# DIPLOMA IN REGISTERED NURSING

# eLEARNING TRAINING PROGRAM

## Course Title: Paediatrics II

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## COURSE AIM:

To equip students with knowledge and skills of paediatric conditions and management of a well and sick child

## COURSE OBJECTIVES

At the end of the course, the students should be able to:

1. Monitor the health of the child
2. Identify various conditions affecting children's health
3. Apply scientific approaches in the management of children and make appropriate referral
4. Apply the appropriate nursing model in the management of patients/clients

## UNIT 1: GENERAL PRINCIPLES OF PAEDIATRICS SURGICAL NURSING

### 1.1 Introduction

### 1.2 Objectives

By the end of the unit, you should be able to;

1. Outline the general principles of paediatrics surgical nursing

### 1.3 The general principles of paediatric surgical nursing

Before you proceed, take the following activity 1

**Activity 1**
List the principles of paediatric surgical surgery
Well done, now you can compare your answers with the information in your note books

i. Make sure that all equipment to be used in a surgical procedure is sterilized or high level disinfected.
ii. All the procedures must be done 30 minutes to an hour after cleaning the operating environment
iii. Start with children first and end with adults
iv. The hours of starvation should be related to the age of a child
v. Start with clean procedures and end with dirt ones
vi. Children should be treated in a suitable environment to their needs with their parents involvement in decisions and with optimal quality of care being delivered
vii. All those involved in children's surgical services should be suitably trained and well supported
viii. Always begin with serious cases then proceed to the non-urgent ones
ix. Wash hands before and after each and every procedure
x. Ensure that you put on protective garments before entering the operating room.

### 1.4 Summary

### 1.5 Self Assessment Test

### 1.6 References

## UNIT 2: COMMON SURGICAL CONDITIONS

### 2.1 Introduction

### 2.2 Objectives

By the end of the unit, you should be able to;

Discuss the following common surgical conditions

- Trauma
- Acute abdomen
- Burns
- Foreign bodies
- Neoplasms
- Wilm's tumour
- Burkirts lymphoma
- Neuroplasms
- Cancrum oris

### 2.3 Trauma

Trauma, according to the WHO (2009), is the sixth leading cause of death worldwide resulting in five million or 10% of all deaths. Trauma can be defined as the physical injury that causes damage to the body structure or alter the body function. Traumatic injuries can be blunt or sharp. Blunt injuries include abrasions, contusions lacerations and fractures. Sharp injuries include incisions and stab wounds.

Abrasions involve localised destruction of the outer skin layers that have directly contacted the offending force. In a contusion, blood extravagates into the tissue. A laceration involves crushing, shearing, tearing or pulling apart of tissue. A fracture involves a break in born or cartilage. An incision follows contact with a sharp object and extends along the surface of the body more than it extends in wards.

**Definitions of trauma**

There are two definitions of trauma as stated below;

i. Trauma is an injury or wound that affects or disrupts the continuity of any tissue caused by external force or violence. It generally results in a physical effect on the body for example pain and loss of function **(Hockenberry, 2004)**.

ii. . Trauma is an emotional or psychological shock that may produce disordered feelings.

The types of injury can involve various systems of the body for example skin, skeleton, nervous, respiratory, cardiovascular digestive, renal, reproductive systems and others. By knowing the normal anatomical structure and physiology of the various systems, it is possible to observe the changes that may occur due to injury to any tissue.

**Types of trauma**

i. **Polytrauma or multiple trauma** - this is a condition of a person who has been subjected to multiple traumatic injuries, such as a serious head injury in addition to a serious burn

ii. **Head injury** - Any injury that results in trauma to the skull and the brain

iii. **Chest trauma** - is any form of physical injury to the chest including the heart and lungs

iv. **Abdominal trauma** - is an injury to the abdomen. It may be blunt or penetrating and may involve damage to the abdominal organs such as the liver, spleen and stomach

v. **Extremity trauma** – these include injuries involving the legs and arms.

vi. **Facial trauma** - also called maxillofacial trauma, is any physical trauma to the face.

vii. **Spinal cord injury (SCI**) - is an injury to the spinal cord resulting in a change, either temporary or permanent, in the cord's normal motor, sensory, or autonomic function.

viii. **Genitourinary trauma** - is an injury to the organs of the genitourinary these include injuries to the kidneys, bladder, ureters and others

ix. **Pelvic trauma** – this includes pelvic ring fractures and acetabular fractures Soft Tissue Injury (STI) - is the injury to any part of the body other than bones or joints. These include injury to muscles, ligaments and tendons

**Management of a child with major trauma**

- A child with a major trauma needs an immediate intervention
- Airway control
- Use basic manoeuvres (suction, chin lift, oropharyngeal airway) to open the airway and apply $O_2$ by face mask. Avoid tilting the head or moving the neck if there is a chance of neck injury. If the airway remains obstructed despite these measures, get expert help and consider advanced maneuvers.
- Oxygen administration
- Provide high flow $O_2$ to Patients who are apnoeic or hypoventilating require assistance by bag and mask ventilation prior to tracheal intubation and IPPV.
- Cervical spine control

This is the first priority in any patient who presents with possible spine injury (e.g. neck pain, loss of consciousness). Provide immediate in-line manual cervical immobilization by placing one hand on each side of the patient's head and holding it steady (without traction) and in-line with the remainder of the spine. Whilst maintaining manual immobilization, ask an assistant to apply an appropriately sized hard cervical collar. Adhesive tape and sandbags may be applied, but may cause problems in certain patients (for example patients who are vomiting or uncooperative patients who have consumed much alcohol).

**Intravenous fluids**

- Insert a cannula in the forearm or antecubital fossae veins. If initial attempts fail, consider a femoral venous line or in a child an intraosseous line. If these fail or are inappropriate, consider a central line or a cut-down onto the long saphenous vein. However, bear in mind the difficulties and potential hazards of attempting central venous access in hypovolaemic patients.
- Commence IV fluids for patients with hypovolaemic shock with 1L of 0.9% saline 20mL/kg in children. If further IV fluid is required, alternate crystalloid with colloid and consider urgent blood transfusion once.

**Analgesia:** Adequate pain relief is often forgotten or deferred. Give morphine IV (diluted in saline to 1 mg/mL) titrated in small increments according to response. Provide an antiemetic (for example cyclizine 50mg IV) at the same time. Consider other forms of analgesia (e.g. regional nerve blocks, immobilization and splintage of fractures).

**Antibiotics:** Give prophylactic IV antibiotics for compound fractures and penetrating wounds of the head, chest or abdomen. Antibiotic choice follows local policy a broad spectrum antibiotic (for example, cefuroxime) is useful.

**Tetanus toxoid administration**

Administer tetanus prophylaxis in all patients

**Advanced Trauma Life Support**

Advanced trauma life support is done in an attempt to improve the immediate treatment of patients with serious injury. Treatment of patients with major trauma passes through the same phases:

- Primary survey
- Resuscitation phase
- Secondary survey
- Definitive care phase

**Primary survey**

On initial reception of a seriously injured patient, life-threatening problems are identified and addressed as rapidly as possible. An approach is adopted, with each of the following aspects being quickly evaluated and treated:

- Airway maintenance with cervical spine control
- Breathing and ventilation
- Circulation and haemorrhage control
- Disability (rapid assessment of neurological status)
- Exposure (the patient is completely undressed to allow full examination)

With optimum staffing and direction, instead of considering each of the above aspects sequentially aims to address these simultaneously.
Resuscitation phase

During this period, treatment continues for the problems identified during the primary survey. Further practical procedures (for example insertion of NG tube, chest drain and urinary catheter) are performed.

**Secondary survey**

This involves a head to toe examination to identify other injuries. This examination is accompanied by relevant investigations (for example X-rays). The patient is monitored throughout any deterioration necessitates a return to the assessment of ABC. Repeated clinical assessment and a high index of suspicion are essential if occult injuries are not to be missed this applies particularly to the severely injured and to those with a reduced conscious level.

**Definitive care phase**

The early management of all injuries is addressed, including fracture stabilization and emergency operative intervention.

**Investigations in major trauma**

Identification of injuries and their sequelae is based upon information gathered from the history, examination and investigations. Select specific investigations according to the presentation of each patient, but bear in mind that all patients with major trauma require: group and save/X-match, BMG, X-rays, ABG.

**Blood tests**

Check U&E, FOREIGN BODYC and glucose on all patients. If there is any possibility of significant haemorrhage, request group and save/X-match. Request baseline clotting screen in patients with major haemorrhage or those at special risk (for example alcoholics or those on anticoagulants)
Obtain serum amylase level in abdominal trauma and cardiac specific enzymes in significant chest trauma.

**X-rays**

Multiply injured patients often require multiple X-rays. Obtain CXR and pelvic X-rays as a minimum (these provide information which guides resuscitation). Obtain a lateral cervical spine X-ray if the patient's condition permits, but remember that an X-ray does not exclude spinal injury. Put on lead aprons and gloves and remain with the patient whilst X-rays are taken in particular, ensure satisfactory immobilization of the cervical spine throughout. Accompany the patient if he needs to be taken to the radiology department for further X-rays, but remember that resuscitation in this unfamiliar environment is difficult.

**Urinalysis**

Test the urine for blood if there is suspicion of abdominal injury.

**ABG**

This provides useful information, including the degree of hypoxia, hypoventilation and acidosis. In critically ill patients (especially those requiring ventilation support or those destined for neurosurgery/ITU) repeat as necessary and consider inserting an intra-arterial line to continuously monitor BP.

**ECG**

Monitor all patients and record an ECG if >50yrs or significant chest trauma.
CT scan

This is being used increasingly to aid evaluation of head, neck, and chest, abdominal and pelvic injuries. Ensure that an appropriately trained doctor accompanies the patient to the CT scanning suite and that monitoring continues. Do not transfer a patient with haemodynamic instability to the CT scanner.

**Complications of trauma**

i. Inflammation
ii. Degeneration
iii. Circulatory disturbances
iv. Infection
v. Haemorrhage
vi. Haematoma formation
vii. Airway obstruction if involving the respiratory tract

**Self Assessment Test**

**Activity 2**

You have come to the end of our topic on trauma. Read on acute abdomen which will be the next topic.

## 2.4 Acute abdomen

The term acute abdomen refers to a sudden, severe abdominal pain of unclear aetiology that is less than 24 hours in duration (Stanfied & Bwino, 2010). The acute abdomen may be defined generally as an intra-abdominal process causing severe pain and often requiring surgical intervention.

Acute abdomen is a serious condition in children frequently encountered in the paediatric emergency. The aetiology of acute abdomen varies depending on the age of the patient. Cause of acute abdomen in children older than 1 year of age, and then followed by traumatic injury. Acute appendicitis was the major and most common. The term '**acute abdomen**' represents a rapid onset of severe symptoms that may indicate life threatening. Acute severe abdomen pain is usually a cardinal feature and it is a symptom of many different types of tissue injury. In this topic you are going to look at the definition of acute abdomen, causes, signs management and complications of acute abdomen.

**Types of abdominal pains**

**i. Visceral pain**

Comes from abdominal viscera, which are innervated by autonomic nerve fibres and respond mainly to the sensations of distensions and muscular contractions .The pain is typically vague, dull and nauseating. It is poorly localised and tends to be referred to areas corresponding to the embryonic origin of the affected structure.

**ii. Somatic pain**

Comes from parietal peritoneum, which is innervated by somatic nerves, which responds to irritation from infections, chemical or other inflammatory processes
It is sharp and well localised.

### iii. Referred pain

This is pain perceived distant from its source and results from convergence of the nerve fibres at the spinal cord.
Examples: scapular pain due to biliary colic, groin pain due to renal colic and shoulder pain due to blood or infection irritating the diaphragm. Myocardial infarction- pain is felt in the neck, shoulders, and back rather than in the chest the site of injury. Referred pain is also called reflective pain

**General causes of referred pain**

**Inflammatory;** peritonitis, appendicitis (perforated)

**Mechanical:** Obstruction of intestines by worms, food bolus, volvulous and intussusceptions.

**Vascular:** Perforated peptic ulcer, perforated diverticulitis, perforated appendix, and perforated bowel

**Neoplastic:** These are growths that obstruct movement of food along the gastrointestinal tract for example, intraluminal intestinal growths.

**Traumatic:** Ruptured spleen, ruptured aorta, ruptured ectopic pregnancy, ruptured ovarian cyst, ruptured urinary bladder among others.

**Congenital defects (**Stanfied & Bwino (2010)

**Signs and symptoms**

Nausea and vomiting due to gastrointestinal disturbance and excessive peristalsis

Boborygymy (exaggerated bowel sounds – mumbling sounds)

- Abdominal tenderness on examination due to the disease process
- Constipation or diarrhoea due to dehydration
- Signs of shock due to excessive vomiting and or diarrhoea
- Hard woody abdomen due to hyper excited peritoneum
- Apparent abdominal contours due to actively peristaltic intestines
- Guarding due to severe pain

**Table 1: Mechanical and non-mechanical signs and symptoms**

The table illustrates mechanical and non-mechanical obstruction signs and symptoms

| Mechanical (Small bowel) | Mechanical (Large bowel) | Non Mechanical |
|---|---|---|
| Colicky pain | Constipation | Diffused abdominal discomfort |
| Nausea and vomiting | Vomiting in a later stage | Abdominal distension |
| Constipation | Constant hypostatic pain | Hiccups |
| Distended abdomen | Nausea | Constipation |
| Borborgymin and rashes bowel sounds | Sudden onset of colicky abdominal pains after constipation | Frequent vomiting (gastric and bowel contents |

| | | |
|---|---|---|
| Abdominal tenderness | Distended abdomen | Decreased bowel sounds |
| Rebound tenderness | Visible loops of large bowels | |
| | Loud, high pitched borborygmi | |

**Specific conditions causing acute abdomen**

**Intestinal obstruction**

**Definition of intestinal obstruction:** Intestinal obstruction is the disruption in the movement of gastrointestinal contents.

It is a common surgical emergency and because of its serious nature, it demands early diagnosis and speed relief. Intestinal obstruction may be complete in which case there is total disruption of the movement of intestinal contents or it can be incomplete where there is partial disruption. The former forms the acute form of the obstruction while the later forms the chronic form (Lewis, 2004)

**Types of Intestinal Obstruction**

**i. Dynamic**

In this type of intestinal obstruction there is an increased peristaltic process that works against an obstruction which may be in the lumen, such as a bolus of incompletely digested food material, solid stood as in constipation, a gall stone in the wall such as an inflammatory malignant stricture, intussusception (invagination of a loop of intestine up on itself) etc.

**ii. A dynamic**

In this type of intestinal obstruction there is cessation of peristalsis and no true propulsive waves are present. This commonly occurs in paralytic ileus (due to dinnervation of the intestinal wall) or mesenteric vascular occlusion.

**Pathology of intestinal obstruction**

At the onset, the intestine above the point of obstruction endeavours to overcome the obstruction by vigorous peristalsis. Increased peristalsis continues for a period of 48 hours to several days; the more distal the point of obstruction, does it remain vigorous. If the obstruction is not relieved a time is reached when increasing distension causes peristalsis to become less and less until it all ceases. The obstructed intestine becomes flaccid and paralyzed. For two or three hours following the obstruction, the intestine below the point of obstruction exhibits normal peristalsis and absorption from it continues until the residue of its contents has passed onwards. Then the empty intestine becomes immobile, contracted and pale so it remains until the obstruction has been overcome or death ensues.

Distension occurs proximal to the obstruction and begins immediately after the obstruction occurs. It is caused by gas mostly swallowed from the atmospheric air (68%), diffusion from the blood into the bowel lumen (22%) and the products of digestion and bacterial activity (10%). The distension also results from intestinal fluid which is made up of various digestive juices.

**Strangulation:** Strangulation of the bowel occurs when a loop of the intestine is trapped by a hernia or band or involved in a volvulus (twisting upon it) or intussusception (invagination) in such a way that its blood supply is progressively interfered with. This is dangerous as it leads to an immediate necrosis of the loop of intestine involved especially if the strangulation is not reversed.

**Clinical Features of Acute Intestinal Obstruction**

**i. Colicky pain**

Colicky pain is the first symptom and it commences suddenly and often without warning. It becomes increasingly severe then passes off gradually only to return at intervals of a few minutes.

**ii. Vomiting**

The type of the vomiting and vomitus depend on the level of obstruction. This is to say that if the obstruction is situated at the level of the duodenum or jejunum the vomiting is usually projectile and gastric contents are characteristic vomits while if it is far beyond the ileum involving the large intestine (colon), faecal matter are characteristic.

**iii. Abdominal distension**

In the early cases of obstruction of the small intestine abdominal distension is often slight or even absent. Centrally placed distension is present in fully established cases of the ileum. Visible peristalsis may be present. Borborygmi are sometimes loud enough to be heard by the unaided ear.

**iv. Constipation**

In complete intestinal obstruction after the contents of the bowel below the obstruction have been evacuated there is constipation and usually neither faeces nor flatus is passed.

**v. Dehydration**

Repeated vomiting and also loss of absorptive power by the distended intestine leads to dehydration so that when the patient is first examined, obvious signs of dehydration that is dry skin and tongue, and sunken eyes and others may be present.

**Peritonitis**

Peritonitis is the inflammation of the peritoneum usually due to an invasion of the peritoneal cavity Marilyn & David 2007). The peritoneum is conveniently divided into *visceral peritoneum* surrounding the abdominal organs and *parietal peritoneunl* lining the rest of the cavity and closer to the abdominal muscles. The parietal portion is richly supplied with nerves and when irritated causes severe pain accurately localised to the affected area. The visceral peritoneum on the other hand is poorly supplied with nerves and pain arising from there is vague and poorly localised.

**Paths of bacterial Invasion**

**a. Direct infection**

i. Via perforation of some part of the gastrointestinal canal
ii. Through a penetrating wound of the abdominal wall
iii. Post operatively

**b. Local extension**

i. From an inflamed organ for example. appendicitis or cholecystitis
ii. Migration through gut wall for example. strangulated hernia
iii. From or via the fallopian tubes

**c. Blood stream**

Part of general septicaemia (haematogenously)

**Clinical Features of Peritonitis**

i. Local peritonitis is bound up intimately with the causative lesion and the initial signs and symptoms are those of the lesion;

- Raised temperature
- Raised pulse rate
- Intense abdominal pain
- Rigidity of the abdominal wall over the area involved

- If the inflammation arises under the diaphragm, shoulder tip pain may be felt (referred)rectal or vaginal tenderness on examination

**ii. Diffused (Generalised) peritonitis**

- Pain is cutting or burning and made worse by moving or breathing
- Vomiting may occur
- Tenderness
- Guarding
- Rigidity
-

**Self Assessment Test**

## 2.5 Acute appendicitis

Appendicitis is inflammation of the appendix (Lewis, (2004).

**Incidence**

Acute appendicitis is rare before the age of two but becomes increasingly common during childhood and adolescence.

**Types of Appendicitis**

**a. Non Obstructive Acute Appendicitis**

In this type of appendicitis the inflammation usually commences in the mucous membranes less often in the lymph follicles like any inflammatory process. It terminates in one of the following;

- Resolution
- Suppuration
- Ulceration
- Gangrene

**b. Obstructive Acute Appendicitis**

In this type of appendicitis obstruction can be in the lumen (due to faecal matter, foreign body or parasites and others.) or in the wall. About one third of cases of acute appendicitis belong to this type.

**Aetiology of Acute Appendicitis**

**i. Race and diet**

Appendicitis is particularly common in the highly civilized European, American and Australian countries while it is rare in the Asiatic, Africans and Polynesians. The rise of appendicitis amongst is due to departure from a simple diet rich in cellulose to one relatively rich in meat.

**ii. Social status**

In England, acute appendicitis is more common among the upper and middle classes than in those belonging to the working class.

**iii. Familial susceptibility**

This unusual but generally accepted fact can be accounted for by a hereditary abnormality in the position of the organ.

iv. **Obstruction of the lumen of the appendix** when an acutely inflamed appendix has been removed, some form of obstruction to its lumen can be demonstrated in a large percentage of the cases. The obstructing agent is usually a faecolith (faecal matter), a stricture, especially a foreign body or round worm or thread worms.

**v. The abuse of purgatives**

It is abundantly clear that the ingestion of purgatives, particularly castor oil by patients with stomach ache and the violent peristaltic action which persists, favours and often determines perforation of an inflamed appendix.

**vi. VI Bacteria cultures**

These reveal that the infection is mixed and there is hardly a pyogenic organism. Common organisms are e-coli, enterococci, non-haemolytic streptococci, anaerobic streptococci and clostridium welchii.

**Clinical Features of Acute Appendicitis**

**a. Non Obstructive Acute Appendicitis**

i. Abdominal pain which shifts, as the first symptom around the umbilicus in epigastrium or it may be generalised

ii. Upset of gastric function; protective pylorospasms occur and this may be manifested by anorexia, nausea, vomiting, brown furred tongue and foul breath, constipation but occasionally there may be diarrhoea

iii. Localised tenderness at the site of the appendix. As soon as the pain has shifted there is localised tenderness either at Mc Burney's point or elsewhere.

b. Obstructive Acute Appendicitis

i. Retrocaecal: - Rigidity is often absent because the caecum is distended with gas. Psoas spasms due to inflamed appendix being in contact with this muscle.

ii. Pelvic: - When the appendix lies entirely within the pelvis there is usually complete absence of abdominal rigidity and often tenderness over Mc Burney's point is lacking as well.

iii. Post Ileal: - Although this is rare it accounts for some of the cases of missed appendix. Here the inflamed appendix lies behind the terminal ileium.

**Management**

Acute abdomen is a surgical emergency and thus it requires quick surgical intervention. All preparations should thus be made within the shortest period.

**Preoperative care**

**Pre-operative care objectives**

- To ensure that Surgery is performed as soon as possible in order to decrease the risk of complications.
- To correct or prevent fluid and electrolyte imbalance and dehydration by giving antibiotics and intravenous fluids before surgery
- To relieve pain by giving analgesia, as patient awaits surgery
- To prepare the patient physically, emotionally, psychologically so as to enable him withstand the effects of surgery and administration of anaesthesia.

**A. Physical preparation**

Investigations should be done as quickly as possible and this includes getting a blood sample for haemoglobin count, bleeding and clotting time, grouping and cross matching. A portable abdominal X – ray should be done within the shortest possible time which might reveal the presence of gases in the abdomen. Do the vital observations for baseline data. Rehydration should be done via intravenous infusion with normal saline solution or ringers lactate to replace the lost fluids and electrolytes. A Ryles (nasogastric) tube should be inserted to deflate the abdomen and for post-operative aspiration. A urethral catheter should be inserted to drain the urinary bladder and to prevent accidental injury to the bladder as it becomes an abdominal organ when full. The patient should be shaved from the nipple line to the end of the

thighs to prevent microorganisms from entering the incision site as hair harbours microorganisms. It is advisable to use a hair clipper for shaving to avoid cuts. If the patient had eaten as there is no time to starve him, the nasogastric tube is important to aspirate the gastric content and deflate the abdomen.

**B. Psychological Preparation**

Depending on the age, emotional care is directed towards allaying anxiety from the patient and relatives. It can be given directly to the patient or to the parents or guardians if under age. Briefly explain the importance of surgery and why it should not be delayed. Ensure that the signs the consent form and if under age, the parent or relative should sign.

**C. Immediate Preparation**

Ensure that prescribed pre medications if any are given within the shortest period. Remove all dentures or prostheses, jewels, label them and keep them safely. Ensure that the laboratory results, radiological results are collected and put together in the patient's file, no time to bath the patient in such as emergency but just wipe or give him a cleansing bath change in theatre gown. Label the patient for identification. Wheel the patient to theatre on a theatre trolley. At the operating theatre hand over to the theatre staff or receiving nurse giving relevant details such as patient's name, age, sex, diagnosis and proposed surgery, what was done being an emergency, the latest observations among others.

**Post-operative nursing management of a client who has undergone surgery following acute abdomen**

Depending on the type of anaesthesia used the patient may be brought onto the ward in an unconscious state or conscious. The aims of postoperative nursing care include the following;

- To prevent postoperative complications
- To promote quick recovery
- To impart knowledge

**Environment**

Nurse the patient in the general surgical ward or acute bay for close monitoring. Ensure that all sources of infection are eliminated. The patient who has undergone spinal anaesthesia should be flat in bed (spine) for a period of 4 – 8 hours when the anaesthesia has completely worn out, this is important to prevent post spinal anaesthesia headache or syndrome which is common in these post-operative patients. Ensure that the patient has no pillow placed to the head. A patient who has undergone general anaesthesia and is till unconscious should be put in the recovery position (left or right lateral) with the head tilted to one side, this ensures that secretions and or the tongue which has a tendency to fall back do not block the airway. Ensure that all the tubings are in the functional position and patent. The room should be well ventilated and well lit to allow stale air out and for easy visibility respectively. In the room there should also be post-operative devises such as suction machine and oxygen machine in case they might be needed. In the first few days a bed cradle may be used to lift off the linen from the incision site. This will allow for easy observation and also to prevent any pressure exerted on the wound.

**Observations**

Establish the baseline data of the vital signs which are; temperature, pulse rate, respiratory rate and blood pressure. These are important to determine the physiology of the body and to detect early any deviation from normal. Initially vital signs should be checked every 15 minutes and if they are stable every 30 minutes for a period of 2 hours and change to 6 hourly in the next 24 hours. When they are normal they should be checked 12 hourly. That is, twice daily. Temperature and pulse rate are important to detect early any post-operative infection. However, it should be noted that anaesthesia can affect the hypothalamus and temperature regulation centre leading to altered body temperature. Checking the blood pressure is important also to detect any hypovolemic shock especially if the patient is bleeding. Continue aspirating the gastrointestinal tract observing and recording the content. Aspiration of the gastrointestinal tract is important to prevent

paralytic ileus and to enhance quick return of the peristalsis. Observe the intravenous line for any infiltration of fluids into the tissues or extravasation as well as signs of phlebitis. Observe the return of peristalsis by auscultating the abdomen. Observe the intake and output of fluid to avoid fluid overload or under hydration, to monitor kidney function, exclude urine retention, consistence of urine and electrolyte balance,` observe the pain using the pain rating scale to determine the amount and what kind of analgesia to give. Observe the incision site for any bleeding by marking the margins about 5 – 10mm away from the site using a marker and if blood reaches the mark it means there is active bleeding hence reinforce with another bandage and inform the surgeon immediately.

**Pain management**

Assess the pain level as mentioned above. Reassure the patient and explain the physiology of pain. Ensure the patient is in a comfortable position to help relieve pain and promote rest. Give diversional therapy depending on the interest of the patient, age, literacy, and degree of pain to divert the patient's minds from focusing on the pain. Warm compresses to promote blood circulation should be done. It is also important to give the patient the prescribed analgesics before he complains of pain. The surgeon may prescribe morphine or pethidine intramuscularly and it should be given accordingly as ordered. Explain the action and importance of these pain medications.

**Nutrition and fluids**

Initially the patient is nil orally and will depend on parenteral fluids. Once peristalsis returns he will be started on surgical diet which starts with sips of plain water or fluids. If he does not vomit on day 1 post operatively start him with semi solid foods. The food should be mild or a bland diets none irritating to the bowels. The nasogastric tube normally will be removed in the next 24 hours or day 1 after surgery if there are no complications. Ensure that the diet is rich in proteins and vitamins especially vitamin C which is important in wound healing. Fluids are also important for hydration and to prevent constipation.

**Wound care**

Ensure that aseptic techniques are observed and used when managing the wound. Drains should be removed by the shortening procedure to prevent formation of fistulae if any. Sutures may be removed by the surgeon or under his order may be removed on alternate days, to prevent gapping of the wound especially in patients who are obese. Use the prescribed solution to dress the wound.

**Hygiene measures**

Initially in the first 24 hours the patient will not be disturbed however, oral care should be done to moisten the buccal mucosa and promote salivation. When the patient has stabilised, bed baths are provided to remove dirt from the body, to refresh the patient and improve his self-esteem. Shower baths can be given as the patient improves.

**Psychological care**

Reinforce the preoperative teaching to the guardians and allow them to verbalize their worries. Ensure that he does exercises when stable such as deep breathing, limb movement, coughing while supporting the incisional wound when appropriate or 1 day after surgery. This is done to prevent post-operative complications as emphasized in the preoperative teaching.

**Elimination**

Ensure that the patient is not constipated and that there is no urine retention. The patient will initially be confined in bed post operatively and hence he will have the indwelling catheter left in situ for a while to aid urinary elimination and as he is still under the effect of the anaesthesia and surgery. Early ambulation though is important as it aids in preventing constipation. Encourage the patient to be taking a diet rich in fibre when he has commenced taking orally to prevent constipation. Employ all the nursing measures to the patient with urine retention as a result of incisional pain.

**Health education on discharge**

i. Avoid weight lifting in the next 6 months to allow maximum healing.
ii. Explain to the patient on the importance of review for close monitoring and ensuring complete recovery. If discharged with sutures, he has to visit the nearest health centre for suture removal.
iii. Explain the importance of good nutrition for quick recovery, wound healing and general restoration of body energy reserves.
iv. Emphasize on the need to be vigilant with hygiene so as to prevent contamination of the wound which may lead to infection and delayed wound healing.
v. Explain on the importance of visiting the health centre as soon as he experiences any abdominal disturbance even before the review date to identify and recommend early treatment.
vi. Explain the importance of drug compliance to ensure completion of the course of antibiotics and prevent drug resistance, prevention of infection and ensure complete recovery

**Complications of abdominal surgery**

i. Haemorrhage
ii. Post-operative wound infection
iii. Evisceration
iv. Pulmonary complications
v. Dehiscence
vi. Deep vein thrombosis
vii. Adhesion formation leading to intestinal obstruction
viii. Paralytic ileus
ix. Hernia

The signs and symptoms of acute abdomen are:

- Nausea and vomiting due to gastrointestinal disturbance and excessive peristalsis.
- Boborygymy (exaggerated bowel sounds – mumbling sounds)
- Abdominal tenderness on examination due to the disease process
- Constipation or diarrhoea due to dehydration
- Signs of shock due to excessive vomiting and or diarrhoea
- Hard woody abdomen due to hyper excited peritoneum
- Apparent abdominal contours due to actively peristaltic intestines
- Guarding due to severe pain

**Self-assessment**

Thank you for being very attentive and participatory. Now answer the following questions:

i. Define acute abdomen

ii. List the conditions that can cause acute abdomen

iii. List the signs and symptoms of acute abdomen

Good, now you can compare our answers with the content below

**Answers to the above questions**

1. Acute abdomen is a condition in which there is sudden severe abdominal pains of unclear aetiology which is less than 24 hours in duration
2. The conditions that can cause acute abdomen are intestinal obstruction, Strangulation, ectopic pregnancy, peptic ulcer and ruptured ovarian cyst

## 2.6 Burns

Burns are a major cause of injuries to human beings. A significant proportion of children who have traumatic injuries are burned. Almost 1% of the population is burned or scalded each year (Stanfied & Bwino 2010).There are different types of burn injuries. The skin is usually damaged and this compromises its function as a barrier to injury and infection.

**Definition of burns**

A burn results when there is injury to the tissues of the body caused by either heat, chemicals, electrical current or radiation, (Wong L. D. (2004).

**Causes and types of burn injury**

**Thermal burns**

Can be caused by flame, scald, flash, and contact with hot objects or cold (cold thermal injury) for example frostbite

**Chemical Burns**

These are caused by acids, alkaline substances and by-products of burning substances for example carbon.

**Electrical Burns**

Result from coagulation necrosis that is caused by intense heat generated from an electrical current. This can also result from direct damage to nerves and vessels causing tissue anoxia and death (Hockenberry, 2004).

**Classification of burns**

Burns are classified on the basis of four criteria

i.The depth of injury

ii.The percentage of surface area involved

iii.The location of the burn

iv.Association with other injuries

**Partial-thickness burn**

**Superficial or 1st degree**

There is erythema, blanching on pressure, pain and mild swelling, no vesicles or blisters.
Although after 24 hours skin may blister and peel off.

**Deep or 2nd degree**

The epidermis and dermis are involved to varying depth.
There is fluid-filled vesicles that are red, shiny, wet; severe pain caused by nerve injury; mild to moderate oedema.
Some skin elements, from which epithelial regeneration can occur, remain viable.

**Full-thickness Burn**

**3rd and 4th degree**
All skin elements and nerve endings are destroyed. There is dry, waxy white, leathery or hard skin; visible thrombosed vessels; insensitivity to pain and pressure because of nerve destruction; possible involvement of muscles, tendons and bones.

**Extent of burn**

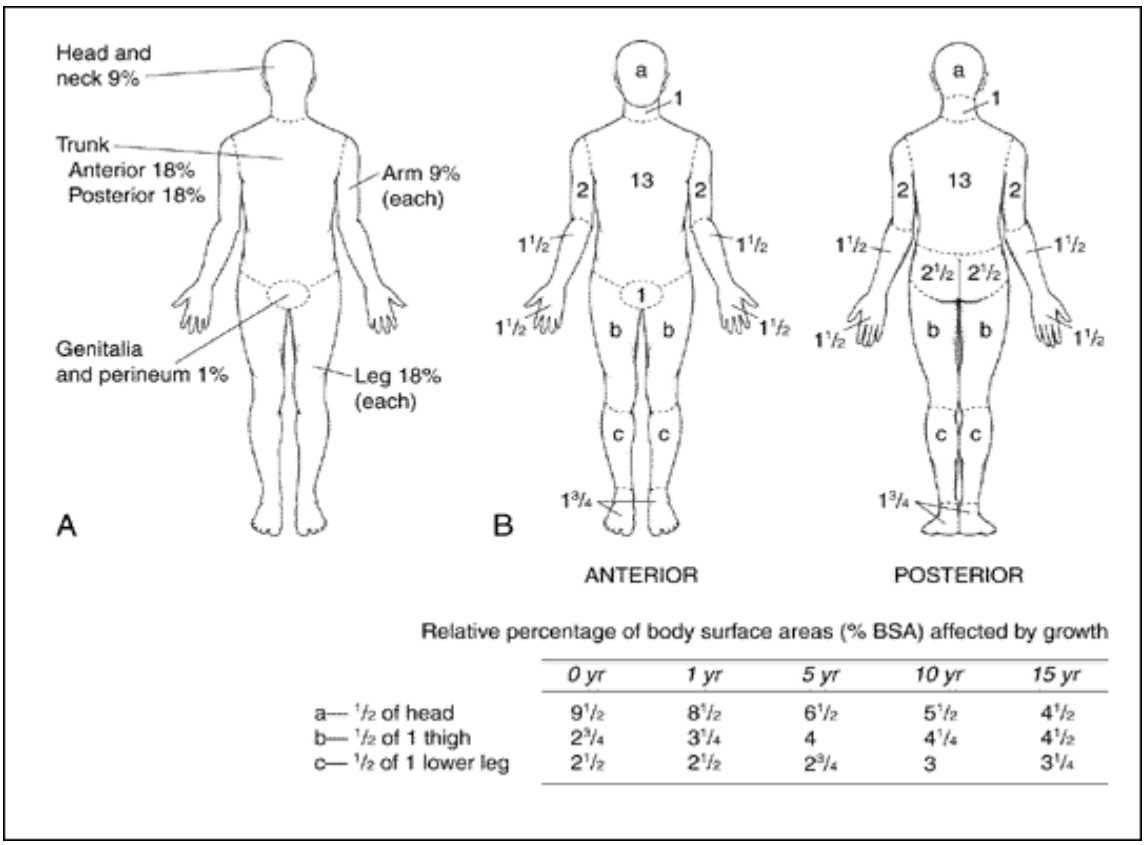
The Lund-Browder Chart and the Rule of nines are the commonly used guides for determining the total body surface area affected or the extent of a burn wound.
**Rule of nines**

**Easy to remember**

Considered adequate for initial assessment of an adult burn patient

- Head & Neck-9%,
- Arms-9%,
- Ant.trunk-18%,
- Post.trunk-18%,
- Legs-18%,
- Perineum-1%.

Total=100%.




Relative percentage of body surface areas (% BSA) affected by growth

| | *0 yr* | *1 yr* | *5 yr* | *10 yr* | *15 yr* |
|---|---|---|---|---|---|
| a— ½ of head | 9½ | 8½ | 6½ | 5½ | 4½ |
| b— ½ of 1 thigh | 2¾ | 3¼ | 4 | 4¼ | 4½ |
| c— ½ of 1 lower leg | 2½ | 2½ | 2¾ | 3 | 3¼ |

**Figure 1: Rule of nine**

## Pathophysiology

May be classified into 3; fluid and electrolyte shifts, inflammation and healing and immunological changes.

## Fluid and electrolyte shifts

Hypovolaemic shock caused by massive shift of fluids out of blood vessels as a result of increased capillary permeability. Colloidal osmotic pressure decreases with loss of protein from the vascular space leading to fluid accumulation in the interstitium. Exudates and blister formation. Signs of intravascular volume depletion for example oedema, reduced BP, and increased pulse. Normal insensible loss of 30 to 50ml/hour may increase to as much as 200 to 400ml/hour in the severely burnt patient.

## Inflammation and healing

- There is coagulation necrosis which results in tissue and vessel damage or destruction.
- Neutrophills and monocytes accumulate at the site of injury.
- Fibroblasts and newly formed collagen fibrils appear and begin wound repair within the 1st 6 to 12 hours after injury.

## Immunological changes

- Burn injury causes widespread impairment of the immune system. Skin barrier to invading organisms is destroyed. Circulating levels of immunoglobins are decreased

- Changes in WBCs quality and quantity occur
- Depression of neutrophil chemotactic, phagocytic and bactericidal activity occur

All of these changes in the immune system can make the burn patient more susceptible to infection (Lewis et al, 1996).

**Clinical Features**

- Shock due to pain and hypovolaemia.
- Pain due to exposed nerve endings.
- Blisters filled with fluid and protein may be present in partial-thickness burns.
- A dynamic or paralytic ileus as a result of the body's response to massive trauma and potassium shifts.
- Unconsciousness or altered mental status due to hypoxia associated with smoke inhalation.
- Hypotension due to hypovolaemia.

**Nursing Management of patient with severe burns**

**Admission**

Admit child in the burn unit containing all the resuscitative equipment like oxygen cylinder, suctioning machine and emergency trolley containing all the emergency drugs. The room should have good lighting system for easy motoring of the patient condition.

**Maintenance of clear airway**

- If there are respiratory problems, intubate or have patient intubated and place him/her on a ventilator.
- Positive end-expiratory pressure (PEEP) may be used to prevent collapse of the alveoli and progressive respiratory failure.
- Administer humidified air and 100% oxygen.
- Place patient in the fowler's position, unless contraindicated.
- Change patient's position every 1 or 2 hours.
- Suction patient's airway if secretions are present.
- Bronchodilators may be administered to prevent bronchospasms.

**Fluid and electrolyte replacement**

The initial fluid and electrolyte support of a burned child is critical. The first priority is to support the circulating blood volume, which requires the administering of intravenous fluids to provide both maintenance fluid and electrolyte replace on going burn losses. Establish an intravenous access that can accommodate large volumes of fluids. Replace fluids using crystalloids (Ringer's Lactate, 5% Dextrose and saline), colloids (albumin, Dextran) or a combination of the two.
The amount of fluid required for resuscitation and on-going losses is considered to be 4ml/ kg/% body surface area involved. Ringer's Lactate solution per kg body weight per % total burnt surface area (TBSA) = Total fluid requirements for 1$^{st}$ 24 hour after burn. The fluid should be given as follows:

- Half of the total to be given in the first 8hours
- Quarter of the total to be given in the second 8hours
- Quarter of the total to be given in the third 8hours

**Wound care**

- Cleanse the wound in a hydrotherapy tub, shower or bed.
- During this procedure loose necrotic tissue is removed.
- Perform the procedures efficiently and quickly.
- You may use tap water of not exceeding $40^0$c to perform this procedure.
- Because pathogenic organisms are present on the burn wound, you may use a disinfectant or cleansing agent. Bath patient twice or once daily to limit the amount of bacterial growth.
- Apply the burnt areas with topical antibiotics (for example salfadizine) and expose or use sterile gauze dressings impregnated with Vaseline or laid over the topical antibiotic.
- Change the dressings two to three times every 24hrs to once in the next three days.
- When patient's wounds are exposed, the staff must wear disposable hats, masks, gloves and gowns. Sterile gloves must be used when applying ointments and sterile dressings on the wound.
- Patient's room must be kept warm for example temperature should be $29^0$c.
- Hand washing should be practiced to prevent cross-contamination of patient's wound.

**Hygiene**

Keep patient's perineum clean and dry the entire time. Catheterise patient to prevent contamination with urine. Practice hand washing each time you are handling the patient to prevent infecting the wound. Change linen PRN to prevent cross infection. Dump dust the cubical where you are nursing the patient to minimize the number of microorganisms that can gain access on the wound thus infecting the wound. Provide frequent perineal and catheter care.

**Relief of pain and drug therapy**

Administer prescribed analgesics or narcotics for pain relief.
Administer tetanus toxoid to all burn patients to prevent the likelihood of anaerobic burn wound contamination. Cleanse the wound and apply topical antibiotics. For example silver salfadiazine or flumazine is commonly used because of its effectiveness. Silver-impregnated dressings may also be used and can be left on up to 3 days.

**Nutrition**

Give patient IV fluids only in the initial phase.
Patients with severe burns develop paralytic ileus, therefore insert a nasogastric tube connected to low intermittent suction for decompression. Initiate oral intake when bowel sounds resume, beginning with clear fluids and progressing to a diet high in proteins and energy.

**Elimination**

Patient with severe burns are prone to development of renal failure.
Catheterise patient to monitor urinary output.

**Psychological care**

Psychological care is very important in this type of patient since they have lost their cosmetic appearance. Reassure the guardians that the medical team is doing everything possible to help the patient.

Explain to the guardian the possibility for skin graft to correct defects on their body and improve the body image. Allow them to ask questions and provide answers truthfully.

**Complications**

- Hypovolaemic shock
- Contractures
- Gangrene
- Respiratory failure
- Infection
- Acute renal failure
- Deformity

Activity 3

Well learners, you have come to the end of our discussion on burns. Now let us do activity 3

1. Define burns
2. List the causes of burns
3. Mention the classification of burns
4. List the complication of burns
5. Good, you can compare your answers with the information in which is in your note books

**Self Assessment Test**

## 2.7 Foreign bodies

Foreign bodies of the ear, nose and throat refer to any object that is placed in the ear, nose or mouth that is not meant to be there and would cause harm with immediate medical attention (www.chkd.org/ent/foreign.htm)

Foreign bodies of the ear, nose and throat are usually common in children. Curiosity to explore the apertures (openings) of the body is usually the reason why children put foreign bodies in their nostrils and ears. However, some objects are inserted intentionally into the ear by adults who may have been trying to clean the external canal or relieve itching, while foreign bodies of the throat may accidentally embed themselves in the throat especially when eating. Intellectually challenged or mentally ill adults are also at increased risk (Berkow et al, 2005).

**Epidemiology**

- Foreign bodies are more common in children than in adults. 75% of foreign bodies of the ear occur in children younger than 8 years.
- These foreign objects are usually asymptomatic and are often an accidental finding.
- Foreign bodies in the airway accounts for nearly 9% of all home accidental deaths in children under five. According to the American Academy of paediatrics, deaths in children occur most often in children younger than

five years. Infants younger than 1 year old account for two-thirds of child choking victims. Insects are more common in children older than 10 years and adults

**Types of foreign bodies**

**The ear**

- Insects such as cockroaches
- Beads
- Vegetative objects such bean seeds, peas and nuts
- Imparted cerumen

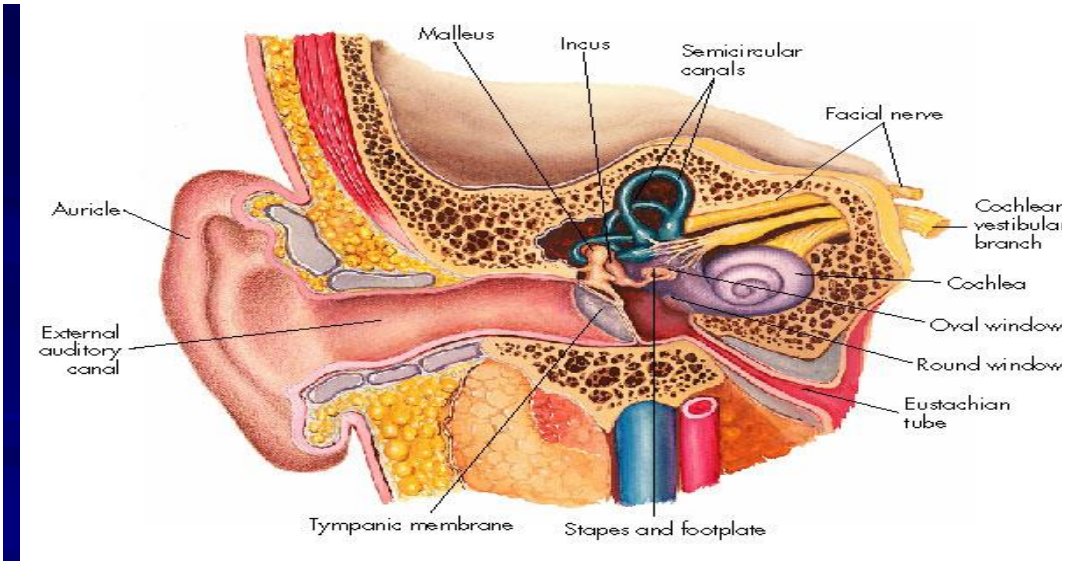



**Figure 2: The ear**

**The nose**

- Buttons and beads
- Wood and cotton
- Paper, cloth toy parts, pebbles and candle wax.

**The throat**

- Dentures
- Fish bones
- Metal pins
- Small batteries
- Seeds
- Coins
- Balloons
- Pieces of deformable plastics
- Food boluses

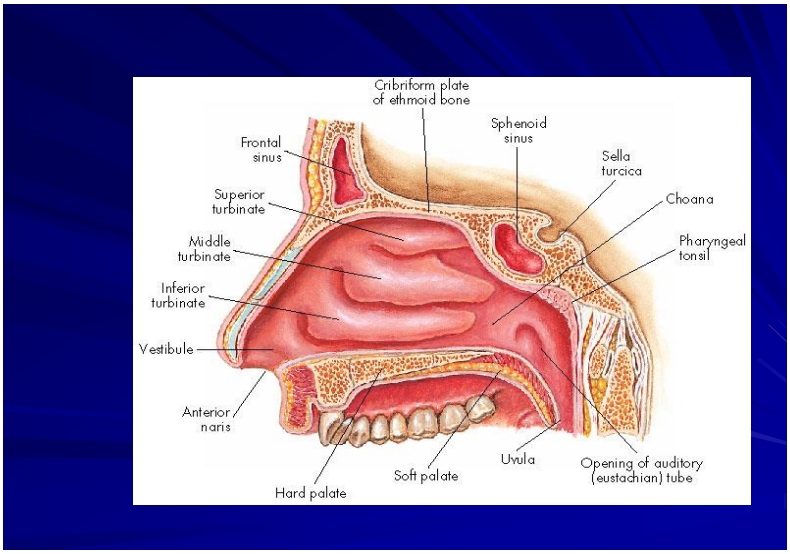



**Figure 3: Nose and throat**

## Clinical presentation

- Pain
- Dysphagia
- Dyspnoea
- Local inflammatory reaction
- Nasal discharge
- Choking
- Coughing
- Wheezing

## Investigations

- X-ray-
- Endoscope
- Audiography

## Assessment of the ear

## History

-Obtain a thorough history of what happened.

## Physical Examination

- -Inspection
- -Palpation
- -Observation (Stanfied & Bwino (2010),

**Management of foreign body in the ear**

- No irrigation when there is a discharge or perforated ear drums.
- Inanimate objects must be killed before removal
- Foreign bodies of vegetative nature must not be flushed out using water.
- Refer to ENT specialist

**Assessment of the nose**

**History**

- Obtain full history
- Find out the type of foreign body

**Physical Examination**

- Inspection
- Palpation

**Management of foreign body of the nose**

- Foreign bodies must be removed through the route of entry.
- Irrigation of the nose or pushing the objects backwards must be avoided.
- Use local anaesthesia in difficult cases.
- Refer to ENT specialists in cases of failed measures.

**Assessment of foreign body in the throat**

- Foreign bodies of the throat are a life- threatening situation, which requires prompt action by specialists.
- If objects obstruct the entire airway, signs of asphyxia are evident and this calls for emergency assessment and management.

**Assessment of the throat**

**History**

- -History is taken to determine the problem

**Physical examination**

- -Inspection
- -Assess vital signs and respiratory status noting signs of respiratory distress

**Signs of respiratory distress**

- Dyspnoea
- Tachycardia

- Inspiratory Stridor
- Restless

**Management of foreign bodies in throat**

- Treatment of the problem varies with the degree of air blockage.
- If the object is visible and large, a forceps is used to remove it.
- If not visible x-ray may be done
- Heeblich manoeuvre is performed.
- If object is in the trachea with respiratory distress, a tracheostomy is performed.

**Complications**

- Airway obstruction
- Laryngeal oedema
- Pushing the foreign body into the sub-glotic space, oesophagus or trachea.
- The object may be pushed completely into the bony portion of canal, lacerating the skin and perforating the tympanic membrane leading to loss of hearing and deafness.
- Secondary infections leading to septicaemia and bacteria (Hockenberry, 2004).

**Health teaching**

- Keep objects out of the ear
- Clean ears with only a washcloth and finger
- Avoid use of bobby pins and cotton tipped applicators to pock the ear.
- Ensure that children are monitored as they play with toys.

**Activity 4**

Well learners, having discussed foreign bodies, now take activity 4

1. Define foreign bodies
2. List the most common foreign bodies in children
3. List the complications of foreign bodies

Good participation. You can now compare your answers with the information in your note books

**Conclusion**

Foreign bodies are common both in adults and children though there is a higher incidence in children. They account for 9% of all home accidental deaths. Those of the airway constitute a medical emergency and the client must seek immediate medical care. Treatment of the air way problem varies with the degree of airway blockage. Delayed removal of the foreign body from the ear may lead to infections like otitis media and deafness. Foreign bodies of the throat are an emergency in that any complete obstruction may lead to severe respiratory symptoms like respiratory distress and death if emergency procedures like tracheotomy are not done

**Self Assessment Test**

## 2.8 Neoplasms

### 2.8.1 Wilm's tumour

Wilm's tumour or Nephroblastoma is cancer or malignant tumour of the kidneys that typically occurs in children and rarely in adults (Hockenberry, 2004).

**Wilms tumour** or **nephroblastoma** is cancer of the kidneys that typically occurs in children, rarely in adults. It is named after Dr Max Wilms, the German surgeon (1867–1918) who first described it. Approximately 500 cases are diagnosed in the U.S. annually. The majority (75%) occurs in otherwise normal children; minorities (25%) are associated with other developmental abnormalities. It is highly responsive to treatment, with about 90% of patients surviving at least five years.

**Symptoms**

Typical symptoms are:-

- An abnormally large abdomen
- Abdominal pain
- Fever
- Nausea and vomiting
- Blood in the urine (in about 20% of cases)
- High blood pressure in some cases (especially if synchronous or metachronous bilateral renal involvement

**Diagnosis**

- The first sign is normally a painless abdominal tumour that can be easily felt by the doctor.
- An ultrasound scan,
- Computed tomography scan
- MRI scan is done first. A tumour biopsy is not typically performed due to the risk of creating fragments of cancer tissue and seeding the abdomen with malignant cells

**Staging**

Staging is determined by combination of imaging studies and pathology findings if the tumour is operable. Treatment strategy is determined by stage:

**Stage 1 (43% of patients)**

For stage I Wilms' tumour, all of the following criteria must be met:

- Tumour is limited to the kidney and is completely excised.
- The surface of the renal capsule is intact.
- The tumour is not ruptured or biopsied (open or needle) prior to removal.
- No involvement of extrarenal or renal sinus lymph-vascular spaces
- No residual tumour apparent beyond the margins of excision.
- Metastasis of tumour to lymph nodes not identified.

**Stage 2 (23% of patients)**

**For Stage 2 Wilms' tumour**, 1 or more of the following criteria must be met:

- Tumour extends beyond the kidney but is completely excised.
- No residual tumour apparent at or beyond the margins of excision.
- Any of the following conditions may also exist:
- Tumour involvement of the blood vessels of the renal sinus and/or outside the renal parenchyma.
- The tumour has been biopsied prior to removal or there is local spillage of tumour during surgery, confined to the flank.
- Extensive tumour involvement of renal sinus soft tissue.

**Stage III (23% of patients)**

For Stage III Wilms' tumour, 1 or more of the following criteria must be met:

- Unresectable primary tumour.
- Lymph node metastasis.
- Tumour is present at surgical margins.
- Tumour spillage involving peritoneal surfaces either before or during surgery, or transected tumour thrombus.

**Stage IV (10% of patients)**

Stage IV Wilms' tumour is defined as the presence of hematogenous metastases (lung, liver, bone, or brain), or lymph node metastases outside the abdomenopelvic region.

**Stage V (5% of patients)**

Stage V Wilms' tumour is defined as bilateral renal involvement at the time of initial diagnosis. Note that, For patients with bilateral involvement, an attempt should be made to stage each side according to the above criteria (stage I to III) on the basis of extent of disease prior to biopsy

**Management**

- Nephrectomy followed by radiotherapy
- Chemotherapy: Vincristine, doxorubicin, cyclophosphamide

**Prognosis**

Most children with Wilm's' tumour can be cured of cancer. Long-term survival rates generally are greater than 90%.

**Self-Assessment**

Thank you for being attentive. You can do self- assessment 1

1. …….. is a concer or malignant tumour of the kidney is called

2. In stage …… of the disease, the Wilm's tumour is limited to the ……

3. Nephroblastoma is common between …… years and rare after 5 years.

4. Wilm's tumour is associated with ……..

**Answers to the above questions**

1. Nephroblastoma or Wilm's tumour

2. Kidney

3. 3-4

**4.** Genetic

### 2.8.2 Burkitts lymphoma

Burkitt 's lymphoma is a tumour of the lymphatic system in which the cancer starts in immune cells called B-cells (Hockenberry, 2004).

Burkett's lymphoma is a form of non-Hodgkin's lymphoma in which cancer starts in immune cells called B-cells. Recognized as the fastest growing human tumour, Burkett's lymphoma is associated with impaired immunity and is rapidly fatal if left untreated. However, intensive chemotherapy can achieve long term survival in more than half the people with Burkitst lymphoma.

Burkett's lymphoma is named after British surgeon Denis Burkett's, who first identified this unusual disease in 1956 among children in Africa. In Africa, Burkitt lymphoma is common in young children who also have malaria and Epstein-Barr, the virus that causes infectious mononucleosis. One mechanism may be that malaria weakens the immune system's response to Epstein-Barr, allowing it to change infected B-cells into cancerous cells. About 98% of African cases are associated with Epstein-Barr infection.

Outside of Africa, Burkitt lymphoma is rare. In the U.S., about 1,200 people are diagnosed each year, and about 59% of patients are over age 40. Burkitt lymphoma is especially likely to develop in people infected with HIV, the virus that causes AIDS. Before highly active antiretroviral therapy (HAART) became a widespread treatment for HIV/AIDS, the incidence of Burkitt lymphoma was estimated to be 1,000 times higher in HIV-positive people than in the general population.

### Types of Burkitt's lymphoma

In the World Health Organization classification, there are three types of Burkitt lymphoma:

- Endemic (African). Endemic Burkitt lymphoma primarily affects African children ages 4 to 7 and is twice as common in boys as in girls. Sporadic (non-African).
- Sporadic Burkitt lymphoma occurs worldwide. Globally, it accounts for 1% to 2% of adult lymphoma cases. In the U.S. and Western Europe, it accounts for up to 40% of paediatric lymphoma cases.
- Immunodeficiency-associated. This variant of Burkitt lymphoma is most common in people with HIV/AIDS. It accounts for 30% to 40% of non-Hodgkin lymphoma in HIV patients and may be an AIDS-defining disease. It also can occur in people with congenital conditions that cause immune deficiency and in organ-transplant patients who take immunosuppressive drugs.

**Symptoms of Burkitt lymphoma**

The symptoms of Burkitt lymphoma depend on the type. The endemic (African) variant usually starts as tumours of the jaw or other facial bones. It also can affect the gastrointestinal tract, ovaries, and breasts and can spread to the central nervous system, causing nerve damage, weakness, and paralysis. Other symptoms associated with Burkitt lymphoma include:

- Loss of appetite
- Weight loss
- Fatigue
- Night sweats
- Unexplained fever

**Diagnosis of Burkitt Lymphoma**

Because Burkitt lymphoma spreads so quickly, prompt diagnosis is essential.-

- If Burkitt lymphoma is suspected, all or part of an enlarged lymph node or other suspicious disease site will be biopsied.
- In a biopsy, a sample of tissue is examined under a microscope, this will confirm or rule out Burkitt lymphoma.

Additional tests may include:

- Computed tomographic (CT) imaging of the chest, abdomen, and pelvis
- Chest X-ray
- PET or gallium scan
- Bone marrow biopsy
- Exam of spinal fluid
- Blood tests to measure kidney and liver function
- Testing for HIV disease

**Treatments for Burkitt Lymphoma**

Intensive intravenous chemotherapy which usually involves a hospital stay is the preferred treatment for Burkitt lymphoma. Because Burkitt lymphoma can spread to the fluid surrounding the brain and spinal cord, chemotherapy drugs also may be injected directly into the cerebrospinal fluid, a treatment known as intrathecal chemotherapy.

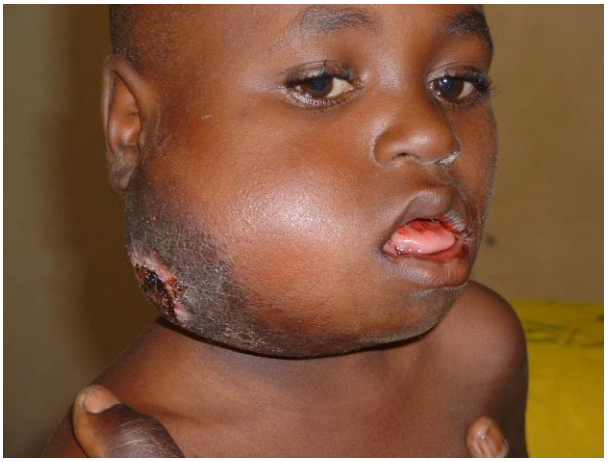
Examples of drugs that may be used in various combinations for Burkitt lymphoma include:

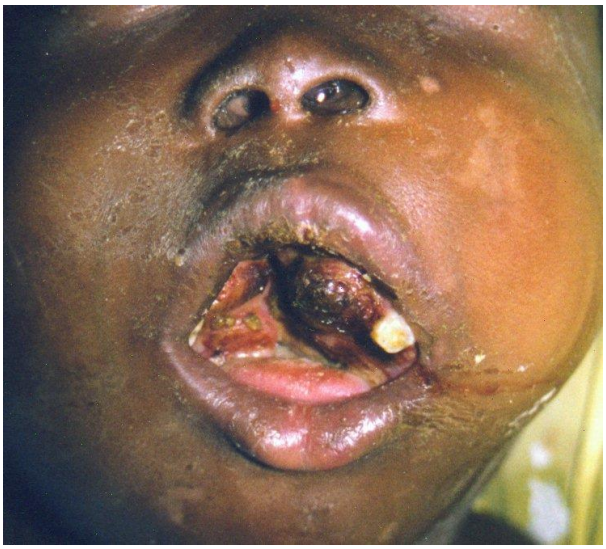
- Cyclophosphamide (Cytoxan)
- Cytarabine (Cytosar-U, Tarabine PFS)
- Doxorubicin (Adriamycin)
- Etoposide (Etopophos, Toposar, vepesid)
- Methotrexate (Rheumatrex)
- Vincristine (Oncovin)

Other treatments for Burkitt lymphoma may include intensive chemotherapy in combination with:

- Rituximab (Rituxan), a monoclonal antibody that sticks to proteins on cancer cells and stimulates the immune system to attack cancer cells
- Autologous stem cell transplantation, in which the patient's stem cells are removed, stored, and returned to the body
- Radiation therapy
- Steroid therapy

In some cases, surgery may be needed to remove parts of the intestine that are blocked, bleeding, or have ruptured.





**Prognosis**

The outcome depends on the stage at diagnosis. It is often worse for adults over age 40, though treatment for adults has improved in recent years. The prognosis is poor in people who have HIV/AIDS. It is significantly better in people whose cancer has not spread.

**Self-assessment 2**

Well learners, you have come to the end of our discussion on burkirts lymphoma. You can now do the following exercise:

Put true or false against each option

1. .....In burkirts lymphoma the cancer starts in immune cells called B-cells.
2. .....Burkirts lymphoma is associated with conditions such as chronic pneumonia
3. ……The two forms of lymphoma are Hodgkin's and non Hodgkin's lymphoma
4. ……Endemic burkirts lymphoma affects children that live in malaria endemic areas
5. ……Burkirts lymphoma is associated with epistern bar virus
6. ……..Chemotherapy is not the best treatment for burkirts lymphoma

Well done, you can compare our answers with the content below

### 2.8.3 Neurblastoma

**Neuroblastoma** is the most common extracranial solid cancer in childhood and the most common cancer in infancy, with an incidence of about six hundred fifty cases per year in the U.S. and a hundred cases per year in the UK.

Nearly half of neuroblastoma cases occur in children younger than two year. It is a neuroendocrine tumour, arising from any neural crest element of the sympathetic nervous system. It most frequently originates in one of the adrenal glands which have similar origins to nerve cells and sit on top of the kidneys, but can also develop in nerve tissues in the neck, chest, abdomen, or pelvis. Neuroblastoma is one of the few human malignancies known to demonstrate spontaneous regression from an undifferentiated state to a completely benign cellular appearance. It is a disease exhibiting extreme heterogeneity, and is stratified into three risk categories

**Definition**

Neuroblastoma is a type of cancer that starts in certain very early forms of nerve cells found in an embryo or fetus Hockenberry M.J. (2004).

**Cause**

- The ethology of neuroblastoma is not well understood.
- About 1-2% of cases run in families and has been linked to specific gene mutations.

**Predisposing factors**

- Parental factors around conception and during gestation.
- Occupation (that is exposure to chemicals in specific industries)
- Smoking, alcohol consumption, use of medicinal drugs during pregnancy and birth factors; however, results have been inconclusive.
- Other studies have examined possible links with atopy and exposure to infection early in life
- Use of hormones and fertility drugs

- Maternal use of hair dye.

**Signs and symptoms**

The first symptoms of neuroblastoma are often vague making diagnosis difficult. Fatigue, loss of appetite, fever, and joint pain are common. Symptoms depend on primary tumour locations and metastases if present:

- In the abdomen, a tumour may cause a swollen belly and constipation.
- A tumour in the chest may cause breathing problems.
- A tumour pressing on the spinal cord may cause weakness and thus an inability to stand, crawl, or walk.
- Bone lesions in the legs and hips may cause pain and limping.

▪ A tumour in the bones around the eyes or orbits may cause distinct bruising and swelling.
▪ Infiltration of the bone marrow may cause pallor from anaemia.

Neuroblastoma often spreads to other parts of the body before any symptoms are apparent and 50 to 60% of all neuroblastoma cases present with metastases.

The most common location for neuroblastoma to originate (the primary tumour) is on the adrenal glands. This occurs in 40% of localised tumours and in 60% of cases of widespread disease. Neuroblastoma can also develop anywhere along the sympathetic nervous system chain from the neck to the pelvis. Frequencies in different locations include: neck (1%), chest (19%), abdomen (30% non-adrenal), or pelvis (1%). In rare cases, no primary tumour can be discerned.

**Diagnosis**

**Biochemistry-** blood and urine test will show elevated levels of catecholamine or their metabolites.

**Imagin-** the mIBG scan (meta-iodobenzylguanidine), which is taken up by 90 to 95% of all neuroblastomas, often termed ‘mIBG-avid. The mechanism is that mIBG is taken up by sympathetic neurons, and is a functioning analog of the neurotransmitter rnorepinephrine. When it is radio-ionated with I-131 or I-123 (radioactive iodine isotopes), it is a very good radiopharmaceutical for diagnosis and monitoring of response to treatment for this disease. With a half-life of 13 hours, I-123 is the preferred isotope for imaging sensitivity and quality. I-131 has a half-life of 8 days and at higher doses is an effective therapy as targeted radiation against relapsed and refractory neuroblastoma.

**Histology-** On microscopy, the tumour cells are typically described as small, round and blue, and rosette patterns may be seen.

**Staging**

The ‘International Neuroblastoma Staging System’ (INSS) established in 1986 and revised in 1988 stratifies neuroblastoma according to its anatomical presence at diagnosis:

- **Stage 1:** Localised tumour confined to the area of origin.
- **Stage 2A:** Unilateral tumour with incomplete gross resection;
- **Stage 2B:** Unilateral tumour with complete or incomplete gross resection.
- **Stage 3:** Tumour infiltrating across midline with or without regional lymph node involvement; or unilateral tumour with contralateral lymph node involvement; or midline tumour with bilateral lymph node involvement.
- **Stage 4:** Dissemination of tumour to distant lymph nodes, bone marrow, bone, liver, or other organs except as defined by Stage 4S.
- **Stage 4S:** Age <1 year old with localised primary tumour as defined in Stage 1 or 2, with dissemination limited to liver, skin, or bone marrow (less than 10% of nucleated bone marrow cells are tumours).

**Treatment**

When the lesion is localised, it is generally curable. However, long-term survival for children with advanced disease older than 18 months of age is poor despite aggressive multimodal therapy (intensive chemotherapy, surgery, radiation therapy, stem cell transplant, differentiation agent isotretinoin also called 13-*cis*-retinoic acid, and frequently immunotherapy with anti-GD2 monoclonal antibody therapy). Biologic and genetic characteristics have been identified, which, when added to classic clinical staging, has allowed patient assignment to risk groups for planning treatment intensity. These criteria include the age of the patient, extent of disease spread, microscopic appearance, and genetic features including DNA ploidy and N-myc oncogene amplification (N-myc regulates microRNAs into low, intermediate, and high risk disease.

The therapies for these different risk categories are very different.

- Low-risk disease can frequently be observed without any treatment at all or cured with surgery alone.
- Intermediate-risk disease is treated with surgery and chemotherapy.
- High-risk neuroblastoma is treated with intensive chemotherapy, surgery, radiation therapy, bone marrow / hematopoietic stem cell transplantation, biological-based therapy with 13-*cis*-retinoic acid (isotretinoin or Accutane) and antibody therapy usually administered with the cytokines GM-CSF and IL-2.

With current treatments, patients with low and intermediate risk disease have an excellent prognosis with cure rates above 90% for low risk and 70%-90% for intermediate risk. In contrast, therapy for high-risk neuroblastoma the past two decades resulted in cures only about 30% of the time. The addition of antibody therapy has raised survival rates for high-risk disease significantly

Chemotherapy agents used in combination have been found to be effective against neuroblastoma. Agents commonly used in induction and for stem cell transplant conditioning are platinum compounds (cisplatin, carboplatin), alkylating agents (cyclophosphamide, ifosfamide, melphalan), topoisomerase inhibitor (etoposide), anthracycline antibiotics (doxorubicin) and vincaalkaloids (vincristine). Some newer regimens include topoisomerase inhibitors (topotecan and irinotecan

Activity

- Define Neuroblastoma
- List the risk factors to Neuroblastoma
- Mention the signs and symptoms of Neuroblastoma
- Thank you for your active participation. You now compare your answers with what is in your note books

**Conclusion**

Neoplasms are new growths that are either cancerous or non-cancerous. They are an abnormal mass of tissue arising from an abnormal proliferation of cells. These growths are Burkitt's Lymphoma, Wilm's tumour and Neuroblastoma. Most of these neoplasms are hereditary in nature. With good treatment, neoplasms can be treated and the prognosis is good. Where they bear poorly managed, the consequences can be fatal.

Read on Cancrum oris which you are going to discuss in our next

**Self Assessment Test**

## 2.9 Cancrum oris

Cancrum oris is a rapidly progressive, polymicrobial, opportunistic infection that occurs during periods of compromised immune function (Hockenberry, 2004).

Cancrum oris is a gangrenous process of the mouth, which starts suddenly, rapidly involves the adjacent tissues of the face, quickly becomes well demarcated, and then spreads no further. The lesion starts inside a child's mouth, in association with acute ulcerative gingivitis, and then spreads to his lips and cheeks. The earliest stage, which is seldom seen, is a painful red or purplish-red spot, or indurated papule, on his alveolar margin, most often in his premolar or molar region. This lesion rapidly forms an ulcer, which exposes his underlying alveolar bone. At this stage, the patient has a sore mouth, a swollen, tender, painful lip or cheek, profuse salivation, and an extremely foul smell, with purulent discharge from his mouth or nose. Within the next 2 or 3 days, a bluish-black area of discolouration appears externally on his lips, or cheek. The gangrenous area is cone-shaped, so that much more tissue is destroyed inside his mouth, than his external wound might indicate. After separation of the slough, his exposed bone and teeth rapidly sequestrate.

### Incidence

Mainly affects children under the age of twelve in the poorest countries of Africa, especially between the 2-6 years old.

### Causes

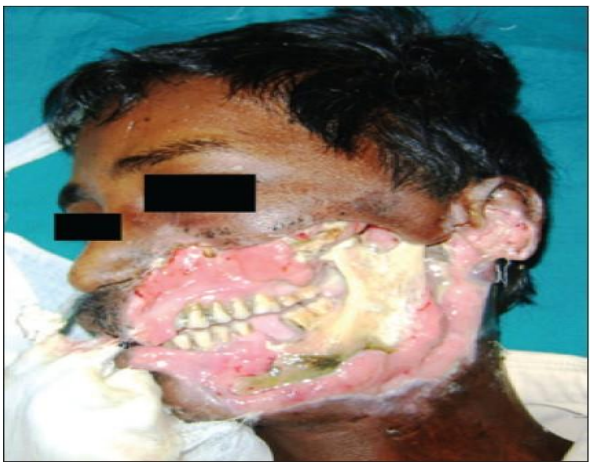
- Fusobacterium necrophorum
- Prevotella intermedia
- Borrelia vincentii
- Porphyromonas gingivalis
- Tannerella forsynthesis
- Treponema denticola,
- Staphylococcus aureus
- Nonhaemolytic Streptococcus

### Predisposing factors include

- Malnutrition (particularly A-and B-vitamins) or dehydration
- Poor hygiene, particularly oral
- Unsafe drinking water
- Proximity to unkempt livestock
- Recent illness
- An immunodeficiency disease, including AIDS

**Clinical presentation**

The mucous membranes of the mouth develop ulcers, and rapid, painless tissue degeneration ensues, which can degrade tissues of the bones in the face

**Management**

In the acute stages of cancrun oris, start emergency treatment immediately, and aim to build up the child's general resistance. If possible admit him. There is no need to isolate him. If admission is impractical, outpatient treatment is possible.

**Feeding and electrolytes**

Correct his protein energy malnutrition by feeding him by mouth. If his mouth is too sore, feed him through a nasogastric tube.

**Antibiotics**

• Give him penicillin in large doses and metronidazole.

**Care for the lesion**

- Repeatedly irrigate it with saline.
- Chewing raw pineapple, or slices of orange, will help to clean his mouth.
- Pack cavities with gauze pads soaked in hypochlorite ("Eusol'), saline.
- Change the dressings often, and keep them moist by adding more solution to the outer layers.
- Avoid Vaseline gauze (which acts like a foreign body), especially when it has been impregnated with antibiotics.
- Reconstructive plastic surgery to repair
- Reconstruction is usually very challenging and should be delayed until full recovery (usually about one year following initial intervention).
- Link the survivors to international charitable organizations
- If he is fit enough for surgery, cut away any separating sloughs, and remove any loose teeth or sequestra. When quite large sequestra are ready to separate, you may be able to remove them under ketamine.
- If he is too ill for surgery, allow the dead tissues to separate spontaneously.

- Sequestra occasionally drop out.
- More often, they have to be removed after 3 or 4weeks, when his condition has improved enough for surgery to be safe.

**Complications**

- Septicaemia
- Aspiration pneumonia.
- Secondary haemorrhage
- Avernous sinus thrombosis
- Permanent deformities

**Activity**

Well learners, having looked at cancrum oris, let us now do activity 2

**1.** Define cancrum oris
**2.** List the causative organisms of cancrum oris
**3.** List the risk factors to cancrum oris
**4.** Mention the complications of cancrum oris

Good, now compare your answers with the information in your note books

**Self Assessment Test**

## 2.10 Summary

Cancrum oris is a gangrenous process of the mouth, which starts suddenly, rapidly involves the adjacent tissues of the face, quickly becomes well demarcated, and then spreads no further. It mainly affects children under the age of twelve in the poorest countries of Africa, especially between the 2-6 years old. The causative organisms are Tannerella forsynthesis, treponema denticola, staphylococcus aureus, Nonhaemolytic Streptococcus, Tannerella forsynthesis, Treponema denticola, staphylococcus aureus and nonhaemolytic streptococcus. It can be predsiosed by factors such as Malnutrition (particularly A-and B-vitamins) or dehydration, poor hygiene, particularly oral, unsafe drinking water andproximity to farm animals among others. Cancrum oris is usually treated with large doses of peinicin and metronidazle and daily cleaning with saline. Reconstructive surgery can also be done to repair the affected area. The complications of cancrum oris are septicaemia, aspiration pneumonia, secondary haemorrhage, avernous sinus thrombosis and permanent deformities.

Cancrum oris is a very devastating disease. Therefore, it is important to link the survivor to charitable organizations.

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