Wound healing and wound care

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Wound healing

Definition: normal body process/mechanism responses to heal a new traumatic wound
เกิดขึ้นต่อทุกอวัยวะของร่างกาย ทุกส่วนของ tissue eg. brain nerve bone muscle skin bowel
ในส่วนนี้จะพูดถึงจะยกในส่วนของผิวหนังมาเป็นตัวอย่างเนื่องจาก เห็นง่ายที่สุด

Phase of wound healing

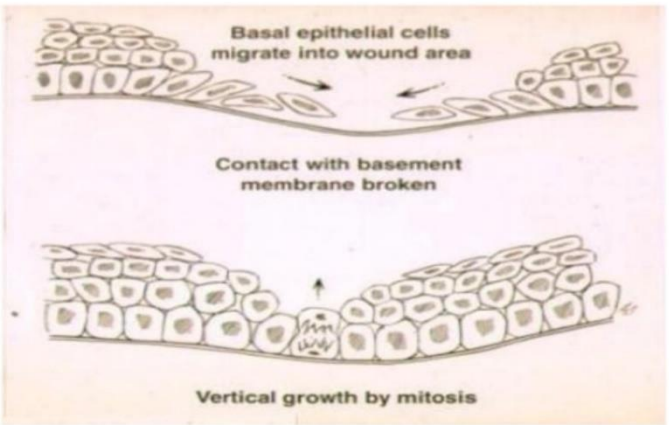
1. Inflammatory phase ระยะการอักเสบ

- Hemostasis การหยุดเลือด*** กลไกสำคัญ***
- vasoconstriction
- coagulation

- Inflammatory processes
- inflammatory cells: PMNs, mononuclear cells, macrophage (antigen presenting cell)**
- cytokines

2. Proliferation phase

- 3 mechanisms:
  Epithelialization: loss of contact > stimuli adjacent cells > basal cells enlarge and dedifferentiation > migration and mitosis > stop when contact > mitosis and differentiate (contact inhibition)
  Contraction: myofibroblast: actin containing cell ยืดหยุ่นได้ หดยืดได้ *** แผลหายเร็วจากบทบาทนี้****



- Extracellular matrix deposition (fibroplasia)
- Fibroblast proliferation: กระตุ้มการสร้างcollagen. (factory of wound healing)

Extracellular matrix

- Collagen (Protein: main component)
- Ground substances
  - Glycosaminoglycans
  - Hyaluronic acid
  - Proteoglycan

- Increase synthesis of extracellular matrix
  - Angiogenesis

Granulation tissue ←

Collagen synthesis:

- collagen fiber ถูกสร้างจาก amino acids: proline, hydroxyprolene and glycine

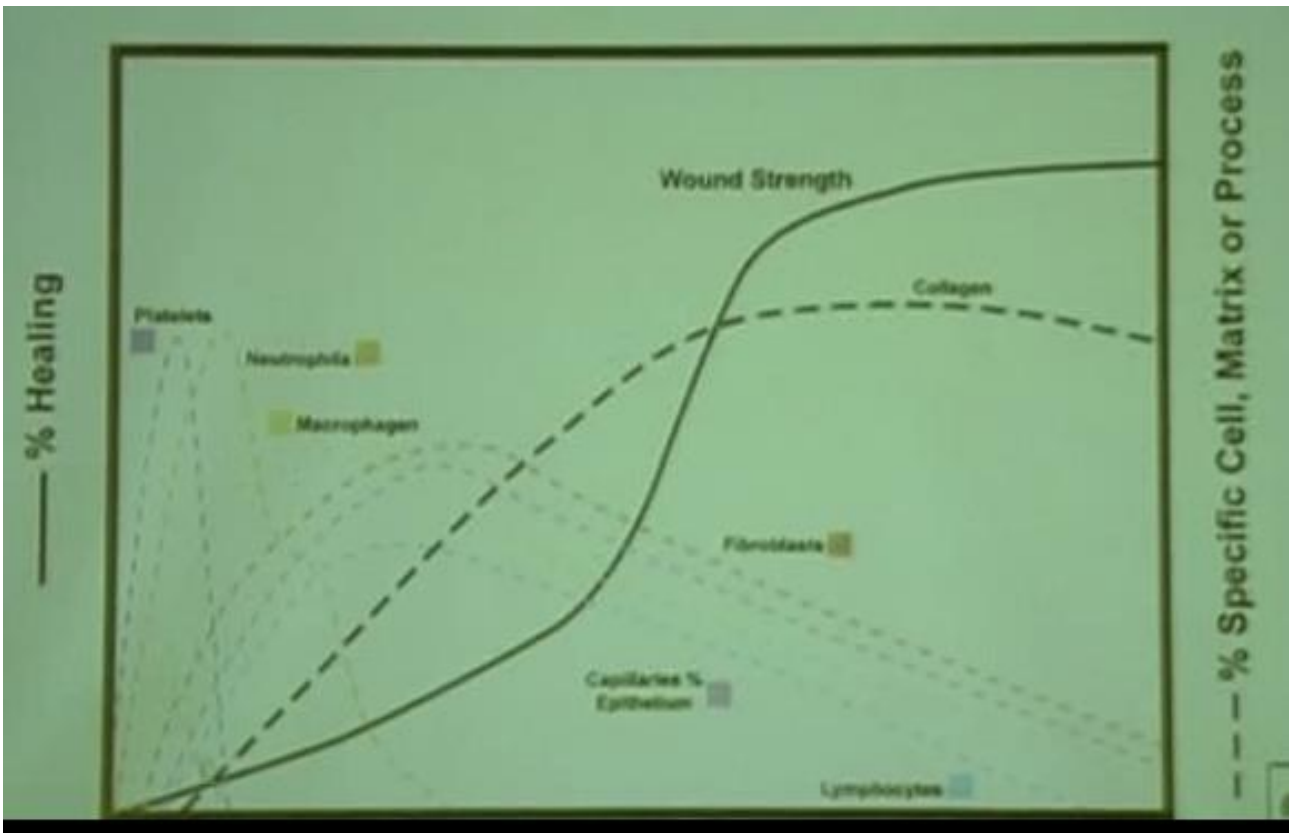
  - Amino acids จะถูกนำมาเรียงเป็นสายโปรตีนอย่างมีระเบียบ
  - สายโปรตีนดังกล่าวจะถูกสฐงให้เป็นรูปเกลียว triple-helix molecule
  - ท่อนเกลียวโปรตีนดังกล่าวจะถูกนำมาเชื่อมและเรียงตัวใหม่
  - กลไกทั้งหมดเกิดขึ้นทั้งภายในและภายนอกเซลล์

- Impact factors to collagen synthesis
  Cytokines eg. TGF-B, FGF
  Specific enzymes e.g. propel hydroxyls, Lysil oxidase
  Vitamin C
  Copper
  Oxygen

3. Remodeling phase (Maturation) : Balance of collagen synthesis, deposition, maturation and degradation (degradation : Metrix-metaloprotease (MMPs) : collagenase)

- Establish vascular network
- กลไกนี้เป็น Dynamic process เป็นการเปลี่ยนแปลงไปเรื่อยๆจนกระทั่งเข้าสู่สมดุลย์ คือแผลหายสนิทกลายเป็น “แผลเป็น”

Time-lapse in wound healing
Wound healing เป็น dynamic process แต่ละ phase จะคาบเกี่ยวกันอยู่ไม่สามารถแบ่งแยกได้อย่างชัดเจน
Inflammatory phase : 1-3 days
Proliferative phase : 2-3 days up to 3 weeks
Remodeling phase : 3 weeks up to year(s)



Wound tensile strength (Skin)

Apply graph to clinical use:

When we'll remove sutures?

- When tensile strength more than breaking strength (depends on activity)

  No need for wound holding materials

  Sutures/staples removal

Wound healing and wound care

Scope:

- Tissue injury and response
- Wound healing phases
- Modes of wound healing
- Abnormal wound healing
- Wound care
- Chronic ulcers

1. Tissue injury and response

- Hemostasis

  Vasoconstriction response

  Platelet response

  Biochemical response

- Tissue repair (wound healing phase)

2. Wound healing phases

- Inflammatory phase (Day 0-6)

  clot formation

  chemotaxis

  defense response (Neutrophils, macrophage)

  - Hemostasis (scab formation)
  - PMN
  - Macrophage

- Proliferative phase (Day 4-14)

  Angiogenesis

  Epithelialization

  Fibroplasia and granulation tissue formation

  Collagen deposition

  Wound contraction

- Maturation and remodeling phase (Day 8 - Year 1)

  Increase tensile strength

  Decrease vascularity

  Downsize of scar

3. Modes of wound healing

- Primary healing (suture wound)

  For minimal tissue loss

  Edges was approximated

  Minimal scar

- Secondary healing (ปล่อยให้แผลหายเอง)

  delayed process (Granulation, contraction, epithelialization)

  large scar results

- Tertiary (delay primary) healing

  for infected wound and foreign body

  require 3-5 cleansing days before delayed closure

- Skin graft (หายแผลหายเร็วขึ้น, ป้องกันการเกิดinfection)
- Flaps

4. Abnormal wound healing

- Hypergranulation
- Hypertrophic scar
- Keloid (นูนพ้นขอบแผล)
- Contracture

Hypertrophic scars and keloids: Abnormal wound healing > abnormal scar

Incidence

- True incidence : unknown
- Dark skin > white skin
- All age groups: More common in 10-30 years old
- Common sites : ear lobes, deltoid areas, sternum areas, Back
- Less common sites : eye lids, perineum, palms and soles.

Etiology

- Unknown
- Fields of study:

  Cytokines, growth factors and inflammatory mediators such as TGF-beta, CTGF, PDGF, IGF-1, VEGF, ECGF, PAI-1, PGE2

  Keloid fibroblast metabolic activity

  Mechanical strain and focal adhesion complexes

  Aberrant anabolic wound healing processes

  Abnormal regulation of apoptosis secondary to gene mutations such as p53, p63, p73

  Keloid epithelial-mesenchymal signaling.

Hypertrophic scars vs Keloid scars

- Natural history
- Management

Hypertrophic scars and keloids: epidemiological, clinical and histological differences.

	Hypertrophic scarring	**Keloids**
Incidence	40% to 70% following surgery, up to 91% following burn injury	6% to 16% In African populations
	Equal in sex distribution with highest incidence in the second to third decade	
Predilection sites	Shoulders, neck, presternum, knees and ankles	Anterior chest, shoulders, earlobes, upper arms and cheeks
	Less affected: eyelids, cornea, palms, mucous membranes, genitalia and soles	
Time course	Within 4 to 8 weeks following wounding, rapid growth phase for up to 6 months, then regression over a period of a few years	Within years after minor injuries or spontaneous formation on the midchest in the absence of any known injury. Persistence for long periods of time. No spontaneous regression
	Low recurrence rates after excision of the original hypertrophic scar	High recurrence rates following excision
Appearance	Do not extend beyond the initial site of injury	Projects beyond the original wound margins
Histological characteristics	Primarily fine, well-organized, wavy type III collagen bundles oriented parallel to epidermis surface with abundant nodules containing myofibroblasts and plentiful acidic mucopolysaccharide. Proliferating cell nuclear antigen (PCNA)/p53-level/ATP expression low	Disorganized, large, thick, type I and III hypocellular collagen bundles with no nodules or excess myofibroblasts. Poor vascularization with widely scattered dilated blood vessels. PCNA/p53-level/ATP expression high

Reference: Clinical, Histology, Biochemical

Hypertrophic Scarring and Keloids: Pathomechanisms and Current and Emerging Treatment Strategies

Gerd G Gauglitz,1,2 Hans C Korting,1 Tatiana Pavicic,1 Thomas Ruzicka,1 and Marc G Jeschke2,3,4,5 Mol Med. 2011 Jan-Feb; 17(1-2): 113–125

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	Hypertrophic scars	Keloid
Borders	Remains within ongoing wound	Grows beyond original wound
Onset	Often develops weeks after surgery	May develop months following injury
Contractures	Present	Absent
Regression	Often partial within 1-2 years	Infrequent
Pruritus/erythema	Present	Present
Extent of scar	Related to initial depth of tissue injury	Can far surpass initial extent tissue injury
Response to surgery	Well, especially with adjunctive therapy	Poor, often worsening

Hypertrophic scars	Keloid scars
Spontaneous regression	Less regression, sometime progression
Response well to treatments	Poor response to treatments
	High recurrence rate
	Multimodality therapy

Management

Prevention

- Avoid injury or incision in keloid-prone persons
- Avoid incision in keloid-prone areas
- Elective incision along relax skin tension lines
- Meticulous surgical techniques
- Good wound care : rapid healing

Treatment

- Surgical excision
- Radiation
- Pressure garment
- Intralesional steroid injections
- Cryotherapy
- Silicone gel dressing

5. Wound care

- General care
- Specific care

- Wound debridement
  A. Surgical (sharp) debridement (Fast&easy, painful, blood loss, surrounding tissue injury)
  B. Mechanical debridement (wet-to-dry dressing, osmotic debridement, pulsatile pressure cleansing, negative pressure therapy (NPT).)
  C. Enzymatic debridement e.g. collagenase > iruxol
  D. Biological debridement (Maggot therapy: apply for local chronic infection, osteitis, burns, acute infections)
  E. Autolytic debridement (hydrocolloids, foams, calcium algenate, hydrofibers, hydrogels)
     - less painful but slower than surgery
     - moist environment
     - endogenous autolytic enzymes
     - macrophage

Hydrocolloids: gel-forming agent (DuoDERM, Cutinova hydro, Urgotul)

- Low to moderate exudate wound (แบ่งเป็น 4 quadrants)
- Clean granulating superficial wound
- Change dressing every 3-7 days
- Cons - surround skin maceration
  - malodor

Foams (Allervyn, Urgocell, Lyofoam, Biatain)

- polyurethane sheet covered by non-adherent hydrophilic semi-permeable membrane
- Absorb and collect exudate in vertical plane
- ดีใน exudate เยอะ
- Less malodor than hydrocolloids

Calcium algenate (Urgosorb, Kaltostat, Sorbsan, Seasorb)

- natural polysaccharides of brown seaweed
- Manuronic acid (autolytic debridement) and guluronic acid (fiber integrity)
- ดีใน exudate เยอะ (high absorption capacity: moisture, hemostasis)

Hydrofibers (Aquacel)

- non-woven CMC spun
- High absorption activity
- ดีใน exudate เยอะ
- No hemostasis effects
- Change dressing every 7 days

Hydrogels (intrasite gel, duodermgel)

- insoluble polymer colloid for
  Low to moderate exudate wounds
  Non-infected slough and necrotic wounds
- change dressing every 1-3 days

- Exudate control

- Infection management
- Wound debridement
- Systemic antibiotics
- Exudate management
- Topical antimicrobials (bactigras, silver sulfadiazine cream, nano crystalline silver dressing; acticoat, urgotul SSD, aquacel Ag)

6. Chronic ulcers

- Venous ulcers
- Ischemic ulcers
- Neuropathic ulcers
- Diabetic ulcers
- Infectious ulcers
- Hematologic ulcers
- Malignant ulcers
- Miscellaneous ulcers

Venous ulcers

- most common leg ulcer (up to 80-90%)
- Pathophysiology
  Gravitational reflux (valve ไม่ดี)
  Compartment pressure
  Leukocyte trapping and activation
- Wound characteristics
  Sloping edge with well granulation
  Typical site at Gaiter area
  Dermatitis
  Eczema
  Hyperpigmentation
  Lipodermatosclerosis

Ischemic ulcers

- Atherosclerotic risk factors: smoking, DM, HT, dyslipidemia
- History of intermittent claudication, rest pain, non healing ulcer or tissue gangrene
- Diminish or absent ankle pulses
- Typical sites
- Tip of toes

  Soles

  Heels

  1st, 2nd, and 5th head of metatarsal bone

  Lateral malleolus

  Pre-tibial area

Neuropathic ulcers

- Pathophysiology

  Motor neuropathy (deformity: in normal population extend IP joint, flex MTP joint)

  Sensory neuropathy

  Autonomic neuropathy

Pressure ulcers:

- Def: injury caused by unrelieved pressure that damages the skin and underlying tissue.
- Definition by European Pressure Ulcer Advisory Panel (EPUAP)

  A pressure ulcer is lcoalized injury to the skin and/or underlying tissue usually over a bony prominence, as a result of pressure, or pressure in combination with shear and/or friction.

Etiology

- The pressure ischemia theory maintains that pressure sores result from constant pressure sufficient to impair local blood flow to soft tissue for an extended period.
- The external pressure must be greater than arterial capillary pressure of 32mmHG to impair inflow and greater than venous capillary closing pressure of 8-12 mmHg to impede the return of flow for an extended time.
- Constant external pressure for 2 hours or more produces irreversible changes in tissues in animal model studies.
- One study demonstrated no histologic changes with pressure release at 5-minute intervals.

TABLE 98.1

NATIONAL PRESSURE ULCER ADVISORY PANEL STAGING SYSTEM, 2007

STAGE	DESCRIPTION
I	Intact skin with non-blanchable redness of a localized area
II	Partial-thickness loss of dermis presenting as a shallow open ulcer with a red pink wound bed
III	Full-thickness tissue loss. Subcutaneous fat may be visible but bone, tendon, and muscle are not exposed
IV	Full-thickness tissue loss with exposed bone, tendon, or muscle

Data from National Pressure Ulcer Advisory Panel. NPUAP Pressure Ulcer Stages/Categories. http://www.npuap.org/resources/educational-and-clinical-resources/npuap-pressure-ulcer-stagescategories/

Classifications of pressure ulcers.

Grabb and Smith's Plastic Surgery7th edition. "Pressure sore",

Factor increase the risk for pressure ulcers:

- Being bedridden or in a wheelchair
- Fragile skin
- Having a chronic condition, such as DM or vascular disease, the prevents areas of the body from receiving proper blood flow.
- Inability to move certain parts of your body without assistance, such as after spinal or brain injury or if you have a neuromuscular disease (eg multiple sclerosis).
- Malnourishment
- Mental disability from conditions such as Alzheimer's disease — the patient may not be able to properly prevent or treat pressure ulcers.
- Older age
- Urinary incontinence or bowel incontinence.

Prevention:

- ตรวจดูลักษณะบริเวณกระดูกบ่อยๆ
- การจัดท่า
- การหนุนหมอน

- การพลิกตัว
- การทำความสะอาด
- ใช้อุปกรณ์เพื่อลดแรงกดทับ

Treatment:

- Conservation(stage 1,2) vs surgical (Stage 3,4)

Conservative

- Remove all necrotic tissue
- Topical antibiotics
- Negative pressure dressing

Stage 1:

- ใช้อุปกรณ์ช่วยลดแรงกดทับรองบริเวณที่พบ
- พลิกตะแคงตัวทุก 2 ชั่วโมง
- จัดท่าที่ถูกต้องดูแลผิวหนังให้สะอาด
- ไม่นวดบริเวณรอยแดง
- ใช้วัสดุปิดแผลเช่น แผ่นฟิล์ม, แผ่นโฟมปิดแผล เพื่อลดแรงกดและป้องกันการเสียดสี

Stage 2:

- ทำแผลด้วย NSS
- ปิดแผลด้วย gauze or hydrocolloid or foam dressing

Stage 3,4:

- ทำแผลด้วย NSS
- ปิดแผลด้วย gauze วันละ 1-2 ครั้ง
- Dressing with hydrocolloid, Foam, or Silver

Pre-operative patient evaluation

- Physical status
- Coexisting medical problems
- Nutritional status must be optimized (albumin level >3.5 g/ml) to reduce risk of adverse outcome.
- Proper support network and pressure- release bed at home.
- Compliant with nonoperative measures.
- The patient’s social situation.

Wound evaluation

- Wound infections - wound reconstruction can be considered once the bacterial load has been minimized to fewer than 100,000 organisms to reduce the risk of infectious complications.
- Osteomyelitis
- Significant fecal soiling into the pressure sore
- Urethral fistulas

Surgical principle for pressure ulcer

- Adequate debridement : bursectomy, osteotomy.
- Obliterate spaces
- Provide cushion good soft tissue paddle and coverage.

Adequate debridement (Bursectomy):

- Debridement of a pressure sore that will be reconstructed is different from debridement of a pressure sore that will be treated conservatively; allowed to heal by secondary intention.)
- A radical bursectomy is performed by placing a methylene blue-moistened sponge in the bursa and exercising the pressure sore circumferentially, removing all granulation tissue, even from the wound base.

Obliterate spaces & provide good soft tissue paddle.

- After the bursectomy, primary closure of the pressure sore is almost always under tension and is doomed to fail.