Paper pathology and microbiology

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Q 1**. WHAT do you know about healing and repair explain**

Ans; HEALING healing is process of restoration of health from un balance diseases damage organism.

The result healing can be took our the case of health challenge but can grow without bring cover or heal without a cure

1. Physical damage or diseases suffer by an organism healing involve the repaired of living tissue.

 Organ and the biological system is a whole resumption of normal function most organ heal using a mixture of both mechanism

2 it is also refer to in the context grieving process

3 in psychiatry in psychology healing is the presses by which neuroses and psychosis are resolve to the degree that the client is able to lead a normal or full filling existence without being overwhelmed

By psycho pathological phenomena this process many involve psychotherapy and pharmaceutical

Treatment

4 healing start BEFORE THE end of inflimmation the three possible outcome of act inflimmation.

* Complete regenerations
* Chronic inflammation
* Fibrosis

**Phases of healing**

There are three of phases of healing if you are injure physical therapy you to accelerate your healing process and increase the likelihood of a complete recovery

**1 reaction phase**

During the first 24 to 72 hour of an injury a trauma response has begun it your injure area. This can include swelling muscles spasm and greedy .it is during this stage of often use pain killer and immobilizes the pain full area. Physical therapy at new highest help you through this phase by using the body nature press.

2 **regeneration phase**

Regeneration typically last six to eight week during this time your lays down new tissue with in injured area and nerve collection to damage tissue

* Prevent of exercise scare tissue but up ;which can lead to recovery injure
* Help in over all recovery no matter is how sever the injury
* Decreases pain
* Increase range of motion

**Remolding phase**

 Remolding phase is the complete restoration of healthy function to the injured area, and may take as long as 3-12 months after your injury. Don’t be fooled into thinking you are healed just because you no longer have pain. Connective tissue heals over time. For example, the true source of a recurring spinal disc injury is often poor health of the connective tissue, and faulty spinal mechanics. The simple act of bending over to pick up a pencil can cause serious back pain. During the Remodeling Phase, we are correcting faulty spinal mechanics to achieve complete restoration of connective tissue health. The benefits provided by physical therapy include:

* Thickening (strengthening) of damaged tissue to prevent recurring injury
* Reduction of scar tissue buildup (accumulation of scar tissue can lead to recurring injury)
* Restoration of full elasticity and flexibility to muscles and joints
* Improving faulty mechanics of your joints and removing compensation patterns
* Improved blood circulation and oxygenation to the entire body
* Minimize development of chronic pain
* Maintaining your range of motion
* Work retraining or work simulated activities that help you stay on the job or return safely

**PROCESS OF HEALING:-**

* **WITHIN 24 HOURS;**
* Neutrophils appear at the margin of the incision moving towards the fibrin clot.
* **In 24 to 48 hours;**
* Movement of the epithelial cells from wound edge from dermis cut margin depositing BM component as they move
* **Day 3;**
* Neutrophils are replaced by macrophages.
* Invasion of granulation tissue invading the incision space.
* Collagen fibers , birding the incision
* Epithelial cell proliferation thickness the epidermal layer.
* **Day 5:-**
* Incision space filled with granulation tissue.
* Neovascularization is maximal
* Abundant collagen fibers, bridging the incision.
* Mature epidermal architecture with surface keratinization.
* **Second week:**
* Continued accumulation of collagen and proliferation of fibroblast.
* Dissapperanceod edema and increased vascularity.
* **End of first month:**
* Scar is made of cellular cut inflammatory cell infiltrate, covered now by intact epidermis.
* Dermal appendages that have been destroyed in the line of incision are permanently lost.

**REPAIR:-**

* Repair is the process by which lost or destroyed cells are replaced by viable cells

**TWO PROCESS OF REPAIR:-**

1. Regeneration
2. Replacement by connective tissue i.e. fibrosis

 **1. Regeneration:-**

* The replacement of the destroyed tissue by the parenchyma cells of the same type is called regeneration.
* The replacement of destroyed cells by proliferation of surrounding undamaged cell of the same type is called regeneration.
* Regeneration refers to the proliferation of cells and tissue to replace lost structures, such as the growth of an amputated limb in amphibians.
* In mammals, whole organs and complex tissue rarely regenerate after injury, and the term is usually applied to processes such as liver growth after partial resection or necrosis, but these processes consist of compensatory growth rather than true regeneration.



**2. REPLACEMENT BY CONNECTIVE TISSUE I.e. FIBROCES:-**

**SCAR FORMATION:-**

* Following are the condition in which tissue repair is achieved by scar formation.
* 1. When resolution (recovery) fails to occur in an acute inflammation
* Then parenchyma cell necrosis cannot be repair by generation because:
* Necrotic cells are perm ant
* Stable cells are destroyed
* Necrosis is so extensive that no cell are available for regeneration
* Three processes that participate in the formation of a scar:
	+ 1. Emigration and proliferation of fibroblast in the site of injury.
		2. Deposition.
		3. Tissue remodeling

**QNO.2 WHAT ARE HEMODYNAMIC DISORDERS? EXPLAIN ANY 3.**

**ANS:-** **HEMODYNAMICS DISORDER:-** The health and well.being of cell and tissue depend not only on in itact circulation to deliver nutrients but also on normal fluid hemostasis. The chapter review the major disturbance involving the hemodynamic system.

**3 HEMODYNAMICS DISORDERS ARE FOLLOWING:-**

**1. Thrombosis:-**

**Definition:** Thrombosis is defined as the formation of a solid or semisolid mass from the constituents of the blood within the vascular system during life.

**Postmortem clot: condensation** of blood composition include (RBCs, WBCs, platelets, and fibrin) inside vascular system after death.

**Postmortem clot Chicken fat clot Thrombus**

After death after death during the life Dark- red in veins Yellow and red in hearts cavities of horse Greenish- red Unattached Attached Homogenous Homogenous Laminated smooth Rough

**Classification of thrombus:-**

According to the intensity of thrombus can be classified as

1- Occluded thrombosis: which closed whole the lumen of blood vessels.

2- objurgating: which closed the lumen of blood vessels partially.

3- Canalized: which closed the canal opening.

**Pathogenesis:**

Platelets leave the blood stream, agglutinate and adhere to the damaged endothelium. They form laminar, which are arranged vertical to the blood stream and called lines of Zhan. Soon, fibrin accumulates around them with red and white blood cells.

**Cases of thrombus:**

There are three factors that predispose to thrombus formation. These factors are called Virchow’s triad:

A: Endothelial injury

B: Stasis or turbulence of blood flow

C: Changes in composition of blood:

**A: Endothelial injury**

It is the most important factor in thrombus formation and by itself can lead to thrombosis. Endothelial injury is particularly important in thrombus formation in the heart

& arterial circulation.

• In hemodynamic stress like severe hypertension & turbulence of flow over scarred valves directly damaging the endothelium.

• Bacterial end toxin.

Endothelial damage may be:

Mechanical, inflammatory, or degenerative the injured endothelium becomes. Swollen with rough surface.

**B: Turbulence or Stasis (Alterations in normal blood flow)**

Under physiologic conditions normal blood flow is laminar, that is, the cellular elements flow centrally in the vessel lumen separated from endothelium by slowing moving clear zone of plasma. Stasis & turbulence therefore:

a. Disrupt the laminar flow and bring platelets in to contact with the endothelium

b. Prevent dilution of activated clotting factors by freshly flowing blood

c. Retard or make a time lag in the inflow of clotting factor inhibitors and permit the buildup of thrombi.

• Stasis is a major factor in the development of venous thrombi while turbulence contributes to arterial & cardiac thrombosis by causing direct endothelial injury or by forming countercurrents & local pockets of stasis.

d) Hypervisicosity syndrome, i.e. an increase in hematocrit in excessive amount due to various reasons such as polycythemia causes stasis in small vessels.

**C- Changes in composition of blood:**

- **↑** platelets e.g. after operations.

- **↑** fibrinogen as in pregnancy.

- **↑** R.B.Cs. (polycythemia) **→ ↑** viscosity of blood **→** stasis **→** thrombosis.

- **↑** W.B.C. as in leukemia **→ ↑** viscosity of blood **→** stasis **→** thrombosis.

**2. Edema:-**

**Definition:** Edema is increased fluid in the interstitial tissue spaces or it is a fluid Accumulation in the body cavities in excessive amount Depending on the site,

**Clinical classification of edema:**

One can also clinically classify edema into localized & generalized types.

**A) Localized**

a) Hydrothorax – fluid accumulation in pleural cavity in a pathologic amount.

b) Hydro pericardium – pathologic amount of fluid accumulated in the pericardial cavity.

c) Hydro peritoneum (ascites) – fluid accumulation in peritoneal cavity**.**

d) Hydrocephalus fluid accumulation in brain.

Example of disease cause localized edema

1) Deep venous thrombosis

2) Pulmonary edema

3) Brain edema

4) Lymphatic edema

**Generalized edema (antisera):** is a sever & generalized edema of the body with profound subcutaneous swelling. **Occurs due to**

a. Reduction of albumin due to excessive loss or reduced synthesis as is caused by:

1) Protein loosing glomerulopathies like nephritic syndrome

2) Liver cirrhosis

3) Malnutrition

4) Protein-losing enteropathy

Pathology lectures 3rd stage

b. Increased volume of blood secondary to sodium retention caused by congestive heart failure

Ex of diseases cause generalized edema

1) Nephrotic syndrome

2) Liver cirrhosis

3) Malnutrition

4) Heart failure

5) Renal failure

**Mechanism of edema formation:**

Approximately 60% of the lean body weight is water, two-thirds of which is intracellular with the remainder in the extracellular compartment.

The capillary endothelium acts as a semipermeable membrane and highly permeable to water & to almost all solutes in plasma with an exception of **proteins**. Proteins in plasma and interstial fluid are especially important in controlling plasma & interstitial fluid volume. Normally, any outflow of fluid into the interstitium from the arteriolar end of the microcirculation is nearly balanced by inflow at the venular end. Therefore, normally, there is very little fluid in the interstitium.

**Edema formation** is determined by the following factors:

1) Hydrostatic pressure

2) Oncotic pressure

3) Vascular permeability

4) Lymphatic channels

5) Sodium and water retention

**3. Hemorrhage:-**

Hemorrhage is extravasations of blood outside the blood vessel.

**Causes:**

• Physical trauma – Stabbing

-Atherosclerosis

-Vacuities

- Gunshot

- Motor vehicle accident

• Inadequacies in blood clotting which can be due to:

A. Too few or poorly functioning platelets (i.e. qualitative & quantitative defect of platelets)

B. Missing or low amount of clotting factors

E.g. Low levels of prothrombin, fibrinogen & other precursors.

Inadequate vitamin K leads to clotting factor deficiency because this vitamin is important in the synthesis of the clotting factors by the liver.

**Type of hemorrhage.**

**A. External hemorrhage**

Escape of blood outside the body.

1- Epistaxis: Bleeding from the nose.

3- Hemoptysis: Coughing of blood.

3- Hematemesis: Vomiting of blood.

4- Melena: Presence of dark digested blood in stools.

5- Bleeding per rectum: passage of red blood with stool

6- Hematuria: Blood in urine.

7- Menorrhagia: Excessive or prolonged menstrual bleeding

8- Metrorrhagia: Irregular uterine bleeding unrelated to menses

9- Bleeding from skin.

**B. Internal hemorrhage**

Bleeding into body cavities.

1- Hem thorax: Hemorrhage into the pleural sac.

2- Hem pericardium: Hemorrhage. Into pericardial sac.

3- Hem peritoneum: Hemorrhage. Into peritoneal sac.

4- Hematocele: Hemorrhage. Into tunica vaginalis sac.

5- Hemoarthrosis: Hemorrhage. Into a joint cavity.

**C. Interstitial hemorrhage**

1) **Hematoma:** Hemorrhage enclosed within a tissue or a cavity.

2) **Petechial:** Minute 1-2 mm hemorrhages occurring in the skin, mucosal membrane, or serosal surface.

3) **Purpura:** Slightly > 3mm hemorrhage occurring in the skin.

4) **Ecchymosed**: Larger than 1-2cm subcutaneous hematoma (bruises). It is typical after trauma.

**Effects of hemorrhage**:

Depend on the rate and amount of blood loss:

• If > 20% the total blood volume is rapidly lost from the body, it may lead to hypovolemic shock & death. Chronic loss of blood leads to anemia.

**QNO.3. WHAT IS RENEWAL AND REGENERATION?**

**ANS: -REGENERATION:-**

Regeneration results in the complete restitution of lost or damaged tissue In mammals, whole organs and complex tissues rarely regenerate after injury, and the term is usually applied to processes such as liver growth after partial resection or necrosis, but these processes consist of compensatory growth rather than true regeneration Tissues with high proliferative capacity, such as the hematopoietic system and the epithelia of the skin and gastrointestinal GI tract themselves continuously and can regenerate after injury as long as the stem cells of these tissue are not destroy.

**RENEWAL:-**

Most often consists of a combination of regeneration and scar formation by the deposition of collagen scar formation is the predominant healings process that occurs when the extracellular matrix framework is damaged by severe Chronic inflammation that accompanies persistent injury also stimulates scar formation because of local production of growths factory they promote fibroblasts prololiferation and collagen the term fibrosis is used to describe the extensive deposition of collagen that occur under these situation Cells in the ECM (fibroblasts, macrophages, and other cell types) produce growth factors, cytokines, and chemokines that are critical for regeneration and repair. Although repair is a healing process, it may itself cause tissue dysfunction, as, for instance, in the development of atherosclerosis.

**Control of Normal Cell Proliferation and Tissue Growth:-**

In adult tissues the size of cell populations is determined by the rates of cell proliferation, differentiation, and death by apoptosis, and increased cell numbers may result from either increased proliferation or decreased cell death.

Differentiated cells incapable of replication are referred to as terminally differentiated cells.

The impact of differentiation depends on the tissue under which it occurs: in some tissues differentiated cells are not replaced, while in others they die but are continuously replaced by new cells generated from stem cells.

Cell proliferation is largely controlled by signals (soluble or contact- dependent) from the microenvironment that either stimulate or inhibit proliferation. An excess of stimulators or a deficiency of inhibitors leads to net growth and, in the case of cancer, uncontrolled growth.

The tissues of the body are divided into three groups on the basis of the proliferative activity of their cells: continuously dividing (labile tissues): Such as surface epithelium quiescent (stable tissues):

Such as liver Non-dividing (permanent tissues): Such as neurons Stem cells are characterized by their self-renewal properties and by their capacity to generate differentiated cell lineages (To give rise to these lineages, stem cells need to be maintained during the life of the organism. Such maintenance is achieved by two mechanisms:

obligatory asymmetric replication, in which with each stem cell division, one of the daughter cells retains its self-renewing capacity while the other enters a differentiation pathway, and stochastic differentiation, in which a stem cell population is maintained by the balance between stem cell divisions that generate either two self- renewing stem cells or two cells that will differentiate.

**QNO.4 WRITES A DETAILED NOTE ON STAPHLYOCOCCUS AND STREPTOCOCCUS?**

**ANS:-STAPHYLOCOCCUS:-**

* Bacteria in the genus staphylococcus are pathogens of man and other mammals.
* Traditionally they were divided into two groups on the basis of their ability to clot blood plasma. The coagulates- positive staphylococci constitute the most pathogenic species s aurous. The coagulates- comprise over 30 other species. The CNS is common commensalism of skin, although some species can cause infections. It is now obvious that the division of staphylococci into coagulate positive and negative is artificial and indeed, misleading in some cases.
* S aurous expresses a variety of extracellular proteins and polysaccharides, some of which are correlated with virulence. Virulence result from the combined effect of many factors expressed during infection. Antibodies will neutralize staphylococcal toxins and enzymes, but vaccines are not available.
* Both antibiotic treatment and surgical drainage are often necessary to cure abscesses’, large boils and wound infections.
* Staphylococci are common causes of infection associated with indwelling medical devices.

**STRUCTURE OF STAPHYLOCOCCUS:-**

* Staphylococci are gram-positive cocci about 0.5 – 1.0 Mm in diameter.
* They grow in clusters, pairs and occasionally in short chains. The clusters arise because staphylococci divided in two planes.
* Streptococci grown on solid medium may appears as clumps. Several fields should be examined before deciding whether clump or chains are present.

**TREATMENT:-**

* Infections acquired outside hospitals can usually be treated with penicillin’s resistant B lactates.
* Hospital acquired infection is often caused by antibiotic resistant strains and can be treated with vancomycin.

**DIAGNOSIS:-**

* Diagnosis is based on performing tests with colonies. Tests for clumping factor, coagulate, hemolytic and thermos table deoxyribonucleic are routinely used to identify S aurous. Commercial latex agglutination tests are available. Identification of S epidermidis is confirmed by commercial linotyping kits.

**STREPTOCOCCUS:-**

* Streptococci are gram-positive aerobic organism that cause many disorder skin infections, sepsis, pneumonia, pharyngitis and endocarditic.
* Symptoms vary with the organ infected. Squealer of infections due to group A beta-hemolytic streptococci may include rheumatic fever and glomerulonephritis strains are sensitive to penicillin, but macrolide-resistant strains have recently emerged.

**CLASSIFICATION OF STREPTOCOCCUS:-**

* They are classified into three types
1. **BETA-HEMOLYTIC STREPTOCOCCI** produce zones of clear hemolytic around each colony.
2. **ALPHA-HEMOLYTIC STREPTOCOCCI** are surrounded by green discoloration resulting for incomplete hemolysis.
3. **GAMMA-HEMOLYTIC STREPTOCOCCI** are no hemolytic.

**DISEASE CAUSED BY STREPTOCOCCUS:-**

The most common acute disease due to GABHS is

* Pharyngitis
* Skin infections
* In addition, delayed, non-supportive complications
* DISEASE caused by other streptococci species is less prevalent and usually involves soft-tissue infection

**SKIN INFECTION:-**

* Skin infections include
* Impetigo
* Erysipelas
* Cellulitis
* **Impetigo** is a superficial skin infection that cause crusting or bullas
* **Erysipelas** is superficial cellulites that also involve the lymphatic. Patients have shiny, red, raised, indurate lesions with distinct margins. It is most caused by GABHS.
* **Cellulites** involve the deeper layers of skin and may spread rapidly because of the numerous lyric enzyme and toxins produced mainly by group a streptococci.

**DIAGNOSES:-**

* Culture
* Sometimes rapid antigen tests or antibody titers
* Streptococci are readily identified by culture on a sheep blood agar plate.

**TREATMENT:-**

* PENICILLIN is the drug of choice for pharyngeal GABHS infection. No isolate of GABHS has shown penicillin resistance clinically.

***THE END***