Wounds

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**Wound** - tissue injury with ***interruption of continuity*** caused by physical means (but also by thermal or chemical injury).

Closed wound, which heals in timely fashion, is *sine qua non* of successful surgeon.

Strengths of normal body tissues:

**Tensile Strength** - load *per cross-sectional area* unit at point of rupture; relates to material nature rather than its thickness.

**Breaking Strength** - load required to break wound regardless of its dimension; more clinically significant measurement!

**Burst Strength** - amount of pressure needed to rupture *viscus*, or large *interior organ*.

Wounding mechanisms:

1. **Shearing** – lacerations by sharp objects; low infection rates (can be closed primarily)
2. **Tension** – blunt objects striking skin at < 90° angles → wound flaps; high infection rate.
3. **Compression** – blunt objects striking skin at 90° angle → tissue crushing, stellate lacerations; high infection rate.

TISSUE INJURY & RESPONSE

* injury / necrosis causes repair by forming ***scar tissue*** (there is no return to preinjury status quo!).
* some animals (e.g. stone crab, salamander) can regenerate amputated parts, but mammals have no such ability (with exception of *bone* and *liver*).
* wound healing is continuum and not series of steps (i.e. healing phases overlap in both time and activity).
* the greater the insult, the longer the reparative process → the greater amount of residual scar.
* **acute wounds** proceed in orderly & timely reparative process to restoration of structure and function; **chronic wound** (e.g. pressure sore, leg ulcer), for some reason, does not proceed to restoration of functional integrity - it is stalled in inflammatory phase and does not proceed to closure.

WOUND HEALING PHASES

Whether surgical intervention occurs or not, whether wound is left opened or sealed, same basic repair mechanisms occur!

1. ***Inflammatory* *(reactive)* *phase*** - body's defenses are aimed at limiting damage and preventing further injury;
   * tissue does not gain tensile strength, but depends solely upon closure material.
2. ***Proliferative*** ***(regenerative or reparative) phase*** - reepithelialization, matrix synthesis, neovascularization (granulation tissue).
3. ***Maturational* *(remodeling)* *phase*** - scar contracture (collagen cross-linking, shrinking, loss of edema and vascularity).

N.B. all three phases may occur simultaneously, individual processes may overlap.

I. Inflammatory (Reactive) Phase

- immediate tissue reaction to injury; in clean wound lasts first few days (3-7 days).

1. **Hemostasis** - stops bleeding, seals wound surface.

* fibrin strands trap red cells, form clot, and seal wound.
* hemostasis also causes localized ischemia → further damage.

1. **Inflammation** - removing necrotic tissue, foreign debris, bacteria.

* begins after hemostasis is achieved (hemostatic vasoconstriction → inflammatory vasodilation).
* causes characteristic wound edema.
* ***neutrophil*** activity is also *destructive to surrounding viable tissue* (e.g. more oxidative bursts occur, more bacteria can be removed, but more tissue destruction occurs) - early wound debridement and bacteria removal will limit inflammation and subsequent scarring.
* ***T cells*** produce **IFN-γ** - can cause glycosaminoglycan synthesis and suppress collagen synthesis (important in chronically open, nonprogressing wounds).
* ***macrophage*** is the one cell that is truly central to wound healing; neutrophil work (to phagocytose necrotic tissue & bacteria) is continued by macrophage.

N.B. without monocyte / macrophage, wound proceeds slowly with inadequate debridement and delayed proliferation of fibroblasts & endothelial cells, todėl kai kas išskiria Migratory Phase (macrophage & fibroblast migration).

* bacterial debris (such as lipopolysaccharide) can activate monocyte to release cytokines that will regulate wound healing.
* **TGF-β** is most potent stimulant of fibroplasia (fibroblasts are directly stimulated to produce collagen and fibronectin → proliferative phase).

II. Proliferative (Regenerative) Phase

- formation of granulation tissue (capillary bed, fibroblasts, macrophages, loose arrangement of collagen, fibronectin, hyaluronic acid, and bacteria); starts from 3rd day.

1. **Angiogenesis**

* necessary to support wound repair.
* as basement membrane of postcapillary venules is degraded (by activated endothelial cells), endothelial cells migrate through this gap → divide and form tubules → basement membrane is deposited → mature new capillary.
* angiogenesis is stimulated and manipulated by variety of *cytokines*; also stimulated by local wound *hypoxia*.

1. **Fibroplasia**

* early wound cleft contains: **fibrin**, **hyaluronic acid** (has large hydration shell - enhances cell migration); **fibronectin**, **laminin**, **tenascin** (all 3 are adhesion glycoproteins - assist in cell attachment & migration).
* normally quiescent and sparse fibroblasts are chemoattracted to site, where they divide and produce extracellular matrix.
* chemoattracted fibroblasts use *hyaluronidase* to cleave this early matrix and deposit larger sulfated GAGs; at the same time, fibroblast is depositing collagen onto fibronectin and GAG scaffold.
* in skin, significant collagen synthesis occurs in **5 ÷ 42 days** (new collagen fibers are present in wound cleft 3 days after injury).

N.B. until collagen, wound strength is maintained by fibrin.

1. **Epithelialization** (prevents *fluid loss* and protects against *bacterial invasion*).

* wound is first sealed by blood clot.
* epidermal cells migrate from:
  1. wound periphery
  2. depth of epithelium-lined skin appendages (if still intact).
* if basement membrane is intact, epithelialization proceeds more rapidly; if basement membrane is not intact, it will be repaired first.
* basal cells are stimulated to migrate, their attachments to adjoining cells and to dermis loosen.
* epithelial cells in leading edge of migration become *phagocytic* and release *collagenase & plasminogen activator* - assist migration through wound cleft.
* cells slightly behind leading edge begin to divide.
* after wound cleft is reepithelialized (contact occurred between advancing epithelial edges), epithelial cells divide and stratify (i.e. become columnar and layered again).

III. Maturational (Remodeling) Phase

* begins when collagen deposition is completed.
* ***all wounds contract*** - pull normal surrounding tissue into wound area (reducing amount of disorganized scar).

N.B. wound contraction can be very *beneficial* (e.g. in buttocks, trochanter) but can be very *harmful* in hand, neck and face (can cause disfigurement); H: skin graft (reduces contraction).

* fibroblasts in contracting wound undergo change to *myofibroblasts*.
* fibroblasts develop linear arrangement in tension line that, when removed, causes cells to round up.
* ***matrix metalloproteases*** are necessary to allow cleavage of attachment between fibroblast and collagen → lattice can be made to contract.
* once wound is healed, it will remodel and mature:
  + fibroblast population decreases.
  + dense network of new capillaries regresses.
  + in skin wound strength increases rapidly within 1-6 weeks; after 6 weeks, any additional gain in tensile strength is due to remodeling (cross-linking) of collagen fibers (rather than collagen synthesis); tensile strength increase will continue for as long as 2 years.

N.B. whereas cross-linking causes further wound contraction and increase in strength, it also results in scar that is more brittle and less elastic than normal skin!

* compared with unwounded skin, **scar tensile strength** is only 30% (NMS Surgery: scar reaches 90% of original tissue strength in ≈ 6 weeks).

scar contraction (normal) vs. scar contracture (abnormal)

* contracture is clinical definition that indicates **limit of function**, e.g.
  + scar crosses joint - prevents flexion
  + scar involves eyelid - causes ectropion.
* contracture results when important area has too much scar to prevent normal wound contraction.

ABNORMAL WOUND HEALING

Ultimately, scar type is dictated by *collagen deposition* and balanced by *collagen degradation*;

if balance is tipped in either direction, result is poor:

1. inadequate scar – ***chronic wound, wound dehiscence***

N.B. chronically inflamed wounds can develop ***squamous cell carcinoma*** (originally reported in chronic burn scars by Marjolin); premalignant state is ***pseudoepitheliomatous hyperplasia*** (if this report is obtained on biopsy, biopsy should be repeated - there may be carcinoma present in other areas).

1. proliferative scar – ***hypertrophic scar, keloid*** žr. dermatology 3003 (4) psl.

N.B. **tension** (that signals formation of activated fibroblasts → deposition of excessive collagen) is important;

* + scar that is *perpendicular* to underlying muscle fibers tends to be flatter, more narrow, with less collagen formation (as muscle fibers contract, they reapproximate wound edges) than that which is *parallel* to underlying muscle fibers.
  + **position of elective scar** must be chosen in such way to make less obvious scar in distant future.

Factors That Inhibit Wound Healing

1. **Infection** - the most common cause! dar žr. surgical infection 2203 psl.

N.B. if ***bacterial count in wound exceeds 106 organisms per gram of tissue***, or if ***any β-hemolytic streptococcus is present***, wound will not heal by any means!

* endotoxins stimulate phagocytosis and release of collagenase → collagen degradation and destruction of surrounding previously normal tissue.
* treatments that decrease bacterial count:
  1. mechanical measures (resections, debridements, irrigations)
  2. antibiotics.

1. **Ischemia** (circulation↓, respiration↓, local tension).

* although hypoxia stimulates angiogenesis, wound will not proceed without ***tissue oxygen levels ≥ 35 mm Hg***.
* **tobacco products** (vasoconstriction) has similar impact on wound healing. H: stop smoking before plastic surgery procedures!
* although **anemia** is considered risk factor for impaired wound healing, but it is not so important as hypoperfusion!
* blood supply in *lower extremity* is worst in body; blood supply in *face & neck* is best.

1. **Immune defects**

* immunodeficiencies (e.g. HIV, chemotherapy) – no protection from infection.
* allergies (to specific suturing materials, metal alloys, latex) - heightened immune response also interferes with healing process.

1. **Diabetes mellitus** impairs wound healing at all stages!

* *exogenous insulin* can improve wound healing, but even if started 5 days before wounding, impairment is not completely reversed.
* increased collagen degradation, decreased collagen deposition (formed collagen is more brittle, probably owing to glycosylation).

1. **Ionizing radiation (to wound site)**

* causes endothelial cell injury → endarteritis (atrophy, fibrosis, delayed tissue repair).
* greatest effect is on rapidly dividing keratinocytes during wound healing process → impaired epithelialization.

1. **Advanced age**

* aging process affects all stages of wound healing (macrophages are particularly impacted!).

1. **Obesity**

* excess fat may prevent securing good closure.
* fat does not have rich *blood supply* - most vulnerable of all tissues to trauma and infection!

1. **Malnutrition** (esp. protein catabolism) in generally does not prevent healing.

* *hypoalbuminemic* patient can experience even wound dehiscence!
* ***protein supplements*** can reverse this effect.
* normal patient does not need nutritional support for proper wound healing if food intake will resume within 7-10 days.

1. **Vitamin deficiencies**
2. **Vitamin C deficiency** – impairs collagen synthesis (previously healed wounds may even open in scorbutic conditions!).
3. **Vitamin A deficiency** - impairs monocyte activation, fibronectin deposition; vitamin A directly counteracts effect of glucocorticoids.
4. **Vitamin K deficiency** - ↓synthesis of prothrombin and factors VII, IX, X.

* vitamin K metabolism is impeded by *antibiotics* - patients with chronic infections should have their *clotting parameters* checked before surgical procedures!

1. **Mineral deficiencies**
   1. **Zinc deficiency** (large burns, severe multiple trauma, hepatic cirrhosis) → decreased bursting strength (zinc is cofactor for RNA polymerase and DNA polymerase).
   2. **Iron deficiency anemia** is debatable cause of wound healing delay.
2. **Exogenous drugs**
3. **Glucocorticoids** – inhibit all phases of wound healing; esp. impair fibroblast proliferation and collagen synthesis, stabilize lysosomal membranes (can be reversed by vitamin A); effect is both time and dose related.
4. **Azathioprine** (but not cyclosporine!) ≈ glucocorticoids.
5. **Doxorubicin (Adriamycin)** - direct potent inhibitor of wound healing (esp. if administered preoperatively); in general, delay any chemotherapy for 7-10 postoperative days.

N.B. NSAID doses in therapeutic range are unlikely to delay healing, but aspirin may cause delayed hematomas!

1. **Bad surgical technique**

* tyrinėta, kokiu **instrumentu** (skalpeliu, elektrokauteriu, ultragarsiniu skalpeliu, lazeriu) padarytos chirurginės žaizdos gyja geriausiai - geriausiai gyja žaizdos, padarytos vienu skalpelio judesiu (todėl nesižavėti elektrokauteriu!).
* vengti **mass ligations**.
* negalima leisti audiniams **išdžiūti** operacijos metu – pastoviai drėkink su saline, apdenk su saline-soaked sponges, etc.
* eliminate **dead spaces** by:

1. sutures
2. drains
3. pressure dressings (postoperatively)

Liver

N.B. **jaundice** per se does not affect wound healing and is not contraindication to operation; liver dysfunction (as cause of jaundice) may be contraindication to operation!

Kidney

It is not clear if **urea** per se inhibits wound healing; probably other toxic substances (eliminated by kidneys) play inhibitory role. H: perioperative dialysis.

FETAL WOUND HEALING

* animals and humans who have undergone surgery in utero, have ***little or no scar*** apparent after birth.
* ability for scarless healing is *gradually lost* late in gestation.
* fetal fibroblasts *do not develop activated state (myofibroblast)* until late in gestation - speculation that this is origin of healing differences.
* fetal wound healing has *limited period for inflammation* - less scarring to occur.
* complications of congenital diaphragmatic hernia and myelomeningocele dictate need to further explore fetal surgery.

NEW HORIZONS

* simply closing wound and allowing it to heal in ***moist environment*** is shown to speed closure.
* clinical trials have shown beneficial effect of chronic wounds treatment with exogenous **growth factors**: TGF-β, basic FGF, PDGF.
* until precise cytokine deficit is known for each wound type, application of growth factor remains costly educated guess.
* chronic wounds have significant increase in *matrix metalloproteases* - some investigators are examining **blockage of MMPs** (N.B. MMPs degrade expensive growth factors before they interact with cellular wound components).
* if application of expensive growth factors allows chronic wound to proceed to closure, then application of **gene responsible for these growth factors**, performed less frequently, holds promise of being less expensive.

WOUND CARE

Tetanus prophylaxis! see 227 (3-4) p. (infection)

Face wounds – see [TrH25 p.](file:///D:\Viktoro\Neuroscience\Neuroscience\TrH.%20Head%20trauma\TrH25.%20Facial%20Trauma%20(GENERAL).doc)

1. **Hemostasis**
2. direct pressure & elevation
3. ligation of large vessels
4. if coagulopathic – pack wound until coagulopathy is corrected.
   * it is first step in wound care!
   * without hemostasis, **hematoma** develops → prolonged healing (even suppuration!)
   * if care is started in prehospital setting, next step is wound coverage with (sterile / clean) dressing\* → contact EMS.

\*avoid thick layers – prevents application of direct pressure, may silently absorb large amounts of blood.

1. **distal neurovascular exam**
2. **anesthesia**
3. clean **wound environment** (matted blood and hair) with *saline-soaked gauze* → *disinfecting solutions*\*:
4. ***povidone-iodine*** – the best
5. ***chlorhexidine*** – second the best
6. ***hydrogen peroxide*** – not recommended (but useful to dissolve dried blood and matted hair on scalp wounds)
7. ***alcohols*** – not recommended + painful
8. ***hexachlorophene*** – not recommended

\*N.B. all these solutions are toxic to wound tissues – use only on intact surrounding skin!

N.B. **hair shaving** around wound is no longer recommended – increases risk of infection!

* + if necessary, clip but do not shave.
  + eyebrows should never be shaved or clipped (sometimes do not regrow!)

1. perform **anesthesia**:
   * 1. ***regional*** – preferable: performed away from contaminated and painful wound skin, may “cover” several wounds, doesn’t distort wound environment.
     2. ***local*** – safe, fast & effective; performed through disinfected *intact skin*.
   * anesthetic can also be injected through *exposed subcutaneous tissue*.
     1. ***topical*** – should be applied at least 20 min before initial wound care; anesthesia success is noted as blanching around wound;

TAC (tetracaine, adrenaline, cocaine)

LET (lidocaine, epinephrine, tetracaine)

N.B. avoid vasoconstrictive epinephrine / cocaine on:

* + - 1. distal vasculature areas (ears, nose tip, penis, digits) – can cause necrosis!
      2. contaminated / infected wounds – weaken wound defense mechanisms.

1. **inspection & exploration**

N.B. wound must be ***adequately anesthetized*** before inspection & exploration.

* **bloodless field** must be obtained (e.g. by tourniquet).
* wound edges are pulled apart with instruments – ***entire wound must be adequately visualized*** (esp. wound bottom).
* examine for ***tracts*** that may lead to other areas of injury (if necessary, wound can be *extended surgically* to allow complete examination!).
* if wound is over *joints* or *tendons*, examine them through entire range of motion.

1. **Cleansing & debridement** – removal of:
2. **foreign materials** – with *forceps, scalpels, brushes*.

* if foreign material is ground into tissue (e.g. gravel in motorcycle accident), tissue needs to be resected.

1. **nonviable or heavily contaminated tissue** – by *resection*.

* there are cumbersome techniques to detect tissue viability (e.g. IV fluorescein, tissue oxygen tension, nuclear medicine blood flow) – unpractical; better leave questionable tissue in wound (in anatomical position) and reinspect after 24 hours.
* after debridement, tissues should look healthy with good blood supply.

1. **microscopic debris and bacteria** –by *high-pressure* (7 psi) *high volume* (200-2000 ml) *irrigation with sterile 0.9% saline* (“the solution to pollution is dilution”); e.g. pulsatile jet lavage system;

* alternative to saline may be *nonionic detergent solutions*.

N.B. skin cleaning & disinfecting solutions should not be used on fresh wounds!

* + - * intact skin can stand very strong disinfecting agents, whereas cells of fresh surgical wound are very susceptible to further damage;
      * scrub solutions (containing iodophors, hexachlorophene) are toxic for use in surgical wounds;
      * contaminated experimental wounds subjected to topical treatment with these scrub solutions developed more infections than wounds subjected to 0.9% saline!
* 1964 study: incisional wounds in guinea pigs were contaminated with contaminants (bacterial cultures, saliva, feces, urine, soil, pus); after inoculation, wounds were copiously irrigated and closed; with exception of wounds contaminated with pus, all wounds healed without significantly increased rate of infection! - since that time, irrigation has become standard step in treatment of contaminated wounds.

N.B. all foreign bodies (devitalized tissues, hematomas, seromas also act as foreign bodies):

* 1. Prevent **apposition** of tissues for healing.
  2. Enhance **infection**.
* debridement goal is to ***remove devitalized and highly contaminated tissues*** while preserving critical structures (nerves, blood vessels, tendons, bone).
* immediately after trauma, surgeon must delineate border between *irreversibly damaged tissue* and *tissue that, when properly managed, will recover*.
* **perichondral hematomas** (in ear, nasal septum) should be drained immediately to prevent permanent cartilage damage.
* debridement timing:
  1. **delayed debridement** - causes progression of necrosis (esp. after burns) due to:
     + wound surface *desiccation* (N.B. nerves, tendons, joints, bone, when traumatized and exposed, progress predictably to surface necrosis!).
     + gradual multiplication of contaminating *bacteria*.
  2. **debridement on admission** (strategy developed during Vietnam conflict) → package with saline gauze → debridements at 48 hours intervals until deemed sufficiently free of devitalized tissue & contamination → closure.
     + desiccation remains problem - water in saline gauze quickly evaporates, effectively converting wet dressing to dry dressing.

1. **Closure** types (choice depends on *m/o content in wound*): about flaps & grafts – žr. 2215 p.

**Primary (s. first-intention) closure** - wounds immediately sealed with suturing (all layers), skin graft, or flap → reduced inflammation → finest possible scarring.

* indicated for **clean** and **clean-contaminated** wounds.
* m/o left in subcutaneous tissue may cause wound infection (normally < 2%).
* exposed cartilage (e.g. ear injuries) must be covered with available soft tissue or topical antibiotic ointment - to prevent chondritis.

**Secondary (s. spontaneous) closure** - no active intent to seal wound\*; such wounds spontaneously close by granulation and slow reepithelialization → broad scar.

* indicated for **infected** wounds (or contaminated small wounds).

\*deep layers may be closed, but subcutaneous layer & skin are left open.

* *excessive granulation tissue* may build up (requires treatment if it protrudes above wound surface, preventing epithelialization).

**Tertiary (s. delayed primary) closure**

* indicated for **contaminated** wounds.
* wound is left open (***monofilament nylon*** sutures are laid but not tied; suture ends are held in place with sterile tapes) and treated as infected wound healing by secondary intention (repeated debridements ± systemic or topical antibiotics).
* after 48 hours open wounds become resistant to new infection.
* wound is assessed at 3-5th day:

1. *subcutis is clean and just beginning to granulate* (wound is ready for ***closure***) → sterile adhesive tapes or surgical intervention (suturing, clips, skin graft, flap) is performed.
2. *purulence in subcutis* → treat further as ***infected wound***.

N.B. every wound (surgical or traumatic) should be considered for closure (layer-by-layer).

N.B. if skin is not closed, wound infection cannot occur!

**Age of injury** also dictates closure type - ***golden period of wound care is < 6-8 hours*** (some extend up to 12 hours); exceptions:

*facial wounds* can be closed up to 12-24 hours;

*contaminated foot wounds* should not be closed after > 3 hours.

Choice of surgeon for wound closure:

**Facial wounds** – plastic surgeon (sometimes ENT surgeons).

**Neck injuries** – trauma / general surgeon, ENT surgeon.

**Chest, abdominal injuries** – general surgeon.

**Hand / forearm wounds** – hand surgeon (alternatives – plastic surgeon, orthopedic surgeon).

**Arm, leg injuries** – orthopedic surgeon.

1. **Dressing**

sutured wound – use **dry dressing**.

* dressing ***absorbs*** wound secretions (so bacteria do not accumulate in wound) and ***protects*** wound.
* epithelium migrates across in 48 hours – after that (if no drainage persists) no dressing is necessary (except for patient comfort).

open wound with necrotic debris – use **wet-to-dry dressing** (moistened thin piece of gauze is placed into wound and covered with dry dressing).

* dressing ***cleans & débrides*** wound (devitalized tissue adheres to gauze as it dries and may be pulled off with dressing – useful complement to surgical debridement).
* periodically changing dressing (1-3 times daily ± irrigation), debris is removed (but also removes viable tissue – slows healing!).

N.B. wet-to-dry dressing changes are debridement technique and not healing promoter!

* although some wounds will heal when treated with repeated saline gauze changes, *controlled hydration dressing* (polyurethane, hydrocolloid, hydrogel) will promote faster healing.
* most advocate use of **antibiotic ointments**.
* enzymatic debridement – ointments with collagenase (e.g. Santyl) – daily application suffices.

clean open wound (e.g. healing by second intention after cleaning by wet-to-dry dressings) – use **wet-to-wet dressings** (gauze is not allowed to dry).

* dressing keeps tissue ***moist***, does not débride healthy tissue, removes exudates.
* to prevent dressing adherence, use petroleum *jelly-impregnated porous gauze* or various topical jellies (e.g. Saf-Gel).

large open dermal wounds (e.g. skin graft donor sites, large abrasions) – use dressing that ***enhances epithelial migration*** by protecting its surface (e.g. nonpermeable **scarlet red dressing** – forms scab under which epithelium migrates in moist environment).

1. **additional measures, Wound healing promotion**
2. keep wound **elevated & immobilized**
3. **vitamin A** supplements (esp. if patient is on steroids)
4. hyperbaric **oxygenation**
5. prophylactic **systemic a/b** – only for infection-prone wounds (e.g. bites, heavy contamination); these wounds must be followed-up closely (at 1, 3, 5, 7 days).
6. **pain medications** (NSAIDs, narcotics) – for every patient!

* **showering** is permitted after 48 hours, but wound submersion in water must be avoided for first 3 days.

Topical Negative Pressure (TNP), s. Wound VAC (vacuum assited closure)

- therapeutic technique used to promote healing in acute or chronic wounds, fight infection and enhance healing of burns.

* typical indications:

1. chronic wounds
2. wounds expected to present difficulties while healing (diabetes, vascular disorders)

* vacuum source is used to create sub-atmospheric pressure in local wound environment:

1. removes excess fluid from wound bed
2. enhances circulation
3. removes waste from lymphatic system.
4. seals wound to prevent dehiscence

* vacuum may be applied ***continuously*** or ***intermittently***.
* two forms of dressing:
* **gauze** may be a better choice - for pain sensitive patients with shallow or irregular wounds, wounds with undermining or explored tracts or tunnels, and for facilitating wound healing;
* **foam** (may be cut easily to fit wound) - better when aggressive granulation and wound contraction is desired goal.

Cochrane Review: *There is no valid or reliable evidence that topical negative pressure increases chronic wound healing*

Application of vacuum pump using foam dressing to wound:

“KCI Acti VAC” pump used to create negative pressure:

Negative pressure wound therapy after amputation for wet gangrene:



WOUNDS & INFECTION

1. **clean wounds (class I)** - padarytos steriliose sąlygose, kai GU, GI, respiratorinė sistemos neatvertos (pvz. hernioplastika, breast biopsy); only skin microflora potentially contaminates wound;

***supūliuoja < 2%*** - treat by primary closure.

N.B. *inflammation* is natural part of healing process and should be differentiated from *infection* in which bacteria are present and produce damage.

**Class ID wounds** - prosthetic device (e.g. mesh or valve) is inserted.

1. **clean-contaminated wounds (class II)** - minėtos sistemos opened under controlled circumstances (without significant spillage of contents), jos specialiai paruoštos (mechaniškai + antibakteriškai) ir jose nėra aktyvios infekcijos (pvz. cholecistektomija, kolektomija po gero išklizmavimo);

***supūliuoti turi < 3%*** - treat by primary closure (dar gali sugyti pirminiu būdu).

N.B. class II wounds in colorectal surgery have infection rates 9-25%

1. **contaminated wounds (class III)**\* - stipriai užterštos chir.intervencijos metu (pvz. cholecistektomija dėl empiemos su turinio išsiliejimu, kolektomija su turinio išsiliejimu, visos trauminės žaizdos, open cardiac massage);

***supūliuoti turi < 5%*** - treat by tertiary (s. delayed primary) closure (microorganisms multiply so rapidly that within 6 hours contaminated wound can become infected!).

1. **dirty wounds (class IV)**\*\* - užterštos dar prieš chir.intervenciją (pvz. traumatic wounds in which significant delay in treatment has occurred and in which necrotic tissue is present, wounds created in presence of overt infection with presence of purulent material, wounds created to access perforated viscus accompanied by high degree of contamination, necrotizing soft tissue infections);

***supūliuoja > 50%*** - leave to heal by secondary intention (vėliau kartais galimas tertiary closure).

\***Contamination** exists when microorganisms are present, but in insufficient numbers to overcome body's natural defenses.

* contamination also depends on wound location (e.g. axillae, perineum have high bacterial counts).

\*\***Infection** exists when contamination level exceeds tissue's ability to defend (generally, > 106 bacteria / gram of tissue in immunologically normal host).

* contaminated wounds tend to become infected when:
  1. ***foreign bodies, hematomas, necrotic / devascularized tissue*** (esp. fascia, muscle, bone) are present – m/o multiply rapidly under these “shelters” (to cause infection only 100 microorganisms may be enough).
  2. ***poor vascularity*** exists (e.g. feet wounds more commonly suppurate than face wounds).

Mikrobinė flora: **A atveju** - pagrinde odos, šnervių flora (stafilokokai).

**B, C, D atvejais** - odos, šnervių flora and/or flora priklausomai nuo šaltinio.

It is well accepted that **single dose of antibiotic** should be administered immediately before commencing surgery for class ID, II, III, and IV wounds.

*Panaudota literatūra*:

Merck Manual 1999

Sabiston Textbook of Surgery 2001

NMS Surgery, Emergency Medicine, Pathological Anatomy, Medicine, Pediatrics