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Research Article

WOUND HEALING- CONCERNED FOR PROSTHODONTICS

Mrudula AG., Rao BL., Satyanarayana TSV., Aditya K., Sravanthi TLG and Padmaja B

Department of Prosthodontics, Lenora Institute of Dental Sciences, NTR University, Andhra Pradesh

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A B S T R A C T

Wound healing is still a difficult clinical problem and wound treatment must be done correctly and efficiently. Wound care has received a lot of attention, with a focus on innovative therapeutic techniques and technology development for acute and chronic wound management. Multiple cell types, the extracellular matrix, and soluble mediators including growth factors and cytokines all play a role in wound healing. Although healing is a continuous process, it can be separated into four phases: (i) coagulation & hemostasis (ii) inflammation; (iii) proliferation and (iv) wound remodelling with the creation of scar tissue.

INTRODUCTION

Wounds remain a difficult clinical challenge in everyday pathology, with early and late consequences causing a high rate of morbidity and mortality.[1] Many efforts have been made to better understand the physiology of wound healing and wound care, with a focus on innovative therapeutic methods and the continued development of technology for acute and long-term wound management.[1]

Wounds have a massive social and economic impact all over the world.Their high rate of occurrence in general, as well as their rising frequency among the elderly. There are many chronic, difficult-to-heal wounds linked with diseases and anomalies that directly or indirectly result in cutaneous covering damage, such as arterial, venous, diabetic, and pressure ulcers, in addition to a huge number of acute wounds. Chronic wounds become more common as people get older. Furthermore, because of the problems that come with acute wounds, if they do not heal in a timely and orderly manner, they might become chronic wounds, which are more difficult to manage.[1]

WOUND INJURY – Is a disruption of anatomic structure and function in any body part.[2]

HEALING – Is the body response to injury in an attempt to restore normal structure and function.[2]

In undamaged skin, the epidermis (surface layer) and dermis (deeper layer) form a protective barrier against the external environment.When the barrier is broken, a regulated sequence of events is set into motion to repair the damage.[2]

Corresponding author:* **Mrudula AG
Department of Prosthodontics, Lenora Institute of Dental Sciences, NTR University, Andhra Pradesh

Healing By First Intention (Primary Union) [2]

Is defined as the wound which has following characteristics:

Clean and uninfected, surgically incised, without much loss of cells and tissue and edges of the wounds are approximated by the surgical sutures

Sequence of Events in Primary Union: [2]

1. Initial haemorrhage
2. Acute inflammatory response
3. Epithelial changes

Epithelial spurs formation (Basal cells from both the cut margins starts proliferating and migrating towards incisional space) followed by scab formation.[2]

By 5th day multi-layered new epidermis is formed which differentiated into superficial and deeper layers

4. Organisation
5. Suture tracks

Each suture track is a separate wound and incites the same phenomena as in healing of primary wound. When sutures are removed around 7th day, much of epithelialized suture track is avulsed andremaining epithelial tissue in the track is absorbed.[2]

However, sometimes the suture track gets infected (stitch abscess) or epithelial cells may persist in the track (implantation or epidermal cyst).[2]

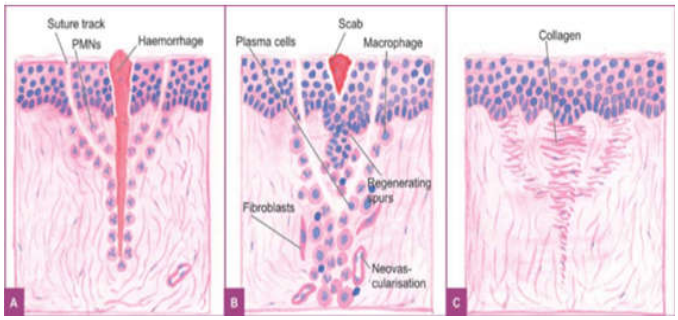

Figure 1 Primary union of skin wounds.

A. The incised wound as well as suture track on either side are filled with blood clot and there is inflammatory response from the margins.
B. Spurs of epidermal cells migrate along the incised margin on either side as well as around the suture track. Formation of granulation tissue also begins from below.
C Removal of suture at around 7th day results in scar tissue at the sites of incision and suture track.

Healing By Secondary Intention (Secondary Union)[2]

Is defined as healing of the wound having the following characteristics:

open with a large tissue defect, at times infected, having extensive loss of cells and tissues & wound which is not approximated by surgical sutures but is left open.[2]

Sequence of Events In Secondary Union.[2]

1. Initial haemorrhage
2. Inflammatory phase
3. Epithelial changes which include epithelial spurs formation and scab formation
4. Granulation tissue formation

Main bulk of secondary healing is by granulations. It is formed by proliferation of fibroblasts and neo vascularisation. Deep red, granular, fragile (newly formed granulation tissue) while pale white (on maturation).[2]

5. Wound contraction

Not seen in primary healing.Due to the action of myofibroblasts present in granulation tissue, the wound contracts to one third to one fourth of its original size.[2]

6. Presence of infection

Bacterial contamination of an open wound delays the process of healing due to release of bacterial toxins that causes necrosis, suppuration and thrombosis. Surgical removal of dead and necrosed tissue, debridement helps in preventing bacterial infection of open wounds.[2]

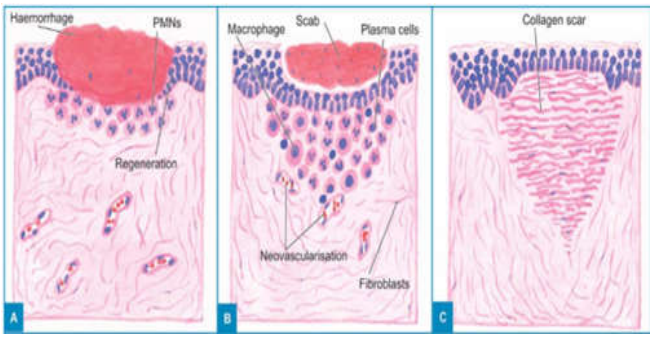

Figure 2 Secondary union of skin wounds.

- A.The open wound is filled with blood clot and there is inflammatory response at the junction of viable tissue.
- B. Epithelial spurs from the margins of wound meet in the middle to cover the gap and separate the underlying viable tissue from necrotic tissue at the surface forming scab.
- C. After contraction of the wound, a scar smaller than the original wound is left.

Difference between Primary & Secondary Union of Wounds[2]

Table 1 showing the difference between primary & secondary healing of wounds

Feature	Primary Union	Secondary Union
Cleanliness of wound	Clean	Unclean
Infection	Generally uninfected	May be infected
Margins	Surgically clean	Irregular
Sutures	Used	Not used
Healing	Scanty granulation tissue	Exuberant granulation tissue
Outcome	Neat linear scar	Contracted irregular wound
Complications	Infrequent, epidermal inclusion cyst formation	Suppuration, may require debridement

Complications of Wound Healing[2]

Infection
Implantation (epidermal cyst)
Pigmentation
Deficient scar formation
Incisional hernia
Hypertrophied scars and keloid formation
Excessive contraction
Neoplasia

Fracture Healing [3]

The process of fracture healing can occur in two ways:

Direct or primary bone healing occurs without callus formation. Indirect or secondary bone healing occurs with a callus precursor stage.[3]

Primary healing [3]

Under few special situations when the ends of fracture are approximated as done by application of compression clamps. In these cases, bony union takes place with formation of medullary callus without periosteal callus formation.[3]
Secondary healing of the bone[3]

Involves classical stages of fracture healing they are:

1. Hematoma Formation
2. Inflammation.
3. Primary soft callus formation
4. Callus mineralization
5. Callus re-modelling

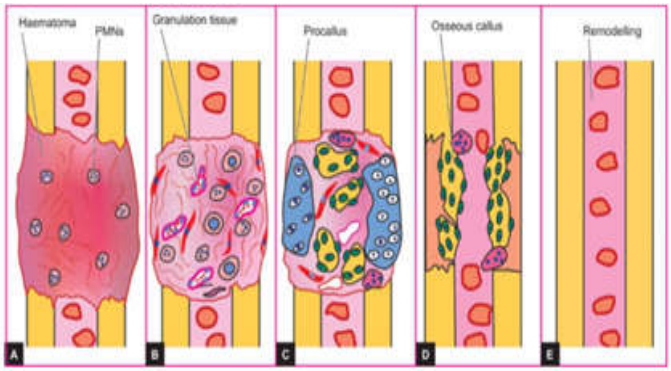

Figure 3 Fracture healing.

A. Haematoma formation and local inflammatory response at the fracture site.
B. Ingrowth of granulation tissue with formation of soft tissue callus.
C. Formation of procallus composed of woven bone and cartilage with its characteristic fusiform appearance and having 3 arbitrary components—external, intermediate and internal callus.
D. Formation of osseous callus composed of lamellar bone following clearance of woven bone and cartilage.
E. Remodelled bone ends; the external callus cleared away. Intermediate callus converted into lamellar bone and internal callus developing bone marrow cavity.

It is divided into 5 stages:
Haemorrhage and clot formation

Figure 4(a) Immediately after tooth extraction; Hemmorhage & blood clot may be seen

Organization of the clot by granulation tissue

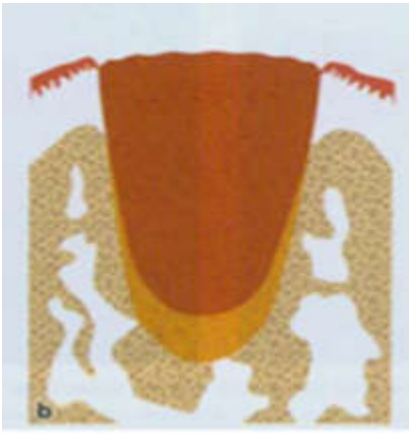

Figure 4(b) 24-48 hrs after extraction; Blood clot & beggining of granulation tissue formation may be seen

Replacement of the granulation tissue by connective tissue and epithelisation of the wound.

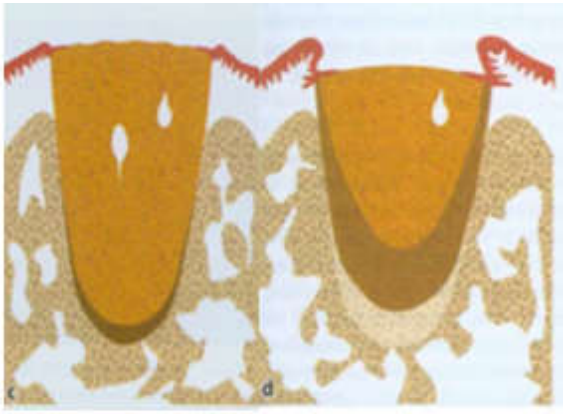

Figure 4(c) 96 hrs after extraction; Residual Blood clot , granulation tissue & epithelial proliferation may be seen
Figure 4(d) 7 days after extraction Young connective tissue, Primary osteeoid formation & epithelial proliferation may be seen Reconstruction of the alveolar process

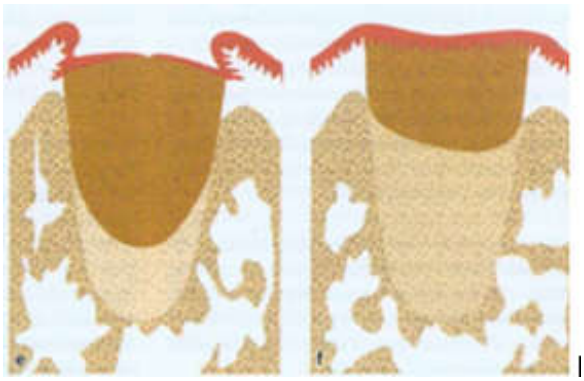

Figure 4(e) 21 days after extraction; Connective tissue may be seen, osteoid starts mineralization & re-epithelialization may be seen
Figure 4(f) 6 weeks after extraction Connective tissue, woven bone, trabeculae & re-epithelialization may be seen Replacement of the immature bone by mature bone tissue

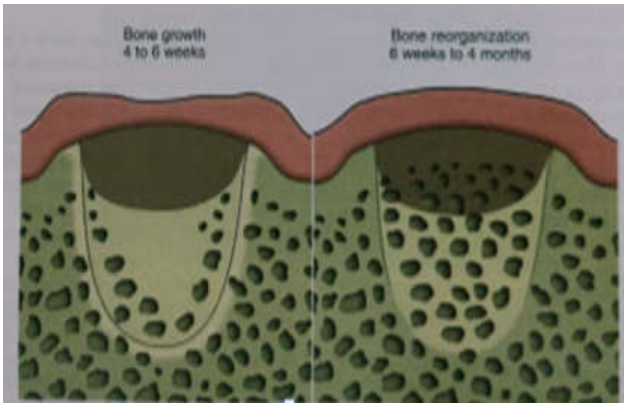

Figure 4(g) Upto 4- 6 weeks Bone growth may be seen
Figure 4(h) Upto 6 weeks – 4 months Bone regeneration may be seen

Post-operative complications of extraction socket [4]

Alveolar osteitis (dry socket)
Haemorrhage
Ecchymosis and haematoma
Swelling
Pain

Dry-socket /alveolitis sicca dolorosa / alveolar osteitis [4]

Clinical features includes-
Delayed post-operative pain of extreme intensity is of radiating type, loss of blood clot
Dirty Gray appearance of clot, Foul odour may be seen. Commonly seen in patients of 40–45 year old. Mandible more

affected than maxilla. The dry socket usually starts by 2-3rd day and was extremely painful. The diagnosis is confirmed by gently passing a probe into extraction wound, in dry socket a bare bone which is extremely sensitive and partially necrotic clot is encountered.[4]

Pathogenesis [4]
Any trauma/ infection to the teeth may cause inflammation of the bone marrow

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Tissue activators such as plasminogen may be seen

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Plasminogen converted to plasmin which causes lysis of fibrin,formation of kinins results in dissolution of the blood clot & pain

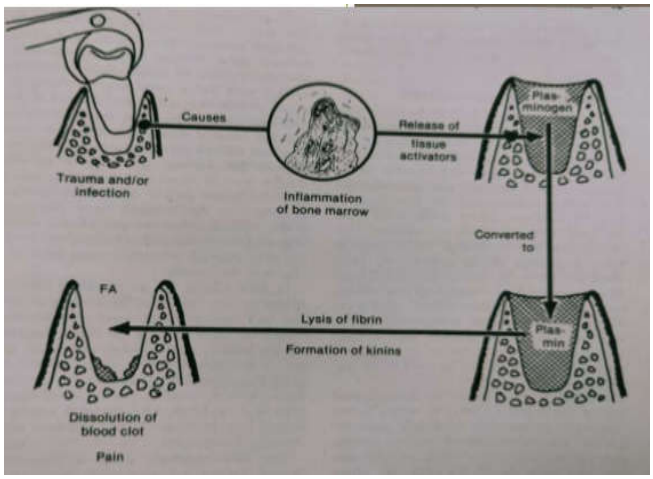


Figure 5 Pathogenesis of Dry socket

Treatment [4]
Irrigation of the socket with warm sterile isotonic solution or solution of dilute hydrogen peroxide; to remove the debris.
Application of an obtundent eg.; Eugenol or a topical anaesthetic: benzocaine followed by analgesics and antibiotics.[4]

Healing After implant placement

Accoding to Jain M et al; [5]
Good healing requires implant stability
Initial stages of implant healing and interface development occurs in 4 stages namely Hemostasis phase, Inflammatory stage, Proliferative stage & Remodeling phase

Table 2 showing stages of implant healing

Hemostasis phase	Proteins adsorption, soft tissue healing, clot formation
Inflammatory stage	Neutrophils, macrophage activation, Release of inflammatory mediators
Proliferative stage	
Early	Angiogenesis, Increase in number of fibroblasts & osteoclasts
Late	Woven bone formation
Remodelling phase	
Early	Reconstruction of woven bone by osteoclasts
Late	Immature woven bone replacement By mature lamellar bone

Stages of Osseo - integration by MISCH [6]

Misch has given below the following stages which are as follows:

Stage 1: woven callus formation: 6 weeks
Stage 2: lamellar compaction – 18 weeks
Stage 3: interface remodelling–18weeks
Stage 4: compact maturation -54 weeks

Despite the fact that oral surgical wounds heal in a similar manner, soft tissue recovery is influenced by the underlying bone tissue. Bone healing occurs in the first place when everything is done correctly & in the conditions where fractures that have been relocated and perfectly stabilised. [7]

When a bone heals with a secondary aim, it is called secondary intention healing.[7] As in extraction, the defect must be filled spontaneously.[8] Healing can be classified as either early or late stage. The healing process is similar to that of a foreign body response. In this stage, implant stability and implant surface effect material and morphology are both important. Osteogenesis allows the host to bridge the space between them.

Early stage involves the bone and the implant surface, culminating in woven bone that hasn't fully matured [8] where as Late stage bone healing entails a modification of both the host and immature bone, resulting in the production of new bone. [8] Developed lamellar bone that lasts for the rest of one's life since it is heavily impacted by mechanical factors. Three distinct systems are involved in graft healing ie; osteogenesis, osteoinduction and osteoconduction are all terms used to describe the processes of bone formation. [8]

Osteogenesis is the production of new bone from osteo competent graft cells.[8] The stimulation of recipient bed mesenchymal cells to produce bone by graft inductive proteins is known as osteoinduction.[8] When bone forms in and around the recipient graft bed, this is called osteoconduction. [8]

All types of bone healing require a fresh blood supply, and proper underlying bone repair requires total coverage by typically mending overlying soft tissue.

Monitoring of Wound Healing [9,10]

After meticulous diet and plaque debridement, wound examination is used to monitor post-surgical wound healing.[9] Wound healing monitoring also includes suture monitoring and removal following a thorough assessment of soft tissue healing progress.[9]

Sutures should be removed according to each individual situation, not after a routine 7-10 day period, because they have been shown to have an adverse effect on flap blood circulation as well as an inflammatory reaction in surrounding tissues.[10] However, early removal may result in wound margin dehiscence.[10]

Influence of Local & Systemic Factors on Wound Healing Process

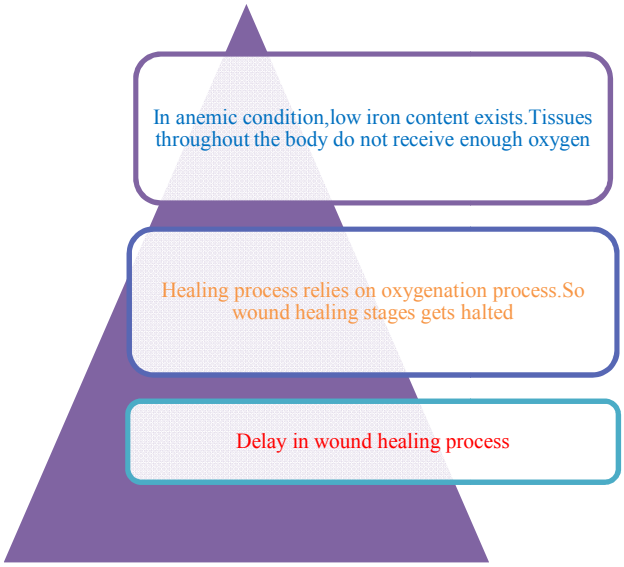
Local factors includes-
Infection
Poor blood supply
Foreign bodies
Exposure to UV light facilitates healing
Exposure to ionizing radiation

Systemic factors includes-
Age
Haematological abnormalities
Nutrition
Administration of drugs eg; Glucocorticoids
Un controlled diabetes

UN Controlled Diabetes – WOUND HEALING.[11]
Long-term hyperglycaemia leads to development of glycation in which glucose bind to proteins and other molecules leads to non - enzymatic process which results in

- neutrophil dysfunction
- diminished vascular perfusion
- progressive nerve damage all of which contribute to poor wound healing

Healing In Anaemic Condition



Micro Nutrients Involved In Wound Healing [12]

Aminoacids

Arginine

Precursor of nitrous oxide and proline. Activates T cells. Essential for synthesis of collagen. Recommended dose is 4.5g/day.[12]

Glutamate- Plays several roles in wound healing via; metabolic, enzymatic, antioxidant and immune properties and involved in inflammatory phase by regulating leukocyte apoptosis, superoxide production, antigen processing and phagocytosis & protects against risk of infectious and inflammatory complications by up regulating expression of heat shock proteins.[12]

Vitamins: [12]

VIT A - Deficiency impairs B cell and T cell function

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Impairs antibody production during inflammatory phase

↓

Decreased epithelialization, collagen synthesis, granulation tissue development.12

VIT A – counteracts delay in wound healing caused by corticosteroids by down regulating TGF- BETA, IGF – 1. In wound patients **vit A** supplementation: 10,000 to 25,000 IU / day.[12]

B – COMPLEX VITAMINS are essential co factors in enzyme reactions involved in leukocyte formation and anabolic process of wound healing. Among these **B COMPLEX VITAMINS**, Thiamine, Riboflavin, Pyridoxine, Cobalamine helps in synthesis of collagen. Hence vit B production indirectly affects wound healing process by impairing antibody production.[12]

VIT C involved in healing with several roles in cell migration and transformation, collagen synthesis, anti-oxidant response & angiogenesis. In inflammatory phase, **vit c** participates in recruitment of cells to wound and their transformation into macrophages. During collagen synthesis, **vit c** forms extra bonds between collagen fibres which increases stability and strength of collagen matrix. Recommended **vit c** supplements – 500mg/day (non-complicated wounds) 2g/day (severe wounds).[12]

VIT E negatively affects collagen synthesis, anti-oxidant response and inflammatory phase. It counteracts the benefits of vit A supplementation in wound management.[12]

Minerals: [12]

Zinc

In inflammatory phase, it promotes immune response. Zinc counteracts susceptibility to infectious complications by activating lymphocytes and producing antibodies. In proliferative and remodelling phase, zinc is essential for collagen production fibroblast proliferation and epithelialisation.[12]

Topical administration of Zn to surgical wounds significantly improves healing process. Recommended zinc supplementation for zinc deficient patients: 40 – 220mg/day for 10 to 14 days.[12]

IRON

Transports oxygen to tissues which is essential for tissue perfusion and collagen synthesis. Hence iron deficiency causes tissue ischemia, impaired collagen production and decreased wound strength in proliferative phase.[12]

CONCLUSION

Understanding of wound healing is as important as knowing the pathogenesis of disease, because satisfactory wound healing is the ultimate goal of the treatment

References

1. Velnar T, Bailey T, Smrkolj V. The Wound Healing Process: an Overview of the Cellular and Molecular Mechanisms. J Int Med Res 2009; 37: 1528 – 542.
2. Mohan H, Mohan S. Essential pathology for Dental students. 4th edition, Jaypee Brothers Medical Publishers, New Delhi 2012; 131-33.
3. Kumar, Abbas, Aster, Robbins, Cotran. Pathologic Basis of Disease Volume 1. 7th edition, Elsevier publications an imprint of Mosby, Philadelphia 2003.
4. Lindhe J, Lang NP. Clinical periodontology and implant dentistry volume 1. 5th edition, Wiley Blackwell publications, Australia 2008.

5. Jain M, Thukral H, Kukreja S, Arora G, Ray A, Arora D. Concept of healing after implant placement. World J Pharma Pharmaceutic Sci 2017;6(8): 1250-257.
6. Misch CE. Contemporary implant dentistry. 3rd edition, Elsevier publications an imprint of Mosby, St Louis Missouri 2008.
7. Sandor GKB, Carmichael RP, Ylikontiola LP. Healing of large dentofacial defects. Endod Topics 2012;25:63-94.
8. Davies JE. Understanding peri-implant endosseous healing. J Dent Educ 2003;67(8):932-49.
9. Burkhardt R, Lang NP. Role of flap tension in primary wound closure of mucoperiosteal flaps: a prospective cohort study. Clin Oral Implants Res 2010;21:10-4.
10. Atterbury RA, Vazirani SJ. Removal of sutures following oral surgery. Oral Surg Oral Med Oral Pathol 1961;14(6):658-61.
11. Shafer, Hine, Levy. Shafer's Textbook of oral pathology. 8th edition, Elsevier publication an imprint of Mosby, St Louis Missouri 2016.
12. Barchitta M, Mauyeri A, Favara G , Lio RMS, Agodi A, Basile G, Evola G. Nutrition and Wound Healing: An Overview Focusing on the Beneficial Effects of Curcumin- A Review. Int J Mol Sci 2019; 20 :1119.

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