓ Give immediate and vigorous treatment.
- ✓ Give oxygen by face mask.
- ✓ Maintain normal blood volume through administration of plasma and intravenous fluids.
- ✓ Prevent development of disseminated intravascular coagulation (DIC). Serious complications can occur.
- ✓ Administer whole blood and fibrinogen.
- ✓ Monitor the patient's vital signs.
- ✓ Deliver the fetus as soon as possible

**OBSTETRIC SHOCK**

- Shock is a critical condition and a life threatening medical emergency.
- Shock results from acute, generalized, inadequate perfusion of tissues, below that needed to deliver the oxygen and nutrients for normal function

## ETIOLOGY

- Hypovolemia (Hemorrhage (occult /overt)
- Hyperemesis
- Diarrhea
- Diabetic acidosis.
- Peritonitis
- Burns
- Sepsis
- Cardiogenic (cardiomyopathies, obstructive structural, obstructive non -structural, dysrhythmias).
- Anaphylaxis
- Distributive (Neurogenic- spinal injury, regional anesthesia

## DIAGNOSIS

There are no laboratory test for shock

- A high index of suspicion and physical signs of inadequate tissue perfusion and oxygenation are the basis for initiating prompt management.
- Initial management does not rely on knowledge of the underlying cause.

## INITIAL MANAGEMENT

- Maintain ABC
- Airway should assured - oxygen 15lt/min.
- Breathing – ventilation should be checked and support if inadequate
- Circulation- (with control of hemorrhage) – Two wide bore canulla – Restore circulatory volume  Reverse hypotentionwith crystalloid. – Crossmatch.
- Arrange and give blood if necessary.
- See for response such as , vital sign

## HYPOVOLEMIC SHOCK

The normal pregnant woman can withstand blood loss of 500 ml and even up to 1000 ml during delivery without obvious danger due to physiological cardiovascular and haematological adaptations during pregnancy.

## ETIOLOGY

Antenatal – Ruptured ectopic pregancy , Incomplete abortion ,Placenta previa – Placental abruption , Uterine rupture

Post partum – Uterine atony, Laceration to genital tract,Chorioamnionitis – Coagulopathy, Retained placental tissue.

## SIGN AND SYMPTOMS

Mild symptoms can include:

- headache
- fatigue
- nausea
- profuse sweating

Dizziness Severe symptoms, include:-

- cold or clammy skin
- pale skin
- rapid, shallow breathing
- rapid heart rate
- little or no urine output
- confusion
- weakness
- weak pulse
- blue lips and fingernails
- Light-headedness
- loss of consciousness

## MANAGEMENT

- Basic shock management then treat specific cause.
- Laparotomy for ectopic pregnancy
- Suction evacuation for incomplete abortion
- Management of uterine atony
- Repair of laceration
- Management of uterine rupture – Stop oxytocin infusion if running
- Continuous maternal and fetal monitoring

i. Emergency laparotomy with rapid operative delivery

ii. Cesarean hysterectomy may need to perform if hemorrhage is not controlled.

iii. Management of uterine inversion. – Replacement of the uterus needs to be undertaken quickly as delay makes replacement more difficult.

iv. Administer tocolytics to allow uterine relaxation. – Replacement under taken ( with placenta if still attached)-manually by slowly and steadily pushing upwards, with hydrostatic pressure or surgically

## CARDIOGENIC SHOCK

- Cardiogenic shock in pregnancy is a life- threatening medical condition resulting from an inadequate circulation of blood.
- Pregnancy puts progressive strain on the heart as progresses.
- Preexisting cardiac disease places the parturient at particular risk.
- Cardiac related death in pregnancy is the second most common cause of death in pregnancy

## SIGN AND SYMPTOMS

- Chest pain
- Nausea and vomiting
- Dyspnoea
- Profuse sweating
- Confusion/disorientation
- Palpitations
- Faintness/syncope

- Pale, mottled, cold skin with slow capillary refill and poor peripheral pulses.
- Hypotension (remember to check BP in both arms in case of aortic dissection).
- Tachycardia/bradycardia.
- Raised JVP/distension of neck veins.
- Peripheral oedema.
- Quiet heart sounds or presence of third and fourth heart sounds.
- Heaves, thrills or murmurs may be present and may indicate the cause, such as valve dysfunction.
- Bilateral basal pulmonary crackles or wheeze may occur.
- Oliguria

**MANAGEMENT**

- Re-establishment of circulation to the myocardium
- Minimising heart muscle damage and improving the heart's effectiveness as a pump.
- Administer Oxygen (O2) therapy to reduces the workload of the heart by reducing tissue demands for blood flow.
- Administration of cardiac drugs such as Dopamine, dobutamine, epinephrine, norepinephrine,

**SEPTIC SHOCK**

- This is sepsis with hypotension despite adequate fluid resuscitation. To diagnose septic shock following two criteria must be met.
- Evidence of infection through a positive blood culture.
- Refractory hypotension- hypotension despite of adequate fluid resuscitation.

**ETIOLOGY**

- Post caesarean delivery
- Prolonged rupture of membranes
- Retained products of conception
- rupture membrane
- Intra-amniotic infusion
- Water birth
- Retained product of conception

- Urinary tract infection
- Toxic shock syndrome
- Necrotizing Fasciitis

## SIGN AND SYMPTOMS

- Abdominal pain – Vomiting – diarrhea
- Signs of sepsis – Tachycardia ,Pallor
- Clamminess – Peripheral shutdown
- Systemic inflammation – Fever or hypothermia
- Tachypnea
- Cold peripheries
- Hypotension
- Confusion
- Oliguria
- Altered mental state

## MANAGEMENT

- Transfer to a higher level facility.
- Invasive monitoring will inevitably but necessary
- Obtain blood culture , wound swab culture and vaginal swab culture.
- Start broad spectrum antibiotics.
- Removal of infected tissues.

## ANAPHYLYTIC SHOCK

A serious rapid onset of allergic reaction that is rapid onset and may cause death

It is a relatively uncommon event in pregnancy but has serious implications for both mother and fetus.

## ETIOLOGY

Pharmacological agent- penicillin group of drugs.

i. Insect stings

ii. Foods

iii. Latex

**SIGN AND SYMPTOMS**

- Cutaneous – Flushing, pruritus, urticaria , rhinitis, conjunctiva erythema, lacrimation.
- Cardiovascular – Cardiovascular collapse, hypotension, vasodilation and erythema, pale clammy cool skin, diaphoresis, nausea and vomiting
- Respiratory – Stridor, wheezing, dyspnea, cough, chest tightness, cyanosis.
- Gastrointestinal – Nausea vomiting , abdominal pain , pelvic pain .
- Central nervous system – Hypotension – collapse with or without unconsciousness, dizziness incontinence – Hypoxia – causes confusion

MANAGEMENT

Immediate

- Stop administration of suspected agent and call for help
- Airway maintenance
- Circulation – Give epinephrine IM and repeat every 5-15min in titrated until improvement. In severe hypotension intravenous epinephrine should be given.
- Rapid intravascular volume expansion with crystalloid solution.

Secondary

- If hypotension persist alternative vasopressor agent should use. – Atropine if persistent bradycardia
- If bronchospasm persist nebulize with salbutamol
- Antihistaminic
- Steroids
- All patient with anaphylactic shock should referred to critical care

**DISTRIBUTIVE SHOCK**

In distributive shock there is no loss in intravascular volume or cardiac function.

The primary defect is massive vasodilation leading to relative hypovolemia, reduced perfusion pressure, so poorer flow to the tissues.

ETIOLOGY

Spinal injuries- Neurogenic shock

**SIGN AND SYMPTOMS**

- Hypotension
- Bradycardia
- Hypothermia
- Shallow breathing
- Nausea vomiting
- No response to stimuli
- Unconscious
- Blank expression of patient

**MANAGEMENT**

- Resuscitation
- Vasopressor agent and atropine may required in management because spinal injury leads bradycardia due to unopposed vagal stimulation.
- Anesthesia -High spinal block
- Basic ABC management – θ Ventilation if needed θ Administer iv fluids θ Iv steroid such as methylprednisolone
- Immobilize the patient to prevent further damage

**UTERINE INVERSION**

It occurs when the placenta fails to detach from the uterus as it exits, pulls on the inside surface, and turns the organ inside out.

Uterine inversion is a potentially fatal childbirth complication with a maternal survival rate of about 85%

The incidence is about 1 in 20,000 deliveries.

## ETIOLOGY

The exact cause of uterus inversion is unclear.

The most likely cause is strong traction on the umbilical cord, particularly when the placenta is in a fundal location, during the third stage of labor.

## DIAGNOSIS

Prompt diagnosis is crucial and possibly lifesaving. Some of the signs of uterine inversion could include:

- The uterus protrudes from the vagina.
- The fundus doesn't seem to be in its proper position when the doctor palpates (feels) the mother's abdomen.
- The mother experiences greater than normal blood loss.
- The mother's blood pressure drops (hypotension).
- The mother shows signs of shock (blood loss).
- Scans (such as ultrasound or MRI) may be used in some cases to confirm the diagnosis

## MANAGEMENT

i. Before shock
ii. Urgent manual replacement
iii. After replacement, the hand should remain inside the uterus until the uterus become contracted by parentraloxytocics.
iv. The placenta should be removed manually only after the uterus becomes contracted.
v. Usual treatment of shock including blood transfusion should be arranged.
vi. After shock
vii. Morphine 15mg IM, dextrose saline drip and arrangement of blood transfusion.
viii. Push the uterus inside the vagina if possible and pack the vagina with roller gauze

ix. Raised foot end of bed.

x. Replacement of uterus under general anaesthesia to be done.

xi. Emergency hysterectomy (surgical removal of the uterus) in extreme cases where the risk of maternal death is high.

**NURSING MANAGEMENT**

- Monitor for signs of hemorrhage and shock and treat shock
- Prepare patient to reposition the uterus to the correct position via the vagina or lapr0tomy if unsuccessful.

**RUPTURE UTERUS**

The most serious complication in midwifery and obstetrics.

It is often fatal for the fetus and may also be responsible for the death of the mother.

**DEFINITION**

Disruption in the continuity of the all uterine layers( endometrium, myometrium and serosa) any time beyond 28 weeks of pregnancy is called rupture of uterus.

**INCIDENCE**

The prevalence widely varies from 1 in 2000 to 1 in 200 deliveries.

**TYPES OF TEAR (RUPTURE) COMPLET E INCOMPLET E**

**Complete rupture:-**

- The peritoneum tears and the contents of the mother's uterus can spill into her peritoneal cavity.
- It is suggested that delivery via cesarean section (C- section) should occur within approximately 10 to 35 minutes after a complete uterine rupture occurs.
- The fetal morbidity rate increases dramatically after this period

Incomplete:-

- The mother's peritoneum remains intact.

- The peritoneum acts as a channel for blood vessels and nerves.
- An incomplete uterine rupture is significantly less dangerous with fewer complications to the delivery process

**ETIOLOGY**

It is further divided into:

- Spontaneous
- Scar rupture
- Iatrogenic

**Spontaneous**

During pregnancy-

- Previous damage to the uterine walls following D& C procedure.
- Manual removal of placental
- Thin uterine wall
- Congenital malformation of uterus.

During labour-

- Obstructive rupture due to obstructed labour
- Non obstructive rupture due to weakening of walls due to repeated previous birth

**Scar rupture**

**Classical caesarean or hysterectomy scar**

- Iatrogenic

During pregnancy-

- Injudicious administration of oxytocin
- Use of prostaglandin for induction of abortion or labour

- Forcible external version
- Fall or blow on the abdomen.

During labour

- Internal podalic version.
- Destructive operation.
- Manual removal of placenta.
- Application of forceps or breech extraction through incomplete dilated cervix.
- Injudicious administration of oxytocin for augmentation of labour+

SIGN AND SYMPTOMS

- Abdominal pain and tenderness
- Shock
- Vaginal bleeding
- Undetectable fetal heart beat
- Palpable fetal body parts
- Cessation of contractions
- Signs of intra-peritoneal bleeding
- The most common sign is the sudden appearance of fetal distress during labor.
- Complete laceration of uterine wall.

i. Sharp pain between contractions - Contractions that slow down or become less intense
ii. Recession of the fetal head (baby's head moving back up into the birth canal)
iii. Bulging under the pubic bone (baby's head has protruded outside of the uterine scar) Sharp onset of pain at the site of the previous scar.
iv. Uterine atony (loss of uterine muscle tone)
v. Maternal tachycardia (rapid heart rate) and hypotension

**DIAGNOSIS**

Ultrasonography is probably the safest and most useful imaging technique during pregnancy. sonographic findings associated with includes:

• Extra peritoneal hematoma

• intrauterine bleed

• free peritoneal blood

• empty uterus

• gestational sac above the uterus

• large uterus mass with gas

- Painful bleeding.
- Loss of FHS

**MANAGEMENT**

Principles for the treatment of uterine rupture includes:

- Intensive resuscitation
- Emergency laparotomy
- Broad spectrum antibiotics
- Adequate post operative care

❖ Intensive resuscitation

- Correct hypovolaemia from- # Haemorrhage # Sepsis #Dehydration
- Intravenous broad spectrum antibiotics #Cephalosporin + Metronidazole combination
- Monitor to ensure adequate fluid and blood replacement
- Blood volume expansion may worsen the bleeding from damaged vessel and so the laparotomy should not be delay, once patient condition has improved.

❖ Surgical options

Hysterectomy -Treatment of choice except any other compelling reasons to preserve the uterus # Total # Sub-total

Rupture repair # Occasionally one may be forced to repair # Repair with sterilization

**NURSING MANAGEMENT**

- Monitor for the possibility of uterine rupture.
- In the presence of predisposing factors, monitor maternal labor pattern closely for hyper tonicity or signs of weakening uterine muscle.
- Recognize signs of impending rupture, immediately notify the physician, and call for assistance.
- Assist with rapid intervention.If the client has signs of possible uterine rupture, vaginal delivery is generally not attempted.
- Monitor maternal blood pressure, pulse, and respirations; also monitor fetal heart tones.

i. If the client has a central venous pressure catheter in place, monitor pressure to evaluate blood loss and effects of fluid and blood replacement.
ii. Insert a urinary catheter for precise determinations of fluid balance.
iii. Obtain blood to assess possible acidosis.
iv. Administer oxygen, and maintain a patent airway.
v. Restore circulating volume using one or more IV lines.
vi. Evaluate the cause, response to therapy, and fetal condition

**CORD PROLAPSE**

There are three clinical types of abnormal descent of the umbilical cord by the side of the presenting part:

- Cord presentation
- Occult prolapse
- Cord prolapse

**Cord presentation**

When cord is slipped down below the presenting part and is felt lying in the intact bag of membranes.

**Occult prolapse**

The cord is placed by the side of the presenting part and is not felt by the fingers on internal examination.

**Cord prolapse**

The cord is lying inside the vagina or outside the vulva following rupture of the membranes The incidence of cord prolapse is about 1 in 300 deliveries

**ETIOLOGY**

- Malpresentation- transverse lie & breech.
- Contracted pelvis
- Prematurity
- Twins
- Hydramnios
- Placental factor- minor degree placenta praevia
- Iatrogenic- low rupture of the membranes, manual rotation of the head.
- Stabilising induction

**DIAGNOSIS**

**OCCULT PROLAPSE**

Difficult to diagnose.

- Persistence of variable deceleration of fetal heart rate pattern.

**CORD PRESENTATION**

Feeling the pulsation of the cord through the intact membrane.

**CORD PROLAPSE**

The cord is palpated directly by the fingers and its pulsation can be felt if the fetus is alive.

Cord pulsation may caese during uterine contraction, however returns after the contraction passes away.

**MANAGEMENT**

Protocol is guided by:

- Baby living or dead
- Maturity of the baby

- Degree of dilatation of the cervix

**CORD PRESENTATION**

- Once the diagnosis is made, no attempt should be made to replace the cord.
- If immediate vaginal delivery is not possible or contraindicated , caesarean section is the best method of delivery.
- A rare occasion when multipara with longitudinal lie having good uterine contractions with cervix 7-8cm dilated without fetal distress- watchful competency and delivery by forcep or breech extraction

CORD PROLAPSE

- Living baby
- Immediate take the mother for Caesarean section.
- Immediate safe vaginal delivery if- head is engaged
- Immediate safe vaginal delivery not possible- First Aid
- First aid
- Bladder filling is done to raise the presenting part off the compressed cord.It is done by 400-750ml of NS with a foley's catheter, the ballon is inflated and catheter is clamped.
- Lift the presenting part off the cord.
- Postural treatment- exaggerated and elevated sims position or trendelenburg or knee chest position.
- Replace the cord into the vagina to minimize vasospasm due to irritation.

**Conclusions:**

It was concluded that obstetric emergencies are more common in unbooked cases and women with low socioeconomic status with poor access to antenatal care.

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# HYPERTENSION IN PREGNANCY

## INTRODUCTION:

Hypertension is one of the common disorders of pregnancy and contributes significantly to the maternaland peri-natal morbidity and mortality. Hypertension may appear for the first time during pregnancy as adirectresultofthe gravid stateor asa sign ofunderlyingpathology, whichmaybepre-existing.

Hypertension is the commonest cardiovascular disorders, posing a major Public Health challenge to population in social-economic and epidemiological transition. It is one of the major risk factors for cardiovascular mortality, which account for 20 to 50% of all deaths.

- Hypertension is one of the most common complicationduring pregnancy
- Increased maternal and peri-natal morbidity and mortality
- It is a sign of an underlying pathology that may be pre- existing or appears for the first time during pregnancy that is why it is also called as TOXEMIA OF PREGNANCY

### DEFINITION OF HYPERTENSION IN PREGNANCY

- Hypertension in pregnancy is diagnosed either from an absolute rise in blood pressure or from a relative rise above measurements obtained at booking. [Nima Bhasker]
- The convention for absolute value is a systolic>140 mm Hg or a diastolic > 90 mm Hg. [AnnamaJacob]

- The definition for a relative rise in blood pressure incorporates either a rise in systolic pressure of > 30 mm Hg or rise in diastolic pressure of >15 mm Hg above blood pressure at booking.

[D.C. Dutta]

- Blood pressure must be elevated on at least two occasions and measurements should be made with the woman seated and using the appropriate cuff size.[Pub Med line]

## INCIDENCE

- 5% to 8% of all pregnancies
- Young women
- First pregnancy
- Twin pregnancy
- Previous pre-eclamptic pregnancy
- Diabetes mellitus
- Chronic hypertension.
- Hypertensive disorders of pregnancy in India-5.38%
- Preeclampsia-44%
- Eclampsia-40%
- HEELP syndrome-7%

## PREVALENCE

- Hypertensive disorders during pregnancy occur in women with preexisting primary or secondary chronic hypertension, and in women who develop new-onset hypertension in the second half of pregnancy
- The present study was undertaken to study the prevalence and correlatesof hypertension inpregnancy in a rural area

- A total of 931 pregnant women were included in the present study. Prevalence of hypertension in pregnancy was found to be 6.9%. Maternal age ≥25 years,
- gestational period ≤20 weeks, history of cesarean section, history of preterm delivery, and history of hypertension in previous pregnancy were found to be significantly associated with prevalence of hypertension in pregnancy

## RISK FACTORS OF PREGNANCY INDUCED HYPERTENSION

- Nulliparity
- Pre-eclampsia in a previous pregnancy
- Age >40 years or <18 years
- Family history of pregnancy-induced hypertension
- Chronic hypertension
- Chronic renal disease
- Anti-phospholipid antibody syndrome or inherited thrombophilia

## CLASSIFICATIONOFHYPERTENSIONINPREGNANCY

1) Pregnancy-inducedhypertension(PIH):

a) Withproteinuriaand/oredema:

- ❖ Preeclampsia
- ❖ Eclampsia.

b) Withoutgrossedemaorproteinuria:

- ❖ Gestationalhypertension..

2) Chronichypertensioninpregnancy.Pregnancyisunrelatedtothehypertensivestate:

i) Essentialhypertension.

ii) Renovascularhypertension.

3) Hypertensionworsenedbypregnancy:
   a) Superimposedpreeclampsia.
   b) Supermiposedeclampsia.

## THENATIONALHIGHBLOODPRESSUREEDUCATIONPROGRAM

Workinggroupclassifiedhypertensivediseasesinpregnancy into4groupschronichypertensionpreeclampsiapreeclampsiasuperimposedonchronichypertensionand gestationalhypertension.

Theclassificationofhypertensivediseasesinpregnancyaccordingtothenhbppworkinggroupis asfollows:

1) Gestationalhypertension.
   a) BPof 140by90 mm HG or greaterforthefirsttimeduringpregnancy.
   b) Noproteinuria
   c) BPreturnstonormallessthan12weeksPostpartum.
   d) FinaldiagnosismadeonlyPostpartum

2) Chronichypertension
   - BP140by90mmHDorgreaterbeforepregnancyordiagnosedbefore20weeksgestationnot attributabletogestationaltrophoblastic disease.
   - Hypertensionfirstdiagnoseafter20weeksgestationandpersistentafter12weeksPostpartum.
3) Preeclampsiaoreclampsia:
   a) BPof149/90mmHgorgreaterafter20weeksgestationinawomanwithpreviouslynormalblood pressure and with protein urea.
   b) Eclampsiaisdefinedassuchthatcannotbeattributedtoothercausesinawomanwithpreeclampsia.
4) Superimposedpreeclampsia(onchronichypertension)
   - New onsetproteinuriainawomanwithhypertensionbutnoproteinuriabefore 20weeksgestation.
   - Asuddenincreaseinproteinuriabloodpressureforplateletcountslessthan1lakhinawoman with hypertension and proteinuria before20weeks.

## ESSENTIALHYPERTENSIONINPREGNANCY

Apart from the specific hypertensive disorder in pregnancy (PIH), essential hypertension is themostcommonhypertensivestateinpregnancy.Itsincidencevariesfrom1to3%.**Diagnosis**

Thediagnosticcriteriaof essentialhypertensionare:

1. Raiseofbloodpressuretotheextentof140/90mmHgormoreduringpregnancypriortothe20th week.
2. Persistenceofbloodpressureevenafter3monthsfollowingdelivery.
3. Commonin multiparaand elderlywomen.
4. Presenceofpre-pregnanthypertensionandoftenfamilyhistory..
5. Presenceofhypertensiveretinopathy.

## EffectsOf PregnancyOnTheDisease:

a) Theremaybeamid-pregnancyfallofbloodpressureinabout50%.However,thebloodpressuretends to risein thelast trimesterwhich mayormaynot reach its previous level.
b) Inabout 50%,thebloodpressuretends toriseprogressivelyaspregnancyadvances.

c) Inabout20%, itissuperimposed bypreeclampsia.

d) Rarely,malignanthypertensionsupervenes.

e) In about 30%, there is permanent deterioration of the hypertension following delivery.**RisksToMotherAndFetus**:Inthemilderform,thematernalriskremainsunaltered,butinthesevereformorwhensuperimposedbypreeclampsia,thematernalriskismuchincreased.Thebabiesare likelytobegrowthretardedduetochronicplacentalinsufficiency.Perinatallossisabout10%inthemilderformwithbloodpressurelessthan160/100mmHg.Whenthebloodpressureexceeds160/100mmHg,theperinatallossdoublesandwhencomplicatedbypreeclampsia,it trebles (Dutta, 2001).

**Management:** In mild cases with blood pressure less than 160/ 100 mm Hg, adequate rest, lowsalt diet and sedative (phenobarbital 60 mg one to three times daily) are given. Weekly or morefrequent checkups are needed up to 28 weeks and thereafter weekly.

In severe cases or in cases of superimposed preeclampsia, the patient should be hospitalized andplaced in the treatment protocol as described under preeclarnpsia. Antihypertensive drugs aregiven only when the blood pressure is raised beyond 160/ 100 mm Hg because the diminishedblood pressure may reduce the placental perfusion which may be detrimental to the fetus.Incases where these drugs have been used before pregnancy, care is taken to adjust the dose duringpregnancy especially during midpregnancy, when the blood pressure tends to fall.

Spontaneous labor at term is awaited in mild cases. In severe or complicated cases, termination isdone after 38 weeks by low rupture of membranes with/ without oxytocin drip or by cesareansection. PGEZ is used to make the cervix favorable before low rupture of membranes. Labor ismanagedin a manner similarto that of preeclampsia.

## GESTATIONALHYPERTENSION:

Gestational hypertension is a sustained rise of blood pressure to 140/90 mm Hg or more on atleast two occasions, 4 or more hours apart beyond 20th week of pregnancy, or during the first 24hours after delivery in a previously normotensive woman. It is associated with a much higherincidence of essential hypertension in later life than preeclampsia. Both are like the two phase ofthesame disorder. Essential features of thiscondition are:

- Absenceof anyevidencefortheunderlyingcause ofhypertension.

- Unassociatedwith other evidencesof preeclampsiasuchasedema orproteinuria.
- The blood pressure returns to normal within 10 days following delivery.

The hypertension may be a stress response. Perinatal mortality remains unaffected. Thesepatients are more likely to develop hypertension with the use of oral contraceptives or insubsequentpregnancies.

## PREECLAMPSIA

### Definition:

Preeclampsia is a multisystem disorder of unknown etiology characterized by developmentofhypertensiontotheextentof140/90mmHgormorewithproteinuriainducedbypregnancy after the 20th week in a previously normotensive and non-proteinuric woman(International Society for Study of Hypertension in Pregnancy, 1988).**Incidence:**

- ✓ About58%ofallpregnancies arecomplicatedbyhypertensionandofthesepreeclampsiaaccountsfor80% (Llewellyn Jones, 1990).
- ✓ Itoccursmorefrequentlyinyoungprimigravida andinmothersover35yearsofage.Itisknown to be associated with hydau'diform mole, multiple pregnancy and maternaldiabetes.

## Etiology:

1) The exact nature of the primary event causing PIH is not known. The following arethought to be the possibilities: There is a relative or absolute deficiency of vasodilatorprostaglandin 12 (PG12), synthesized in vascular endothelium and increased synthesis ofthromboxaneA2 ($TxA_2$),apotent vasoconstrictorin platelets.
2) ThereisanincreasedvascularsensitivitytothepressoragentangiotensinII.Thesensitizingsubstances areyet to beexplored.
3) Nitricoxide,whichnormallyrelaxesvascularsmoothmuscle,inhibitsplateletaggregationand preventsintervillousthrombosis,isfounddeficientinpreeclampticclients. Hence, preeclampsia is characterized by complex endothelial cell dysfunction(Dutta,2001).
4) 1n preeclampsia, trophoblastic invasion of the spiral arteries is thought to be inhibited bysomeimmunological mechanism (Roberts andRedman, 1993).

The cause of excessive accumulation of fluids in the extracellular spaces is not clear. Excessiveretention of sodium in the edematous state is probably due to the increased aldosterone, out ofactivationofcorticosteronebyangiotensinII.Diminishedrenalbloodflow,decreasedglomerular filtration rate and increased tubular reabsorptionare responsible for retention ofsodium.

The probable events that contribute to proteinuria are spasm of the afferent glomerular arterioles,anoxic damage to the endothelium of the glomerular tuft, increase capillary permeability andincreasedleakageof proteins tubularreabsorption issimultaneouslydepressed.

## PREDISPOSINGFACTORS:

Thereisincreasedassociationofpre-eclampsiawiththefollowing:

a) Elderlyandyoungprimigravidae.
b) Familyhistoryofpre-eclampsia, eclampsiaor hypertension.
c) Poorandunderprivilegedsector-moreduetoneglectinantenatalcareratherthannutritionalcause.
d) Pregnancycomplicationssuchashydatidiformmole,multiplepregnancypolyhydramnios,Rhincompatibility.
e) Medicaldisorders-hypertension,nephritis,diabetes.
f) Newpaternity.
g) Hereditary-thoughttobesinglerecessivegenedisorder.Cigarettesmokingreducestherisk.

## PATHOPHYSIOLOGY:

The pathological changes that occur in the various organs in severe preeclampsia and eclampsiaarewelldocumented:

1) **Uteroplacental bed:** There is an increased evidence of premature aging of the placenta.Areasof occasionalacuteinfarcts arevisible onthematernal surfaceof theplacenta.
2) There is acute atherosis of spiral arteries with obliteration of the lumen. Intervillouscirculation is impaired to the extent of about one-third, secondary to changes in thematernal blood vessels. This results in placental changes, which are responsible for fetaljeopardy.
3) In the kidneys, there is reduced blood flow and glomerular filtration rate, and

impairedtubularreabsorption or secretaryfunctions.

4) In the blood vessels, there is intense vasospasm.Circulation in the vasa vasorum isimpairedleadingto damageofthevascularwalls includingtheendothelialintegrity.

5) Hemorrhagic necrosis occurs in the liver due to thrombosis of the arterioles. Hepaticinsufficiency seldom occurs because of the reserve capacity andregenerative ability oftheliver.

## CLINICALCLASSIFICATIONOFPREECLAMPSIA:

The clinical classification of preeclampsia is principally dependent on the level of blood pressureformanagement purpose.

i) **Mild preeclampsia** is diagnosed when there is sustained rise of blood pressure of morethan140/90mmHg,butlessthan160systolicor110diastolicwithoutsignificantproteinuriaon two occasions of 6 hours apart.

ii) **Severe preeclampsia** is diagnosed when the blood pressure exceeds 160/100 mm Hg,when there is an increase in the proteinuria (75 g/day) and where edema is marked. Thewoman may complain of frontal headaches, visual disturbances and upper abdominal painwithorwithoutvomiting(imminenteclampsia).Reducedplateletcount(lessthan100,000/pL), elevated liver enzymes, retinal hemorrhages or papilledema, pulmonary edemaandintrauterinegrowth retardation ofthefetus arealso seen.

## CLINICALTYPES:

The clinical classification of pre-eclampsia is arbitrary and is principally dependent on the levelof blood pressure for management purpose. Proteinuria is important, however, level of bloodpressureis considered moresignificantto predict maternal and fetal outcome.

- **Non-severe:**This includes cases of sustained rise of blood pressure of more than 140/90mm Hg but less than 160 mm Hg systolic or 110 mm Hg diastolic without significantproteinuria.
- **Severe:**

i. Apersistentsystolicbloodpressureaboveorequalto160mmHgordiastolicpressureabove 1 10 mmHg

ii. Proteinuria-present

iii. Oliguria(<4OOmL/24h

iv. Plateletcountlessthan100,000/mm3

v. Elevatedliverenzymes

vi. Cerebralorvisualdisturbances

vii. Persistentsevereepigastricpain

viii. Retinalhemorrhages, exudatesorpapilledema

ix. Intrauterinegrowthrestrictionofthefetus.

x. Pulmonaryedema.

xi. Serumcreatinine>].lmg/dL.

## CLINICALFEATURES:

- Preeclampsiafrequentlyoccursinprimigravida (70%).
- Itisoftenassociatedwithobstetrical-medicalcomplicationssuchasmultiplepregnancies,polyhydramnious, pre-existinghypertension,diabetes, etc.
- Theclinicalmanifestationsusuallyappear afterthe20th week.
- The onset is usually insidious and the symptoms run a slow course. On rare occasions,however,the onset becomes acuteandfollows arapidcourse.
- Edema is seen in approximately 80% of women with preeclampsia. It may appear rathersudden and be associated with a rapid weight gain. Clinical edema may be mild or severein nature and the severity is related to the worsening of the preeclampsia. The edema pitson pressure and may be found in the following anatomical areas such as face, hands,lowerabdomen, vulva, sacralarea, pretibial region,ankles andfeet.
- Elevated blood pressure: More than 140/90 mm Hg in mild cases and above 160/ 110mm Hgin severepreeclampsia.

## AlarmingSymptomsAndSigns

The following symptoms and signs may be evident either singly or in combination. These areusuallyassociated with acute onset ofthesymptoms:

1) Headacheovertheoccipitalorfrontalregion.
2) Disturbedsleep.
3) Diminished urinaryoutput (less than 500mLin 24 hour).
4) Epigastricpainassociatedwithvomiting,attimescoffeecoloredduetohemorrhagicgastritisor subcapsular hemorrhagein theliver.
5) BlurringordimnessofVisionorattimescompleteblindhess(visionisusuallyregained4-6h

followingdelivery)

6) Arapidgaininweightofmorethan2.5kg(51b)amonthormorethan500g(1lb.)aweekin thelater months of pregnancy.

7) Visibleedemaovertheanklesonrisingfromthebedin themorning.Suddenandgeneralizededema mayindicate imminent eclampsia.

8) Scantyliquororgrowthretardationofthefetus.Preeclampsiaisprincipallyasyndromeofsigns andwhen symptoms appear it is usuallylate.

## INVESTIGATIONS:

a) **Urine:**Proteinuria is the last feature of pre-eclampsia to appear. It may be trace or attimes copious so that urine becomes solid on boiling (10315 g/L). There may be fewhyaline casts, epithelial cells or even few red cells. 24 hours urine collection for proteinmeasurementis done(described earlier).

b) **Ophthalmoscopic examination:**In severe cases there may be retinal edema, constrictionof the arterioles, alteration of normal ratio of vein: arteriole diameter from 3:2 to 3:1 andnickingof theveinswherecrossed bythe arterioles. Theremaybe hemorrhage.

c) **Blood values:**The blood changes are not specific and often inconsistent. A serum uricacid level (biochemical marker of pre-eclampsia) of more than 4.5 mg/ dL indicates thepresence of pre-eclampsia. Blood urea level remains normal or slightly raised. Serumcreatininelevelmaybemorethan1mg/dL.Theremaybethrombocytopeniaandabnor malcoagulationproblemofvaryingdegrees.Hepaticenzymelevelsmaybeincreased.
Coagulation profile is not necessary when platelet count and liver enzymes arenormal.

d) **Antenatal fetal monitoring:** Antenatal fetal wellbeing assessment is done by clinicalexamination, daily fetal kick count, ultrasonography for fetal growth and liquor pockets,cardiotocography,umbilical arteryflowvelocimetryand biophysical profile.

e) **Renal:**Urine quantity reduced. Proteinuria of > 300 mg/ 24 h.Urinedipstick >1 +.

f) **Blood:**

- ✓ Protein/creatinineratio> 0.3.
- ✓ Serumcreatinine>1.2mg/dL.
- ✓ Serumuricacid >5.6mg/dL.

- ✓ Plateletcount <100,000/mm3.

- ✓ ElevatedPT orPTT.
- ✓ Decreeasedfibrinogen.
- ✓ Peripheralsmear-abnormal.Indirectbilirubin>1.2mg/dLLactatedehydrogenase >60U/L.

**g) Liverenzyme:**

- ✓ SerumAST>70U/L.

## COMPLICATIONS:

**I. Immediate:**

**i. Duringpregnancy:**

- ❋Eclampsia2% (morein acutecases).
- ❋Placentalabruptionandintrauterinefetaldeath.
- ❋ Oliguriaandanuria.
- ❋Dimnessofvision and blindness.
- ❋Pretermlabor.
- ❋Hemolyticanemia,elevatedliverenzymes,lowplateletcount(HELLP)syndrome.

**ii. Duringlabor:**

- ❋Eclampsia.
- ❋PostpartumhemorrhagemayberelatedWithcoagulationfailure.

**iii. Puerperium:**

- ❋Eclampsia(usuallyoccurswithin 48 hour).
- ❋Shock(relatedto reducedsodium andchloride).
- ❋Sepsis(duetoincreasedincidenceofinductionandoperativedeliveryandlowvitality).

**II. Remote:**

i. Residualhypertension:Thehypertensionmaypersistevenafter6monthsfollowingdeliveryin about 50% ofcases.

ii. Recurrentpreeclampsia:Thereis25%chanceofpreeclampsiatorecurinsubsequentpregnancies.

## PREVENTIVEMEASURES:

Preeclampsiaisnotaverypreventabledisease.However,somespecific‘high-risk'factorsleadingto preeclampsiamaybeidentitied in individuals. Theseare:

- Primigravida,especiallyyoungandelderly.
- Poornutrition.
- Lowlevelofeducation.
- Presenceofcomplicatingfactorslikepre-existinghypertension,twins,polyhydramnios,clinicalor latent diabetes andnephritis.
- History of preeclampsia or hypertension in the familyorin previous pregnancy.
- Abnormalweightgain.
- Risingserumuricacidlevel.

Thefollowingregimeisenforcedinsuchpatientsinanattempttopreventortodetectearlymanifestations of preeclampsia:

- Regular antenatal check-up at frequent intervals from the beginning of pregnancy todetectattheearliest,therapidgaininweightoratendencyofrisingbloodpressureespeciallythe diastolic pressure.
- Advise to take adequate rest in bed on her left side at least for 2 hours in the afternoonfromthe20th weekof pregnancyonwards.
- Low dose aspirin (60 mg) daily, beginning early in pregnancy to potentially high-riskwomen.It selectivelyreduces platelet thromboxaneproduction.
- Calciumsupplementation(2g/day) reducestheriskofpreeclampsia.
- Antioxidants,vitamin Cand Efrom 16to 22weeks onwards.
- Well-balanceddiet,which isrich inprotein.

## MANAGEMENTANDNURSE’SROLE:

Astheetiologyofpreeclampsiaremainsunclear,themanagementismostlyempiricalandsymptomatic.

Objectivesofcareareto:

- Provide rest and a tranquil environment
- .Monitorthecondition .
- Preventeclampsia andothercomplications.

- Deliver a healthy baby in optimal time with minimum maternal morbidity.

**a) Rest:**

- The woman should be in bed preferably in left lateral position as much as possibletolessen theeffects of venacavalcompression.
- Rest is to be continued until all the preeclamptic manifestations subside. Whenproteinuria develops in addition to hypertension, the risks to the mother and fetusareconsiderablyincreased.
- Admission to the hospital is required at this stage to monitor and evaluate thematernalandfetal condition.
- Rest increases the renal blood flow causing increased diuresis, increases uterinebloodflowcausingimprovedplacentalperfusionandreducesthebloodpressure.

**c) Diet:**

- Asforanypregnantwoman,adietrichinprotein,fiberandvitaminsarerecommended.
- Saltisneitherrestrictednorforced.
- Omission of salty food and extra salt in the dish is desirable. Fluids need not berestricted.
- Total calorie maybe approximately1,600 perdaywithabout100gproteins.
- There is some evidence to suggest that prophylactic fish oil in pregnancy may actasanantiplateletagent,therebypreventinghypertensionandproteinuricpreeclampsia(Redmanand Roberts, 1993).
- Calcium supplementation is also thought to be helpful as low serum calciumlevelis associated with hypertension.

**d) <u>Antihypertensive</u>Therapy:**

- Bloodpressureusuallycomesdown with adequaterest and sedatives.
- Antihypertensive have limited value in controlling the rise of blood pressure duetopreeclampsia.
- They are used to prevent increase in blood pressure and the development ofsevere preeclampsia, especially when the diastolic pressure is over 110 mm Hg,andwhen associated with proteinuria.

- The common oral drugs used are either methyldopa (Aldomet) 0.5-2 g/day or anadrenoceptor antagonist (aand Ba blocker)--labetalo] 200 mg 6-8 hourly. If bloodpressure is not under control, nifedipine, a calcium channel blocker 10-20 mgtwiceadayor hydralazine 25 mgtwicedailyareadded.
- ***Sedatives:***Mildsedativesaregiventoreducetheemotionalfacto;thatcontributes to elevation of blood pressure. Phenobarbltone 60 mg or diazepam 5mgat bedtimeor morefrequentlyare given.
- Laxative If the woman is constipated, a mild laxative like milk of magnesia fourteaspoonsat bedtime maybegiven.

**e) AbdominalExamination:**

- Abdominal examination is to be carried-out daily for women admitted to thehospital. Any discomfort or tenderness must be recorded and reported to thephysicianimmediatelyas this maybeindicativeofplacentalabruption.
- UpperabdominalpainishighlysignificantandindicativeoftheHELLPsyndrome.

**f) FetalAssessment:**

- Assessment of the fetalwell-being must be done by the use of kick charts,cardiotocographmonitoringand serial ultrasound scans.

g) **Assessment of Treatment:** The effect of treatment should be evaluated by maintainingrecordsof:

- ✓ Bloodpressurefourtimesaday.
- ✓ Statusofedemaand dailyweight.
- ✓ Volume offluid intake and urinaryoutput.
- ✓ Dailyexamination of urinaryprotein.
- ✓ Blood values such as hematocrit, platelet count, uric acid, creatinine and liverfunctiontest onceaweek.
- ✓ Ophthalmoscopicexaminationonadmissionandlaterasneeded.

**FAVORABLE SIGNS:**In favorable cases, there is fall of blood pressure and weight Withsubsidence of edema. Urinary output increases withdiminishingproteinuria.

## TREATMENT:

The definitive treatment for preeclampsia is termination of pregnancy. The aim of treatment is tocontinue the pregnancy if possible, without affecting the maternal prognosis until the fetusbecomesmatureenoughtosurviveintheextrauterineenvironment.Thus,thedurationoftreatmentdepends on:

1. Severityofpreeclampsia.
2. Durationofpregnancy.
3. Responsetotreatment.

Those women who respond to treatment, if pregnancy is far from term, the treatment iscontinued with weekly assessment. If the pregnancy is near term, the woman is kept in thehospital until completion of 37th week. Thereafter, a decision is taken either to terminate thepregnancy or to wait for spontaneous onset of labor by the due date.For women whose blood pressure remains high, if the pregnancy is beyond 37 completedweeks,terminationisconsidered.Iflessthan38weeks,expectanttreatmentmaybecontinued with careful monitoring of maternal and fetal well-being.

For those women who do not respond to treatment or develop additional symptoms, such asheadache, epigastric pain and oliguria, termination of pregnancy is to be done irrespective ofthe period of gestation. Such patients may need prophylactic anticonvulsant therapy withmagnesium sulfate. Methods of termination are:

A) Induction.

B) Cesareansection.

## IndicationsForInduction:

- Aggravationofpreeclamptic symptomsinspiteofmedicaltreatment.
- Persistenceofhypertensioninspiteofmedicaltreatmentwithpregnancyreaching38weeksormore.
- Acutefulminatingpreeclampsiairrespectiveoftheperiod ofgestation.
- Tendencyof pregnancyto overruntheexpected date.
- Recurrentpreeclampsia with previoushistoryofintrauterine fetaldeath..

- If the cervix is ripe, surgical induction with low rupture of the membranes is the methodof choice. Oxytocin infusion may be added to accelerate the process in selected cases. Ifthe cervix is unripe and the termination is not urgent, intracervical prostaglandin gel(PGEZ) may be inserted to make the cervix ripe when low rupture of membranes can beperformed.Inseverepreeclampsia,sedativesandantihypertensivesareusedduringinduction.

## IndicationsForCesareanSection

- Urgentterminationisindicated,butthecervixisunfavorable(unripeandclosed)forsurgicalinduction.
- Severepreeclampsiawith atendencyto prolongthe induction deliveryinterval
- Associatedcomplicatingfactorssuchaselderlyprimigravida,contractedpelvis,malpresentation,etc.

## MANAGEMENTOFLABOR:

### I) FirstStage:

- The midwife should remain with the pregnant women throughout the course oflabor. Blood pressure tends to rise during labor and convulsions (intrapartumeclampsia)mayoccur.
- Itisessentialtodocumentbloodpressureandurinary output.Fluidbalanceshouldbemonitored carefully.
- The patient should be in bed and sedatives (injection pethidine) should be given atintervals.Marked deviations shouldbenoted andmedicalassistancesought.
- The pregnant women should be made as comfortable as possible by providinggeneralnursingcare.
- Vital signs: Blood pressure and pulse are measured half hourly. Measurement ofthe mean arterial pressure (MAP) is recommended because of the hemodynamicchangesineclampsia.Respiratoryrateandlevelofconsciousnessmust beassessed periodically. Examination of the optic fundi can give an indication ofcerebraledema.Cerebralirritabilitycanbeassessedbythedegreeofhyperreflexiaor

the presenceof clonus.

- Fluid balance: Intravenous fluids are administered using infusion pumps and therecommended infusion rate is 85 mL/h. Because of the reduced intravascularcompartmentinpreeclampsia,poorlycontrolledfluidbalancecanresult incirculatory overload, pulmonary edema, adult respiratory distress syndrome andultimately death.A urinary catheter isinsertedandurine outputis measuredhourly. A quantity about 30 mL/h reflects adequate perfusion. Urinalysis to detectthepresenceofprotein, ketones andglucoseis done4 hourly.
- Pain relief: Epidural analgesia may procure the best pain relief and reduce thebloodpressure.If cesareansectionshouldbedone,epiduralanesthesiaisthebest.
- Fetal condition: The fetal heart rate (FHR) should be monitored continuously anddeviationsfrom the normal must be reportedand actedon.

## II) SecondStage:

- Whenthesecondstagecommences,theobstetricianandpediatricianshouldbenotified.Themidwifewill continueto care forthemother.
- Duration of the second stage is usually shortened by the application of a forceps orventouse.Dependingonthebloodpressurereading,thewomanissedatedimmediately following delivery of the baby with intramuscular morphine to preventpostpartumeclampsia.
- Usuallyblood pressuredropsafter delivery.

## III) ThirdStage:

- ErgometrineandSyntometrineshouldnotnormallybeusedastheycancauseperipheralvasoconstriction and increaseblood pressure.
- In the presence of severe hemorrhage, Methergine intramuscularly or Syntocinon inthedrip maybegiven.

## IV) Puerperium:

- The maternal condition should continue to be monitored at least every 4 hours for thenext48 hours, the period duringwhich convulsions usuallyoccur.
- Tabletphenobarbitone60mginrepeateddosescanproduceeffectivesedation.Hypotensive drugs may be prescribed, if the diastolic pressure is raised beyond 100mm Hg. Thewoman iskept in the hospital until the blood pressure reaches a safeleveland

proteinuriadisappears.

## SignsOfImpendingEclampsia:

Thefollowingsignsandsymptomswillalert the midwifetotheonsetofeclampsia:

- Asharprise inbloodpressure.
- Diminishedurinaryoutput which is dueto acutevasospasm .
- Increaseinproteinuria.
- Headachewhichisusuallysevere, persistentandfrontalinlocation.
- Drowsinessorconfusionduetocerebraledema.
- VisualdisturbancessuchasblurringofvisionorflashinglightsduetoretinaledemaoEpigastricp ain due to liveredemaand impairmentof liverfunction.
- Nauseaandvomiting.

Themidwifeshould bealert to anyoftheseand summon medical help immediately.

## ECLAMPSIA:

Preeclampsia when complicated with convulsion and/or coma is called eclampsia. The termeclampsia is derived from a Greek word meaning 'like a flash of lightning'. It may occur quiteabruptly without any warning manifestations. In majority (over 80%), the disease is preceded byfeatures of severe preeclampsia. Thus, it may occur in women with preeclampsia or in

womenwhohavepreeclampsiasuperimposedonessentialhypertensionorchronicnephritis.**Incidence**:

- ❋The incidence varies widely from country to country and even between different zones inthesame country.
- ❋In the developed countries, its prevalence is estimated to be around 1 in 2,000 deliveries.In the developing countries, particularly in the rural areas, it contributes significantly tothematernal deaths.
- ❋The hospital incidence in India ranges from 1 in 500 to 1 in 30. It is more common inprimigravida (75%), five times more common in twins than in singleton pregnancies andoccursbetween the 36thweek andterm in morethan 50%(Dutta,2001).

**Pathophysiology**: Since eclampsia is a severe form of preeclampsia, the histopathologicalandbiochemicalchangesaresimilar althoughintensifiedthan thoseof preeclampsia.

**OnsetOFConvulsions**:Convulsionsoccurmorefrequentlybeyond36thweek.Onrareoccasions,conv

ulsion mayoccurinearlymonthsas in hydatidiform mole:

1. Antepartum(50%):Fitsoccur before the onsetof labor. More often, labor startssoonafter and attimes, it isimpossible todifferentiate it fromintrapartum fits.
2. Intrapartum(30%):Fitsoccurforthefirsttimeduringlabor.
3. Postpartum (20%): Fits occur for the first time in puerperium usually within 48 hours ofdelivery.Exceptonrareoccasions,aneclampticpatientalwaysshowspreviousmanifestations of acutefulminating preeclampsiacalledpremonitorysymptoms.

**EclampticConvulsions**:Theconvulsionsare epileptiformandconsistoffourstages.

1) **Premonitory stage the patient becomes unconscious:** There is twitching of the musclesof the face, tongue and limbs. Eyeballs are rolled or turned to one side and become fixed.Thisstagelasts for about30 seconds.
2) **Tonic stage:** The whole body goes into a tonic spasm. The trunk opisthotonus, limbs arefixed and hands clenched.Respiration ceases and the tongue protrudes between the teeth;Cyanosisappears. Eyeballsbecome fixed. Thislasts forabout30 seconds.
3) **Clonicstage:**Allthevoluntarymusclesundergoalternatecontractionandrelaxation.Thetwitchingsstartinthefaceandtheninvolveonesideoftheextremitiesandultimately the whole body is involved in the convul= sion. Biting of the tongue occurs,breathing becomes stertorous and blood-stained frothy secretions fill themouth.Cyanosis graduallydisappears. This stagelasts for1-4 minutes.
4) **Stage of coma:**Following the convulsion, the patient passes on to the stage of coma. Itmay last for a brief period or may persist until another convulsion. At times, the patientappears to be in a confused state following the fit and fails to remember the happenings.Rarelycoma occurs without convulsion.

Thefitsusuallyaremultiple,recurringatvaryingintervals.Whenitoccursinquicksuccession, it is calledstatus eclampsia.Following convulsions, the temperature usuallyrises; pulse, respiration rates and blood pressure are also increased. The urinary output ismarkedlydiminished,proteinuriais pronouncedand the serum uricacid is raised.

**MANAGEMENT:**The patient, if at home or in the peripheral health centers, should be shiftedurgently to the referral hospitals. The patient must be heavily sedated before moving her

to thehospital.Theaims of immediatemanagement in thehospital areto:

- ✓ Clearand maintaintheairway.
- ✓ Preventhypoxia .
- ✓ Preventinjury.
- ✓ Arrestconvulsions.
- ✓ Effect deliveryin 6-8 hours.

The midwife must remain with the mother constantly. In the first instance, all effort is devoted tothepreservation ofthe mother's life:

1. The patient should be placed in a railed cot in an isolated room, protected from noxiousstimuli, which might provoke further fits. The patient is to be positioned in semi proneposition in order to facilitate drainage of saliva and vomit. Side lying position helps tominimize' vena cava compression. If the patient is unconscious, the position should bechanged at intervals to prevent hypostatic pneumonia and bedsore. Airway is maintainedandoxygen administeredto prevent severehypoxia.
2. Detailed history is to be taken from the relatives relevant to the diagnosis of eclampsia-durationofpregnancy,numberof fitsandthemedications administered outside.
3. After the patient is properly sedated, thorough, but quick general, abdominal and vaginalexaminations are done. A self-retaining catheter is introduced and the urine is tested forprotein.Continuousdrainageisestablishedformeasurementoftheurinaryoutput,periodic urinary analysis and for prevention of soiling of the bed due to incontinencelikelyto occur duringfits.
4. Vital signs check (pulse, respiration and blood pressure) is to be done at every 30 minutesand recorded. Progressof labor andFHR mustbe monitored.Urinary output is to benotedhourly.
5. Fluid balance: Crystalloid solution (Ringer's lactate) is started as aiirst choice. Totalfluids should not exceed the previous 24 hours urinary output plus 1,000 mL (insensiblelossthroughlungsandskin).Normally,itshouldnotexceed2L in24hours.Inpreeclampsiaandeclampsia,although thereishypovolemia,thetissuesareoverloaded
6. Anticonvulsanttherapyisgiventocontrolthefitandtopreventitsrecurrence.Magnesiumsulf

ateisthedrugofchoice.Itreducesmotorend-platesensitivitytoacetylcholineandtherebyreducesneuromuscularirritability.Magnesiumsulfateinduces

cerebralvasodilation,dilatesuterinearteriesandinhibitsplateletactivation.Ithasnodetrimental effects on theneonate withintherapeutic level.

**ADMINISTRATIONOFMAGNESIUMSULFATE:**Theregimensgivenbelowmayvarybetweenhospitals:

1) Pritchardregimen:
   - ❖ Loadingdose:4g[Vover3-4minutes(20mLof20%solution)andthen10gdeepintramuscular (1M)
   - ❖ Maintenancedose: 5gIM in alternate buttock4hourly(10 mLof 50%solution).
2) Zuspanregimen:
   - ❖ Loadingdose:4 gIVover 5-10minutes.
   - ❖ Maintenancedose:2 g/hIVinfusion.3
3) Sibairegimen:
   - ❖ Loadingdose:6gIVover20minutes
   - ❖ Maintenancedose:2g/hourIVinfusion.

Repeated injections are given only if the knee jerks are present, urine output exceeds 30 mL/ hand the respiration rate is more than 12 per minute. The therapeutic level of serum magnesium is4-7 mEq/L. Magnesium sulfate is continued for 24 hours after the last seizure. For recurrence oftits,further2 gIVbolus is given over 5minutes in theaboveregimens.

Magnesiumsulfateisnowtherecommendeddrugofchoiceforroutineanticonvulsantmanagement of woman with eclampsia rather than diazepam or phenytoin (Robson and Duley,1996).Magnesium sulfate is superior to all other drugs because it does not depress maternah fetalrespiration within the therapeutic level. The mothg remains conscious and hence cooperation inchildbinandnursingare easier.Complicationsofdeepsedatioandimmobilization areavoided.

High levels of the drug can be toxic and therefore the patellar reflex and respiratory rate oroxygen saturatior levels (pulse oximetry) should be measured hourly In women with oliguria,regular monitoring of serum magnesium level is necessary. Calcium gluconate is the antidote formagnesium toxicityandshould bereadilyavailable.

**ANTIHYPERTENSIVESANDDIURETICS:**

In spite of anticonvulsant and sedative regimen,“ blood pressure remains more than 160/110 mmHg, antihypertensive drugs are administered. Hydralazinei mg IV is given slowly and repeatedafter20minuteswith10mg,ifthereisnoresponse.Thebloodpressureshouldbemonitoredat

every 5 minutes. Hydralazine is repeated whenever the diastolic pressure rises to 110 mm Hg.Alternatively, labetalol is given by slow IV route 20 mg/ h [0' smooth control of blood pressure.Presence of pulmonary edema requires diuretics.In such cases, frusemideis administered indosesof20-40 mgintravenouslyal1drepeated at intervals.

Roleofthe midwife:

1) Thewomanshouldbeplacedinasoundprotectedroomtominimizeauditorystimulation.
2) Eyepadstobeappliedtominimizeopticstimulation.
3) Theroomshould bewell-lighted soasnot tomiss thedevelopment of cyanosis.
4) Bedrailingsto bepaddedin orderto minimizephysicalinjuryduringconvulsion.
5) Patienttobeplacedinsemipronepositionandthepositiontobechangedatevery2hours,ifthepatientisheavilysedatedorindeepcomatoavoidhypostaticpneumoniaandbedsores.
6) KeepFoley's catheterintheurinarybladderandmakechart ofurinaryoutputeveryhour.
7) Minimalhandlingandstimulationinordertoreducetheriskofoccurrenceofanotherconvulsion.
8) MaintainanIV linepatent preferablyina centralvein.
9) Keep atracheotomytrayavailable.
10) Applyathromboelastic stockingto prevent deep vein thrombosis.

**COMPLICATIONSOFECLEMPSIA**:

- ✓ **Injuries:**Tonguebite,injuries duetofallingoutofbed.
- ✓ **Cardiovascular:**Vasospasm,pulmonaryembolism.
  - ✓ **Renal**:Oliguria,renalfailure.
- ✓ **Hematological:**Hypovolemia,hemoconcentration, thrombocytopenia,DIC.
- ✓ **Neurological:**Cerebraledema,cerebralhemorrhage.
- ✓ **Hepatic:**Subcapsularhematoma,Hepaticnecrosis.
- ✓ **Respiratory:**Pneumonia(aspiration,hypostaticorinfective).
- ✓ **Sensory:**Disturbed vision duetoretinal edemaordetachment (usuallyreversible).
- ✓ **Puerperal:**Sepsis,psychosis.

✓ **Fetal:**Placentalabruption,intrauterinegrowthretardation,fetaldistress,intrauterinedeath.

**Mortality:Maternal**mortalityisveryhighinIndia andvariesfrom2% t0 30%, andmore inruralhospitals. Causes for maternal death are:

- Cardiacfailure.
- Pulmonaryedema.
- Aspirationand/orsepticpneumonia.
- Cerebralhemorrhage.
- Anuria.
- Pulmonaryembolism.
- Postpartum shock.
- Puerperalsepsis.

**Prevention:** In majority of cases, eclampsia is preceded by a severe preeclampsia. Hence, theprevention of eclampsia rests on early detection and effective institutional management withindicioustermination ofpregnancy.However,eclampsiaCanoccurbypassingthepreeclamptic state and as such, it is not always a preventable condition. Adequate sedationand/or prophylactic anticonvulsant therapy soon after delivery of the baby in preeclampsiaandmeticulousobservationfor24-48hoursaredelinitestepstopreventpostpartumeclampsia.

**FutureManagement:**ThereisnoindicationthatPIHcauseslaterhypertensivedisease,butitcanleaveaninherentdispositiontowardhypertension.Womenwithahistoryofpreeclampsia before 32 weeks gestation have 5% risk of recurrence by this gestational ageand15%risk of recurrenceoverall.

Usually the blood pressure returns t7o normal within several weeks, but the proteinuria maypersist for a longer period. The obstetrician will examine the mother for about 6 months afterdelivery and if all is well, she will be advised to seek medical advice as soon as a subsequentpregnancyoccurs.

## HELLPSYNDROME:

The syndrome of hemolysis (H), elevated liver enzymes (EL) and low platelet count (LP) is arare complication of PIH. The condition was first described by Weinstein in 1982. It isgenerallythoughttorepresentavariantofthepreeclampsia-eclampsiasyndrome.Thissyndrome is manifested by nausea, vomiting, epigastric or right upper quadrant pain alongwithbiochemicalandhematologicalchanges.Itisobservedin10-15%ofthosewithpreeclampsia and eclampsia. Treatment is immediate delivery and management as that foracute fulminantpreeclampsia.Pregnanciescomplicated by HELLPsyndrome havebeenassociated with both poor maternal and 'poor fetal outcome. Serious maternal morbidityincludesdisseminatedintravascularcoagulation(DIC),a/juterenalfailure,pulmonaryedema, subcapsular liver hematoma and retinal detachment (Sibai et al., 1993). Infants whosemothers have the syndrome are often small for gestational age and at risk for perinatalasphyxia. The affected lowbirth weight babies have relatively high incidences of leukopenia,neutropeniaand thrombocytopenia (Harms et aL,1995).

## Diagnosis

The variety of signs and symptoms makes diagnosis difficult. Hypertension and proteinuriamay be absent or slightly elevated. Upper abdominal pain is a common manifestation of thedisorderwhichshouldbeinvestigatedOccasionally,thepresenceofthissyndromeisassociated with hypoglycemia leading to coma, severe hyponatremia and cortical blindness.Laboratoryfmdings mayshow the following:

1. **Hemolysis**:AbnormalbloodpictureIncreasedbilirubin(>20umol/L)Increasedlacticdehydrogenase(LDH) >600IU/L).
2. **Elevatedliverenzymes:**Increasedserumglutamicoxaloacetictransaminase(SGOT)/aspartateaminotransferase(AST)>72MILIncreasedlacticdehydrogenase >6001U/L.
3. **Low platelets: <**100,000/L

**Complications:**Sub capsular hemorrhage of the liver is a rare, but potentially fatal complicationof the HELLP syndrome. The condition usually presents with severe epigastric pain that maypersistforseveralhoursandinaddition,thewomanmaycomplainofneckandshoulderpain.

**Treatment**: Women with HELLP syndrome should be admitted to a hospital with facilities forintensive care. In pregnancies less than 34 weeks gestation, conservative treatment is given usingplasmavolumeexpandersandvasodilators.Intermpregnanciesandwherethereisadeterioratingmaternal orfetal condition, immediatedeliveryis recommended.

## NURSING THEORY

## Ramona T Mercer's Maternal Role Attainment Theory

## INTRODUCTION

Ramona T mercer is the only theorist whose work has been exclusively concerned with understanding the process of child bearing who is included in a collection considering the work of map: theorists in nursing Reva Rubin was her Mentor and she looked the role attainment beyond the period to 12 months postpartum. Memer has undertaken theory building and research in two main areas, they are:

1. 'Ihe effects of antepartum stress
2. Attainment of the maternal role.

**The effects of ante partum stress on the family:**Ante-partum stress is described as resulting from a combination of negative life events and the level of risk associated with pregnancy. Ante partum stress is defined as a complication of pregnancy or at risk condition and negatively perceived life events. The family is defined as systems which include subsystems-individual within the overall family system. 'These models consider antenatal stress in relation to individual and family functioning. By testing this model regarding variables which are of particular importance in predicting the experience of antenatal stress on family functioning may be utilized in determining priority areas for action by midwives.

**Attainment of the maternal role**: Mercer has systematically researched the field of maternal role attainment and developed a complex model about factors impacting on maternal role development over a time. In maternal role there is an interactional and developmental process occurring over a period oftime. doting which the mother becomes attachment with her infant and care taking tasks involved. Mother will develop confincidence. competence, in the maternal role by supportive system of family and social. Here mother will identify herself as in maternal role

Core self. mothering role. active participation, and self identity are the main assumptions in her research Model used in her theory: Mercer's model of maternal role attainment is placed within Bronfenbrenner's

{1979}Nested circles of the macro system, exosystem, and mircosystem.

Macro system is generally explained about cultural consistencies.

Exosystem is including day care, parents work setting and school.

Microsystem is close to maternal role attainment. It includes mate relationship [Father, mother, relationship],

family functioning, social support and stress.

**Concepts used in her theory:**

**Person**. Mercer refers a person here as a self or core self. It has the concepts of empathy. self concept. maturity, flexibility, attitudes, and pregnancy role attainments.

**Health:** It is a mother's and father's perception of their health, current health' health outlook m susceptibility to illness, health worry concern, sickness orientation. and rejection of the sick role. The health

status of the family is negatively affected by ante partum stress.

**Environment**: Mercer does not define environment. She does however. address the individual, culture, mate and family and or support network as it relates to maternal role attainment.

**Nursing**:According to mercer obstetrical nursing is the diagnosis and treatment of women's and men's response to actual or potential health problems during pregnancy, child birth &post partum period

## SUMMARY:

By the end of the seminar the students will be able gain in depth knowledge about

hypertensivedisorders in pregnancy, develops positive attitude towards the management of preeclampsia,eclampsia, HELLP syndrome and put in practice the treatment for hypertensive disorders inpregnancy.

## CONCLUSION:

By the end of the session the students will have positive attitude towards hypertension one of thecommon disorders of pregnancy and contributes significantly to the maternal and perinatal morbidity andmortality.

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# CERVICAL DYSTOCIA

## INTRODUCTION

### DYSTOCIA

- Difficult labor
- Slow labor progess

In cervical dystocia ,the cervix fails to dilate during labour. Failure of cervical dialation can be to previous cone biopsy or cauterization for cervical dysplasia. Other reason for failure to dilate include truma .sometimes ,if there are unco-ordinated uterine contractions then the failure of cervical dilation may be secondary to this and this should respond to oxytocin if dystocia continues despite this then the infant will need to be delivered by caesarean section .

❖ **DEFINITION**

❖ ACCORDING TO WILLIAM C.SHIEL

1) Failure of the cervix to dilate within a reasonable time in spite of good regular uterine contractions

**OR**.

2) Cervical dystocia is a condition where the external OS fails to dilate in spite of the normal behavior of the uterine contractions and where all other causes of dystocia are excluded

**OR**

3) Cervical dystocia is a difficult labor and delivery caused by mechanical obstruction at the cervix .dystocia comes from the greek word "dys' meaning 'Difficlt, painfull disordered ,abnormal'+'tokos'meaning 'birth'

❖ **INCIDENCE**

- Overall incidence varies between 53 times (16.7% related to the total number of births) .this kind o f birth disorder is regularly followed by a caesarean section ($p < 0.001$).

❖ **TYPES OF CERVICAL DYSTOCIA**

1. **PRIMARY CERVICAL DYSTOCIA (FUNCTIONAL)-=**
   - In spite of the absence of any organic lesion and the well effacement of the cervix ,the external OS fails to dilate.
   - This may be due to lack of softening of the cervix during pregnancy or cervical spasm resulted from overactive sympathetic tone

2..**SECONDARY CERVICAL DYSTOCIA (ORGANIC)** –

- Cervical stances as a sequel to previous amputation ,conebiopsy,extensive cauterization or obstetric trauma.
- Organic lesions as cervical myoma or carcinoma

❖ **CAUSES OF CERVICAL DYSTOCIA**

❖ **MATERNAL CAUSES**

- Cephalo pelvic disproportion
- Injudicious of oxytocin
- Uterine inrtia
- Small pelvic size
- Failure of cervical dilation
- Uterine torsion

❖ **FETAL CAUSES**

- Large sized fetal (macrosomia)
- Congenital anomalies;-

-Fetal ascites

-Hydrocephalus

-Fetal tumors

❖ **RISK FACTORS**

**a)MATERNAL RISK**

- Abnormal anatomy of pelvis
- Maternal age
- Short maternal height
- Overweight
- Obesity
- Smoking
- Prolonged labour
- Labour dystocia

**b)FETAL RISK**

- Age of the mother ,height, weight before and during pregnancy
- Body mass index of the mother
- Weight gain during pregnancy
- Fundal height
- Bith weight and foot length of the mother

❖ **SIGN AND SYMPTOMS OF SHOULDER DYSTOCIA**

▪ **SIGN**

- Prolonged labour
- Small size of uterus
- Small size of pelvis

▪ **SYMPTOMS**

- Heavy severe pain due to uterine contractions
- Dry and oedematous vagina
- Mother becomes tired and restless due to continue pain and discomfort
- Features of maternal ketoacidosis
- Abdominal palpation
  -fetal part may be not well defined
  -fhs usually absent

❖ **MANAGEMENT**

▪ **MEDICAL MANAGEMENT**

▪ **PHARMACOLOGICAL MANAGEMENT**



1) HYPERCONTRACTLITY – Induce by oxytocins can be managed tocolytics (Terbutaline 0.5 mg subcutaneous ,and salbutamol 2 mg orally gien )
2) Oxytocin infusion 5 IU in 500 ML solution ,2to 8 drops in 1to 4 minutes

3) **CAESAREAN SECTION –** Caesarean section is done in majority of the cases specially when obstruction is suspected.

## ❖ NON-PHARMACOLOGICAL MANAGEMENT

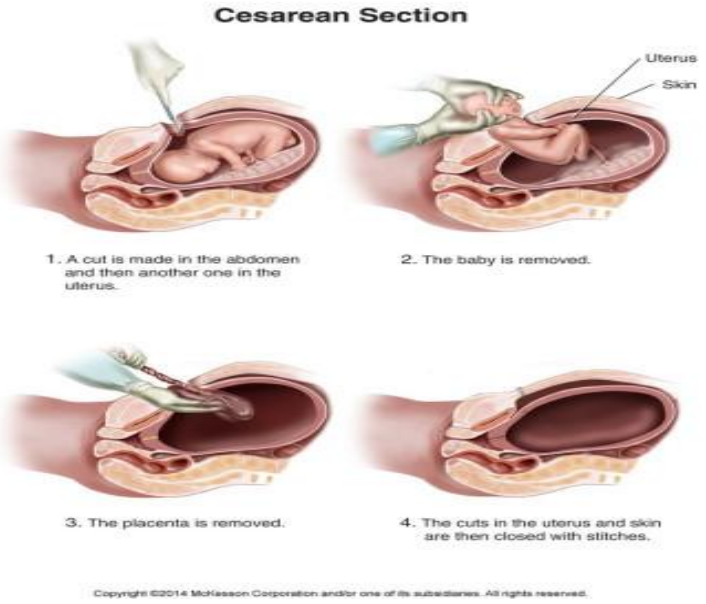
- To provide immediate newborn care and maternal care or KMC care after child birth.
- To check vital signs immediately both mother and newborn.
- To assess newborn condition. .
- To check if any palsy present in newborn.
- To check cervical condition of the baby .
- To check level of cervical dystocia.
- **RESPIRATORY EXERCISE** – To guide the patient about breathing exercise ,as the uterine contractions began ,to inhale and exhale ,breathing through the mouth slowly.
- **Muscle relaxation :-** teached the patient about muscle relaxaing exercise ,instruct the patient for loosing her arms and legs until the contractions stopped.
  (these exercise used were 6 cm ,8 cm and 9 cm of cervical dilation in the active phase of labour, from the beginning of the uterine contraction until its relaxation.)

## ❖ SURGICAL MANAGEMENT

### ➢ CAESAREAN SECTION :-

Caesarean section also known as C-Section or Caesarean delivery, is the use of surgery to deliver babies.A caesarean section is often necessary when a vaginal delivery would put the baby or mother at risk. A caesarean delivery may be performed based upon the shape of the mothers pelvis or history of previous c-section.A trial of vaginal delivery after saesarean section possible.

"An operative procedure to deliver a viable foetus or more (e.g. after 28 weeks or 20 weeks according to the ACOG) through an abdominal and uterine incision.)




**INCIDENCE** :- In 2000 of year 14.5% and 2006 of year 22.7% of incidence included.

## ❖ INDICATION :-

- ❖ Cephalo-pelvic disproportion
- ❖ Fetal mal presentation
- ❖ Previous caesarean section
- ❖ Fetal distress
- ❖ Placenta praevia
- ❖ Abruption placentae ( with live fetus)
- ❖ Dystocia
- ❖ Cord prolapse
- ❖ Multiparity

## ❖ CONTRAINDICATION –

There is no absolute contraindications ,but caesarean section is better to be avoided in cases of fetal demise,major anomalies incompatible with life,and in severe maternal disease as coagulopathy.

## ❖ TYPES OF CAESAREAN SECTION

### ➢ ACCORDING TO TIMING

- Elective caesarean section
- Selective caesarean section

### ➢ ACCORDING TO THE SITE OF UTERINE INCISION

- Upper segment caesarean section
- Lower segment caesarean section

➢ **ACCORDING TO NUMBER OF OPERATION**

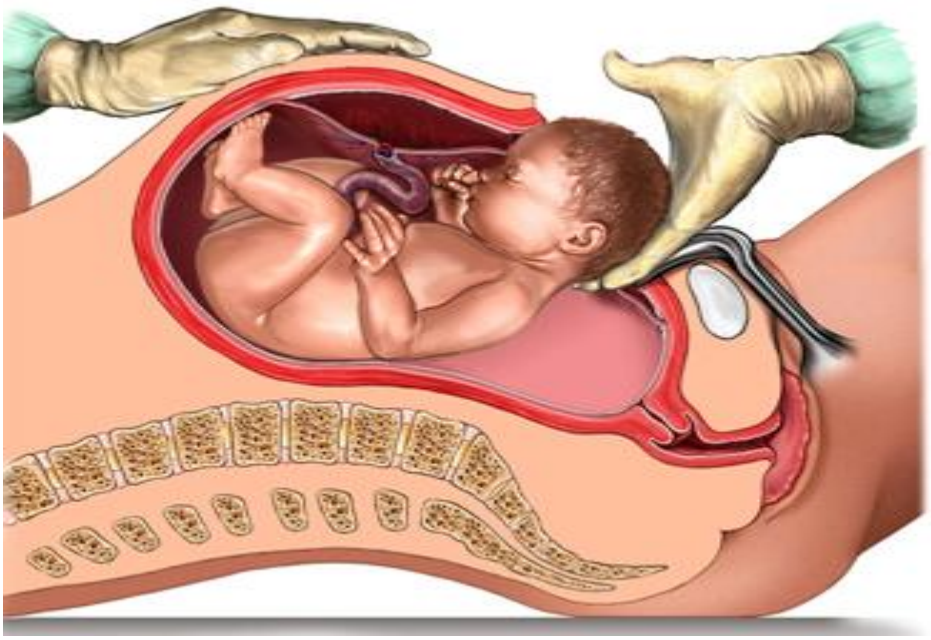
- Primary caesarean section
- Repeated caesarean section

➢ **ACCORDING TO OPENING OF THE PERITNEAL CAVITY**

- Trans-peritoneal
- Extra-peritoneal

❖ **Pre operative care of the patient :-**

- Explain procedure to the mother
- Follow general principles of basic care and infection prevention
- To check vital signs and FHR before surgery
- To instruct the patient for empty bladder before caesarean section .
- To take consent before surgery for caesarean section .
- Injection ranitidine 50 mg IV half to one hour before the surgery
- Injection metoclopramide 10 mg IV half to one hour before surgery
- Bladder should be catheterized
- Fetal presentation,position and FHS should be checked .





❖ **Procedure of lower segment caesarean section**

**ANAESTHESIA** – General inhalation anaesthesia with nitrous oxide + oxygen (The most commonly used),epidural ,spinal or rarely local infiltration anaesthesia .

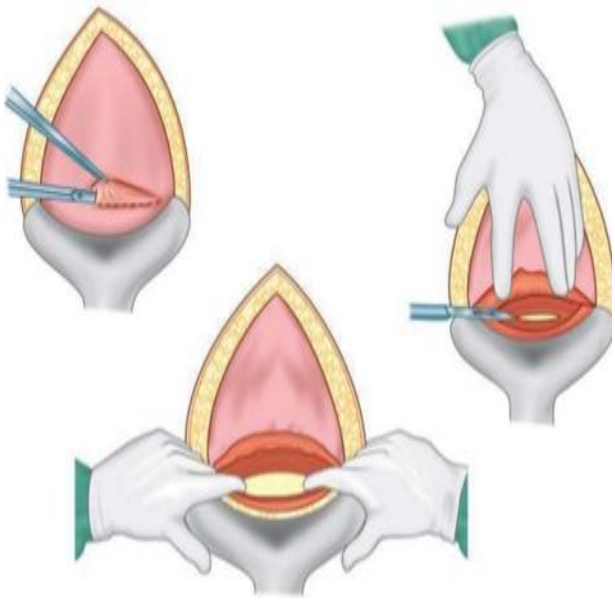
**POSITION**- tilting the patient 150 to the left in the dorsal position minimize the aorto-caval compression.

A).

- The loose peritoneum on the lower segment is cut transversely

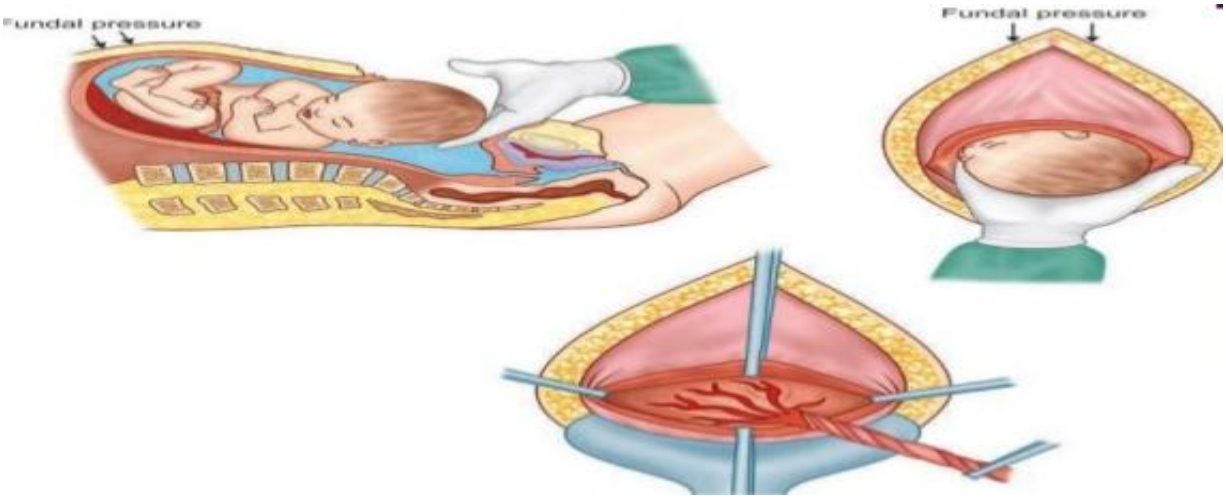
❖ A short incision is made in the midline down to the membrane

❖ The incision of the lower segment is being enlarged using index finger of both hand



B)

- Sagittal section showing insinuation of the fingers between the lower uterine flap and the fetal head until the posterior surface is reached
- Methods of delivery of the head
- Placenta is being delivered.

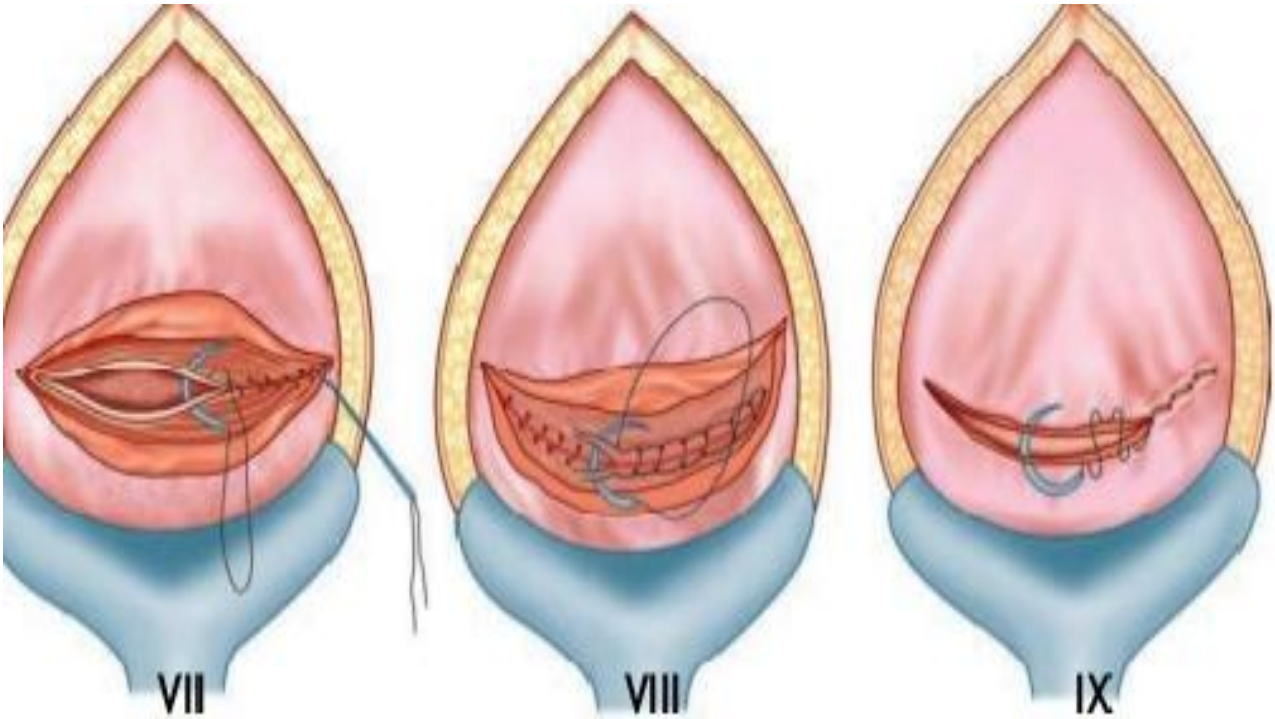



C)

- Inserting the continuous suture taking deeper muscles excluding the deciduas

❖ Similar method of continuous suture taking superficial muscles and facia down to the first layer of suture

❖ Continuous peritoneal catgut suture




❖ **<u>POST-OPERATIVE CARE</u>**

- Palpate the uterine fundus
  -Location
  -Consistency
- Encourage early breath feeding
- Oral fluids after 24 hours
- Discharge from hospital after96 hours
- Stitch removal on 7th post operative day
- To avoid exertion for 4-6 weeks
- Contraceptive advice

## ❖ HEALTH EDUCATION OF THE MOTHER IF BABY BORN WITH SHOULDER DYSTOCIA

- To give emotional and psychological support of the patient.
- To educate the patient about chances of postpartum hemorrhage after baby birth
- To educate the patient if any complication
- To educate the patient about personal hygiene and newborn care.
- To educate the patient foe avoid hard work after delivery of the baby with caesarean section for prevent injury
- To advice the patient for intake healthy diet and liquid diet for better improvement of health status .
- To teach about effective breast techniques of the mother .
- To advice the patient for perform postnatal exercises if possible..
- To advice the patient for intake plenty of water for maintain electrolyte balance

## ❖ SUMMARY

- Cervical dystocia cannot be reliably predicted in the antenatal period
- Clinical estimation of macrosomia is as accurate as ultrasound .
- No consistent patterns of labor or delivery reliably predict cervical dystocia
- Caesarean section is recommended



## ❖ PROGNOSIS

In many cases cervical dystocia is not completely preventable permanently in vaginal delivery but after performance of caesarean section 50% chances of prevents cervical dystocia and newborn death.

## ❖ NURSING MANAGEMENT:

1. Fluid electrolyte imbalancerelated to excessive loss o**f** bodyfluidandbloodlossfromthebody
2. Risk for maternal and fetal injury related to prolonged labor due to cervicaldystocia
3. Ineffective airway clearance related to obstrution in respiratory tract and muconium aspiration
4. Altered body temperature related to intrauterine to extrauterine environment
5. Risk for infection related to surgical procedure
6. Impaired mother and child bonding related to situational crisis and ineffective breast feeding techniques
7. Anxiety related to death of fetus
8. Activity intolerance related to cervical dystocia of the baby
9. Ineffective individual coping related to inadequate support system
10. Risk for infection related to surgical procedure

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# ABNORMAL LABOUR AND ITS MANAGEMENT

INTRODUCTION

Usually the fetal head engages in occipito anterior position and then undergoes a short rotation to be directly occipito anterior in the mid cavity. Malpositions and malpresentation are the abnormal positions of the vertex of the fetal head relative to the maternal pelvis.

**DEFINE THE TERMS:**

1) **LABOUR:** A series of events that takes place in a genital organ in efforts to expel the viable products of conception out of the womb through the vagina into the outer world is called labour.
2) **NORMAL LABOUR (EUTOCIA):** A labour is called normal if it fulfils the following criteria;
    - Spontaneous in onset and at term
    - With vertex presentation
    - Without undue prolongation

- Natural termination with minimal aids
- Without having any complications affecting the health of the mother and the baby.

3) **ABNORMAL LABOUR (DYSTOCIA):** Any deviation from the criteria's of normal labour is called abnormal labour.
4) **MECHANISM OF LABOUR:** It is a series of movements that occur on the fetal head and the trunk in the process of adaptation, during its journey through the pelvis.
5) **ENGAGEMENT:** The widest presenting transverse diameter of the fetal part has passed through the brim of the pelvis is called engagement.
6) **LIE:**Lie refers to the relationship of the long axis of the fetus to the long axis of the centralized uterus.
7) **PRESENTATION:** The part of the fetus which occupies the pelvic brim or in the lower pole of the uterus.
8) **PRESENTING PART:**Presenting part is defined as the part of the presentation which overlies the internal os.
9) **ATTITUDE:** Attitude is the relationship of the fetal head into its trunk.
10) **DENOMINATOR:**It is an arbitrary bony fixed point on the presenting part which comes in relation with the various quadrants of the maternal pelvis.
11) **POSITION:** Position refers to the relationship between the denominator of presenting part and the four quadrants of the maternal pelvis.
12) **MALPOSITION:** The relationship between the denominator (occiput) of the presentation (vertex) and the point on the pelvis is other than iliopecteneal eminence.
13) **MALPRESENTATION:** The fetus presents in maternal pelvis other than vertex, longitudinal lie and flexion attitude.



## MALPOSITIONS & MALPRESENTATIONS

**1) POSITIONS IN VERTEX PRESENTATION**

| POSITION | OCCIPUT POINTS TOWARDS | SAGITTAL SUTURE OF FETUS IN MOTHERS PELVIS |
|---|---|---|
| Left Occipito Lateral | Left Iliopecteneal Line | Transverse diameter |
| Right Occipito Lateral | Right Iliopecteneal Line | Transverse diameter |
| Left Occipito Posterior | Left Sacro Iliac joint | Left Oblique diameter |
| Right Occipito Posterior | Right Sacro Iliac Joint | Right Oblique diameter |
| Direct Occipito Anterior | Symphysis Pubis | Anterio –Posterior diameter |
| Direct Occipito Posterior | Sacrum | Anterio – posterior diameter |

**2) POSITIONS IN BREECH PRESENTATION**

| **Positions** | **Sacrum of the fetus in relation to the mothers pelvis** |
|---|---|
| Left Sacro Lateral position | Left iliopecteal line |
| Right sacro Lateral position | Right iliopecteneal line |
| Left Sacro Posterior position | Left sacro iliac joint |
| Right Sacro Posterior position | Right sacro iliac joint |
| Direct Sacro Anterior position | Symphysis pubis |
| Direct Sacro Posterior position | Sacrum |

**3) POSITIONS IN FACE PRESENTATION**

| **Positions** | **Mentum in relation to pelvis** |
|---|---|
| **Left mento anterior** | |
| **Left mento posterior** | |
| **Left mento transverse** | |
| **Right mento anterior** | |
| **Right mento posterior** | |
| **Right mento transverse** | |

**4) BROW PRESENTATION**

**5) TRANSVERSE LIE**

**6) SHOULDER PRESENTATION**

**7) UNSTABLE LIE**

**8) COMPOUND PRESENTATION**

## OCCIPITO POSTERIOR POSITION

Occipito posterior positions are common type of malpostion of the occiput and occur in approximately 10% of labours. A persistent Occipito posterior position results from a failure of internal rotation prior to birth. This occurs in 5% of birth.The vertex is presenting, but the occiput lies in the posterior rather than the anterior part of the pelvis. Hence the fetal head is deflexed and the larger diameters of the fetal skull present.

**CAUSES**

- Unknown
- Abnormal shape of pelvis
- Contracted pelvis
- Position of the placenta
- prematurity

**ANTENATAL DIAGNOSIS**

Abdominal examination

1. Listen to mother
    - Complain about back ache.
    - Mother feels that baby's bottom is high up against her ribs.
    - Quickening feels across both sides of her abdomen.
2. On inspection
    - Saucer shaped depression at or just below the umbilicus
    - The outline created by the high, unengaged head can look like a full bladder
3. On palpation
    - Breech easily palpated at the fundus.
    - Fetal back is difficult to palpate as it is well out to the maternal side.
    - Fetal limbs can be felt on both sides of the midline.
    - The head is usually high because of non engagement of head (large presenting diameter)
4. On auscultation
    - FHR can be heard in the midline.
    - Or it may be heard more easily at the flank on the same side as the back.



**DIAGNOSIS DURING LABOUR**

1. Continuous severe back ache worsening with contractions.
2. In-coordinate contractions.
3. Spontaneous rupture of membrane at an early stage of labour.
4. Slow decent of head.

## MECHANISM OF LABOUR IN RIGHT OCCIPITO POSTERIOR POSITION

- The lie is longitudinal.
- The attitude of the head is deflexed.
- The presentation is vertex.
- The position is right occipito posterior.
- The denominator is occiput.
- The presenting part is anterior or middle area of parietal bone.
- The occipitofrontal diameter is 11.5 cm, lies in right oblique diameter of the pelvic brim.

## STEPS OF MECHANISM

1. **Engagement**: Engagement occurs in right oblique diameter of the pelvis.
2. **Flexion**: Decent occurs with increasing flexion. The occiput is the leading part.
3. **Internal rotation of the head**: The occiput reaches the pelvic floor first and rotates forwards 3/8 of the circle along the right sides of the pelvis to lie under the symphysis pubis. The shoulders follow, turning 2/8 of a circle from the left to right oblique diameter.
4. **Crowning:** The occiput escapes under the symphysis pubis and crowning occurs.
5. **Extension of head**: The sinciput, face and chin sweeps the perineum and head is born by a movement of extension.
6. **Restitution:** Occiput turns 1/8$^{th}$of a circle to the right and the head realigns itself with the shoulders.

7. **Internal rotation of the shoulders**: The shoulder enters in the pelvis in right oblique diameter, the anterior shoulder reaches the pelvic floor first and rotates forwards 1/8$^{th}$ of a circle and lie under symphysis pubis.
8. **External rotation of the head**: At the same time the occiput turns a further 1/8$^{th}$ of a circle to lie under symphysis pubis.
9. **Lateral flexion**: the anterior shoulder escapes under the symphysis pubis, posterior shoulder sweeps the perineum and the body is born by a movement of lateral flexion.

## THE POSSIBLE COURSE AND OUTCOME OF LABOUR

1. Long internal rotation
2. Short internal rotation
3. Undiagnosed face to pubis
4. Deep transverse arrest
5. Conversion to face or brow presentation

## COMPLICATIONS

1. Obstructed labour
2. Maternal trauma
3. Neonatal trauma
4. Cord prolapsed
5. Cerebral haemorrhage
6. Risk of infection
7. Prolonged labour

## MANANGEMENT OF LABOUR



**Principles:**

- Early diagnosis

- Strict vigilance with watchful expectancy
- Judicious and timely interference

**Steps of management**

1) Diagnosis and evaluation
2) Early caesarean section

**During first stage:**

- If there is no complication labour is allowed to proceed similar to normal labour.
- Anticipating prolonged labour- start intravenous RL.
- Check the progress of labour.
- Weak pain, persistence of deflexion and non rotation of the occiput are too often coexistent start oxytocin infusion for augmentation of labour.
- Check the indications for caesarean section. (Arrest of labour, incoordinate uterine actions, fetal distress).

**During second stage:**

- In majority of the cases, anterior rotation of the occiput is completed and the delivery is either by spontaneous or can be low forceps or ventouse.
- In minority (unrotated or malrotated): Take watchful expectancy for the completion of anterior rotation and decent of the fetal head.
- In occiput sacral position spontaneous delivery occurs(face-to- pubis)
- In face to pubis delivery liberal episiotomy should be done to prevent complete perineal tear.

**During third stage:**



- Prophylactic intravenous ergometrine 0.2mg after the delivery of anterior shoulder to prevent PPH.

- Inspection of birth canal to detect any injury.

**ARRESTED OCCIPITO POSTERIOR POSITION**

If there is failure to progress inspite of good uterine contraction,

1) Per abdominal examination: Size of the baby, engagement of the head, amount of liquor, FHS.
2) Per vaginal examination: Station of the head, position of the sagittal suture and the occiput, degree of deflexion of the head, degree of moulding and caput formation, and assessment of the pelvis.

**ARREST IN THE OCCIPITO TRANSVERSE OR OBLIQUE OCCIPITO POSTERIOR POSITION:**

1) Ventouse or vacuum extraction
2) Alternative methods
    - Manual rotation followed by forceps extraction
    - Forceps rotation and extraction
    - Caesarean section
    - Craniotomy

**OCCIPITO SACRAL ARREST**

- If the occiput below the Ischial spine -Forceps application in unrotated fetal head followed by extraction as face to pubis.
- If the occiput remains at or above the level of ischial spine – caesarean section.

**DEEP TRANSVERSE ARREST**

**Definition:**

The head is deep in to the cavity; the sagital suture is placed in the transverse bispinous diameter and there is no progress in the descent of the head even after ½ to 1 hour following full dilatation of the cervix.

**Causes:**

- Faulty pelvic architecture
- Deflexion of the head
- Weak uterine contraction
- Laxity of the pelvic floor muscles

**Diagnosis:**

- The head is engaged
- The sagital suture lies in the transverse bispinous diameter
- Anterior fontanelle is palpable
- Faulty pelvic architecture may be detected.

**Management:**

Based upon fetal condition andfindings of pelvic assessment.

1) In case of big baby and inadequate pelvis- Caesarean section.
2) If there is no contraindications for vaginal delivery
    - Ventouse.
    - Manual rotation and application of forceps.
    - Forceps rotation and delivery with kielland.

**NURSING MANAGEMENT OF MALPOSITIONS**

Management during I$^{st}$ stage of labour

- Continuous support from midwife (massage, posture and positions)
- Oxytocin infusion to correct in coordinated uterine contraction.
- Provide relaxation to avoid early urge to push (change in position, breathing techniques, inhalational analgesia)

Management during II$^{nd}$ stage of labour



- Vaginal examination to confirm the full dilatation of cervix, moulding, and formation of caput succedaneum.

- Provide upright position. This may shorten the length of second stage of labour and reduce the need for operative delivery.
- Oxytocin infusion to stimulate adequate uterine contractions and achieve advance of the presenting part.
- Close observation of maternal and fetal condition.

## BREECH PRESENTATION

In breech presentation fetus lies longitudinally with buttocks in the lower pole of the uterus. The presenting diameter is bitrochanteric- 10 cm and the denominator is sacrum.

### INCIDENCE

Occurs in approximately, 15% at 30 weeks and 3% at term.

### CAUSES

1) **Fetal**
    - Prematurity
    - Multiple pregnancy
    - Malformations
    - Congenital dislocation of hip
2) **Liquor**
    - Polyhydramnios
    - oligohydramnios
3) **Uterine**
    - Bicornuate uterus
    - Fibroid uterus
4) **Placental**
    - Placenta previa
5) **Pelvic**
    - Any tumours obstructing the pelvic canal

## TYPES OF BREECH PRESENTATIONS

1) **Complete breech:** Breech fully flexed. In this type of breech presentation both fetal thighs and limbs are flexed. Incidence is 25% of breech presentation.
2) **Incomplete breech: This is due to varying degrees of extension of thighs or legs at the podalic pole.**

   **Types:**

   - **Frank breech:** It is breech with extended legs. Fetal thighs of both limbs are flexed and both lower limbs are extended at the knee. Incidence is 65% of breech presentation.
   - **Footling breech:** This is incomplete breech occurs in 10% of breech presentation. One or both fetal thighs are extended and one or both knees or feet lie below the buttocks.
   - **Knee presentation:**Thighs are extended but the knees are flexed, bringing the knees down to present at the brim.

**Clinical types:**

1) **Uncomplicated:** Breech presentation is not associated with any obstetrical complications.
2) **Complicated:** Breech presentation associated with obstetrical complications and which adversely affect the prognosis.

## POSITIONS IN BREECH PRESENTATION

| **Positions** | **Sacrum of the fetus in relation to the mothers pelvis** |
|---|---|
| Left Sacro Lateral position | Left iliopecteal line |
| Right sacro Lateral position | Right iliopecteneal line |
| Left Sacro Posterior position | Left sacro iliac joint |
| Right Sacro Posterior position | Right sacro iliac joint |
| Direct Sacro Anterior position | Symphysis pubis |
| Direct Sacro Posterior position | Sacrum |



## DIAGNOSIS

**Antenatal diagnosis**

**Abdominal examination**:

1) **Listen to mother:**
    - Something hard and uncomfortable under her ribs
    - breathing difficulty
    - may feel very hard kicks on her bladder
2) **Palpation**
    - At fundus –Hard round mass.
    - Soft irregular mass at lower pole of the uterus
3) **Auscultation**
    - Usually FHR heard above the umbilicus
    - In case of extended legs FHR heard at lower level.
4) **Ultrasound examination**
5) **X-ray examination**

**DIAGNOSIS DURING LABOUR**

1) Abdominal examination
2) Vaginal examination
    - Soft irregular mass without sutures
    - Anus may be felt
    - Meconium stained fingers
    - External genitalia is very evident in case of extended legs.
    - If a foot is felt differentiate it from the hands.-
        - Toes are all the same length, shorter than the fingers and the big toe cannot oppose to each other.

- The foot is at right angles to the leg and heel has not equivalent in the hand.

- Confirmatory diagnosis by USG and X-ray

**ANTENATAL MANAGEMENT**

At 36 weeks gestation or later

1) External cephalic version.
2) Assessment for vaginal birth.
3) Fetal size.
4) Pelvic capacity.

**MECHANISM OF LABOUR**

- The lie is longitudinal.
- Attitude is one of complete flexion.
- The presentation is breech.
- The position is left sacro anterior.
- The denominator is the sacrum.
- The presenting part is the left buttocks.
- The presenting diameter is bitrochanteric, 10 cm. Enters the pelvis in left oblique diameter of the brim.the sacrum points to the left iliopecteneal eminence.

**STEPS OF MECHANISM**

1) **Compaction**: Decent takes place with increasing compaction, owing to increased flexion of the limbs.
2) **Engagement:** Engagement of hips takes place in an LSA position with sacrum in the left anterior portion on the mothers pelvis, and the bitrochanteric diameter is in the left oblique diameter of the mothers pelvis.

3) **Internal rotation of the anterior buttocks:** The anterior buttocks reaches the pelvic floor first and rotates forwards 1/8 of a circle along the right side of the pelvis to lie underneaththe symphysis pubis. The bitrochanteric diameter is now in the antero posterior diameter of the pelvis.
4) **Lateral flexion of the body:** The anterior buttocks escapes under the symphysis pubis, the posterior buttocks sweeps the perineum and the buttocks are born by a movement of lateral flexion.
5) **Restitution of the buttocks:** The anterior buttock turns slightly to the maternal right side.
6) **Internal rotation of the shoulders:** The shoulder enters the pelvis in the left oblique diameter. The anterior shoulder rotates forwards 1/8 of a circle along the right side of the pelvis and escapes under the symphysis pubis, the shoulder sweeps the perineum and the shoulders are born.
7) **Internal rotation of the head:** the head enters the pelvis with the sagittal sutures are in transverse diameter of the brim. The occiput rotates forwards along the left side and the suboccipital region(nape of the neck)impinges on the underneath of the symphysis pubis.
8) **External rotation of the body:** At the same time the body turns so that the back is uppermost.
9) **Birth of the head:** The chin, face and sinciput sweeps the perineum and head is born in a flexed attitude.

## COMPLICATION

### Risks to the mother



1. Risks of operative procedure
2. Risks of general anaesthesia

3. Perineal and cervical laceration
4. Bleeding due to laceration
5. infection

**Risks to the baby**

1) **Mortality due to**
   - Intracranial haemorrhage
   - Cord compression
   - Asphyxia
   - Damage to liver, suprarenal gland and kidney
   - Fracture of cervical spine
2) **Morbidity due to**
   - Fracture –femur, humerous and clavicle.
   - Damage to the brachialplexus.
   - Haematoma.
   - Physical and mental handicap.
   - Risks of external cephalic version.

**MANAGEMENT**

**Antenatal management:**

- Identification of the complicating factors
- External cephalic version
- Formulation of the line of management



**During vaginal delivery:**

**Preliminaries:**

1. Confirm the complete dilatation of the cervix
2. Check the adequacy of the pelvis
3. Emptying of the bladder
4. Adequate explanation to mother for effective pushing effort.
5. Readiness for a full- scale new born resuscitation effort.
6. Proper positioning of the articles.
7. Notification and presence or immediate availability of the consulting physician, anaesthesiologist and paediatrician.

**First stage:**

- Place the women in lithotomy position.
- Perform vaginal examination to exclude complications.
- Sited intravenous line with RL, avoid oral intake.
- Provide adequate analgesia.
- Monitor fetal condition and progress of labour.
- Oxytocin infusion for the augmentation of the labour.
- Encourage the women to push with contractions.
- Check the indications for caesarean section.

**Second stage: There are three methods for vaginal breech delivery**

1) **Spontaneous breech delivery:** Delivery occurs with little assistance from the attendant.
2) **Assisted breech delivery:**The buttocks are born spontaneously, but some assistance provided for the delivery of extended legs or arms and the head.
3) **Breech extraction:** Manipulative delivery performed by an obstetrician to hasten the delivery in an emergency.

## ASSISTED BREECH DELIVERY

**PRINCIPLES:**

1) Never to rush.
2) Never pull from below but push from above.
3) Always keep the fetus with the back anteriorly.

**Preliminaries:**

1) Anaesthetist
2) An assistant
3) Instruments and suture materials for episiotomy
4) A pair for obstetric forceps
5) Appliances for resuscitation of the baby
6) Neonatologist

**STEPS:**

1) Patient brought to the table when the anterior buttocks and fetal anus are visible.
2) Place the woman in lithotomy position when the posterior buttock distends the perineum.
3) Tilt the woman $15^{o}$ laterally using a wedge under the back to avoid aortocaval compression.
4) Pudendal block anaesthesia done along with perineal infiltration.
5) Episiotomy: should be done in all cases of primigravide and selected multigravide. Best time for episiotomy is when the perineum is distended and thinned by the breech as it is climbing the perineum.

   **Advantages:**

   - To straighten the birth canal.
   - To facilitates intravaginal manipulation for forceps delivery.
   - To minimise the compression of the after coming head.

6) Patient is encouraged to bear down as the expulsive forces from above ensure flexion of the fetal head and safe decent

7) Soon after the trunk up to the umbilicus is born,

- The extended legs are to be decomposed by pressure on the knees ( poplitial fossa) in a manner of abduction and flexion of thighs.
- The umbilical cord is to be pulled down and to be mobilised to be one side of the sacral bay to minimise compression.
- If the back remains posteriorly, rotate the trunk to bring the back anteriorly.
- The baby is wrapped with a sterile towel to prevent slipping when held by the hands and facilitates manipulation.

8) **Delivery of the arms**: The assistant is to place the hands over the fundus and keep a steady pressure during contractions to prevent extension of the hands. The arms are delivered one after the other only when one axilla is visible, by simply hooking down each elbow with a finger. Baby should be held by the feet over the sterile towel while the arms are delivered.

9) **Delivery of the after coming head:** Most crucial stage of delivery. The time between the delivery of umbilicus to delivery of mouth should be preferably 5- 10 minutes.

**Methods:**

- **Burns –Marshall Method:**
  - ❖ The baby is allowed to hang by its own weight. The assistant is asked to give suprapubic pressure with the flat of hand in a downward and backward direction, the pressure is to be exerted more towards the sinciput.
  - ❖ When the nape of the neck is visible under the pubic arch, the baby is grasped by ankles with a finger in between the two.
  - ❖ Maintaining a steady traction and forming a wide arc of a circle, the trunk swung in upward and forward direction.

- ❖ Meanwhile, the left hand to guard the perineum, face and brow successively slipped off from the perineum.
- ❖ When the mouth should be cleared off the vulva, there should be no hurry. Mucus of the mouth and pharynx is cleared by mucus sucker.
- ❖ Depress the trunk to deliver the rest of the body.

- **Forceps delivery**
  - ❖ Forceps can be used as a routine. The head must be in the cavity.
  - ❖ When the occiput lies against the back of the symphysis pubis an assistant raises the legs of the baby to facilitate the introduction of the blade from below.
  - ❖ Head should be delivered slowly to reduce compression – decompression forces as that may cause intracranial bleeding.
- **Malar flexion and shoulder traction (Modified Mauriceau -Smellie –Veit technique)**
  - ❖ The baby is placed on supinated left forearm with the limbs hanging on either sides.
  - ❖ Place the middle and index fingers of the left hand over the malar bones on either sides. (index finger was introduced in the mouth)
  - ❖ The ring and little fingers of the pronated right hand are placed on child's right shoulder, index finger placed on child's left shoulder and the middle finger placed on suboccipital region.
  - ❖ Apply traction in downward and backward direction till the nape of the neck is visible under the pubic arch.
  - ❖ At the same time ask the assistant to provide suprapubic pressure to maintain flexion.
  - ❖ Carried the fetus in upward and forward direction towards the mother's abdomen releasing the face, brow, and depress the trunk to release the occiput and vertex.

10) **Resuscitation of the baby:**

11) **During third stage of labour**: Prophylactic ergometrine 0.2 mg with the crowning of head.

## MANAGEMENT OF COMPLICATED BREECH DELIVERY

**Delay in descend of the breech:**

**Causes:**

- Big baby with extended legs
- weak uterine contraction
- Rigid perineum
- outlet contraction

❖ **Arrested at the outlet:**

**Management:**

- caesarean section

❖ **In the absence of outlet contraction and feto-pelvic disproportion**

**Management:**

- Liberal episiotomy with or without groin traction.

❖ **Arrest of the breech at or above the level of ischial spine**

**Management:**

- Caesarean section

❖ **Frank breech extraction. ( Pinard'smaneuvere)**

- Is done by intrauterine manipulation to convert frank breech to a footling breech.
- The middle and index fingers carried up to the popliteal fossa. It is then pressed and abducted so that the fetal leg is flexed.
- Grasp the fetal foot at the ankle and extract the breech.



## EXTENDED ARMS

One or both arms may be fully stretched along the sides of the head or lie behind the neck.

**Cause:** Faulty techniques in delivery**.**

**Diagnosis:**

- Winging of the scapula
- Absence of flexed limbs in front of the chest.

**Management:**

Urgent delivery of the extended arms.

**Principles of management:**

- Because of the curved birth canal, when the anterior shoulder remains above the symphysis pubis, the posterior shoulder will be below the level of sacral promontory.
- If the fetal trunk is rotated keeping the back anterior and maintaining downward traction, the posterior shoulder will appear below the symphysis pubis.

**Methods of delivery**

❖ **Classical method:**

- First the posterior forearm is delivered followed by anterior forearm.
- Introduce left arm along the curve of sacrum while the baby is pulled slightly upward.
- Apply firm pressure on humerus, the posterior arm is pushed over the baby's face.
- The extended anterior arm is delivered from the anterior aspect by introducing the right hand in the same manner, while the baby's trunk is depressed towards the perineum.

❖ **Lovset'smaneuver:**

- Baby is wrapped in warm towel and grasped, using both hands by femoro pelvic grip keeping the thumbs parallel to the vertebral coloumn. The maneuver should start only when the inferior angle of the anterior scapula is visible underneath the pubic arch.

**Step 1:** The baby is lifted slightly to cause lateral flexion. The trunk is rotated through $180^0$ keeping the back anterior and maintaining down ward traction. This will bring the posterior arm to emerge under the pubic arch which is then hooked out.

**Step 2:**The trunk is then rotated into reverse direction keeping the back anterior to deliver the posterior shoulder while anterior shoulder under the symphysis pubis.

**ARREST OF THE AFTER COMING HEAD**

- **At the brim:**

**Causes:**

- Deflexed head
- Contracted pelvis

**Management:**

- Delivery of the head by forceps
- In deflexed head- malar flexion and shoulder traction

- **In the cavity**

**Causes:**

- **Deflexed head**
- Contracted pelvis

**Management:**

- Delivery of the head by forceps

- **At the outlet:**

**Causes:**

- Rigid perineum
- Deflexed head



**Management:**

- Episiotomy followed by forceps application

- Malar flexion and shoulder traction

1. **Delivery of the head through an incomplete dilated cervix:**

   **Causes:**

   - Premature baby
   - Macerated baby
   - Footling presentation
   - Hasty delivery of the breech before the cervix is fully dilated

2. **Occipito posterior position of the head:** Usually occurs in spontaneous vaginal delivery.

   - Rotation of the fetus.

## FACE PRESENTATION

**INCIDENCE :**1 in 500 deliveries.

- 60% mento anterior
- 15% mento transverse
- 25% mento posterior

### MECHANICS OF PRESENTATION

Characterized by extreme extension of the fetal head so the face presents to the birth canal. The occiput of the fetus will be in contact with its spine.

### TYPES OF FACE PRESENTATION

**Primary face presentation**: Face presentation before the labour.

**Secondary face presentation**: Face presentation develops during labour from vertex presentation with the occiput posterior.

Possible positions:

1) Left Mento Anterior
2) Left Mento Posterior
3) Left Mento Transverse

4) Right Mento Anterior
5) Right Mento Posterior
6) Right Mento Transverse

## CAUSES

### a) Maternal:

- Multiparity with pendulus abdomen
- Lateral obliquity of the uterus
- Contracted pelvis
- Pelvic tumours

### b) Fetal:

- Congenital malformations
- Twist of the cord around the neck
- Increased tone of the extensor group of neck muscle.

## DIAGNOSIS

Generally diagnosed on vaginal examination in labour.

## ABDOMINAL FINDINGS:

**Inspection:** 'S' shaped spine, no visible bulging on flanks.

**Palpation:**

| Grips | Mento- Anterior | Mento- posterior |
|---|---|---|
| Lateral grip | 1) Fetal limbs are felt anteriorly<br>2) Back is on the flank and is difficult to palpate.<br>3) The chest is thrown anteriorly against uterine wall and is often mistatken for back. | 1) Back is felt to the front and better to palpated only towards the podalic pole because of extension of spine. |
| Pelvic grip | 1) Head seems good and is not engaged. | 1) Same |

| | | |
|---|---|---|
| | 2) Cephalic prominent is to the side towards which back lies.<br>3) Groove between head and neck is not so prominent. | 2) Same<br>3) The groove is prominent. |
| Auscultation | FHS is distinctly audible anteriorly through the chest wall of the fetus towards the side of the limb. | FHS is not so distinct and is audible on the flank towards the side of the limb. |

**Vaginal examination:** Diagnostic features are,

Palpating the mouth with hard alveolar margins, nose, malar eminence, supraorbital ridges and mentum. The examination should be conducted gently to avoid injury to the eyes. Assessment of the pelvis to check the adequacy.

**Distinguishing features:**

1) Mouth and malar eminence are not in a line.
2) Sucking effect of mouth.
3) Hard alveolar margins.
4) Absence of meconeum staining on the examination fingers.

## MECHANISM OF LABOUR

- Lie –longitudinal
- Attitude –Extension of head and back
- Presentation –Face
- Position –LMA, LMP, LMT, RMA, RMP, RMT.
- Denominator –Mentum
- Presenting part –Left malar bone

## STEPS OF MECHANISM

1) **Engagement:**The engaging diameter of the pelvis is oblique diameter. Right in LMA and left in RMA, with the mentum related to one iliopubic eminence and the glabella to the opposite sacro iliac joint.

The engaging diameter of the fetal head is submento bregmatic 9.5 cm.

2) **Extension:** Decent takes place with increasing extension occurs till the chin touches the pelvic floor.

3) **Internal rotation:** when the chin reaches the pelvic floor resistance it rotates.

    a) Rotation of the chin anteriorly

        - $1/8^{th}$ of a circle for RMA and LMA to MA
        - $2/8^{th}$ of a circle for RMT and LMT to MA
        - $3/8^{th}$ of a circle for RMP and LMP to MA

    b) Rotation of the chin posteriorly

        - $1/8^{th}$ of the circle for RMP and LMP to MP

4) **Delivery of the head:** The head is born by flexion, delivering the chin, face, brow, vertex and lastly the occiput.

5) **Restitution:** Restitution occurs through $1/8^{th}$ of a circle opposite to the direction of internal rotation. External rotation occurs further $1/8^{th}$ of a circle to the same side of restitution then the face directly facing towards left maternal thigh in LMA and right maternal thigh in RMA.

6) **Internal rotation of shoulders:** The shoulders enters the pelvis in the oblique diameter and the anterior shoulder reaches the pelvic floor first and rotates forwards $1/8^{th}$ of a circle along the left side of the pelvis in the RMA position and along the right side in LMA position.

7) **Birth of the shoulder and body by lateral flexion:** The anterior shoulder escapes under the symphysis pubis, the posterior shoulder sweeps the perineum and the body is born by a movement of lateral flexion.

**POSSIBLE COMPLICATIONS**

- Obstructed labour.
- Cord prolapse.
- Facial bruising.

- Cerebral haemorrhage.
- Maternal trauma.

**MANAGEMENT:** Overall assessment of-

- Pelvic adequacy
- Size of the baby
- Of the complicating factors
- Congenital malformation
- Position of the mentum
- Check the indications of early or elective caesarean section

**VAGINAL DELIVERY:**

**MENTO-ANTERIOR:**

**Principle:**

Wait and watch policy.

**First stage:** Labour is conducted in the usual procedure.

**Second stage:** Perineum should be protected with liberal mediolateral episiotomy.

**MENTO- POSTERIOR:**

**First stage:** In uncomplicated cases, vaginal delivery is allowed with strict vigilance hoping for spontaneous anterior rotation of the chin.

**Second stage:**

**1)** If anterior rotation of the chin occurs, Spontaneous or forceps delivery with episiotomy is needed.

**2)** In incomplete or malrotation- Should take early decisions for method of delivery soon after the full dilatation of the cervix.

- Caesarean section
- Manual rotation of chin anteriorly followed by immediate forceps extraction.

**NURSING MANAGEMENT**

1) Recognition of the face presentation and notification of the physician about the mal presentation.
2) Should not apply fetal scalp electrode. Care should be taken not to infect or injure the eyes during vaginal examination.
3) Close monitoringof themechanism of labour and informing the physician if rotation is to a direct mentum.
4) Immediately following the rupture of membranes, a vaginal examination should be performed to exclude cord prolapsed.
5) For delivery of the head:
    - Application of pressure on the brow.
    - Exertion of head control.
6) If there is extensive oedema of the nose, neck and mouth and impaired respiratory condition request the paediatrician to assist the delivery.
7) Reassuring the parents, family and significant others that the position of head and neck of the baby and extensive swelling of the features normally disappear in a few days.**BROW PRESENTATION**

Rarest variety of cephalic presentation, where the presenting part is the brow and the attitude of head is extension. The presenting part is bounded by the anterior fontanel and orbital ridges. The presenting diameter is mentovertical.

**INCIDENCE:**i in 1000 births.

**DIAGNOSIS:** Not usually detect before onset of labour.

**Abdominal palpation:**

Head is high, unduly large and does not descent into the pelvis in spite of good uterine contraction.



**Vaginal examination**

1) Difficult to reach the presenting part.

2) Anterior fontanel may be felt on one side of the pelvis and the orbital ridges on the other side.

**Sonography** is confirmatory and to exclude the bony congenital and malformations of the fetus.

**MANAGEMENT**

**During pregnancy**

1) Exclude the contra indications for vaginal delivery.
2) In case of persistent brow presentation associated with complicating factors –elective caesarean section is needed.

**During labour**

1) In uncomplicated cases –if spontaneous correction to either vertex or face fails to occur easily in labour caesarean section is ideal management.
2) Manual correction
3) craniotomy

## TRANSVERSE LIE

**DEFINITION:**

1) When the long axis of the fetus lies perpendicularly to the maternal spine or centralised uterine axis, it is called transverse lie.
2) When the fetal axis placed oblique to the maternal spine and is then called oblique lie.

**POSITIONS:**

**Determined by the direction of the back.**

1) Dorso –anterior
2) Dorso -posteror
3) Dorso –superior
4) Dorso –inferior



**Incidence**: 1 in 200 births.

**Causes:**

1) Multiparity
2) Prematurity
3) Twins
4) Hydramnios
5) Contracted pelvis
6) Placenta praevia
7) Pelvic tumours
8) Congenital malformations of the uterus
9) Intra uterine death

**DIAGNOSIS:**

**Abdominal examination:**

**Inspection:**Uterus looks broader and often asymmetric and not maintaining the pyriform shape.

**Palpation:**

- **Fundal height:** Less than the period of amenorrhoea.
- **Fundal grip**: Fetal pole (Breech / Head) is not palpable.
- **Lateral grip:**
    a) Soft, broad and irregular breech is felt to one side of the midline and smooth, hard globular head is felt on the other side.
    b) Back is felt anteriorly across the long axis in dorso anterior or the irregular small parts are felt anteriorly in dorso posterior.
- **Pelvic grip:** The lower pole of the uterus is found empty.

**Auscultation:**

- In dorso –anterior position: FHS is heard below the umbilicus.
- In dorso posterior position FHS heard in high level.



**Ultrasonography:** To confirm the diagnosis.

**Vaginal examination:**

**During pregnancy:** The fetal part is so high and is difficult toidentify.

**During labour:**

1) Distinguishing features are, acromion process, scapula, clavicle and axilla.
2) Characteristic landmarks are, feeling of ribs and intercostals spaces.
3) Rarely the arm or leg may be prolapsed, loop of cord may found alongside of the prolapsed arms.

**COMPLICATION:**

1) Premature rupture of membrane.
2) Obstructed labour
3) Neglected labour
4) Dehydration
5) Ketoacidosis
6) Shock and sepsis

**FAVOURABLE EVENTS**

1) Spontaneous rectification or version
2) Spontaneous evolution
3) Spontaneous expulsion

**MANAGEMENT OF SHOULDER PRESENTATION:**

**ANTENATAL**

–External cephalic version, should be provided in all cases beyond35 weeks provided if there is no contraindications.

**If version fails or is contraindicated**



- The patient is to be admitted at $37^{th}$ week

  Method of delivery- elective caesarean section.

- Vaginal delivery may be allowed to continue in a dead or congenitally malformed fetus. Labour allowed to continue under supervision till the full dilatation of the cervix. Delivery completed by internal version.

**PATIENT SEEN IN LABOUR**

**Early in labour:**

- External cephalic version.
- Caesarean section.

**Late labour**

- **Baby alive**

–If the baby is mature and fetal condition is good caesarean section is preferable.

-Internal version

- **Baby dead**

-Caesarean section

-Destructive operation

## UNSTABLE LIE

**DEFINITION:** This is the condition where the presentation of the fetus is constantly changed even beyond 36$^{th}$ week of pregnancy when it should have been stabilised.

**CAUSES:** The causes are the conditions those preventing the presenting part to remain fixed in the lower pole of the uterus.

1) Grand multipara with lack of uterine tone and pendulus abdomen
2) Hydramnios
3) Contracted pelvis
4) Placenta praevia
5) Pelvic tumour



**COMPLICATIONS:**

1) Cord entanglement
2) Cord prolapsed
3) Perinatal death

**MANAGEMENT**

**ANTENATAL:**

1) Check the presentation and lie at each antenatal visit.
2) If there is no contraindications –ECV.
3) Hospitalisation –The patient is to be admitted at $37^{th}$ weeks.
4) After admission, the investigation (sonography) is directed to exclude –placenta praevia, contracted pelvis, congenital malformations.

**FORMULATION FOR THE LINE OF TREATMENT:**

- Elective caesarean section
- Stabilising induction of labour

–external cephalic version is done after 37weeks
–Oxytocin infusion to initiate the uterine contraction
–Low rupture of membrane
–Monitor the labour process

## COMPOUND PRESENTATION

**DEFINITION**

When a cephalic presentation is complicated by the presence of a hand or a foot or both along the side of the head or presence of one or both hands along the side of the breech, it is called compound presentation. The commonest one is being the head with hand and the rarest one being the presence of head hand and a foot.

**INCIDENCE:** 1 in 600

**CAUSES:**

- Prematurity
- Contracted pelvis
- Pelvic tumours

- Multiple pregnancy
- Macerated fetus
- High head with premature rupture of membrane
- Hydramnios

**DIAGNOSIS:**

If the cervical os is sufficiently dilated – feel the limb by the side of the presenting part, specially after the rupture of membrane.

**MANAGEMENT**

**Management based on,**

1) Stage of labour
2) Maturity of the fetus
3) Singleton or twins
4) Pelvic adequacy
5) Associated cord prolapse

**A) Caesarean section:**

Indication –mature singleton fetus associated with contracted pelvis or cord prolapsed with the live fetus.

**B) Expectant treatment**: Basesd on wait and watch policy.

Elevation of the prolapsed limb during uterine contraction.

**APPLICATION OF NURSING THEORY**

**HILEGARD E. PEPLAU -THEORY OF INTERPERPERSONAL RELATION**

**Details of the Hildegard**

♦ Born in Pennsylvania in 1909. Started her carrier from a diploma nursing program in 1931.

♦ 1947 MA in psychiatric nursing from Colombia University, Newyork.

♦ 1953 Published book on interpersonal relationship in nursing.



♦ In 1974 got retired & was recognized all over as a nurse.

♦ Nursing can be viewed as an interpersonal process as it involves interaction between two or more individual with a common goal.

♦ For developing interaction between nurse and patient the nurse has to choose sequential pattern and different skills and assume various roles etc.

♦ In 1974 got retired & was recognized all over as a nurse.

♦ Nursing can be viewed as an interpersonal process as it involves interaction between two or more individual with a common goal.

♦ For developing interaction between nurse and patient the nurse has to choose sequential pattern and different skills and assume various roles etc.

**NURSING DIAGNOSIS:**

1) High risk of infection related to disease condition
2) High risk of acquiring an opportunistic infection related to poor nutritional status
3) Impaired family coping related to Maternal out come
4) Anxiety related to disease condition
5) Knowledge deficit related to disease condition.
6) Fatigue related to lack of food intake
7) Altered nutrition less than body requirement related to anorexia
8) Anticipatory grieving related to disease condition

**RECENT MODALITIES IN MANAGEMENT OF ABNORMAL LABOUR**

Treatment:
Induction of labour
Amniotomy
Pelvimetry
USG
X-ray
Diet
Activity
Medication

**SUMMARY:**

Today we have discussed about certain terminologies related to abnormal labour, mal-positions and mal-presentations, demonstrated the mechanism of abnormal labour, management of abnormal labour, nursing management of abnormal labour, recent modalities in management of abnormal labour and nursing diagnosis of abnormal labor.



**CONCLUSION:**

Usually the fetal head engages in occipito anterior position and then undergoes a short rotation to be directly occipito anterior in the mid cavity. Mal positions and malpresentation are the abnormal positions of the vertex of the fetal head relative to the maternal pelvis. There is no known test that can differentiate normal from abnormal labor. The diagnosis and optimal management of abnormal labor combine science and art. The management of abnormal labor taxes one's clinical skills under the best of circumstances. Such cases are complicated by patients who demand and expect a painless and fast delivery resulting in a perfect infant. The possibility of malpractice litigation further complicates the issues—a thorough documentation of events, especially in the face of maloccurrence, cannot be stressed enough. A key to the optimal management of abnormal labor remains intensive observation with conservative, well-chosen, and carefully executed interventions.

Appropriate management during antenatal and intranatal period helps to tackle the complications during the time of labour.

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