NURSING LEVE III

NTQF III

LEARNING GUIDE#35

Unit of Competence: Undertake basic wound care

Module Title : Undertaking basic wound care

Code: HLT NUR3 M07 LO1 – LG33

TTLM Code: HLT NUA3 TTLM 0919v1

LO1: Undertake wound assessment

Instruction sheet -1	learning guide 1

This learning guide is developed to provide you the necessary information regarding the following content coverage and topics:

Introduction to wound care

- Chain of infection
- Wound
- Types /Classification

This guide will also assist you to attain the learning outcome stated in the cover page. Specifically, upon completion of this Learning Guide, you will be able to:

- Aacquire Necessary medical information from appropriate person.
- evaluated Wound condition in accordance with the standard procedures.
- Provide Feedback on the condition of the wound to patient.
- follow Client cooperation in secured using the medical manual.
- Utilize Strategies to minimise cross-infection during assessment and implementation.
- Record Findings appropriately
- Identify Wound care , after the approval of registered nurse, is agreed for implementation

Learning Instructions:

Read the specific objectives of this Learning Guide.

1, Follow the instructions described in number 3 to 7.

2, Read the information written in the “Information Sheets ”. Try to understand what are being discussed. Ask your teacher for assistance if you have hard time understanding them.

3, Accomplish the “Self-check ,in page 22,31,58, Operation sheet on page 60,61 and LAP62, Reference 62

4, Ask from your teacher the key to correction (key answers) or you can request your teacher to correct your work. (You are to get the key answer only after you finished answering the Self-check 1).

5, If you earned a satisfactory evaluation proceed to “Information Sheet ”. However, if your rating is unsatisfactory, see your teacher for further instructions or go back to Learning Activities

6, Submit your accomplished Self-check.This will form part of your training portpholi

Information Sheet-1	Introduction to wound care

1.1. Introduction to wound care

Patient assessment

Wound healing is determined by the general health of the patient. The assessment of the patient as a whole is critical for the planning and evaluation of care and should include:

- Medical history
- Cause of tissue damage
- Medication/Allergies
- Other diseases such as:
  - Diabetes
  - Vascular disease
  - Immune compromise
- Inadequate nutrition
- Lifestyle/Environment
  - Obesity
  - Tobacco/Alcohol abuse
- Impaired mobility
- Inadequate social network, caregiver support
- Psychological problems

Wound assessment

Wound assessment is not an exact science, but requires the skills and assessment of trained professionals. The following need to be assessed and carefully recorded at each dressing change:

- Cause: determine etiology
- Local wound characteristics:
  - Location
  - Size (length x width x depth)
  - Wound bed (black, yellow, red, pink, undermined)
  - Exudate (copious, moderate, mild, none)
  - Wound edge (callus and scale, maceration, erythema, edema)
  - Odor (absent, present)

• Patient concerns: pain (persistent, temporary)

• Condition of surrounding skin (normal, edema, warmth, erythema)

• Clinical signs of critical colonization/local infection and infection

Assessment of the wound is a prerequisite to the selection of an appropriate dressingCaring for patient's wounds is a large part of many nurses' jobs. If you are a flight or emergency room (ER) nurse you see fresh trauma wounds; if you work on a floor in a hospital you may well be in charge of taking care of surgical wounds. Knowing how to assess a wound is key to taking care of your patient.

Nurses commonly assess both "untreated" and "treated" wounds. "Untreated" wounds are those found at the scene of an accident or in the Emergency Department. Assessing in the field or the ER starts with basic emergency care, in

other words, your A-B-Cs. Once you have determined the victim has a clear airway, is breathing adequately and has a pulse then you look at the wound.

Assessing Untreated Wound

1. Check the size and severity of the wound. If you are in the field arrange for transport, if you are in an ED arrange for a physician.
2. Inspect for bleeding. How much blood depends on the wound type and location.
   penetrating wounds may cause internal bleeding.
3. Look for foreign bodies such as soil, broken glass, shreds of cloth or other substances.
4. If the wound is contaminated with foreign material, determine when the client last had a tetanus shot.
5. Assess for associated injuries such as fractures, internal bleeding, spinal cord injuries, or head trauma.

Guidelines for Care of the Untreated Wound

Control severe bleeding by applying direct pressure over the wound and elevating if it is on an extremity.

Prevent infection by cleaning or flushing abrasions or lacerations with water and covering the wound with a clean or sterile dressing, if possible. When applying a dressing, wrap the wound tightly enough to apply pressure and approximate the wound edges, if possible. If bleeding saturates the first dressing, apply a second layer without removing the original dressing.

Removing it may disturb clots that have already formed and increase bleeding. Apply ice to the wound to reduce swelling and pain. If bleeding is severe or internal bleeding is suspected assess the patient for signs of shock.

Assessing Treated Wound

"Treated" wounds are usually assessed to determine the progress of healing. They may be inspected during a dressing change, however if the wound itself cannot be directly inspected, the dressing is inspected and other data, such as pain, assessed.

These days many "treated" wounds are covered with a transparent occlusive dressing that permits observation of the wound with complete exposure. You will be assessing using the following guidelines

Appearance – Inspect color of wound and surrounding area and approximation of wound edges.

Size – Note size and location of dehiscence, if present. For wounds healing by "secondary intention" measure the length, width, and depth in centimeters.

Drainage – Observe location, color, consistency, odor, and degree of saturation of **dressings**. Note number of gauzes saturated or diameter of drainage on gauze.

Swelling – Wearing sterile gloves, palpate wound edges for tension and tautness of tissues. A small to moderate amount of swelling is normal in the early stages of healing.

Pain – Expect severe to moderate postoperative pain for three to five days; persistent severe pain or sudden onset of pain may indicate hemorrhaging or infection.

Drains or Tubes – Inspect drain security and placement, amount and characteristics of drainage. Make sure drainage apparatus is working, if present.

Surgical wounds follow a standard sequence when healing. The nurse can expect:

1.There should be an absence of bleeding and the appearance of a clot binding the wound edges. The wound edges are well approximated and bound by fibrin in the clot within the first few hours after a surgical closure.

2.There should only be inflammation at the wound edges for the first one-to-three days.

3.As granulation tissue starts to bridge the wound there should be a reduction in inflammation as the clot diminishes. The wound should be closed with seven-to-10 days. Increases in inflammation, fever, and drainage likely indicate an infection of the wound site. The wound edges will appear brightly inflamed and swollen.

4.Collagen synthesis starts four days after injury and continues for six months or longer, forming the scar.

5.Scar size will lessen over a period of months or year. An increase in scar size indicates keloid (irregularly shaped scars that progressively enlarge) formation.

Wound Care Nursing

A whole specialty in nursing has grown up around wound care and management. Wound care nurses work with a patient's medical team to monitor a variety of wounds and their healing process. They also care directly for the patient, promoting healthy and rapid healing of a wide variety of wounds

1.1.1Definition of tremens

Pathology : The common pathology terms used in dermatology are:-

Hyperkeratosis:- Incised thickening of stratum corneum.

Parakeratosis :- Premature of immature nucleated cell in the stratum corneum.

Dyskeratosis :- Premature keratinisation of individual epidermal cell.

Acanthosis:- Increase in thickness if prickle cell layer due to stimulation of basal layer

Atrophy:-Consists of thinning of all the layers of the epidermis and is accompanied by flattening of the papillae

A cantholysis:-The loss of chhernce between epidermal odr epithelial ells.

Spongiosis(intercellular oedema) Accumulation of fluid between the epidermal cells

Hydropic degeneration of Basal cells:-A type of degeneration causing vacuolization of basal cells.

Caseation necrosis: Necrosis associated with formation of caseation of caseation a plae eosionophilic either granular material.

Granulama:-A chronic proliferative lesion , consisting of mononuclear cells and either epithliod cells or multinucleated giant cell both .

Langhans Giant cell :-Alarg multinucleated cell formed by mononuclear cells and either epithelioid cells the arrangement of nuclei are in a horseshoe pattern.

Grenz zone:-Anarrow clear zone that may be found between the epidermis and dermal lesion.

Foam cell: Macrophage with foamy appearance containing high proportion of dead lepra- bacilli

Definition of terms (common terms associated with microbiology)

Asepsis:- practices that minimize or eliminate microbes that causes infections or disease

Infection :- The infection and proliferation of microbes in body tissues with a resultant reaction to their presence and/or their toxins

primary infection :- the first or original disease or infection

Secondary infection :- pathogen(infection) that only invades a person with a lower resistance usually caused by a primary infection

latent infection: - a stage of disease in which there are no symptoms

Contamination:- anything that is not sterile; to make unclean

Disinfectant :- substances which destroys most pathogens(infections) but not their spores. Used for dirty surfaces.

Sterilization:- process that destroys all microbes including spores. (Autoclaving)

Antiseptic ;- substance that inhibits the growth of microbes without destroying them. Used on clean surfaces.

Pathogenic ;- micro-organisms that can cause infectious diseases.

Isolation :- state of being separated from others due to infectious disease

Germicide: an agent that kills germs especially pathogenic ones

Culture: - The study of microbes growth or the growth of microbes in special media conducive to their growth

Communicable disease :- a disease whose causative agents may pass or be carried from one person to another directly or indirectly

Inflammation :- A localized protective response elicited by injury or destruction of tissues, which serves to destroy, dilute, or wall off both the injurious agent and the injured tissue. Pain, redness, heat, and swelling are included.

Concurrent disinfection :- continuous decontamination of an area while an individual with infection is present.

Terminal disinfection :- disinfection of a sick room and its contents at termination of an infectious disease

Virulence :- pathogens strength to cause disease

Vector ;- a living carrier of a pathogen

Reservoir :- any place where microbes can survive before moving to a place where they can multiply

Contagious ;- capable of being transmitted from one individual to another quickly and easily

Epidemic:- occurring suddenly in numbers clearly in excess of normal expectancy.

Center for Disease (CDC) an agency of the US department of health and human services. they study infectious diseases and how to control them

inanimate objects:- non-living objects.

Incubation period;- the interval of time required for development, especially the time between invasion of the body by a pathogenic organism and appearance of the first symptoms of disease.

Prodromal ;- a symptom indicating the onset of a disease; disease progressing.

Convalescence:- stage the stage of recovery from an illness,operation, or injury.

Nosocomial :- pertaining to or originating in a hospital; hospital acquired infection

Definition of terms by Types of skin lesion

The morphology of skin lesion is the essential element for the clinical diagnosis of the disease. There is difference in opining about the size and depth of the lesions.

Flat lesions: These lesion are neither raise from surface nor depress from the surface .

Macul: It is a flat. Circumscribed discolouration of the skin and mucus membrane without change in texture up to1cm in its longest .it. May be hyper pigmented .hypo pigment . depigmented or erythema-tous. Macule larger than 1 cm is described as patch or area.

Patch: Macule larger than 1cm is described as patch or area.

Purpura: It is exavasation of RBC in the skin on application of pressure with a glass slide redness will not disappear. Small pinpoint purpuric spots are Known as petechiae and large. Bruise like purpure spots are Known as ecchymosed.

Telanguectasia: They are permanent dilations of capillaries that may or may not disappear with application of pressure.

Papule: A solid elevated lesion of the skin or mucus membrane up to 1 cm in its longest diameter. Papules may have verity of shoes and colour.

Plaque :It is a flat solid lesion above the surface of adjoining skin with horizontal dimensions much more than the vertical ones.

Nodule : It is a solid palpable lesion like papule but differ in depth of involve-ment and or/ and substantives palpability , rather than diameters.

Tumour : It is a solid e elevated lesion of the skin or mucus membrane with the added dimension of depth in the tissue and greater than 1cm its longest diameter.

Wheal :A weal is that topped oedematous; elevated clear lesion with sharply demarked margins which is evanescent in nature, disappearing within hours.

Vesicle :A vesicle is circumscribed, elevated clear fluid containing lesion which have size of 0.5 cm diameter.

Pustule : It is same as vesicle but it contains purulent exudates instead of clear fluid.

Abscess : It is a localized accumulation of purulent material deep in dermis or subcutaneous tissue.

Crust : Crust is a layer of dried-up secretion (serum, blood or purulent exudates).

Lichenification : Lichenification is areas refer to thickened plaque with markings.

Ulcer : In this types of the lesion, break in continuity of skin involving epidermis and at least upper part

It means thinning of the layer of the skin.

Bulla: - sac filled with fluid, its size is > 5cm. e.g.:- partial thickness burn.

1.1.2Physiology of immune system

Immunology: -It is a science it deals with immunity. Immunity refers to all the mechanisms used by the body as protection against environment agent environmental agents that are foreign to the body or it is an "enhanced state " of responsiveness to a specific substance induced by prior contact with that substance. Immunity may be divided in to **two** major types innate (natural or non specific)

Acquire (adaptive or specific) immunity Innate immunity is present form birth and is non specific

- It consists of various barriers to external insults including the skin, mucous membranes, cilia of the respiratory tract and cells like macrophages, mono cytes, neutrophills, eosinophils and the contents of these cells. Others include the HCL of the stomach, flushing activities of urine and the spermine.
- Acquired immunity is created after exposure to a given substance and is specific.
- It is more specialized than innate immunity.
- It consists of humeral and cell mediated immunity and supplements the protection provided by innate immunity
- Unlike the innate immunity there is immunologic memory and it acts with increased intensity on the second exposure.

None-specific host defense mechanism

- The innate (non specific) immune defense consists of a formidable barrier to entry and second line defense by phagocytes and circulating soluble factors.

The body has both "innate" and "adaptive" immune defenses.

- When an organism infects the body, the defense systems already in place may well be sufficient to prevent replication and spread of the infectious agent, there by preventing developing disease. The established mechanisms are referred to as constituting the “innate” immune system how ever, should innate immunity be insufficient to carry the invassion by the infectious agent the so called “adaptive” immune system.

- The main feature distinguishing the adaptive response from the innate mechanism is that specific memory of infection is imprinted on the adaptive immune system.

Innate → adaptive → Specific

Immunity immunity immunologic memory

↓ ↓ ↓

Disease recovery no disease

Comparison of innate & adaptive immune system

Innate immune system		***Adaptive immune system***
Soluble factors	Lysozyme, complement	Antibody
Acute phase proteins		
Cells	Phagocytes	
T-lymphocytes		
Natural killer cells		
Response to		
Microbial infection	+	++
- Resistance not improved by repeated contact		- resistance improved by repeated contact

Prevention of invasion of micro-organisms

- Before an infectious agent can penetrate the body it most over come bio chemical and physical barriers that operate at the body surfaces.
- One of the most important of these is the skin which is normally impermeable to the majority of infectious agents.
- Many bacteria fail to survive for long on the skin because of the direct inhibitory effects of lactic acid and tatty acids present in sweat and sebacous secretions and the lower PH to which they give rise.

However, should there be skin loss, as can occur in burns for example infection becomes a major problem.

- The membranes lining the inner surfaces of the body secrete mucus, when acts as a protective barrier, inhibiting the adherence of bacteria to the epithelial cells, thereby preventing them from gaining access to the body.

- Microbial and other foreign particles trapped with in this adhesive mucus may be removed by mechanical means such as ciliary action, coughing and sneezing.

The flushirng actions of tears, saliva and urine are other mechanical strateges that help to protect the epithelial surfaces. In addition, many of the secrted body fluids contain microbicidal factors.

Specific immune response

Humeral immunity- The bcells, circulating antibodies and the complement cascade mediate the humeral immunity.

- Serum antibodies secreted by activated B-cells mediate humeral immunity. Antibodies are heterogeneous mixtures of serum globulin.

They recognize and bind specifically to a unique structural entity on antigen. they perform common biological functions after combing with antigens. Another important element in the humeral immunity is the complement system, which cause either lysis of target or enhances phagocytosis by phagocytic cells.

- ***Cells mediated immunity***- The T-cells that release various cytokines (that affect T-cells and other cells) mainly medicate the cellular immunity.

- The antigen specific arm of cell-mediated immunity consists of the T- lymphocytes.

T-cells have many identical non-secreted receptors composed of several molecules.

The T-cells circulate directly to the site of antigens and perform their function when interacting properly with the antigen.

The functions of T-cells include

Cooperation with B-cells to enhance the production of antibodies

They are involved in delayed type hyper sensitivity reactions.

They have direct cytotoxic effect for different micro-organisms tumor cells and altered self cells.

They have T-cells can suppress the immune response leading to down ward modulation. this is important to prevent self cells from destruction.

They signal via cytokines-T-cells exert numerous effects on many cells, lymphoid and non lymphoid, through many d/t cytokines that they release.

Immunoglobulins – Are proteins, they can also be antigens & their variability creates different antigenic determinants on molecule based on the structure and antigencity of their heavy chains.

There are five immunoglobulin classes. Those are igG, igM, igA, 1gD and IgE.

Immunoglobulin G (IgG)

IgG makes up about 80% of the immunoglobulin in normal serum.

It consists of two heavy chains and two K or light chains it is also found in tissue spaces.

IgG is the only immunoglobulin that crosses the placental barrier

The transferred igG. across the placenta temporarily protects the new born

Baby against infectious disease to which the mother has antibodies It coats the antigen in preparation for phagocytosis, inactivates toxins and is involved in hyper sensitivity reactions It has four subclasses IgG1, IgG2, IgG3& IgG4

Immunolobulin M

Igm accounts for 5-10% of all serum immunoglobulins

It is not ordinarily found in the extra vascular spaces.

It is the largest of the immunoglobulins, existing primarily as pentamer (five

IgG like molecules bound by the j-chain)

it can not cross the placenta

Igm is the first immunoglobulin to be synthesized during primary response and by neonates. The secondary response primarily mediated by an IgG.

immunoglobulin A (IgA)

IgA consists of 10-15% of the total immunoglobulins in the serum

It is a principal ig found in external secretions

It occurs as monomer in serum and as adimer or tetramer in external secretions.

The secretory piece facilitates the transport of igA across mucosal surfaces and

Prevents igA from proteolytic enzymes and acts as a local immune system epithelial cells of mucous membranes are the sources of the secretory piece.

Immunoglobulin D (igD)

IgD makes up of about 0.2% of total serum immunoglobulins

Mature B-cells express IgD on their surfaces.

It acts as a receptor on b lymphocytes. It has no biological effector function.

Immunoglobulin E (igE)

IgE is found in small amount is serum and can be found in higher cevels in allergic conditions and parasitic disease.

It binds to mast cells and basophils on their fragment crystallize (Fc) receptors leading to degranulation of these cells. Up on degranualtion, these cells release chemicals of the inflammatory response.

Hyper sensitivity reactions

Under some conditions immunity, rather that providing protection produces damaging and sometimes fatal results. Such deleterious reactions are hyper sensitivity or allergic reactions.

- Antigens that commonly cause hyper sensitivity or allergic reaction are allergens.
- Therefore, hyper sensitivity is an exaggerated or in appropriate immune response that leads to damages of the host.

There are four types of hyper sensitivity reactions type-1 hyper sensitivity (anaphylactic reaction) Allergen like protein, plant pollens, drugs or parasite antigens induce type-1 hyper sensitivity reactions these reactions are immediate in that they develop usually in **5 to 30minutes** after exposure to antigens. The individuals may have been sensitized and up on re-exposure to antigens, symptoms develop.

The term allergy usually refers to type-hyper sensitivities.

The allergic conditions can be systemic anaphylaxis or local reactions.

The systemic anaphylaxis is Injection The antibiotics & venoms of sects Air way obstruction & asphyxiation

Spasm of bronchiols Vascular collapse Systemic anaphylaxis can be life threatening

Local allergy of typ1- it refers to a variety of chronic or common allergic states like rhenitis (hay fever), asthma & food allergens. they are not usually life threatening

The allergen evokes response by contact with mucosal surfaces rather than skin injection as in systemic anaphylaxis. Allergy testing can check sensitivity of individual to certain allergic.

Most commonly used skin test

Introduction of allergic to which the patient is allergic lead with in minutes to reddening and swelling of the site locally such tests must be done before injecting allergens systematically. e.g skin testing before injection of tetanus antitoxin (TAT)

Prevention of clinical allergies includes avoiding the allergen type 2 hyper sensitivity reactions

IgG.Igm mediate type 2 hyper sensitivity reactions are cytolytic or cytotoxicreactions which occur when IgG or igm antibody binds to antigen on the surface of cells and activate the complement cascade. t his process culminates in the destruction of cells. in these reactions antigens may be part of the patient's own cells, soluble foreign antigen or antigen-antibody complexes that attach to cells.

The type 2 hyper sensitivity reactions include blood transfusion reactions, Rh in compatibility reactions and drag-induced hemolytic anemias.

Type 3-hyper sensitivity reactions (immune complex disease)

Type 3 hyper sensitivity is an immune complex disease.

An immune complex is any antigen-antibody complex found in the body. Normally the immune system removes these complexes with no damage to the host. there are conditions in which the immune complex leads to hypersensitivity and damage to host tissue.

Clearance of the immune complex appears to depend on their size Immunoglobulin class and relative concentration of antigen and antibody.

The inability to remove the immune complexes leads to deposition mainly On the endothelium of blood vessels basement membrane of kidneys and on the synovial tissue.

A soluble antigen injected in to the skin at intervals progressively leads to the formation of an ever increasing inflammatory focus that becomes necrotic. The injected antigen will make a complex with antibody locally & form insoluble antigen-antibody complex.

If excessive amount of antigen occurs in circulation, then the immune complex will deposit systematically. this was originally described in the form of serum sickness in which a rash, fever, arthritis and glomerulo nephritis followed the injection of antidiphterial horse. similar reactions can follows the administration of drugs (pencillin, sulfonamides) and following various infections.

Type 4-hyper sensitivity reactions (cell mediated hyper sensitivity)

Cell mediated immunity (CMI) involves immune responses intiated primarily by antigen specific T-cells

The antigen eliciting this type of response may be foreign tissue, intracellular organisms (like viruses, mycobacteria and fungi)a soluble protein and chemicals capable of penetrating the skin and coupling with antibodies (as carriers)

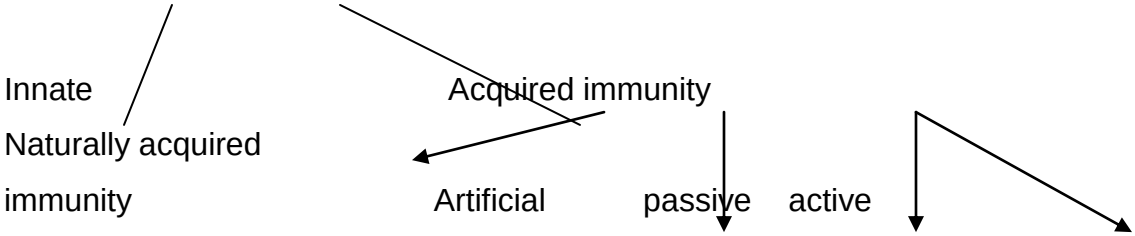
The type 4 hyper sensitivity reaction is a delayed or cell mediated hyper sensitivity reaction which can not be transferred by serum but by T-cells. It takes more than 12 hrs to appear.

e.g. Tuberclin test- a purfied protein derivative when injected induces indurated inflammatory reaction that reaches greator intensity after 24-72hrs.

Allergic contact dermatitis due to soaps, cosmetics and 0 in# ements that make contact with skin.

Vaccination & immunology

Immunity



Naturally acquired immunity:- Is due to exposure to antigen or contact with an antigen un intentionally. usually in this case the antigen is an infectious agent.

Artificially acquired immunity:-Arises when the antigen or antibodies are introduced by artificial means by using a vaccine or antiserum respectively.

Active immunity:- The individual produces antibodies as a result of the infections (natural) or by injecting vaccines.

Passive immunity:- The antibodies is injected either in the form of antiserum or immuno globulins that were obtained from animal or other humans. this is artificially acquired passive immunity.

Naturally acquired passive immunity:- The antibodies may have been transmitted under natural conditions as in the transfer of maternal antibodies across the placenta to the fetus or the transmission of antibodies via colostrums.

The passive immunity status is of short duration (weeks to months)

Vaccination:- Aims to prime the adaptive immune system to the antigens of particular microbe so that a first infection induces a secondary response.

The method of conferring immunity to prevent infection is immuno prophylaxis. e.g. Small box is eradicated by immunization or vaccination.

Vaccine is a material that when deliberately introduced evokes the active immune state.

The type composition of vaccines may vary widely.

Toxoids: - The exotoxins of tetanus and diphtheria can be converted in laboratory to anon toxigenic form by treating with formalin or other modalities. These are toxoids and can be given as vaccines.

Whole cell killed vaccines:- Some vaccines consists of suspension of in activated intact micro-organisms.

e.g. the whooping cough, typhoid fever and plague vaccines

Attenuated vaccines living organism, through laboratory processing, can still multiply in the host but lack ablilty to cause diseases. This process is attenuation.

- Attenuated vaccines induce higher and longer lasting levels of immunity than do non-living organisms.
- Unlike the killed vaccines, the need for booster doses may be lessened.
- The attetulation process commonly involves adapting micro organisms to conditions they do not face in the host.

e.g. goring poliovirus in monkey tissue culture (polio does not infect monkeys naturally)

Yellow fever and influenza viruses in embryonated hen's eggs.

Purified antigens:- Intact micro-organisms have various antigens. The immunity very often is directed against predominant antigen (only one of a few antigenic determinants of that antigen) identifying and when ever possible, purifying the antigen gives an effective vaccine

e.g. Hemophilus influenza & neisseria meningitidis vaccine

Recombinant vaccines-Purifying the protein antigen of micro-organisms beyons the sub unit stage is possible by isolating the genetic material those codes, for the antigen.

The segement of stage is possible by isolating the genetic material inserted into bacteria, yeasts or animal cells produce large quantity of purified antigen for e.g. the gene in am immunogenic as hepatitis B virus has been C coned in yeast cells.

- The vaccine is an immunogenic as hepatitis B surface antigen obtained from human plasma.

Principles of booster doses

Repeated exposure of an individual to an antigen results in the prompt and elevated production of antibodies. This is an anamnestic response.

The rapidity of anamnestic response to are encounter with an antigen provides the host with potential protection up on a repeated exposure to an infectious agent it is particularly important in those infections with a relatively longer incubation period

- Booster dose is also important to achieve successively higher levels of antibodies in serum.

ABO system & RH incompatibility

Based on the presence or absence of various antigens blood is categorized into different blood groups, which in a given blood group there may be two or more different blood types.

There are at least 24 blood groups and more than 100 antigens that can be detected on the surface of red blood cells here we can discuss two major blood group ABO & RH.

ABO blood group

The ABO blood group is based on two glycol lipid antigens called A and B. people whose RBCs display only antigen A have type A blood. Those who have only antigen B are type B.

Individuals who have both A and B antigens are type AB. Those who have neither antigen A nor B are type 0.

Blood plasma usually contains antibodies called agglutinogens that react with the A or B antigens if the two are mixed those are the anti-A antibody which reacts with antigen A and the anti B antibody which reacts with antigen B.

Transfusions

A Trans fusion is the transfer of whole blood or blood components (RBC only or blood plasma only) in the blood stream or directly in to the red bone marrow.

Ina an incompatible blood transfusion antibodies in the recipient's plasma bind to the antibodies on the donated RBCs, which causes agglutination or clumping of the RBCs.

Agglutination is an antigen-antibody response in which RBCs become cross linked to one another.

In essence complement molecules make the plasma membrane of the donated RBCs leaky causing hemolysis (rupture) of the RBCs and the release of haemoglobin in to the blood plasma.

Summary of BO blood Group interactions

Characteristic	Blood type			
	A	B	AB	O
- Agglutinogen (antigen)on RBCs	A	B	Both A and B	Neither A nor B
- Agglutinin (antibody) in plasma	Anti B	Anti A	Neither anti A nor anti B	Both anti A and anti B
- Compatible donor blood	A,O	B,O	A,B,AB,O	O

types (nohemolysis)				
Incompatible donor blood types (hemolysis)	B,AB	A,AB	-	A,B,AB

People with type AB blood do not have anti A or Anti B antibodies in their blood plasma. They are universal recipients because they can receive blood from donors of all four blood types.

People with type O blood have neither A nor B antigens on their RBCs and are called universal donors. Because they can donate blood to all four ABO blood types.

Rh blood group

The Rh blood group is so named because the antigen was discovered in the blood of the Rhesus monkey.

The alleles of ***three*** genes may code for the Rh antigen people whose RBCs have Rh antigens are designated Rh^+(Rh positive) those who lack Rh antigens are designated Rh-(Rh Negative)

Rhesus factor is made up of several antigens, C,c ,D,d, E,e C,D, and E are dominant, C,d and e are recessive. Those who are Rh positive carry the D antigen.

1.1. 3. Skin

Anatomy and physiology of Skin

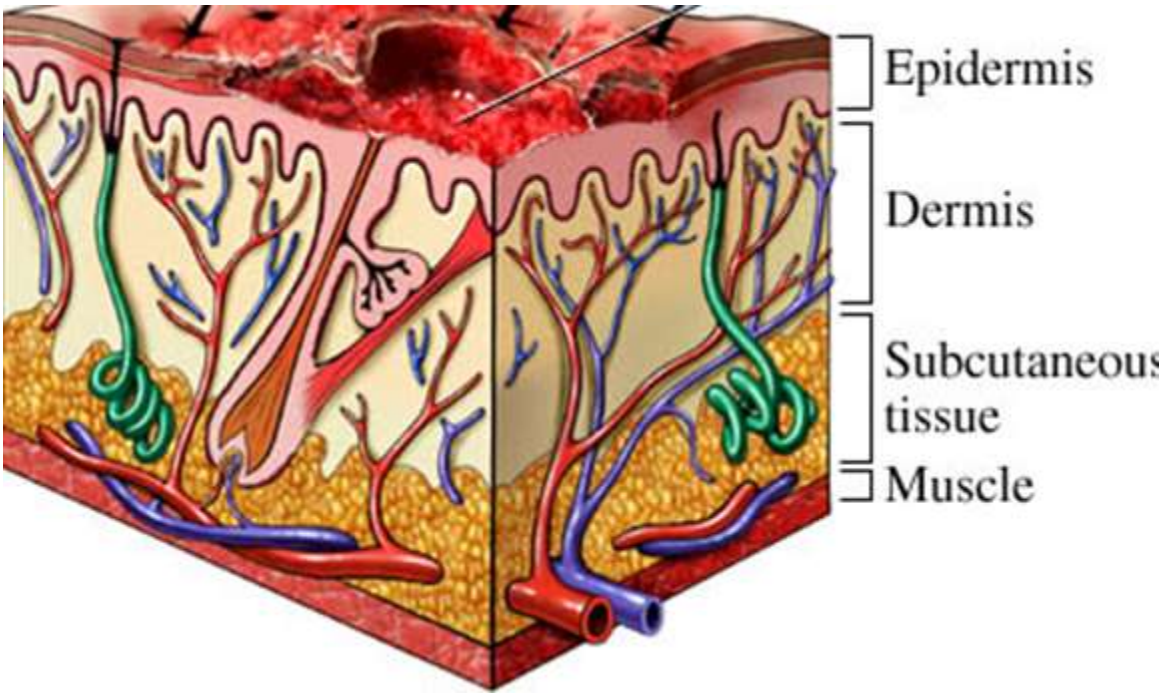
The skin is a highly underestimated organ. It performs many vital functions and has a complex structure which most people are unaware ofits function.

The skin is an organ because it consists of different tissues that are joined to perform specific activities. It is one of the largest organs of the body in surface area and weight. In adults, the skin covers an area of about 2 square meters, and weighs 4.5 to 5 kg. It ranges in thickness from 0.5 to 4.0 mm, depending on location. The skin is not just a simple, thin coat that keeps the body together and provides protection. It performs several essential functions. Dermatology (der'-ma-TOL-o-je; dermado = skin; logos = study of) is the medical specialty that deals with diagnosing and treating skin disorders.

Anatomy of the Skin

Structurally, the skin consists of two principal parts. The outer, thinner portion, which is composed of epithelium, is called the epidermis. The epidermis is attached to the inner, thicker, connective tissue part called the dermis. Beneath the dermis is a subcutaneous (subQ)

layer. This layer, also called the superficial fascia or hypodermis, consists of areolar and adipose tissues. Fibbers from the dermis extend down into the subcutaneous layer and anchor the skin to it. The subcutaneous layer, in turn, attaches to underlying tissues and organs.



Physiology of the Skin

Skin serves several functions, which are introduced here. Regulation of body **1,temperature**:-In response to high environmental temperature or strenuous exercise, the evaporation of sweat from the skin surface helps lower an elevated body temperature to normal. In response to low environmental temperature, production of sweat is decreased, which helps conserve heat. Changes in the flow of blood to the skin also help regulate body temperature

2, Protection:-The skin covers the body and provides a physical barrier that protects underlying tissues from physical abrasion, bacterial invasion, dehydration, and ultraviolet (UV) radiation. Hair and nails also have protective functions.

3, Sensation:-The skin contains abundant nerve endings and receptors that detect stimuli related to temperature, touch, pressure, and pain.

4. **Excretion**:-Besides removing heat and some water from the body, sweat also is the vehicle for excretion of a small amount of salts and several organic compounds.

Immunity:-Certain cells of the epidermis are important components of the immune system, which fends off foreign invaders.

5, Blood reservoir:-The dermis of the skin houses extensive networks of blood vessels that carry 8 to 10% of the total blood flow in a resting adult. In moderate exercise, skin blood flow may increase, which helps dissipate heat from the body. During hard exercise, however, skin blood vessels constrict (narrow) somewhat, and more blood is able to circulate to contracting muscles.

6, Synthesis of Vitamin D:-Vitamin D is a group of closely related compounds. Synthesis of vitamin D begins with activation of a precursor molecule in the skin by ultraviolet (UV) rays in sunlight. Enzymes in the liver and kidneys then modify the molecule, finally producing calcitriol, the most active form of vitamin D.Calcitriol contributes to the homeostasis of body fluids by aiding absorption of calcium in foods. According to the synthesis sequence just described, vitamin D is a hormone, since it is produced in one location in the body, transported by the blood, and then exerts its effect in another location. In this respect, the skin may be considered an endocrine organ.

Self-Check -1	Written Test

Directions: Answer all the questions listed below. Use the Answer sheet provided in the next page

1, what does it mean Tumor?

2, What does it mean Wheal?

3, Write the difference between Naturally acquired immunity And Artificially acquired immunity?

4, Mention anatomical structure of the skin?

5 ,List the function of the skin ?

Note: **Satisfactory rating - 3 points** **Unsatisfactory - below 3 points**

Answer Sheet

Score = _____

Rating: _____

Name: _____ Date: _____

Short Answer Questions

1,_____

_____

2,_____

3_____

4,_____

5, _____

Information Sheet-2	chain of infection

1.2. chain of infection

There are six in the chain of infection

1.the etiologic/Pathogenic agent or micro organism.

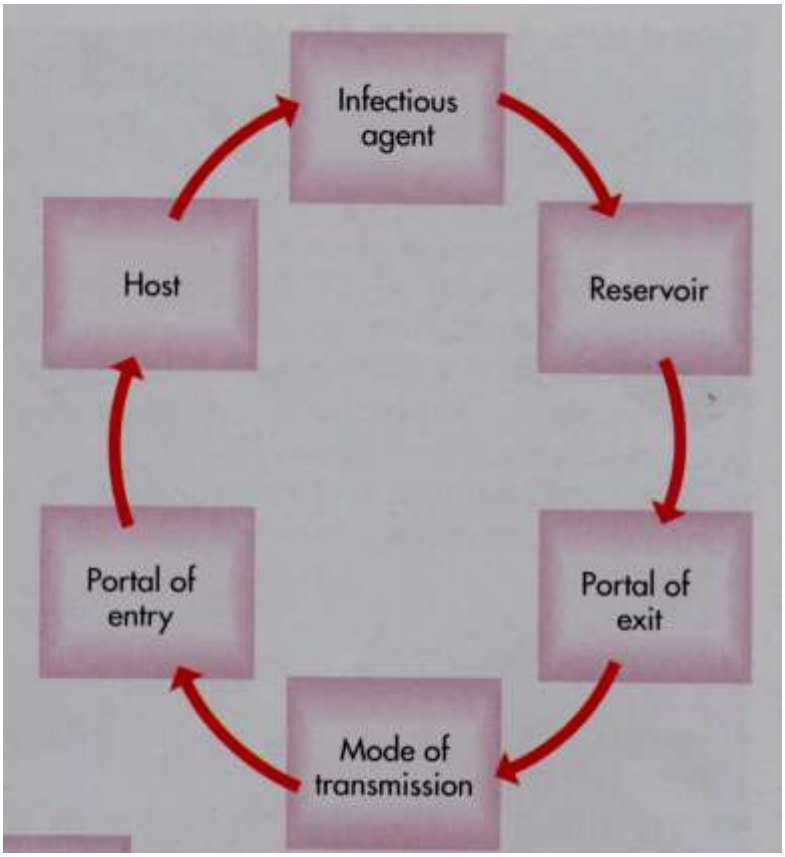
2. the reservoir , the place where the organism naturally resides.

3. portal of exits from reservoir

4. method of transmission

5. portal of entry in to the host

6. Susceptibility of the host



If any of these links is lacking, disease will not developed through the remaining five factors may present

A, Pathogenic agent

etiologic agent:Is an organism capable of causing infection or infectious diseases Some epidemiological characteristics of them are :

Infectivity: ability of an agent to cause infection

Infection rate = #infected people/# of susceptible exposed people exposed

Pathogenicity: ability of pathogenic agent to induce disease

Pathogenicity = # of clinical case/ # of sub-clinical case (# persons infected)

Virulence: ability of the agent to cause severe out of the disease

Virulence = # of death from disease/#of case of the disease

Different strains of the same agent may vary in virulence

Resistance: ability of the agent to survive adverse/unfavorable environmental condition during the transmission from one host to another.

Number of the agent: very small number (dose) may cause no infection or may cause sub-clinical infection

The diseases agents are classified as follow

Biological agent: all living organisms. Like bacteria, rickettsiae, fungi, protozoa, helminthes, viruses. Etc Chemical agents:

1, Endogenous: chemical produced in the body. e.g. urea – uremia, ketones – ketosis, uric acid – gout, calcium carbonate – kidney stones

2, Exogenous: arising outside of human body (host) e.g. gases, insecticides, etc

3, Physical agent: like mechanical force/friction that may produce injury as well as atmospheric abnormalities such as heat, cold, radiation, electricity, etc

4..Genetic agents: transmitted from the parents to child through genes

5. Nutrient agent: specific basic dietary components that we need to survive

E.g. protein, carbohydrate mal-nutrition resulted in PEM

Of these agents biological agent is the concern of CD

Mode of action of infecting organism

Organism may invade/attack the host either:

Through the process of direct invasion

Through the production of toxin substance, this may poison the body. E.g. tetanus

B, The reservoir

Also called source of etiologic agent

Is the place where etiologic agents usually grow and multiply or remain harbored.

It can be living or non-living things

It depends primary for survival

A disease can have more than one reservoir.

Reservoir can be classified as follow:

Human reservoir – the most important reservoir

Frank cases:

hose person obviously ill with a disease.

Generally less dangerous to the community than mild cases;

because disease is easily recognized and people prevent themselves

b. Sub-clinical /Unapparent infection/ missed/abortive cases Person in whom the symptoms are so vague that the patient doesn't seek medical attention The infection is mild enough to escape recognition It is hazardous to the community

c. Carriers: an infected person without clinical manifestation of disease but capable of transmitting disease to other. Are usually unaware of the condition It doesn't give rise to any symptoms No way of recognizing it other than bacteriological or laboratory methods Can circulate freely in the community More dangerous to the community than sub-clinical cases

Types of carrier

Incubatory carrier: - transmits the infection during the incubation period.

E.g. measles

Convalescence carrier: transmits the infection during convalescence - from the time of recovery until the time the agent stops being shedding the agents.

E.g. typhoid fever

Asymptomatic carrier: transmitting the infection without ever showing clinical manifestation of the diseases.

E.g. poliomyelitis

Chronic carrier: continue to shed an agent for a long period of time.

E.g. typhoid fever

II. <u>Animal reservoir</u>:

2nd largest reservoir of organism capable of infecting man.

The principal animal reservoirs are our domestic animal or rodents.

E.g. anthrax – cattle, rabies – dog

III. <u>Non-living things as reservoir</u> :

can harbored pathogenic agents. E.g. soil for tetanus

C. <u>Portal of exit</u>

of escape of organism (infectious agent) from reservoir

There should be a way of escape of agent to bring about the spread of infectious diseases.

The venue of escape from the reservoir is dependent upon the site of parasitic growth in the body of host.

The venue of escape can be classified as follow:

Respiratory tract:

Most common venue of scape Most dangerous ways of escape and difficult to control

Agent escape from respiratory tract through: Exhalation, sneezing, coughing, talking, singing, expectoration drive out, etc.

TB, common cold, pertussis,

2. Genito- urinary tract**:**

Aside from the fact that man is in general less careful in the discharge of urinary discharges, the epidemiological aspect of escape of infectious agents the urinary tract are identical with those modes of infectious agents from the intestinal tract.

Fortunately the urine less frequently carries pathogenic organisms than do feces.

E.g. Schistosoma hematobium, venereal diseases, etc

3. Intestinal tract:

Escapes through discharges through feces.

E.g. typhoid fever, cholera, amoebiasis, shigellosis

4. Open lesion:

open wound or discharges on the surface of the body.

5. Mechanical escape:

through external forces.

Most frequently happens through biting or sucking by insects. E.g. malaria

D. <u>Mode of transmission</u>

Means of transfer from the reservoir to new host.

After the infecting organism has escaped from the reservoir it can cause new infection only if it Reach/finds ways to a new host.

Types:

Direct,

Indirect

Direct: immediate transfer of the agent from the reservoir to host

Organisms pass from the reservoir to the new host without the intervention of intermediate objects.

Actual physical of the reservoir and host is not must.

Direct contact/touch: contact of diseased part with healthy,

E.g. venereal diseases

Contact with soil: direct exposure of susceptible host to a diseases agent in soil.

E.g. Hooke worm larvae, tetanus BiteDroplet: direct projection of droplet synergy of saliva and mass-pharyngeal secretions on the mouth during sneezing, coughing, talking, singing,

E.g. common cold, pertussis of animals: e.g. rabies, insect correspondence diseases.

Transplacental: transmission of diseases through placenta.

E.g. HIV/AIDS

2. Indirect: transfer of infectious agent without close relationship between reservoir and a new host. Indirect transmission to occur: The organism becapable of survival for a period of time outside of body There must be <u>some vehicle</u> which will transmit organism from one place to another The vehicle could be animate or inanimate .

1, Animate (living) vehicle: referred to as vector, e.g. insects.

There could be transfer of unchanged organism or effecting biological change in them.

2. Inanimate (non-living) vehicle: could be through contamination with infectious agent. To be effective, such vehicle should permit survival of etiological agent long enough for the transfer to be accomplished.

Some of the inanimate vehicles are:

Water – contamination with human feces, e.g. surface water, Milk and Other foods

Air, considered as vehicles for some respiratory tract infections

Fomites - inanimate objects capable of carrying germs from an infected person to another person, includes all inanimate vehicles other than water, milk, food and air, e.g. clothes or bedding.

Soil: under certain conditions soils may serve as a vehicle, e.g. tetanus

E. Portal of Entry

Mere arrival of the etiologic agent at the new host is not enough to cause infection.

The mode of entry corresponds roughly with the mode of exit (escape) from the reservoir and with parts of the body to be 1st affected.

It could be: -

Respiratory tract,

Genitor-urinary tract,

Direct infection of membranes (mucous membranes), e.g. trachoma, gonorrhea

F, New Host /Susceptible Host

Transmission to be completed the existence of susceptible host is essential.

Level of susceptibility depends on:

age, nutritional status, stress, environment,

pre-existing medical condition, immune status,

genetic factors, host behavior like personal hygiene, food handling,

diet, occupation, utilization of health resource, etc.

Agents, Host, Environment interaction

Any health problem is the result of an interaction between a numbers of specific or associated risks which can be classified as ‘agent’ ‘host’ and ‘environment’ factors**.**

Self-Check -2	Matching

Directions: Answer all the questions listed below. Use the Answer sheet provided in the next page

‘’ A”	‘’B”
1. the etiologic agent	A, Site of escape of infectious agent from reservoir
2. the reservoir	B, Mere arrival of the etiologic agent at the new host
3. portal of exits from reservoir	c, an organism capable of causing infection
4. method of transmission	d, age, nutritional status and stress are factors
5. portal of entry in to the host	e , Means of transfer from the reservoir to new host.
6. Susceptibility of the host	f, Also called source of etiologic agent
g, Non disease causing agent	
	h, Strengthen of the host

Note: **Satisfactory rating - 3 points** **Unsatisfactory - below 3 points**

Answer Sheet

Score = _____
Rating: _____

Name: _____Date: _____

Matching Answer

1,_____

2_____

3_____

4, _____

5, _____

6, _____

Information Sheet-3	definition of WOUND and its type /classification

1.3. definition of WOUND and its type /classification

Definition :Wound is a break in the continuity and protective function of the tissue of the body internal or external

Caused by surgery, accident , chemicals, heat/ cold, friction/ shear force, pressure or as a result of disease

wounds can generally be grouped into two categories or types .

Open wound:- when the skin or mucus membrane surface is broken.

It may bleed with tissue damage

High risk for infection

Example: Abrasion, Laceration, Puncture, Missile injuries, Bites…

Figure 8: Abrased Wounds

Closed wound:- if the tissue traumatized without a break in the skin may have internal injury and bleeding

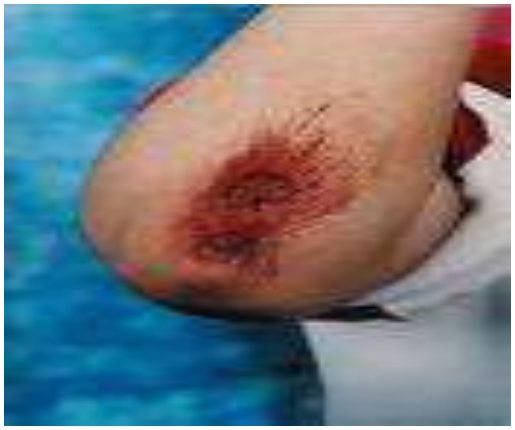
Example: Contusion, Bruise, Hematoma

Examle of open wound explained as follow:

1.Abrasion:- The outer layers of the skin are damaged. It usually results when the skin is scraped against a hard surface.

Bleeding is limited

Sign of contamination and infection is high



2.Excoriation:-In common with Abrasion, this is caused by mechanical destruction of the skin, although it usually has an underlying medical cause

3.Hematoma:- it is closed type Caused by damage to a blood vessel that in turn causes blood to collect under the skin.

4.Laceration:- It is jagged irregular or blunt breaking or tearing of the soft tissues and is usually caused when great force is exerted against the body

Bleeding may be rapid and extensive

Distraction of tissue is greater in a lacerated wound than in a cut.

Deep contamination of the wound increases the chance for later infection.

Figure 10: Laceration

5.Incision: - It frequently occurs when body tissue is cut on knives, rough edges of metal broken glass or other sharp objects.

Bleeding may be rapid and heavy.

Deep cuts may damage muscles tendons and nerves.

Incision

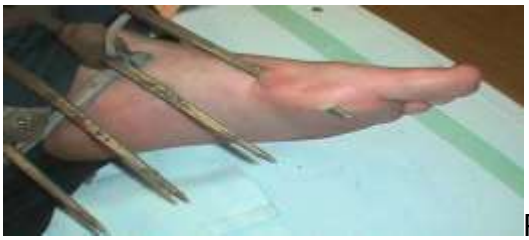
6.Puncture Wound :- An object piercing skin layer creating a small hole in the tissues produces a punctured wound. E.g. bullets, pins, nail.

External bleeding is usually quite limited.

Internal damage may have resulted to the organs causing internal damage.

The hazard or infection is increased because the flushing action of external bleeding is limited

. Tetanus may develop



Puncture wound

7.Avulsions

It results when tissue is forcibly separated or torn of the victim's body An incised wound a lacerated wound or both will usually occur when a body part is avulsed. There will be heavy and rapid bleeding. It occurs in accidents such as motor vehicle destruction wrecks gunshots, explosions, animal bites and other crushing injuries. An avulsed body part may be reattached to a victim's body by a surgeon

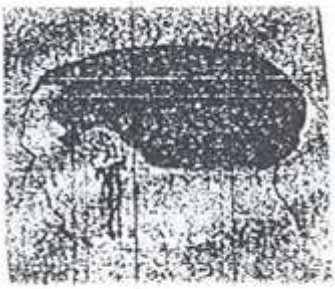


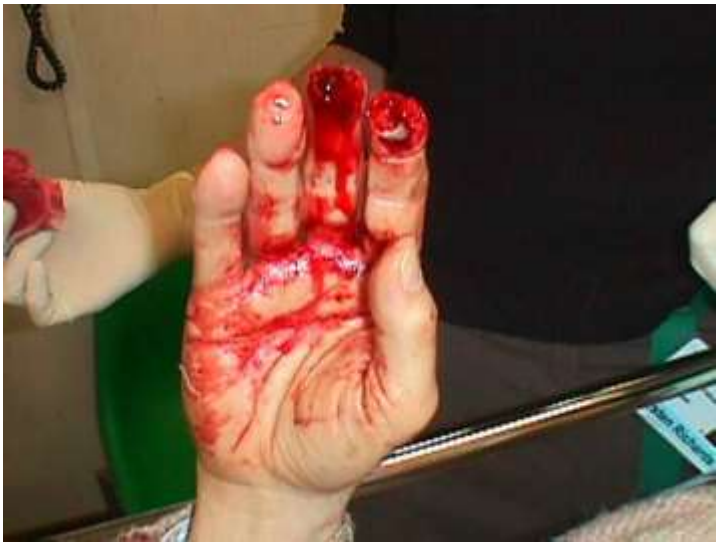
Figure 12: Avulsions

Avulsion Wound S

8.Contusion :- it is closed type Also known as a bruise, this is a blunt trauma damaging tissue under the surface of the skin

9.Crushing injuries:- Caused by a great or extreme amount of force applied over a period of time.

10.Amputation: Cutting or tearing off of a body part such as a finger, toe, hand, foot, arm, or leg



Amputation

Depending on the wound contamination

Clean wound= No break in sterility

Clean contaminated=Minor break in sterility

Contaminated =Major break in sterility

Dirty or infected=Heavy contamination & evidence of infection

1.3.1Cause of wound

Injuries / trauma
Infection of –bacteria
– Fungous
- surgical incision
5, Buren
6, Virus
7, Pressure ulcer
8Diabetes ulcer

A) Wounds caused by bacterial

a). Boils(staphylococcusaurous) —wound infection Is an acute inflammation arise deep in one or more hair follicles and spread into the surrounding dermis deeper form of felicitis(furnculosis) refers tomultiple or recurrent boils

Etiology:- staphylococcus aurous or streptococcus

Painful hard deep follicular absce and overlying skin is hot to touch The area become soft & open to discharge pus ,fever Common sites of lesion buttack,face,neck& auxiliary

b) . Closterdiumtetani

Closteriudmtetani results from neurotoxin (tetanospasmin) produced by organism in wound toxin genes are plasmid encoded.

The organism is non invasive, but the toxin spreads from site of infection via blood stream and acts by binding to ganglio side receptors and blocking release of inhibitory nerurotransmitters, causes convulsive contractions of voluntary muscles.

Transmission- contaminated soil in to wound.

Diseases-Tetanus (cock jaw), severe disease characterized by tonic muscle spasms and hyper flexia, trismus, opisthotonos and convulsions

Laboratory diagnosis

- cultures

-clinical presentation

Treatment, prevention and control

Treatment requires debridement of serious wounds and use of metrondazole.

Prevention is possible by passive immunization with antitoxin globulin and vaccination consists of three doses of tetanus toxoid followed by booster every 10 years.

gas gangrene,

c). ClosterdiumBotulinum

- This vegetative cells is extremely, it produces neurotoxin which is the most potent toxin known. Botulinum toxin is similar in structure and function to tetanus toxin differing only in the target neuraicell..

Disease

1.Food borne botulism is associated with home-canned foods & preserved fish

2. Infant botulism is more common and associated with the consumption of foods (particularly honey) contaminated with botulinum spores.

Wound botulism occurs when spores contaminate a wound, germinate a wound, germinate and produce toxin at the site

Laboratory diagnosis-Botulism is confirmed by isolating the organism, detecting the toxin in food products or the patient's faces or serum.

Treatment, control & prevention

- Administration of metrondazole or pencillin
- Maintain food in an acid PH, by high sugar content or by storing foods at 4^{O}c or colder
- Toxin is heat labile so can be destroyed heating of food at 80^{O}c for 20 minutes.
- Infant botulism is associated with consumption of contaminated foods (particularly honey) And motor cycle accidents and septic abortion. Reheated foods especially meat dishes cab grow the organisms which cause food poisoning.

Diseases: - Diseases include soft tissue infections (cellulites, supportive myositis, myo necrosis or gas tissue Proteus — wound infections

B . Wounds caused by fungus

a). Tineapedis (athlete's foot)

is most fungal infection of skin it is commonly affects teenagers and young adults although it can occur in any age group is more common in those communal shower it involves the intredigital of foot it may appear as acute or chronicinfection on the sole of the feet or between toes

b). Tineacapitus (ringworm of scalp)

-it is contagious fungal infection of the hair shaft and commonly caused hair loss in children

-Round patches of redness and scaling with small pustule or papuls at the edges

-Hair brittle, break easily at scalp

C). Wounds caused by virus

a). Herpes simplex viruses (HSV)

- There are two distinct herpes simplex viruses, type1 (cold sores) Herpes simplex II (genital herpes) (HSV_1,HSV_2)

- The to viruses cross-react serologically but some unique proteins exist for each type.

- Primary infection commonly occurs in children 2-4 years of age.

New born infants can contract the infection from the birth canal or in utero.

The incubation period a primary herpes infection is from 2-20 days depending up on the infected size and the infecting strain of virus

- The virus multiplies locally and in the mucous membrane or a barded skin causing vesicular lesions, which may change to shallow ulcers scars form and lesions heal with out scarring.

- Latent infection (recurrent lesion)-From primary lesion the virus travels through nerves and then remains latent in the trigeminal ganglia (HSV-1) or the sacral ganglia (HSV_2) where the virus persists for the life time of the host.

Provocative stimuli that reactivate the virus include fever, physical or emotional stress exposure to strong sunlight or menstruation clinical findings.

Oropharyngeal disease:- Primary HSV, infections usually symptomatic disease frequently in small children (1-5 years of age) and involve the buceal and gingiral mucosa of the mouth.

- In adult it commonly causes pharyngitis and to nsilitis localized lymph adenopathy any occur.

- Besides HSV1, HSV2 can cause recourent disease characterized by vesicles/blisters/colder**.**

Kerato conjunctivitis:- The intial infection with HSV-1 may be in the eye, producing severe kerato conjunctivitis recurrent lesions cause corneal ulcers with permanent blindness.

Genital herpes:- This is usually caused by Hsv2 but Hsv1 can also cause the disease. the disease is chareacterized by vesiculoulcerative lesions of the penis of the male or the cervix,

vulva, vagina, and perineum of the female. it is associated with fever, dysuria and inguinal lymph adenopathyreccurences are common and tend to be mild.

. Neonatal herpes:- HSV infection of the new born may be acquired in utero, during birth or after birth

sore/ at the border of the lip.

The most common route of infection is during birth by contact with hergetic lesions. to avoid infection, delivery by cesarean section has been used About 75% of neonatal herpes infections are caused by Hsv_2.

Encephalitis:- Hsv1 infections are the most common cause of sporadic and fatal encephalitis. About half of the patients appear to have primary infections and the rest reappear to have recurrent abortions.

Skin infections:- Localized lesions caused by Hsv-1 or Hsv2 may occur in abrasious that become contaminated with the virus (traumatic herpes) severe and life threatening cutaneous infections may occur in individuals with eczema or burns.

Infections in immuno compromised patients:-Immuno compromised patients are at in creased risk of developing severe Hsv infections.

Herpes lesions may spread and involve the respiratory tract, esophagus and intestinal mucosa and eyes (keratitis)

Epidemiology:- Hsv1 is primarily associated with oral disease and Hsv2 with genital disease they differ in their mode of transmission, Hsv1 spreads through the salwa, by contact or droplets, where as Hsv-2 is transmitted sexually or to the new born during birth. these result in different clinical forms of infection.

Diagnosis

1. Isolation-Specimens by swab or fluid form vesicles, skin, saliva, conjunctivas corneal scrapings, brain biopsz are inoculated on cell cultures.
2. Serology-Detection of local antibody produced in CSF
3. Microscopy

Prophylaxis:- Vaccines May help in preventing primary herpes, but are of little use in preventing recurrences which occur in the presence of antibody.

Treatment:-Agclouir is a non toxic drug and a specific in hibitory action on herps simplex virus replication.

c). Varicella-zoster virus (vzv)

This typical herpes virus causes both varicella (chicken pox)and herpes zoster (shingles) varicela is the primary illness.

Properties of the virus: - The vaticella-zoster virus is morphologically identical to herpes simplex virus. The virus properagate in cultures of human embryonic tissue and produces typical intracellular in cufsion bodies.

The virus causes chicken pox and zoster.

Clinical features: - 1-varicella (chciken pox)- chicken pox is usually a mild self limited illness in children.

The virus enters the respiratory tract where it brgins to replicate the virus invades local lymphnodes and causes a primary virema.

After 14 to 21 days in cubation period, fever develops followed by a popular rash of the skin and mueous membranes, which starts on the trunk & spreads to the limbs and face. the papules rapidly become vesicular and beign to itch but remain painless(in contrast to the rash in zostert

Zoster: - This disease occurs primarily as a reactivation of VZV infection in adult with circulating antibodies.

This disease develops from an inflammatory stimulation of a sensory gaglia of spinal or cranial nerves. The virus appears to remain latent in gaglionic nerve cells and following activation, travels back along the nerve fibre to the skin.

Zoster presents as a unilateral painful vesicular rash along the affected sensory nerve-it affects mainly the turnk and neck. it may be accompanied by fever and malaise. The rash may last for 2-4 weeks or months . Clinically episodes of zoster may be intiated by truma, drugs, neoplastic diseases or immuno suppression.

Epidemiology:-Varicella is a contagious found predominantly in children

The disease is transmitted by air born route

Zoster is a pirarily an adult disease resulting form reactivation of virus

Diagnosis: - Serlogy, microscopy & culture

Prophylaxis: - A living attenuated varicella vaccine

Treatment: - Agclovir, famcioclovir

d . Commonly seen wounds as a result of acute/chronic conditions may include:

•Diabetic ulcers(*DIABETIC FOOT ULCERS*)

Foot complications in people with diabetes are common, accounting for almost half of all diabetes-related admissions in the UK.

In community-based surveys, prevalence of foot ulceration has been shown to be 3 – 4 %, whilst the overall incidence of foot complications in the diabetic population is 5-10%.

Amputation affects 1.3% of all patients with diabetes and diabetic foot complications are responsible for 50% of all non-traumatic amputations.

Burns

Introduction

In caring for the patient with a burn injury it is important to remember that many factors Impact on the care that we are able to deliver. This is of particular relevance in the area ofwound care. Access to costly wound products is not an option in many settings. In thesesituations, creativity and innovation have led to many excellent alternatives being developed.

In some instances sophisticated products are available but lack of clinical experience makes them difficult to use. Wound care needs to be undertaken in the context of the local environment.

THE BURN WOUND

It is appropriate to highlight the functions of the skin as they underpin the management of the burn wound:

• Protection
• Immunological
• Fluid, protein and electrolyte homeostasis
• Thermoregulation
• Neurosensory
• Social – interactive
• Metabolism

A burn injury results in either the loss or disruption of some or all of these functions. The burns nurse must assess all of these factors when deciding on an appropriate nursing

management of pain.

Thermal energy effects

The three mechanisms that energy transfers by, are :-

- ✓ conduction,

- ✓ convection and
- ✓ radiation.

All of these mechanisms affecting heat transfer may deliver heat to, or away from, living tissues. Sustained temperatures result in cellular dysfunction and early denaturation of protein.

-As the temperature or the time of exposure increases, then cell damage increases.

-concur with other studies in demonstrating the beneficial effects of cooling on reducing tissue damage and wound healing time. The question often asked, is how long after the burn injury, is it still worthwhile to commence Cooling conclude from various authors that although immediate cooling is

preferable, even a 30 minute delay in application of cooling is still beneficial to the burnwound**.** The same authors do point out that the application of cooling 60 minutes after injury, does not demonstrate any benefit.

Impairment of blood flow in the zone of stasis can occur from shortly after the burn injury up to 48 hours post-burn1. If blood flow is compromised, this may lead to the eventual necrosis of cells in this zone. Clinical management that will promote the recovery of this zone includes:

• Wound dressing chosen to aid moist wound healing

Burn inflammation

• The use of topical antimicrobial agents

• Adequate fluid resuscitation / hydration

• Elevation of burnt area to minimiseoedema

• Advising patient to avoid / minimise smoking

• Management of systemic diseases such as diabetes – monitor&stabilise blood sugar levels.

Burn wound oedema

It is important to have an understanding of the timeframe of oedema development and resolution. The ability of the tissues to receive oxygen and nutrients is reduced during this time, while susceptibility to infection is increased.1 The impact of this on clinical management is that strategies to aid recovery of the zone of stasis must extend until oedema resolution has occurred. Inflammation becomes prominent at 7-10 days post injury. It is at this time that blood flow in the burn wound is at its maximal level. Surgery therefore, may be hazardous due to an

increased risk of blood loss. This is one of the reasons that early burn wound excision, is favoured by many burn surgeons.1

Burn wound depth

Burn depth in Australia is most frequently described using the following classification system:

- Epidermal
- Superficial Dermal
- Mid-Dermal
- Deep Dermal
- Full thickness

Other classification systems refer to 1st, 2nd and 3rd degree injury. This system however is subjected to personal interpretation of the classification. The advantage of the descriptive system is that it removes any ambiguity. This is especially useful when discussing burn wound management over the phone.

4.1.2. wound irrigation

It is directed flow of solution over tissue which forms infection with in cavity

Purpose

To cleaning the area of pathogens.

To remove debris and forming free drainage of infected wound

To facilitate healing of wound

Equipment

Sterile	**Clean**
Galipot and kidney dish	- Rubber and draw sheet
Cotton ball and gauge	- Receiver two
Forceps -3	- Solution (H_2O_2,N/S)
Syringe 20cc or bagwith set.	- Scissor
Irrigation catheter	- Bandage or plaster

4.1.3. Wound debridement

When a wound is covered with black, dead tissue or thick gray/green debris, dressings alone may be inadequate. Surgical removal- sharp debridement– is necessary to remove the dead tissue to allow healing.

Technique

• Sedation or general anesthesia may be required. However, usually the dead tissue has no sensation, so debridement may be done at the bedside or in the outpatient setting.

• Using a forceps, grasp the edge of the dead tissue and use a knife or sharp scissors to cut it off of the underlying wound.

Bleeding tissue is healthy, so cut away the dead stuff until you get to a bleeding base.

• The patient may only tolerate this for a short period of time. Additionally, you don't want to cut off tissue that may be viable. So, you may have to do this a little at a time, and repeat this procedure as needed until all of the necrotic tissue has been removed.

A, Basic Elements of Wound Care

Cleanse Debris from the Wound Possible Debridement bsorb Excess Exudate

Promote Granulation and Epithelialization When Appropriate Possibly Treat Infections Minimize Discomfort Indicated for Mechanical Debridement ONLY Causes Injury to New Tissue Growth Is Painful Predisposes Wound to Infection Becomes a Foreign Body Delays Healing Time Goal is to minimize the frequency of dressing change Daily dressing changes increase chances of infection and disrupts the healing of tissue Optimal wear time is 3-7 days

B. Solutions

Various solutions are appropriate for wound care. These same solutions can be used to cleanse the wounds at the time of dressing change.

Solution	Preparation	Notes
Povidone iodine	Comes pre-made in containers. Best diluted for dressings: 1 part povidone iodine to at least 3 or 4 parts	Toxic to healthy tissues; best used in diluted form for only a few days- then change to a milder solution.

	saline or sterile water.	Safe on the face and around the eyes.
Saline	Comes pre-made, but easy to make yourself. To 1 liter of water add 1 tsp salt. Boil the solution for at least 60 seconds and allow to cool. Store in a closed, sterile container and refrigerate if possible. Good for several days.	Safe anywhere on the body.
Sterile water	Boil a liter of water for at least 60 seconds and allow to cool. Store in a closed, sterile container and refrigerate if possible. Good for several days.	Safe anywhere on the body.
Dakin's solution	Some pharmacies keep Dakin's solution in stock, but it is easy to make. To 1 liter of saline solution, add 5-10 cc of liquid	Better antibacterial agent than saline-so a little harsher on normal tissue. Do not use around the eyes. Makes wounds smell

bleach. Store in a closed, sterile container and refrigerate if possible. If your pharmacy carries Dakin's solution, it's best used diluted: 1 part Dakin's solution mixed with 3-4 parts saline.

better.

4.1.4. Phase of wound healing

Three phases are usually identified in the normal wound healing process:-

1). Inflammatory phase:- vascular & cellular responses occur immediately when tissue is cut or injured.

Vasoconstriction of vessels occurs & fibrin platelets clot forms in attempt to control bleeding .This lasts for 5-10 minutes & is followed by vasodilatation of the vessels. Due to the damage of microcirculation blood elements such as proteins, antibodies, electrolytes &so on will extravagated to the tissue. This causes cardinal sign &symptom of inflammation [oedema (tumour), warmth (color), redness (rubber) & pain (dollar)]. This stay for 2 to 3 days tumercalorrubar dollar (cardinal s/s of inflammation.

2). Proliferotive phase:- lasts from 3 - 21 days.

fibroblasts multiply & form a lattice framework for migrating cells. Epithelia cells form buds at the edges of the wound:these buds develop into capillaries; the nutritional source for the new rganuation tissue. Fibrebles synthesis matrix (collagen) is the 1^0 component of replaced connective tissues. This decrease number of capillaries. Formation of granulation tissue (beefy red tissue w/n is composed of newly formed collagen & b/d vessels) From day 5 to 15 there is progressive increase in the tensile strength blse of increased matrix production. After 2 wks, the wound has only 3-5% of original skin strength. By the end of a month 35-59% finally 70 – 80%

3). Maturation phase

About 3 weeks after injury, fibroblasts begin to leave the wound. The scar appears large, until collagen fibrils recognized into tighter positions. This along with dehydration, reduce the scar but increases its strength. Such tissue maturation continues & reaches maximum strength in 10 or 12 weeks, but it never reaches the original strength of the prewound tissue.

Forms of Wound Healing

Healing by first intention (primary union)

– Wound made aseptically with a minimum of tissue destruction & properly closed as with sutures; heal with little tissue reaction by first intention.

Granulation tissue is not visible & scar formation is minimal

Healing by second intention (granulation) Pus formation (suppuration) has occurred or in which the edges have not been approximated the process of repair is less simple & takes longer.

After the dead cells are removed, the abscess cavity fills with ared, soft, sensitive tissues that bleed very easily.

This tissue is composed of minute, thin walled capillaries & buds that later form connective tissue.

The cells surrounding the capillaries change their round shape to become long. Thin cells (epithelium) grow over these granulation. This method of healing is called healing by granulation & it takes place whenever pus is formed or when lass of tissue has occurred for any reason.

Healing by third intention (secondary suture) if a deep wound either has not been sutured early or breaks sown & then is resutured later, two apposing granulation surfaces are brought together. This result in deeper & wider scar.

4.1.5. Factor affecting wound healing

Local factors

Infection:- non infected wound heal faster than infected wound Presence of any foreign material in the wound interferes with healing of wound e.g. necrotic (dead) tissues

Oedema:- interfere with b/d supply & possibly couse suture to rapture

Local pressure:- impair b/d flow to the area

SYSTEMIC FACTORS

1). NUTRITION:-important nutrient for wound healing are

water-prevent DHN and support physiologic function

protein

vitamins and minerals:- vitamin –C requires for creation of normal collagen

2). **Vascular status:-**inadequate perfusion

smoking causes vasoconstriction and interferes with healing

3). **Systemic disorders:-**depress cell function

- ✓ hemorrhagic shock
- ✓ renal failure
- ✓ diabetic mellitus
- ✓ hepatic disease
- ✓ sepsis

4). **Medication**;-

steroid

anticoagulants

5). Immunosuppresed state

Types of dressing material

- Pick up forceps in a container
- Sterile bowl or kidney dish
" cotton balls
" gallipots
" gauze
- Three sterile forceps /two sterile forceps & one pair of
- Rubber sheet with its cover

- Antiseptic solution as ordered
- Adhesive tape or bandages
- Scissors
- Ointment or other types of drugs as needed
- Receiver
- Spatula if needed
- Benzene or ether

4.1.7. Wound suturing and removal

Suturing

Objective: At the end of this lesson, the learner will be able to

Define suturing and suture

Describe the purpose of the different types of sutures

Re-demonstrate suturing

Definitions

Suturing is the technique of uniting parts of the body by stitching them together.

Sutures are threads used to sew body tissue together which can be **absorbable (Chromic Cat gut)** and **non absorbable (**silk, cotton, linen, clips and wire nylon).

Purpose

-To approximate wound edges until healing occurs.

-To speed up healing of wound.

-To minimize the chance of infection

For aesthetic purpose

Indication

Open intentional and unintentional wound

Contraindication

-Edema of the wound margins
-Infection
-Puncture wounds
-Animal bites

Tendon, nerve, or vessel involvement
Wound more than 12 hours old (body) and 24 hrs (face)

Precautions

Check that the patient gets TAT before he leaves the hospital.

Do not suture puncture (deep) wound.

Before you suture any wounds make sure it is free of any foreign body.

The completed knot must be tight, firm, and tied

To avoid wicking of bacteria, knot should not be placed in incision lines

Knots should be small and the ends cut short (2-3mm)

Avoid excessive tension to finer gauge materials as breakage may occur

Avoid using a jerking motion, which may break the suture

Do not tie suture too tightly as tissue ischemia may occur.

Equipments

Sterile field set

- Tray or trolley covered with a sterile towel
- Sterile needle holder
- Sterile suture scissors
- Sterile round needle (2)
- Sterile cutting needle (2)
- Sterile hole towel (fenestrated towel)
- Sterile kidney dish(2)
- Sterile needle and syringe
- Sterile gloves
- Sterile tissue forceps(2)
- Sterile cotton swabs in gallipots
- Sterile gauze
- Sterile artery forceps(3)
- Sterile needle holder (2)

Clean tray

- Silk and catgut
- Antiseptic solution with its container
- Adhesive plaster
- Local anesthesia
- Light bulb

Stitch removal

Objective: At the end of this lesson, the learner will be able to

Define stitchremoval

Describe the purpose of stitchremoval

Assemble the necessary equipments for stitchremoval

Re-demonstrate stitchremoval

Definition

Stitch removal is the process of removing suture threads

Purpose

- To prevent further contamination
- To comfort the patient
- To relieve tension from wound site.

Precaution

Keep wound clean and dry for the first 24 hours.

Bathing is allowed after 48 hours.

Stick to the day stitches should be removed**.**

Keep wound clean and make dry dressing if no discharge.

Change bandages often.

Equipment

Sterile field set

- Sterile gauze
- Sterile cotton balls
- Sterile kidney dish
- Sterile forceps (2)

- Toothed tissue /pick up/pin forceps (1)
- Sterile stitch scissors/surgical blad

Clean tray

- Rubber sheet and its cover
- Antiseptic solution
- Receiver/ waste container
- Adhesive tape/ bandage
- Plaster and scissor

1.3.8. Complications of woundhealing

A). hematoma(hemorrhage)

B). infection (wound sepsis)

cellulitis;-is bacterial infection that spread in to tissue planes

abscess:-localized bacterial infection characterized by collection of pus

lymphangitis:-infection of lymphatic system

C). dehiscence and evisceration

dehiscence:-disruption of surgical incision

evisceration:- protrusion of wound content

D). keloid- These are over growths of the scar tissue in tumor form

E).constraction

F). Septicemia: - bacteria in the blood

G). Disfigurement:- More if the wound is Saround the face.

H). Osteomylitis:- which can cause fractur

Self-Check -3	Multiple choice And short answer

Directions: Answer all the questions listed below. Use the Answer sheet provided in the next page

multiple choose

1, one of the following is not an example of open wound

A. Abrasion, C. Laceration,

B. Puncture D. Bruise

2, when tissue is forcibly separated or torn of the victim's body is said to be

A, Puncture C. Laceration

B, Abrasion D. Avulsion

3, One of the following Does cause wound

A, Injuries / trauma C. Fungous

B, Virus D, All of the above

4, _____ is contagious fungal infection of the hair shaft and commonly caused hair loss in children

A, Tineapedis (athlete's foot) C, Tineacapitus (ringworm of scalp)

B, Closterdiumtetani D, None of the above

5, _____ is vascular & cellular responses occur immediately when tissue is cut or injured.

A, Proliferotive phase C. Inflammatory phase

B, Maturation phase D. None of the above

6, What are the difference between open and closed wound?

7, Write the purpose of wound suturing?

8, Write the purpose of wound irrigation?

9, List solution used for wound care?

10 , List factors affecting wound healing?

Note: **Satisfactory rating - 6 points** **Unsatisfactory - below 6 points**

Answer Sheet

Score = _____

Rating: _____

Name: _____ Date: _____

Answer

1,_____

2_____

3_____

4, _____

5, _____

6,

7,

8,

9,

10,

Operation Sheet -1	wound irrigation

Procedure of wound irrigation

- Explain the procedure to the patient
- Wash your hand
- Assemble necessary equipment
- Positioning the patient to drain drainage properly
- Put rubber sheet under part.
- Remove outer and inner layer dressing as usual
- Put receiver under area to receive out flow
- Use syringe of bag with desired amount of solution fitted with catheter.
- Use forceps to direct catheter in to wound
- First inject H_2O_2 at body temperature gently and wait for flow. This must be followed by normal saline for rinsing.
- Make sure wound is cleaned and dried properly
- If drainage tube ordered, secured it to centre of the wound and slipping down out of sight the drainage tube.
- Cut the gauze towards its center to fit around drainage tube ,so that it fits properly around tube thus preventing discomfort.
- Dress the wound and check if it is covered completely
- If necessary , attached drainage tube to bottle or bag.
- Secure dressing in place by adhesive tope or bandage
- Leave patient comfortable
- Record state of wound
- Clean and return equipment to its place

Operation Sheet -2	Wound suturing

Procedure of wound suturing

- Check the order for suturing
- Great the patient, introduce yourself and explain the purpose of the procedure to the patient.
- Wash your hands
- Clean trolley or tray, assemble sterile equipment on one side & clean items on the other side and make - sure that the sterile equipments are properly covered.
- Adjust light
- Put on sterile gloves.
- Clean the wound thoroughly.
- Drape the wound with the hold sheet
- Infiltrate the edge of the wound to be sutured with local anesthesia.
- Approximate the edge of fascia with the help of the tissue forceps and using the round needle and catgut.
- Suture the facial layer first followed by muscles. Suture the facial layer and muscle with chromic catgut and.
- Using cutting needle and silk suture the outer layer of skin approximating the edges with the help of the tissue forceps
- Clean wound area with iodine
- Dress the wound with sterile gauze.
- Remove the whole sheet.
- Make the patient comfortable.
- Remove all equipment wash and return to its proper place or send for sterilization.
- Wash your hands
- Record the state of the wound.

LAP Test	Practical Demonstration

Name: _____ Date: _____

Time started: _____ Time finished: _____

Instructions: Given necessary templates, tools and materials you are required to perform the following tasks within --- hour.

Task 1. wound suturing

Task2. wound irrigation

References

_Perry AG & Potter PA. 2006. Fundamental of Nursing. 6th Edition: Elsevier Mosby .

_Rosdahl& Kowalski's. 2003. Textbook of Basic Nursing, Procedure Checklists 8th Ed: Lippincott Williams & Wilkins.

_Ruth F.cravan&Hirnle J. 2007. Fundamental of Nursing: Human health and function:5th Edition: Lippincotwillams&wilkins.

_Suzanne C. O'Connell Smeltzer, & Brenda G. Bare. (2004). Brunner and Suddarth's Text Book of _Medical-Surgical Nursing. 10th Edition: Lippincott Williams and Wilkins. Pp 249-282.

_Clement I. 2007. Basic concepts on Nursing procedures 1st edition: Jaypee Brother

Pripared By

No	Name	Educational Back grund	LEVEL	Region	College	Email	Phaone Number
1	Middega Jbril	Nursing	B	oromia	Nagelle HSC	midhagadhangago@gmail.com	0091318425
2	Biratu Ebessa	Nursing	A	BGRS	Pawi HSC	biratuebs004@gmail.com	0915926607
3	Ali Adan Mohamed	Nursing	A	Somali	Jigjiga HSC	alishide120@gmail.com	0912866022
4	Tariku Abebe	Nursing	A	oromia	Mettu HSC	gessessetariku@gmail.com	0917831032
5	Birhanu Tessisa	Nursing	B	oromia	Nekemte HSC	birhanutessisa3@gmail.com	0913327601
6	Eskender Birhanu	Nursing	B	Harari	Harar HSC	amenaesender@gmail.com	0933259187
7	Ferhan Abubeker	Nursing	A	Harari	Harar HSC	Feru_ab@yahoo.com	0915742083