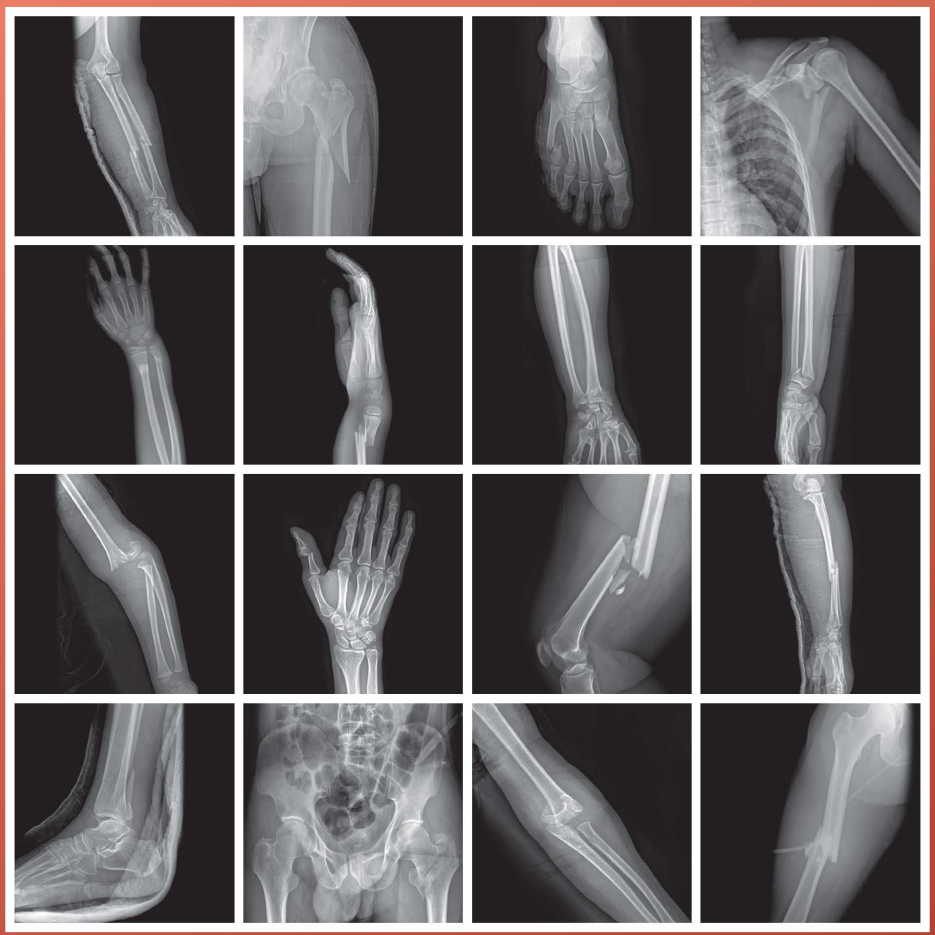
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Handbook of Fractures
The Current Concepts

Editor-in-Chief
Abdul Ghani

Editor
Samarth Mittal

Forewords
DD Tanna
Mandeep S Dhillon

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CHAPTER 1.7

Fracture Healing Process and Determinants

Amandeep Singh Bakshi

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PHASES OF FRACTURE HEALING

Fracture healing involves three primary overlapping phases:

1. *Inflammatory phase*: This phase begins immediately after the fracture. Hematoma formation at the fracture site acts as a scaffold for the influx of inflammatory cells, including neutrophils, macrophages, and lymphocytes. These cells release proinflammatory cytokines [e.g., interleukin-1 (IL-1), IL-6, and tumor necrosis factor-alpha (TNF-α)] that promote angiogenesis and recruit mesenchymal stem cells (MSCs). The inflammatory response is essential for clearing necrotic tissue and setting the stage for subsequent repair processes. Clinically, this phase is characterized by pain, swelling, and reduced mobility.
2. *Reparative phase*: During this phase, soft callus forms as chondrocytes and fibroblasts increase, creating a cartilaginous matrix that bridges the fracture gap. This phase progresses to hard callus formation through endochondral ossification, where the soft callus is gradually replaced by woven bone. The timing and quality of callus formation depend heavily on mechanical stability. In clinical practice, this phase is when radiographic evidence of callus formation becomes apparent, indicating progression toward healing.
3. *Remodeling phase*: The final phase involves the gradual replacement of immature woven bone with mature lamellar bone, aligning its structure according to Wolff's law. Osteoclasts resorb poorly organized bone, while osteoblasts lay down new bone along lines of mechanical stress. The remodeling phase is critical for restoring the bone's original shape, strength, and function. It can continue for months to years, depending on patient-specific factors and the nature of the fracture **(Figs. 1 and 2)**.

FACTORS INFLUENCING FRACTURE HEALING

Biological Factors

- *Age*: Pediatric patients have a remarkable capacity for bone healing due to the abundance of stem cells and growth factors, while older patients may experience delayed healing due to reduced cellular activity, impaired angiogenesis, and a diminished inflammatory response. Orthopedic strategies may include more aggressive fixation and pharmacological support in older patients.
- *Nutrition*: Adequate intake of calcium, phosphorus, and essential vitamins (D, C, and K) is paramount. Vitamin D enhances calcium absorption and bone mineralization, while vitamin K

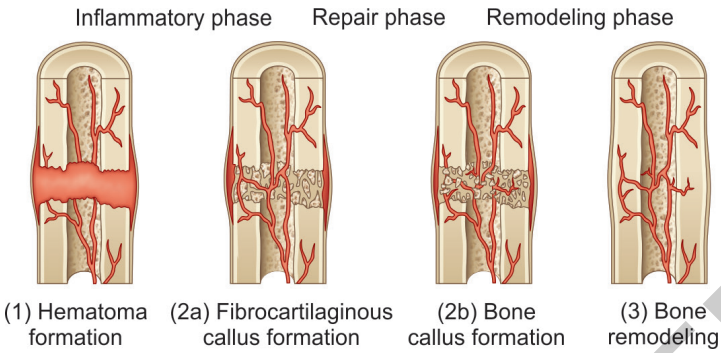


FIG. 1: Stages of secondary bone healing (endochondral ossification). The stages of fracture healing are partially overlapping. (1) Coagulation and acute inflammatory response—initial stabilization, recruitment of inflammatory cells and cytokines. (2) Repair—(a) Revascularization, soft cartilage callus. Chondroblasts derived from MSCs deposit cartilage, mature to chondrocytes that express VEGF, inducing neovascularization; (b) Hard bony callus—hyperproliferative chondrocytes transdifferentiate to osteoblasts and osteocytes, tissue invaded by osteoblasts; (3) Remodeling—deposition of lamellar bone and restoration of preinjury anatomic dimensions.

supports the carboxylation of osteocalcin, crucial for binding calcium in bone tissue. Malnutrition or deficiencies may necessitate dietary supplementation or intravenous administration in cases of severe deficits.

- *Hormonal influence*: Estrogen plays a key role in maintaining bone homeostasis by inhibiting osteoclast activity. Postmenopausal women, who are estrogen-deficient, are at increased risk for delayed union or nonunion. Parathyroid hormone (PTH), particularly in its intermittent form, has an anabolic effect on bone and may be considered as a therapeutic agent to enhance fracture healing in selected patients.
- *Genetic factors*: Genetic predispositions can influence bone density, healing speed, and fracture risk. Understanding these factors can guide personalized treatment strategies.

Mechanical Factors

- *Fracture stability*: Fracture healing is highly dependent on mechanical stability. Absolute stability, achieved through methods such as plate fixation, favors primary bone healing, characterized by direct remodeling without callus formation. Relative stability, obtained through intramedullary nailing or external fixation, promotes secondary healing with visible callus formation. The choice of fixation technique should balance stability needs against the biological environment of the fracture.
- *Mechanical loading*: Weight-bearing and mechanical stimulation are critical modulators of bone healing. Controlled mechanical loading stimulates osteogenic activity via mechano-transduction pathways, enhancing callus formation and maturation. However, excessive or premature loading can lead to nonunion or implant failure. Therefore, the postoperative rehabilitation protocols must be carefully tailored to optimize these mechanical forces.

Systemic and Environmental Factors

- *Smoking*: Nicotine and other toxins found in cigarettes have been shown to reduce perfusion, impair osteoblast function, and decrease collagen synthesis, leading to delayed union or nonunion. Smoking

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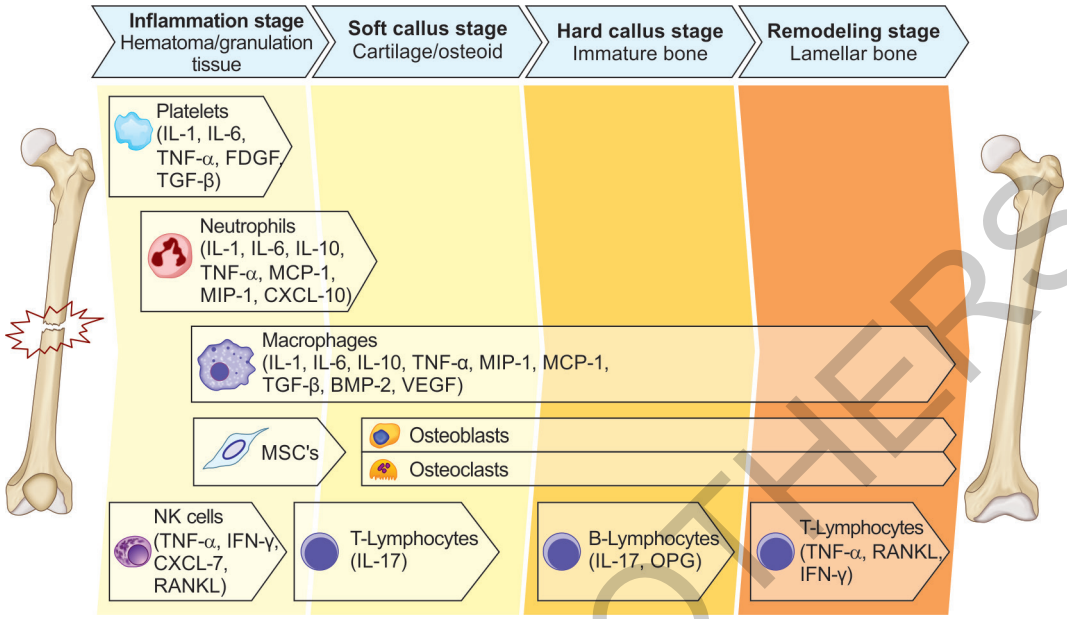


FIG. 2: Immune cells of the fracture healing cascade for each stage of bone healing. The cellular contributions to fracture healing are described. Starting with platelets, a cascade of events that transition the bone from an inflammatory period to a proliferative period noted by soft and hard callus formation and predominated by reparative cells such as osteoblasts and osteoclasts. This phase then transitions to the remodeling phase where the healing site is structurally reinforced. Macrophage, being present throughout the healing cascade, release NO and play an important role throughout all stages of repair. Each stage of bone healing has a different spectrum of immune cells present at the fracture site.

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cessation should be strongly advocated in patients undergoing fracture treatment.

- *Medications*: Nonsteroidal anti-inflammatory drugs (NSAIDs), commonly used for analgesia, inhibit prostaglandin synthesis, which is crucial for the inflammatory phase of bone healing. Chronic use of corticosteroids can suppress osteoblast differentiation and activity. Conversely, medications like teriparatide (a PTH analog) and bisphosphonates can enhance fracture healing in specific contexts.
- *Comorbid conditions*: Diabetes, osteoporosis, rheumatoid arthritis, and chronic kidney disease are examples of conditions that can adversely affect fracture healing. Diabetic patients, for instance, often exhibit impaired angiogenesis and collagen production, necessitating close monitoring and potentially more aggressive therapeutic interventions.

MOLECULAR AND CELLULAR MECHANISMS IN FRACTURE HEALING

Fracture healing is orchestrated by a complex interplay of growth factors, cytokines, and signaling pathways. Key elements include:

- *Bone morphogenetic proteins (BMPs)*: Particularly BMP-2 and BMP-7, these are potent osteoinductive agents that promote MSC differentiation into osteoblasts and chondrocytes. Clinically, BMPs are used to enhance healing in

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challenging cases, such as nonunions and critical-sized defects.

- *Transforming growth factor-beta (TGF-β)*: It stimulates the recruitment of MSCs and their differentiation into bone-forming cells. It also plays a crucial role in modulating the inflammatory response. Understanding TGF-β signaling can help develop targeted therapies to optimize fracture healing.
- *Vascular endothelial growth factor (VEGF)*: It is crucial for angiogenesis, a process vital for providing nutrients and removing waste products from the fracture site. Enhanced VEGF expression has been linked to improved healing, and therapeutic angiogenesis may represent a future treatment avenue.

COMPLICATIONS IN FRACTURE HEALING

Orthopedic surgeons must recognize and address complications such as delayed union, nonunion, and malunion:

- *Delayed union*: Defined as slower-than-expected healing. Early identification through clinical and radiological assessment allows for timely intervention, which may include bone grafting, dynamization, or the use of biologics.
- *Nonunion*: Classified as atrophic (biologically inactive) or hypertrophic (biologically active but mechanically unstable). Management strategies depend on the type and may involve restabilization, bone grafting, or the use of BMPs or MSCs.
- *Malunion*: Occurs when the fracture heals in a misaligned position. Corrective osteotomy may be necessary, especially in cases where the malalignment affects function or causes significant pain.

ADVANCED STRATEGIES TO ENHANCE FRACTURE HEALING

- *Pharmacological interventions*: Anabolic agents like teriparatide have been shown to accelerate healing in fractures at high risk of delayed union or nonunion. BMPs and growth factor therapies are becoming more mainstream, especially in complex or nonhealing fractures.
- *Biophysical stimulation*: Techniques such as low-intensity pulsed ultrasound (LIPUS) and electrical stimulation have shown promise in enhancing fracture healing, particularly in cases of delayed union. These modalities stimulate cellular responses that promote bone formation.
- *Stem cell therapy and tissue engineering*: The use of MSCs, combined with scaffolds and growth factors, is an emerging field aimed at improving outcomes in complex fractures. Ongoing research into the optimal cell types, delivery methods, and combination therapies holds promise for future advancements.

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Handbook of
Fractures
The Current Concepts

The *"Handbook of Fractures: The Current Concepts"* is a valuable resource for orthopedic surgeons, residents, and trauma specialists, presenting the latest in fracture care across a comprehensive spectrum. This book is designed for quick reference and combines foundational knowledge with up-to-date, practical insights for effective patient management.

- Quick & Easy guide to streamline fracture assessment and treatment
- Concise review on fixation options
- Rich visual support with clear illustrations and radiographs to highlight key anatomy and fracture characteristics

This book is a ready reckoner to mastering common and complex fracture care. It shall equip the reader to deliver better outcomes of fracture treatment.

Abdul Ghani, the chief editor of this book, did his MS (Orthopedics) from AIIMS, New Delhi, and then went to the United Kingdom, where he worked for more than 5 years and did MRCS from Edinburgh. He is Professor and Head of Unit, Department of Orthopedics, Government Medical College, Jammu & Kashmir, India. He is also the convenor of the Jammu Trauma Society, BLS and Emergency Trauma Course. With extensive expertise in orthopedic trauma and fracture management, Dr Ghani is deeply involved in medical education, contributing valuable insights into contemporary approaches to fracture care. This book reflects his commitment to sharing cutting-edge practices with aspiring and seasoned orthopedic professionals. He has already authored various books, namely (1) Musculoskeletal Pain Management, (2) Secret of Scoring More Marks and Passing Exams Easily, (3) How to Unleash Your Potential and Achieve Success: The Key Soft Skills, and (4) Competency-based Logbook and Clinical Manual in Orthopaedics for MBBS students.

Samarth Mittal did his MS (Orthopedics) from the prestigious AIIMS, New Delhi, and completed fellowships at various places, including St. Mary Hospital (Hong Kong), Aberdeen Royal Infirmary (Scotland), Nuffield Orthopaedic Centre (Oxford, UK), St. Vincent Hospital (Indianapolis, USA), Schulthess Klinik (Zürich, Switzerland), and AO Trauma fellowship at Memorial Hermann in Texas Medical Center, USA. Currently, he is working as an Additional Professor at JPN Apex Trauma Centre, AIIMS, New Delhi. With more than 100 indexed publications, over 10 book chapters, and over 250 guest lectures, he is an Advanced Trauma Life Support (ATLS) instructor and a regular faculty at AO Trauma courses, both national and international. With teaching and research at the forefront, in addition to numerous publications, he is also a reviewer for many international and national journals and also has an editorial role in the Indian Journal of Orthopaedics (IJO), Journal of Orthopaedics (JOO), and Journal of Clinical Orthopaedics and Trauma (JCOT).

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