University Surgical Hospital Split Thoracic surgery department

WOUNDS

Prof. PHD. Nenad Ilić M.D.

Definition

A wound is an interruption of structural and functional integrity of any tissue

We differentiate wounds according to:

- 1) Cause of occurance traumatic wounds (injuries) VS. surgical wounds (incisions)
- 2) Period of healing acute VS. chronic wounds

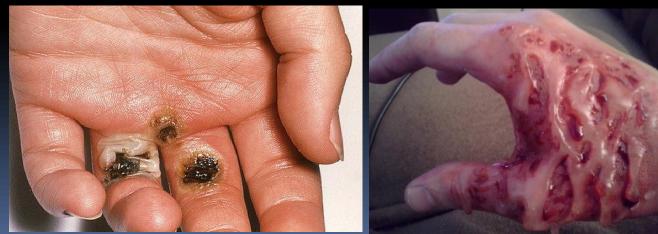
Differentiation of wounds by their origin

 Traumatic wounds (injuries) – due to the effects of external forces upon biological tissue.
 Can be caused by physical, thermic, chemical agents, electric energy as well as ionising radiation.









Differentiation of wounds by their origin

- Surgical wounds (incisions) are made by meticulous tissue dissection following a strict aseptic protocole in order to gain access to the tissue (organ) of interest.
- In such conditions the risk of infection is radically reduced.

 Wound edges are regular and vital, and the wound is being closed layer by layer ensuring the necessary contitions for primary woud healing (reparation).



Differetiation of wounds according to time of healing

- Acute wounds— heal in an orderly and timely reparative fashion to achieve sustained restoration of structure and function.
- Chronic wounds do not proceed to restoration of functional integrity. They are stalled in the inflammatory phase as a result of variety of causes and do not proceed to closure. The prime examples include decubital, arterial and hypostatic ulcers as well as chronic diabetic ulcers.

N.B. All wounds which fail to heal, in spite of best possible treatment, within 3 months are classified as chronic wounds.

Traumatic wounds - INJURIES

- Injuries are the repercussion of an external force applied to the target organs or tissues. The classification is based upon the integrity of the skin or the superficial membrane (capsule) of the injured organs.
- Thus we differentiate between OPEN and CLOSED wounds.
- Classification:

1) CLOSED (the skin or the superficial organ lining (capsule) is intact)

2) OPEN (a breach of skin or organ capsule is present)

A) Penetrating (penetrated body cavity) B) Perforating (opened hollow organ)

A) Non penetrating B) Non perforating

Classification of closed wounds

(injuries)

- a) Contusions
- b) Haematomas
- c) Distensions
- d) Commotions
- e) Crush injuries
- f) Luxations
- g) Fractures
- h) Blast injuries







- Excoriations (Excoriatio) The damage is limited to the epidermis (the basal lamina is undamaged).
- 2) Superficial cut (Vulnus Scissum) caused by a sharp object upon the surface of the skin (knife, scissors, glass, tin). The damage is limited to the skin and subcutaneous layers without injuries to the underlying neurovascular structures or the muscule-tendom apparatus.
- 3) Slash (Vulnus Secatum) grater transfer of energy than with a cut (knife, axe, sickle, scythe). Injuries to the underlying neurovascular structures or the muscule-tendom apparatus are likely







1) Puncture wound (V.Punctum seu

ictum) —Injury to the underlying neurovascular structures or the muscule-tendom apparatus is likely

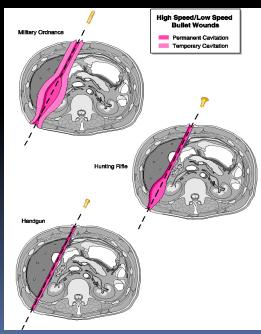
- 4) Laceration (V.Lacerum) caused by blunt force which exceeds the elasticity of the skin. Wound edges are irregular and uneven, prone to infection
- 5) Laceration with contusion (V. Lacerocontusum) – as with laceration, but with larger transfer of force resulting in a greater damage to the tissue



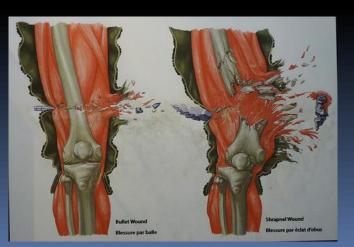
- 7) Crush injury (Conquasatio) due to compression of tissue (limbs). Excessive damage to all tissues types. Risk of Compartment Sy. And Crush Sy.development.
- 8) Gunshot wound (V.Sclopetarium) caused by a firearm projectiles of different velocities.







9) Explosive wound (V.Explosivum) – caused by the blast wave and shrapnel being released at the point of detonation. Massive tissue damage



10) Avulsion (Avulsio)

11) Defect (Defectus)

12) Bite wound (V.Morsum)

V.M.Canis – dog,V.M.Fellis – cat, V.M.Ixodus ricini – tick, V.M.Viperae - snake, V.M.Hominis – human, V.M.Suis – pig, V.M.Equis – horse.







Wounds caused by termal energy transfer:

Burns (Combustio)

- Frost bite (Congellatio)
- Other injuries



- Caued by electrical energy (Combustio electrica) besides skin burns, damage to other organs is likely.
- Radiation burns (Combustio e irridationem) caused by ionic radiation.
 Develops through four stages : redness, blisters, atrophy i scars.
- Chemical burns caused by various chemical compounds: acids, bases, fenols and other corrosive agents.

WOUND HEALING PHASES



WOUND REPAIR (HEALING) IS THE EFFORT OF INJURED

TISSUE TO RESTORE ITS NORMAL FUNCTION AND

STRUCTURAL INTEGRITY

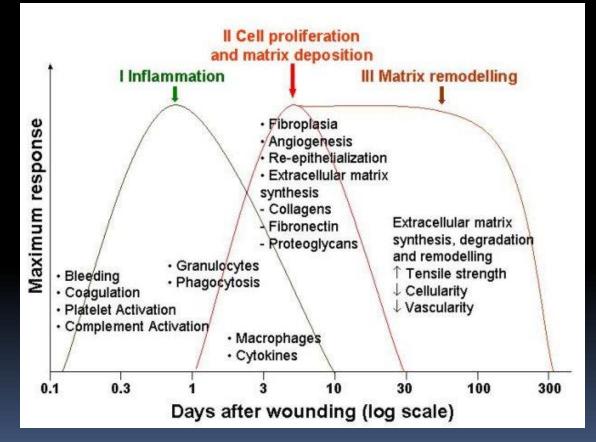
WOUND HEALING PHASES

I. Haemostasis and inflammation

II. Proliferative phase

III. Maturational phase

IV. Contraction

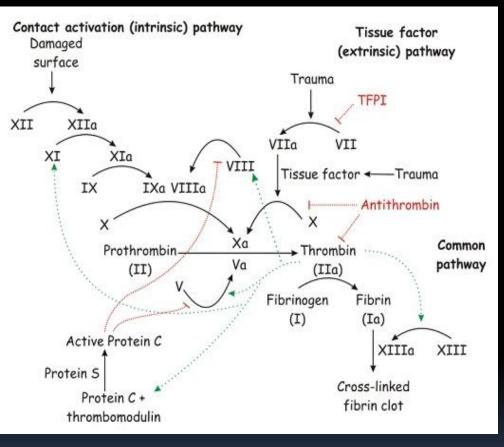


Inflammatory phase

This phase represents an attempt to limit damage by stopping the bleeding, sealing the surface of the wound and removing any necrotic tissue foreign debris or bacteria present.

Hemostasis

- Initiated immediately after injury
- Blood vessel damage results in initial vasoconstriction prevents blood loss
- Erythrocytes and and platelets adhere to the damaged capillary endothelium resulting in plugging of capillaries.
- The clotting cascade is initiated through intrinsic and extrinsic pathways
- PLATELETS play a pivotal role in initiation of inflammatory phase of healing.
- The newly formed blood cloth provides a framework (scaffold) for endothelial cells, inflammatory cells and fibroblasts to migrate across the wound cleft.

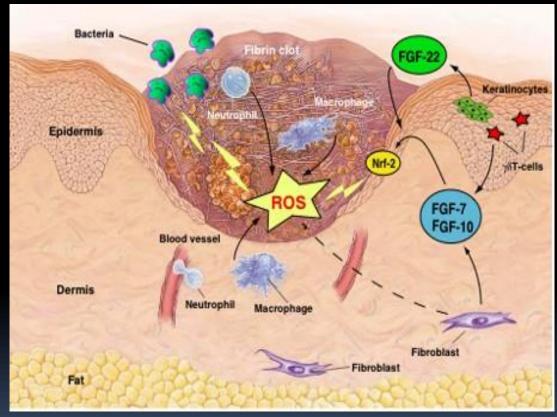


Inflammatory phase

Inflammation

- Lasts up to the fourth day after injury.
- Characterised by classic clinical signs of inflammation (*calor, tumor, dolor, rubor*). Within the first 6 hours the prime cells found in the wound cleft are polymorphonuclears (PMN)
- After 24 to 48 hours the predominance of cells in the wound cleft shifts to mononuclear cells - which are crucial to wound healing
- Stimulated by cytokines, fibronectin, bacterial products, complement degradation monocytes transform into <u>tissue macrophages</u> which orchestrate the further release of cytokines and stimulate other subsequent processes of wound healing

NOTE: PMN are not essential for wound healing. Sterile surgical incisions will heal without the presence of PMN.



Proliferative phase

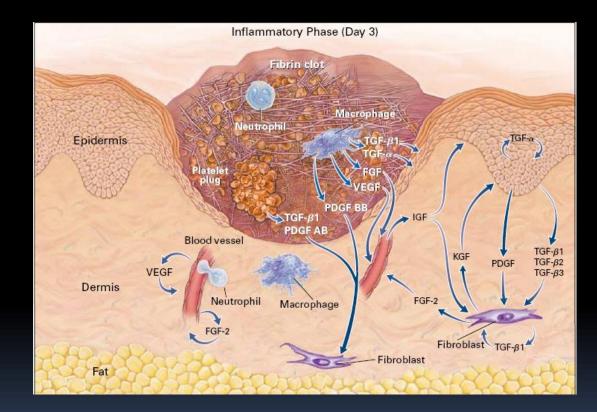
Granulation tissue:

- 1. Newly formed capillary bed
- 2. Macrophages and fibroblasts
- 3. Loose arrangement of collagen, fibronectin and hyaluronic acid
- Glycosa-aminoglican and proteoglycan molecules form a gel – like ground substance between fibrin fibres, permitting diffusion of nutrients, metabolites and hormones



Proliferative phase

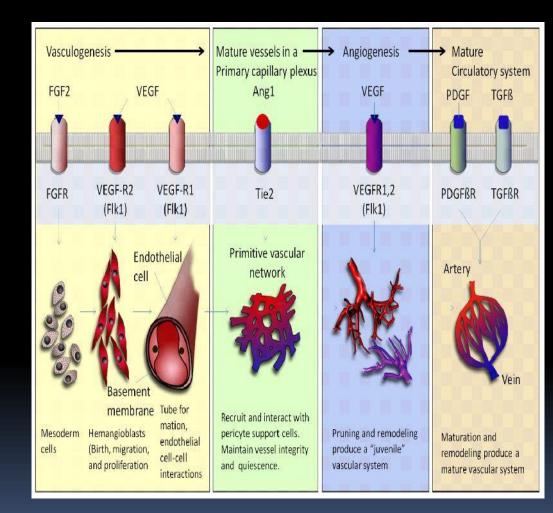
- Angiogenesis
- Activation, proliferation and migration of fibroblasts
- Epithelization (activation, proliferation and migration of keratinocytes)



Proliferative phase – Angiogenesis

Angiogenesis – process of new blood vessel formation necessary to support healing.

- Is initiated within few hours after injury and is stimulated and manipulated by a variety of cytokines produced by macrophages and platelets (FGF 2, TNF-α, PDGF,VEGF)
- Initial angiogenic stimulus within the first three days is provided by FGF 2 released out of distrupted parenchymal cells



Proliferative phase - Fibroblasts

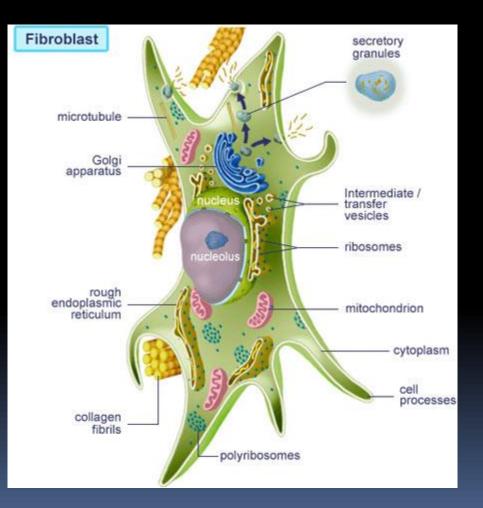
Fibroblasts are specialised cells that differentiate from resting mesenchymal cells in connective tissue

They arrive in the wound cleft by diapedesis form circulating cells

The primary function of fibroblasts is to synthesize collagen

The time required for undifferentiated mesenchymal cells to differentiate into fibroblasts accounts for the delay between injury and the appearance of collagen in the wound (usually 3 to 5 days)

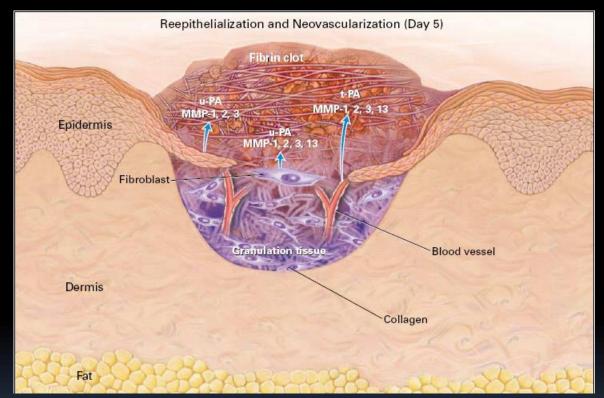
Fibroblasts develop actin-myosin complexes (6th day after injury) – myofibroblasts – wound contraction



Proliferative phase - epitelization

- Begins within hours of injury
- The absence of neighbouring cells at the wound margin provides the initial stimulus for migration and proliferation of epidermal cells (EGF, TGF-α, KGF)
- The migrating keratinocytes dissect the wound by separating the dissicated eschar from viable tissue. Keratinocytes are also phagocyitic and remove bacteria and cellular debris in their path





Maturational phase

- After three to four weeks the wound enters a phase of maturation which can last for months and even years
- Wound contraction occurs by centripetal movement of the whole thickness of the surrounding skin and reduces the amount of disorganised scar
- The main effector cells are myofibroblasts. Wound contraction is initiated around the 5th day after injury and continues up until the 4th week (when fibroblast apoptosis begins to occur)

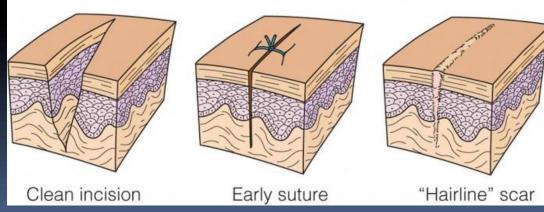
Maturational phase

- During maturation and remodelling phase glycoprotein and mucopolysaccharide levels decrease, formed capillary network regresses and collagen fibres reorganise and are aligned along the major axis of tension.
- Wound strength increases rapidly within 1 to 6 weeks and reaches a plateau up to the 1st year after injury.

Types of wound healing

We differentiate between three types:

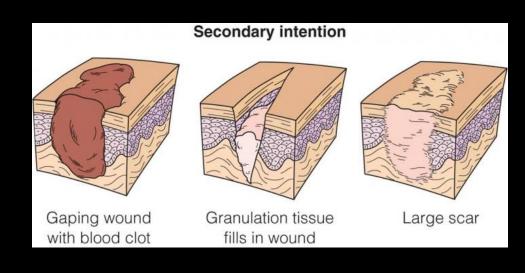
Primary intention — occurs in surgical wounds and injuries without major tissue damage if they had been appropriately surgically processed within the first six to eight hours



Types of wound healing

Secondary intention – occurs when major tissue damage or defect is present (explosive, gunshot, crush wounds) or if an infection has developed within the wound. This type of healing continues for months and results in a large scar formation

Regenerative – results in restoration of normal tissue architecture and function



Factors that inhibit wound healing

Local factors	Systemic factors	External factors	
Infection	Sepsis/SIRS	Antibiotics	
Foreign objects within the wound	Disseminated malignant disease	Antituberculotics	
Wound ischemia – circulation, respiration, local tension	Genetic diseases of the connective tissue	Cytostatics	
Haematoma	Malnutrition	Glucocorticosteroids	
Seroma	Hypoalbuminemia < 2g/dl	Anticoagululatory drugs	
Inadequate wound immobilisation	Vitamin deficiencies A,K,C	Physical damage	
	Mineral deficiencies Zn, Fe	Ionizing radiation	
	Endocrine diseases		
	Liver diseases		
	Advanced age		
	Blood diseases		

Complications of wound healing

 Hemorrhage/Haematoma – inadequate surgical haemostasis



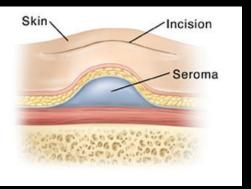
Dehiscence – partial or complete separating of a sutured wound





Complications of wound healing

Seroma formation



Infection - localized (cellulitis / gangrene) or systemic (sepsis)







Clinical examination:

- Location, shape, size, depth, wound edges
- Examination of motor function
- Examination of neurocirculatory status
- X ray analysis if there is doubt of bone damage or residual material within the wound (i.e. glass)

First aid

- 1. Expose the wound completely (remove clothing)
- 2. Disinfect the area around the wound in a centripetal fashion (NaCl 0.09%, ethanol, iodine, chlorehexidine)
- 3. Irrigate the wound with saline (NaCL 0.09%)
- 4. Apply sterile dressing and bandage / transport to hospital

WOUND MANAGEMENT

THE PRIMARY GOAL OF INITIAL SURGICAL

WOUND MANAGEMENT IS TO ESTABLISH THE

CONDITION NECESSARY FOR HEALING BY

PRIMARY INTENTION

Surgical classification of wounds

Wound type	Description	Example	Incidence of infection
I.	Clean	Elective surgery on skin, CNS and cardiovascular system	< 2%
II	Clean / contaminated	All injuries (without major tissue disruption) which have been surgically managed within the first 6 hours and elective surgical procedures on respiratory, digestive and genitourinary tracts	< 30%
111	Contaminated	Wounds which manifest signs of infection at the time of first examination an Surgical procedures in which introduce a large inoculum of bacteria into a normally sterile body cavity	< 60%
IV	Infected	Evident purulent secretion with clear signs of infection (lok i syst)	> 60%

Every wound needs to be surgically managed, revised and eventually closed.

- 1) PRIMARY WOUND MANAGEMENT: if the wound has been surgically dealt with within the first 6 to 8 hours after injury. The goal is to facilitate healing by primary intention
- 2) SECONDARY WOUND MANAGEMENT: applied if the wound is older than 8 hours. The goal is to facilitate healing by primary or by secondary intention.

PRIMARY WOUND MANAGEMENT

STEPS:

- 1) <u>Surgical field preparation</u>
- 2) <u>Application of local anaesthetics</u>
- 3) <u>Wound irrigation</u>
- 4) <u>EXPLORATION</u>
- 5) Debridement
- 6) Suturing
- 7) Dressing
- 8) Antitetanic profilaxis
- 9) Antibiotic protection

NOTE: the first four steps are mandatory when managing any wound type. Steps 5 – 9 are arbitrary.

Surgical site preparation

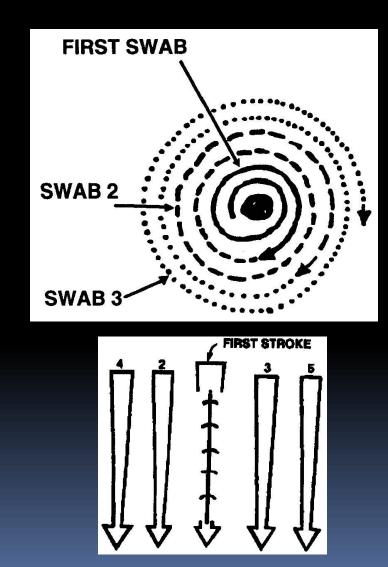
 Surgical site is usually cleaned using topical antiseptics (chlorhexidine, ethanol, 10% iodine)





Surgical site preparation





Surgical site preparation



Local anaesthetics

Lidocaine ("Xylocaine") – commonly used in concentrations of 1% and 2%. The maximal dose for local application is <u>4.5 mg/kg</u>. Anaesthetic effects are immediate and last about 2 hours. If combined with epinephrine the maximal dose of locally given lidocaine can be increased. Bupivacaine ("Marcaine"): 3 mg/kg. Takes effect after 3 to 5 minutes, and lasts about 4 hrs.

Procaine ("Novocaine"): 15 mg/kg. Takes effect after 3 to 5 minutes, and lasts about 1 hrs. The effects of local anaesthetics can be increased with the addition of epinephrine (1:100000). Because epinephrine causes vasoconstriction its use is contraindicated when managing wounds on fingers, ears, nose or male genitalia as there is high potential risk of necrosis.

Wound irrigation

- Irrigation –3% H₂O₂ and saline fluid (NaCl 0.09%)
- Betadine (povidon-iodine) can also be used for irrigation, but its effects are corrosive and have a negative effect on the wound healing process





Debridement and haemostasis

<u>**Debridement**</u> – removal of damaged, or infected tissue to improve the healing potential of the remaining healthy tissue.

<u>Necrectomy</u> - identical process to debridement, except necrotic tissue is being removed. Necrotic tissue does not bleed when cut.

Haemostasis – compression, electrocoagulation, ligatures

Wound drainage – drainage is placed if haematoma or seroma formation is expected. If the wound is without clinical signs of infection the drainage is removed on the 3rd day.





WOUND CLOSURE GUIDELINES

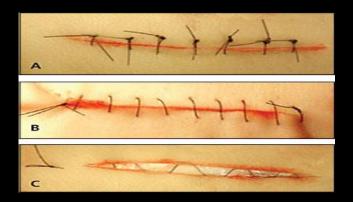
Closure type	Time of closure	Indication
Primary closure	Immediately following primary wound management	 All wounds on the head regardless of time elapsed All serosal tears (dura mater, joint capsule, pleura, peritomeum) All traumatic wounds (excluding bite wounds)
Primary - delayed	3-6 days after initial examination and management	 Bite wounds Traumatic wounds with extensive tissue damage (expl, gunshot, crush) Other traumatic wounds older than 8 hrs at first examination
Secondary closure	7 days after initial examination and management	 Wounds that have become infected after primary or primary-delayed closure Wounds that manifest sure clinical signs of infection at first examination

WOUND CLOSURE

Suturing the wound

Closure with metal clips

Closure with tissue glue







POSTOPERATIVE MANAGEMENT

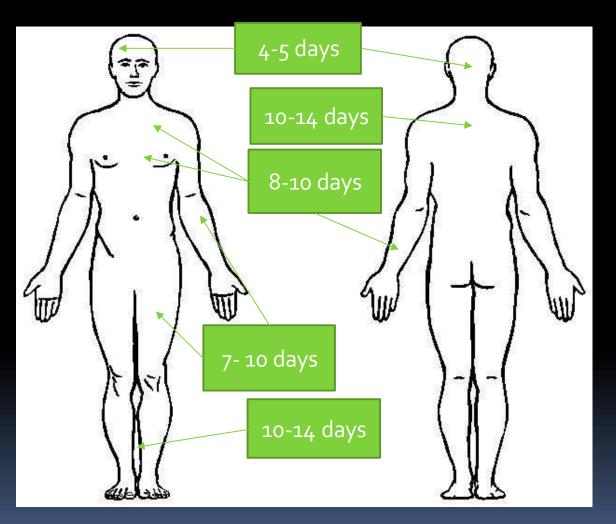
- After closure a sterile dressing is applied to the wounded site
- Role of wound dressings:

- Provides mechanical protection
- Prevents fluid loss
- Prevents secondary infection
- Removes excess exudate
- Creates a moist environment
- If there are not any signs of inflammation present, the wound is inspected and the dressing is changed on the 3rd day.
- Next inspection is scheduled when stitches are to be removed. This depends on the wounded site.

POSTOPERATIVE MANAGEMENT

Suture removal

- Wounds on the head and neck 4-5 days
- 2) Wounds on the back and limbs 10
 -14 days
- 3) Other localisation 7- 10 days



POSTOPERATIVE MANAGEMENT

POSTOPERATIVE WOUND INFECTION

- Open and examine the suspicious portion of the wound
- Irrigate

- Debridement
- Leave the wound open and apply local wound care
- Antibiotics only if systemic signs of infection are present or there is erythema spreading beyond the wound edges



Antitetanic protection

 Antitetanic protection is achieved by – <u>primary wound</u> management, passive (HiG) and active immunisation (Ana-Te)

 Human antitetanus imunoglobuline (HiG) - passive protection, contains antitetanus antibodies. The dose of 250 i.u. is applied into the muscle and has to be administered within the first 24 hrs. The vaccinated person is protected for up to 3 months.

 Antitetanus vaccine (Ana-Te) - active protection, contains tetanus anatoxin which stimulates production of specific antibodies. The vaccine is applied muscularly in a dose of 0,5 ml.

Antitetanic protection

The person is not vaccinated or vaccination records are unavailable	250-500 i.u. HIG + first dose Ana-Te 0.5 ml (next two dose are scheduled one month apart)
Persons vaccinated within the last 5 years	VACCINATION NOT NECESSARY
Persons vaccinated more than 5 years ago	Booster dose with a single Ana-Te vaccine

<u>ACUTE WOUND INFECTION</u> – All wounds with active purulent secretion with clinical signs of inflammation (heat, redness, pain, swelling) along with a positive culture swab Most infections are caused by skin flora inoculated into the wound Therefore the most common pathogens are Staphylococcus species, E.Coli.

ANTIBIOTIC USE

The presence of bacteria in the wound does not necessarily mean infection. It is important to distinguish between:

Contamination – bacteria are present within the wound but are not multiplying

Colonization – multiplication of bacteria without inflammation Infection – inflammation with bacterial multiplication

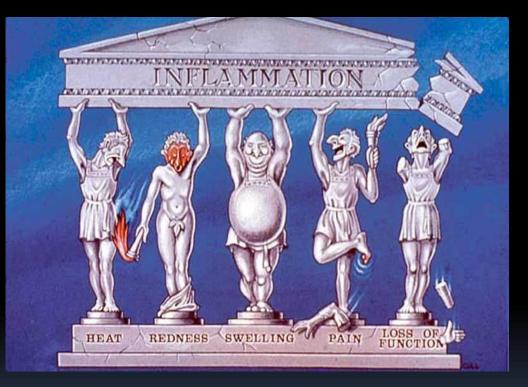
The use of antibiotics is justified if the wound is large and contaminated with clear clinical signs of infection. Wounds without substantial tissue damage, which had been surgically managed within the first 6 to 8 hours usually do not require antibiotic protection Since skin flora (Staphylococcus Epidermidis, S.Auerus) is the most common cause of wound infection β -laktam and macrolide antibiotics are indicated.

Definition:

- Infections that require surgical therapy or are a consequence of surgical treatment
- Ethiology
 - Endogenous bacterial flora (skin, respiratory tract, alimentary tract and genito-urinary tract)
- Surgery's inherent invasiveness creates portals of entry for pathogens to invade the host through natural epithelial barriers. (injured tissue, incisions, puncture sites, indwelling catheters)

Signs and symptoms:

- Local: oedema (tumor), erythema (rubor), pain (dolor), warmth (calor), functional defects (functio laesa).
- General: fever, tachycardia, tachypnea, muscle and joint pain, loss of appetite, nausea and vomiting, diarrhoea, headache, with leucocytosis, neutrophilia or thrombocytopenia.



- The most common surgical infections are:
 - Bacterial infections

- Acute infection of the skin and soft tissues (foliculitis, furuncle, phlegmone, hidradenitis suppurativa, necrotising fasciitis, paranitium, paronychia, erysipelas)
- Acute bodily cavity infection (peritonitis, empyema, intra-abdominal abscess, mediastinitis)
- Anaerobic infections (tetanus, gas gangrene)
- Fungal infections (C. Albicans, Aspergillosis, Actinomycosis)
- Viral infections (HIV, CMV, HV)
- Parasitic infections
- Surgical implant infections

Surgical infections (skin and soft

tissue) • Folliculitis





Carbuncle





Surgical infections (skin and soft tissue)

Hidradenitis suppurativa



Panaritium

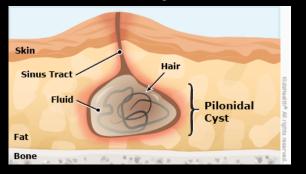
Paronychia





Surgical infections (perianal region)

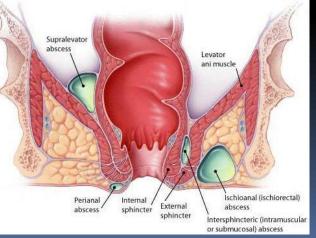
Sinus pillonidalis







Abscessus perianalis





Surgical infections (dangerous)

Necrotizing fasciitis



Acute body cavity infections

- Peritonitis the surface of the peritoneum equals 40% body surface area
 - Primary direct spread of bacteria from blood or lymph vessels to the peritoneal cavity (ascites)
 - Secondary perforation of a hollow viscus, penetrating and perforating wounds, bleeding
 - Clinical signs: signs of peritoneal irritation, abdominal pain, absence of bowel signs, laboratory and imaging studies
 - Therapy: laparotomy, peritoneal irrigation and antibiotic prophylaxis

Intraabdominal abscessus

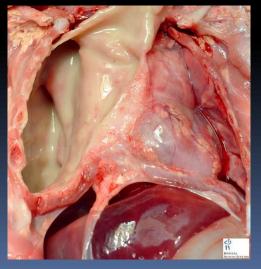
- Limited infaection subdiaphrahmatic, subhepatic, paracolic.
- C.M: remittent and intermmitent i feaver, local and systemic signs of infection
- Th: surgical exploration, drainage under CT drainage

Acute body cavity infections

- Pleural empyema suppurative infection of the pleural space
 - Develops after chest tube placement, surgical procedures on lungs and the oesophagus and as a complication of pneumonia
 - Clinical signs: fever, tachypnoea, dyspnea, tachycardia, hypotension, lab studies, X / ray and MSCT.
 - Therapy: thoracotomy, aspiration, decortication, thoracic drainage.







Other infections

- Mediastinitis perforations of the oesophagus
- Infections after heart surgery mediastinitis
- Infection of orthopaedic, vascular or other implants
- Central and peripheral line infections arterial and venous lines (change of catheter 72 hours)

<u>The main principal of treating surgical infections is to eliminate the</u> <u>cause of infection along with adequate drainage of the infected</u> <u>site</u>

Principals of Antibiotic therapy

- Therapeutic and prophylactic use
- Therapeutic immediate empirical and later in accordance with microbiological analysis.
- Antibiotic Prophylaxis
 - An appropriate narrow spectrum of coverage of relevant pathogens
 - Little or no reliance on the agent for therapy of infection
 - Administration within 1 hour prior to surgery and continued for a maximum of 24 to 48 hours

IT'S JUST & FLESH WOUND