

Healing of Fractures

Hematoma → callus → woven bone → remodeling

Pathology of skeletal repair

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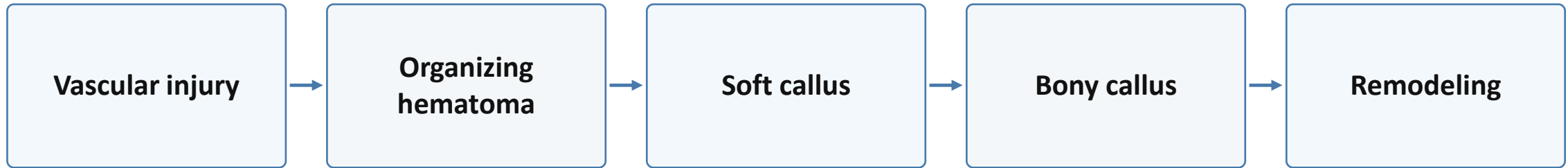
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Learning objectives

- Explain the orderly transitions from hematoma to soft callus, bony callus, and lamellar bone.
- Identify the contributions of vessels, osteoprogenitors, osteoblasts, osteoclasts, and chondrocytes.
- Relate the mechanical environment to callus maturation and remodeling.
- Predict how displacement, instability, infection, neoplasia, and comorbidities disrupt union.

The repair sequence



Repair begins with a fibrin-rich provisional scaffold.

Callus bridges the fracture before it can provide full rigidity.

Woven bone is ultimately reshaped into lamellar bone.

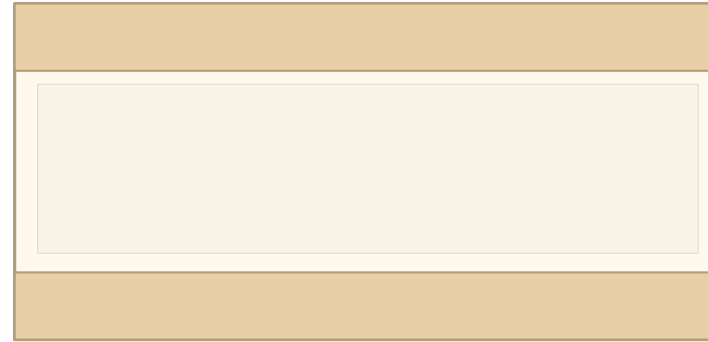
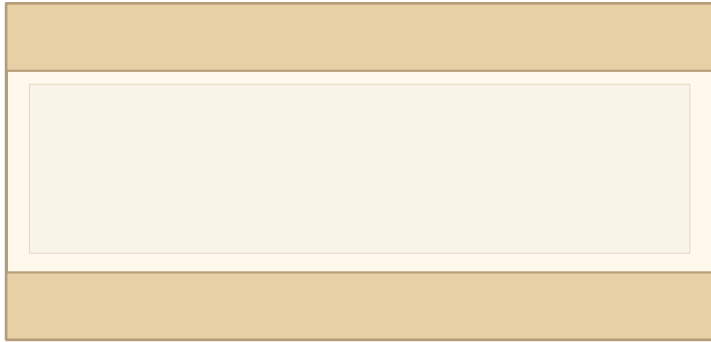
Bone can restore its original architecture

- Fracture repair is a regenerative sequence with production and removal of bone matrix.
- Adequate immobilization supports progression from a weak bridge to weight-bearing strength.
- In uncomplicated simple fractures without major comorbidity, near-perfect union is usual.

Restoration of the medullary cavity marks completion of the process.

Repair occurs across cortex and marrow

Simplified long-bone cross-section at a fracture site



Schematic

Peripheral cortex surrounds the central medullary compartment.

The periosteal and medullary regions both contribute repair cells.

Core cellular functions in skeletal repair

Osteoblast

Produce osteoid matrix

Promote matrix mineralization

Osteoclast

Resorb bone matrix

Remove excess callus

Chondrocyte

Produce cartilage

Enable endochondral bone formation

Cells are coordinated—not interchangeable—during repair.

Woven bone and lamellar bone

Woven bone

- Deposited rapidly during fracture repair
- Haphazard collagen arrangement
- Lower structural integrity

Lamellar bone

- Ordered, parallel collagen fibers
- Forms during maturation/remodeling
- Restores organized mechanical architecture

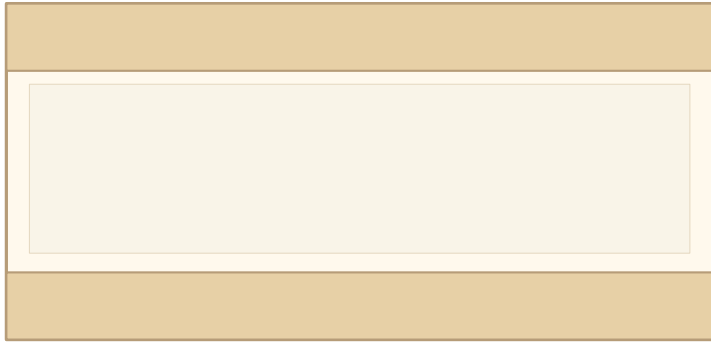
The conversion from woven to lamellar bone is a defining maturation step.

Fracture descriptors affect the healing context

Descriptor	Definition	Healing relevance
Simple	Overlying skin intact	Favorable if aligned and immobilized
Compound (open)	Communicates with skin	Greater concern for infection
Displaced	Ends are misaligned	May heal with deformity
Comminuted	Multiple bone fragments	Often complicates restoration
Pathologic	Bone weakened by disease	Abnormal substrate may inhibit healing

Displacement means loss of fragment alignment

A fracture with misaligned ends changes the geometry of the repair bridge.



Schematic

Displaced fractures frequently leave residual deformity.
Fracture alignment and callus maturation are related but distinct issues.

Comminution increases the geometric challenge

- Comminuted fractures comprise multiple fragments rather than a single break.
- The eventual repair must consolidate fragmented segments into one load-bearing structure.
- Displaced and comminuted fractures as frequent causes of residual deformity.

Closed and open fractures

Simple (closed)

- Skin remains intact
- No fracture–skin communication by definition
- Usual healing requires adequate stability

Compound (open)

- Fracture communicates with the skin
- Fracture-site infection is a major complication
- Infection is a serious obstacle to union

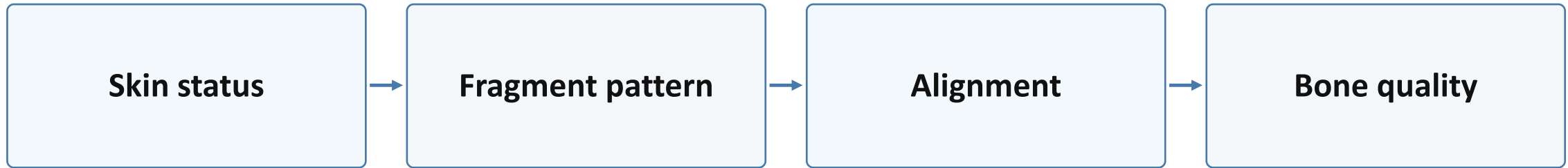
Stress and greenstick fractures: Different patterns

- A stress fracture develops gradually after repetitive loading associated with increased activity.
- A greenstick fracture extends only partway through bone.
- Greenstick fractures are frequent in young children because their bones are relatively soft.
- These descriptors concern fracture configuration—not a separate molecular repair program.

A pathologic fracture begins in diseased bone

- The fracture occurs in bone weakened by an underlying disorder, including neoplasm.
- Primary and metastatic bone tumors can inhibit otherwise normal repair.
- Defining the bone substrate matters in addition to recognizing the fracture line.

Fracture classification is multidimensional



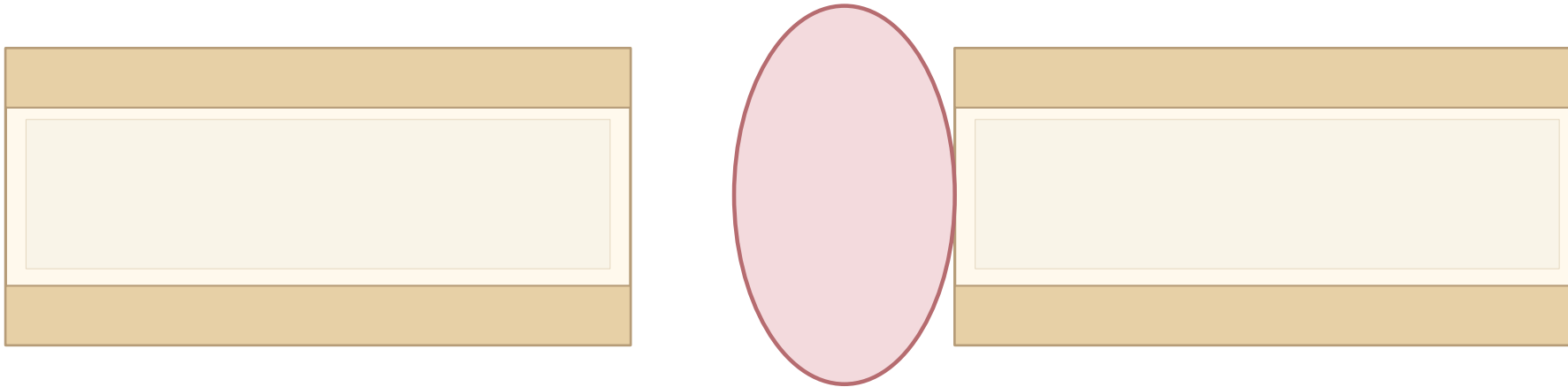
Simple and compound describe skin communication.

Comminuted and greenstick describe the break pattern.

Displaced describes alignment; Pathologic describes diseased substrate.

Immediate response: Vascular rupture

Injury tears local vessels and causes bleeding into and around the break.

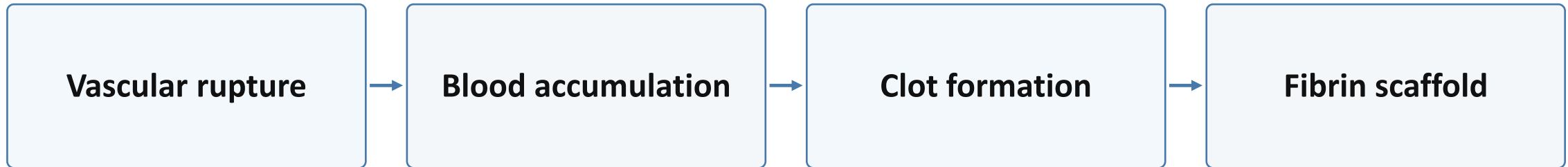


Schematic

Blood accumulates within and around the fracture site.

A hematoma becomes the earliest visible repair environment.

Blood becomes a provisional repair matrix



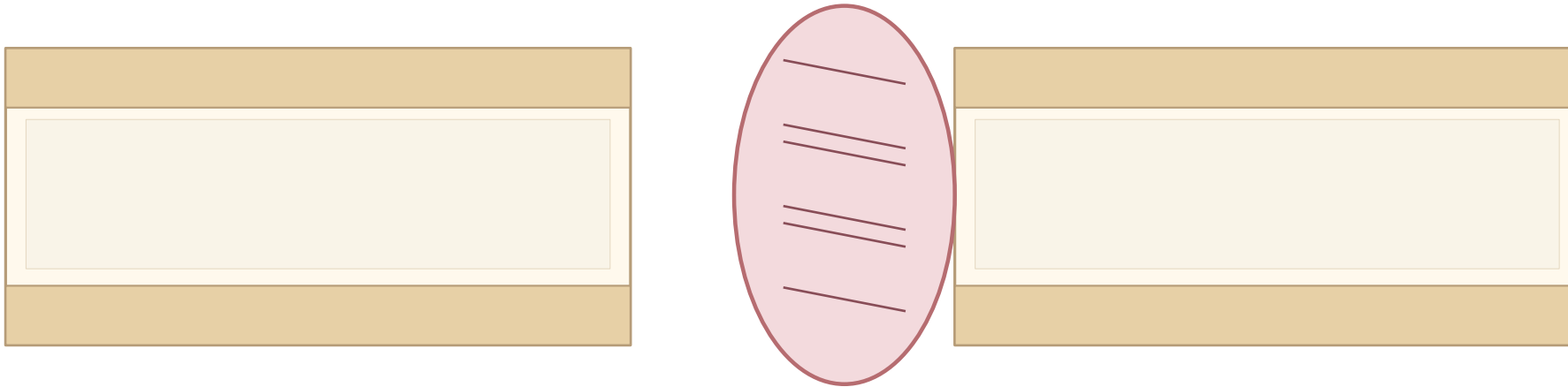
The clot helps seal the injury site.

Fibrin forms a substrate for incoming cells and neovessels.

The earliest repair matrix is not mineralized bone.

Organizing hematoma is a cellular scaffold

Fibrin links the two sides of the injury while cells enter the clot.



Schematic

Fibrin supports cellular and capillary ingrowth.

The clot is progressively reorganized—not the final structural bridge.

Three major ingrowth components

Inflammatory cells

Migrate into clot

Release activating signals

Fibroblasts

Grow into fibrin

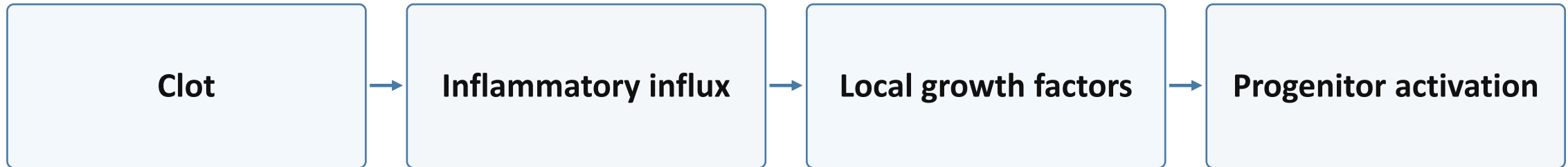
Contribute to provisional tissue

New capillaries

Grow into injury

Support organizing repair tissue

Inflammation drives reparative recruitment



Platelets degranulate while inflammatory cells migrate.

Mediators help recruit and activate bone-forming precursors.

Early inflammatory signals are part of normal healing.

The source highlights three growth factors

PDGF

Platelet-derived growth factor

Released locally

TGF- β

Transforming growth factor β

Released locally

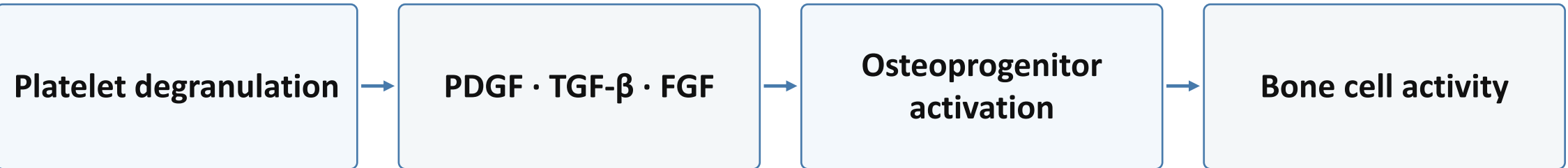
FGF

Fibroblast growth factor

Released locally

These factors contribute to osteoprogenitor activation in the healing milieu.

Growth factors couple injury to cell activity



Signals from platelets and migrating inflammatory cells.

The downstream response includes osteoblastic and osteoclastic activity.

Where osteoprogenitor cells originate

Periosteum

Outer bone envelope

Subperiosteal repair

Medullary cavity

Internal bone compartment

Internal repair matrix

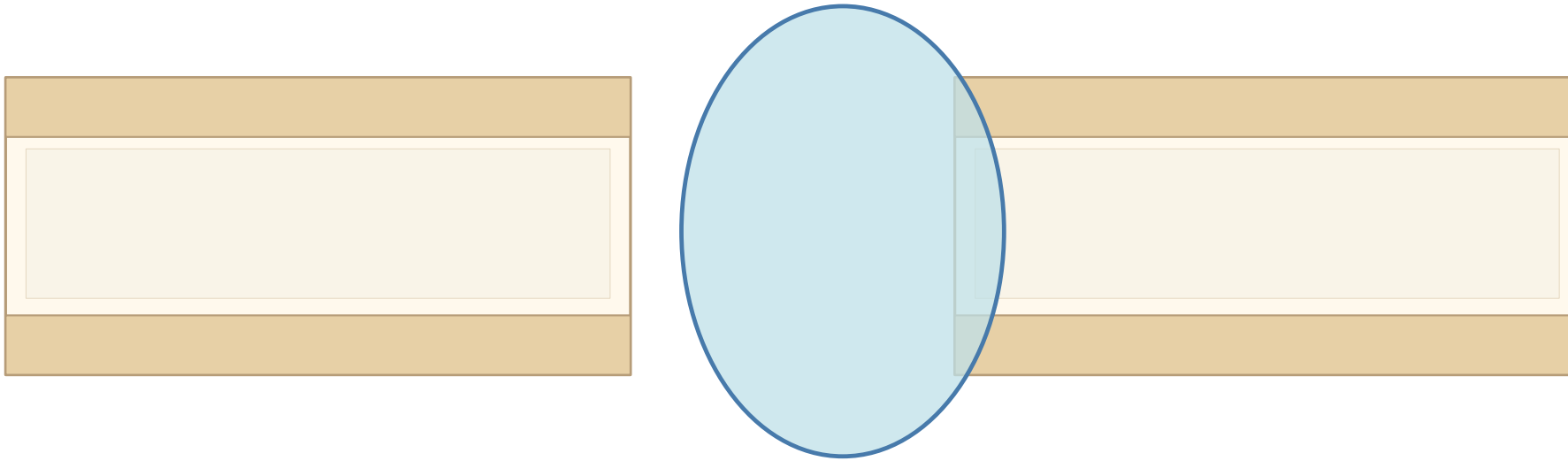
Soft tissues

Adjacent mesenchymal cells

May adopt chondrocytic fate

Periosteal and internal repair are coordinated

The repair response surrounds the fracture and extends within bone.



Schematic

Progenitor populations are not confined to a single surface.

The eventual callus can span periosteal and medullary spaces.

Formation and resorption are simultaneous activities

Bone formation arm

- Osteoprogenitors are activated
- Osteoblasts synthesize osteoid
- Woven bone is deposited

Bone resorption arm

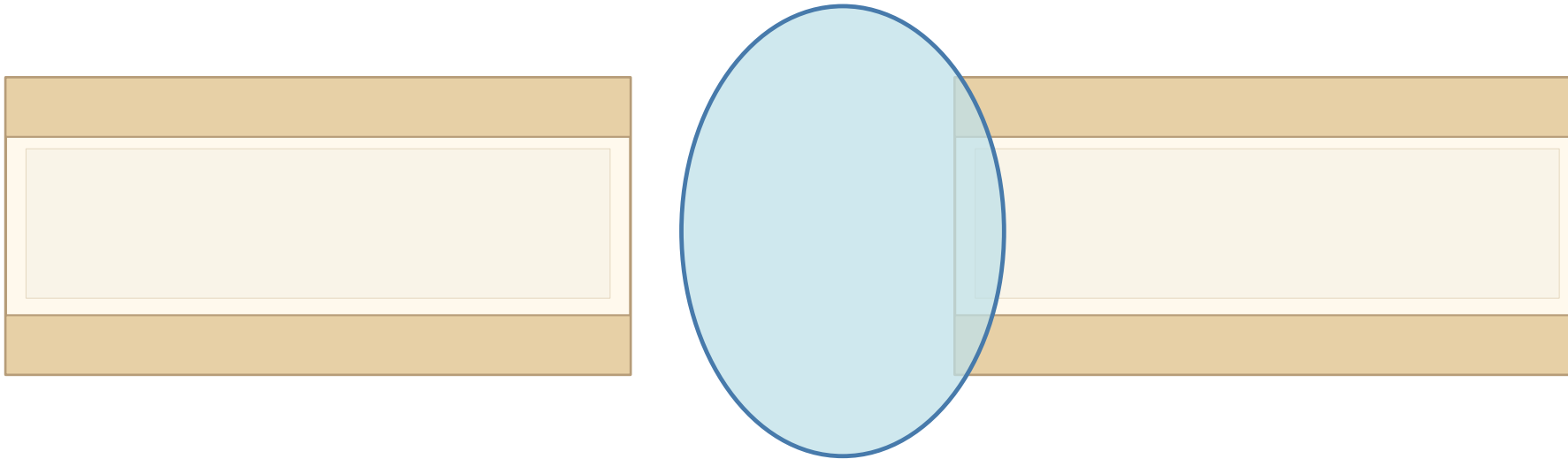
- Osteoclasts are activated
- Bone matrix can be removed
- Later remodeling eliminates excess tissue

Early repair is a transition—not an isolated stage

- Hematoma formation, inflammatory-cell influx, and fibrovascular ingrowth overlap.
- Osteoprogenitor activation begins before the callus becomes mechanically strong.
- The source describes a continuous sequence rather than mutually exclusive compartments.

By the first week: Soft callus bridges the ends

Predominantly uncalcified procallus begins anchoring the fragments.



Schematic

The bridge increases local continuity across the injury.

The bridge is not yet sufficiently rigid for weight bearing.

Soft callus is a provisional bridge

- Soft callus is also called procallus.
- It consists predominantly of uncalcified reparative tissue.
- It joins fracture ends enough to provide some anchorage.
- It lacks the structural rigidity needed to withstand substantial loading.

Anatomical bridging and mechanical competence do not emerge simultaneously.

Anchorage is not equivalent to strength

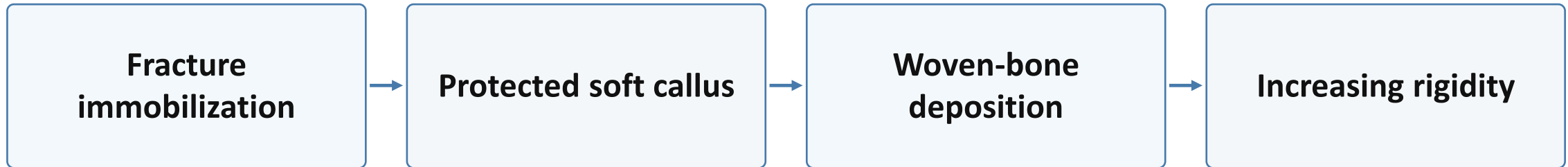
What soft callus provides

- Continuity between bone ends
- Some provisional anchorage
- Substrate for further ossification

What it does not yet provide

- Mature lamellar architecture
- Sufficient load-bearing rigidity
- Restored medullary cavity

A stable environment permits callus maturation

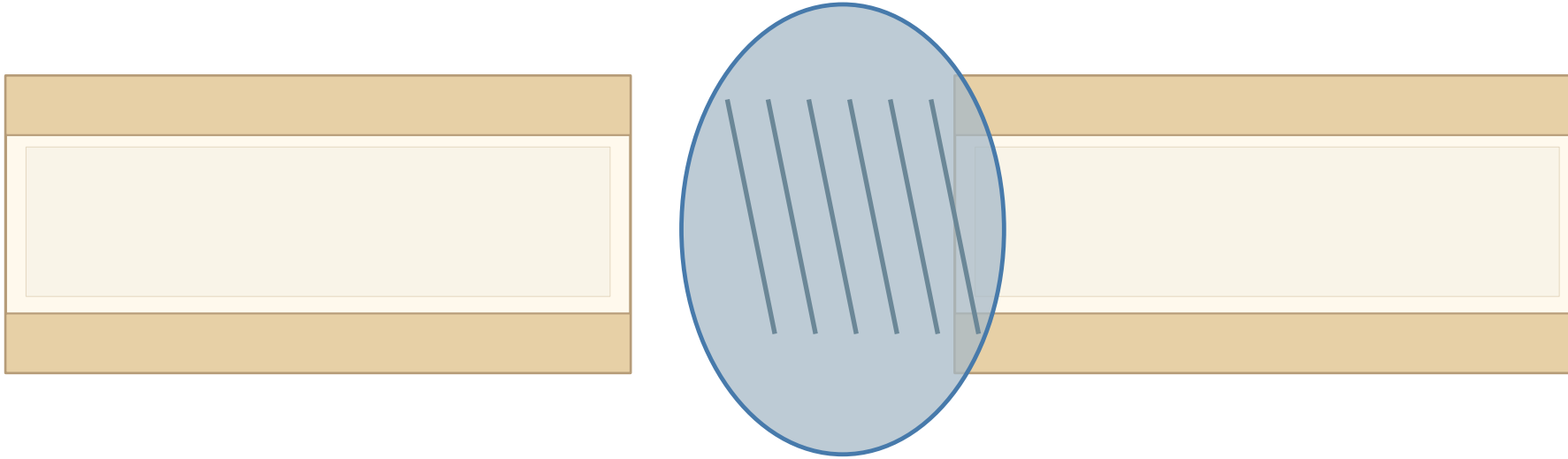


Movement within the callus disrupts normal maturation.

Inadequate immobilization may cause delayed union or nonunion.

Around two weeks: Bony callus begins to form

Woven bone is deposited within the previous procallus.



Schematic

Osteoblast-derived bone begins mineralizing the repair bridge.

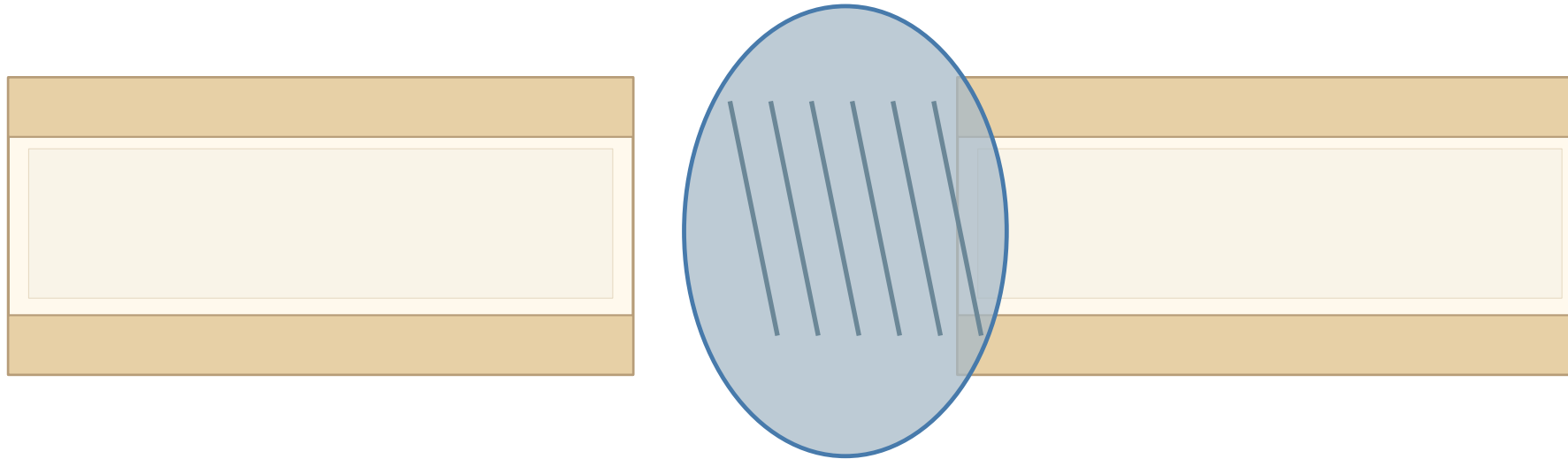
The healing tissue is becoming stronger, but not yet fully remodeled.

Woven bone is the first bony framework

- Conversion of procallus into bony callus at approximately two weeks.
- Activated osteoprogenitors deposit new trabeculae of woven bone.
- The relatively disordered woven architecture is suited to rapid deposition rather than final structural organization.

Subperiosteal trabeculae bridge the fracture

Woven-bone trabeculae form within the periosteal repair zone.



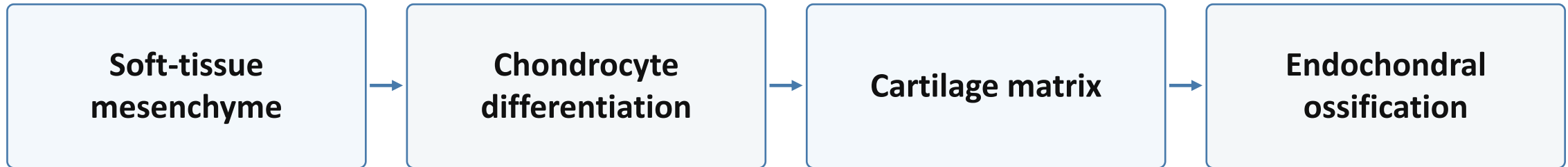
Schematic

The described trabeculae are oriented perpendicular to the cortical axis.
External callus formation reinforces continuity around the cortical break.

The medullary cavity also participates

- Activated osteoprogenitors can deposit trabeculae in the medullary compartment.
- New woven bone is not restricted to an external callus.
- The medullary space is ultimately reconstituted during late remodeling.

A second route uses cartilage as a precursor



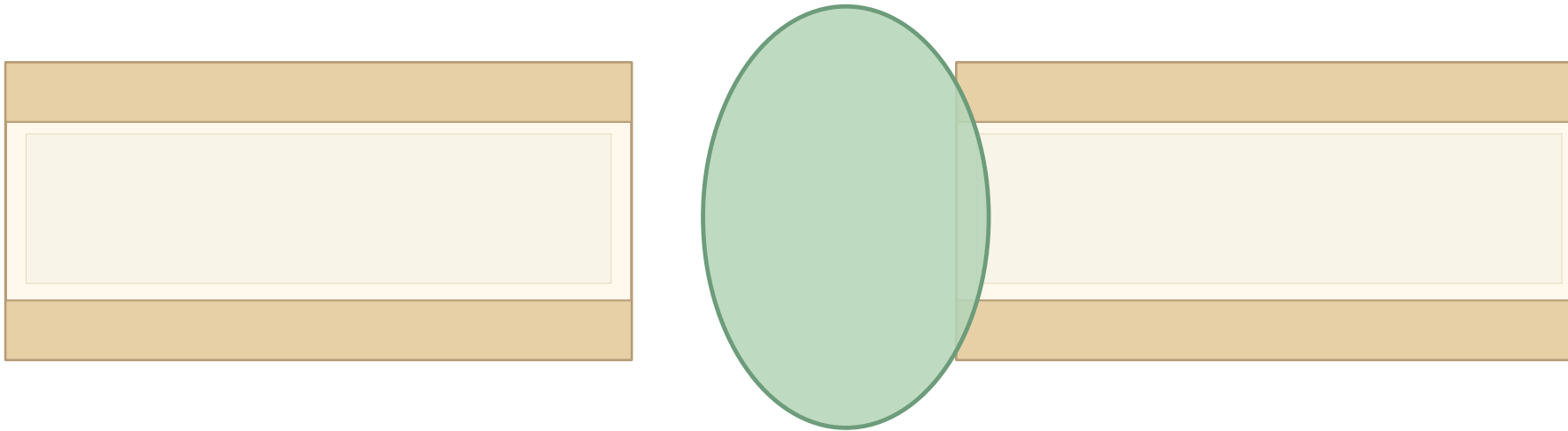
Some activated mesenchymal cells differentiate into chondrocytes.

Fibrocartilage and hyaline cartilage may occur in the callus.

Cartilage is eventually replaced by new bone.

Cartilage may coexist with early bone deposition

Simplified cartilaginous portion of the bridging callus.



Schematic

Chondrocytes produce fibrocartilage or hyaline cartilage.

This component can undergo endochondral ossification.

Two pathways make the new bony bridge

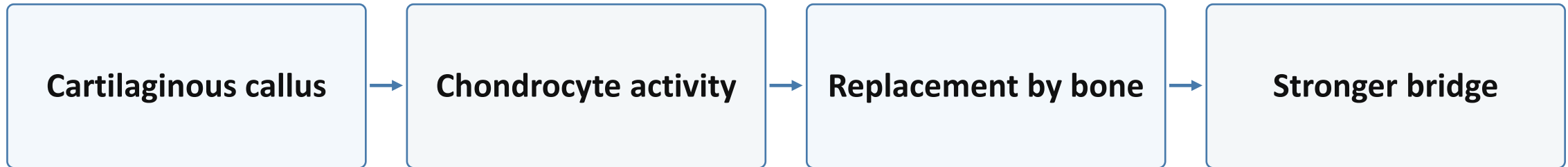
Direct woven-bone deposition

- Activated osteoprogenitors form osteoblasts
- Osteoblasts deposit woven trabeculae
- Subperiosteal and medullary sites

Cartilage-mediated deposition

- Mesenchymal cells differentiate to chondrocytes
- Fibrocartilage / hyaline cartilage forms
- Endochondral ossification yields bone

Endochondral ossification links cartilage to bone

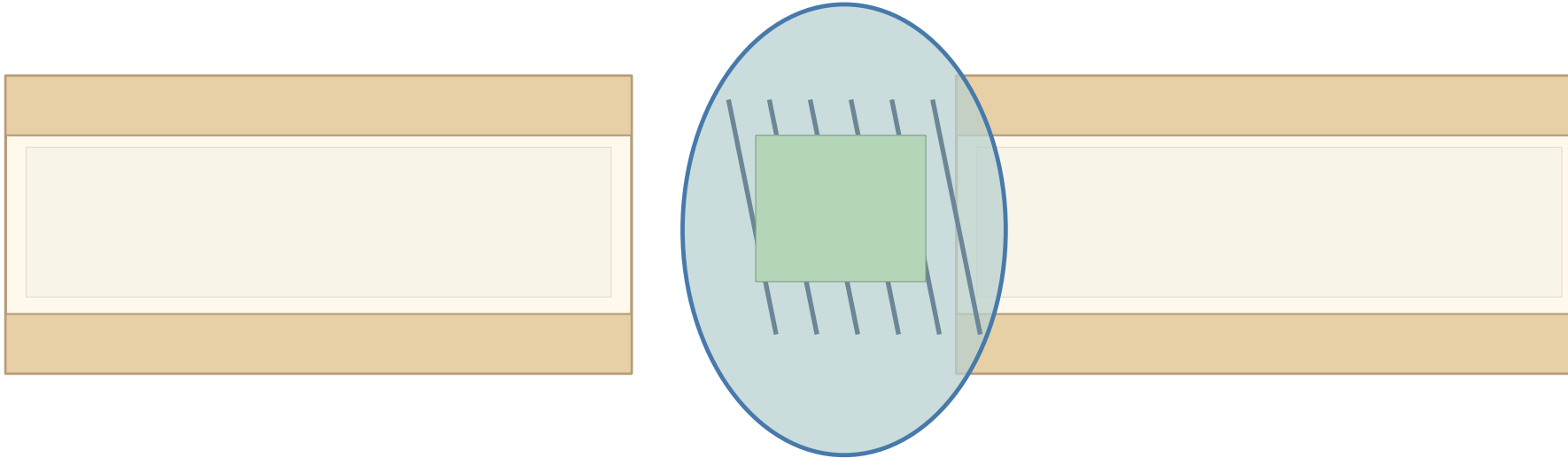


The mechanism reprises a developmental pathway used by the skeleton.

The fracture source identifies cartilage as a temporary component in some cases.

Early callus is compositionally heterogeneous

Fibrous tissue, cartilage, and woven bone may coexist.



Schematic

The reparative mass initially exceeds final structural requirements.
Maturation depends on mineralization and subsequent remodeling.

Progressive mineralization increases strength

- Mineral deposition makes the bony callus stiffer.
- As stiffness and strength increase, the bridge becomes better able to transmit mechanical load.
- Mineralization is necessary but is not itself the final remodeling step.

Woven-bone formation produces a functional bridge; Remodeling refines it.

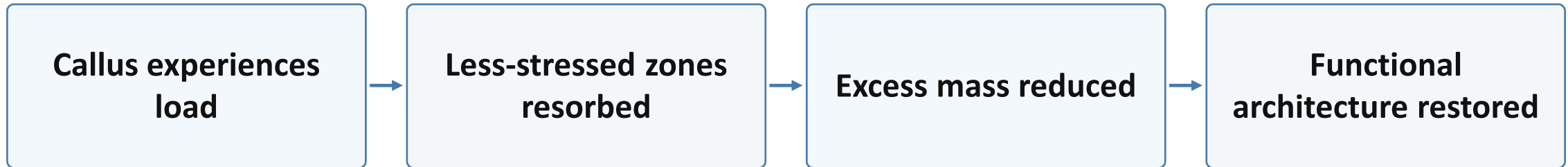
Compare the early and bony callus

Feature	Soft callus / procallus	Bony callus
Typical period	By end of first week	Around 2 weeks onward
Dominant matrix	Predominantly uncalcified	Deposited woven bone
Mechanical role	Anchorage without rigidity	Increasing stiffness and strength
Cell processes	Fibrovascular and progenitor activity	Osteoblastic deposition; Cartilage replacement

Early callus is deliberately exuberant

- The early repair mass contains excess fibrous tissue, cartilage, and woven bone.
- The initial goal is to bridge the defect, not immediately recreate original contours.
- Excess material can later be removed as loading clarifies mechanical requirements.

Mechanical loading guides late remodeling



Robbins specifies resorption of regions not subjected to physiological stress.

Mechanical input is a determinant of callus shape.

Formation and removal reshape the callus

Early repair

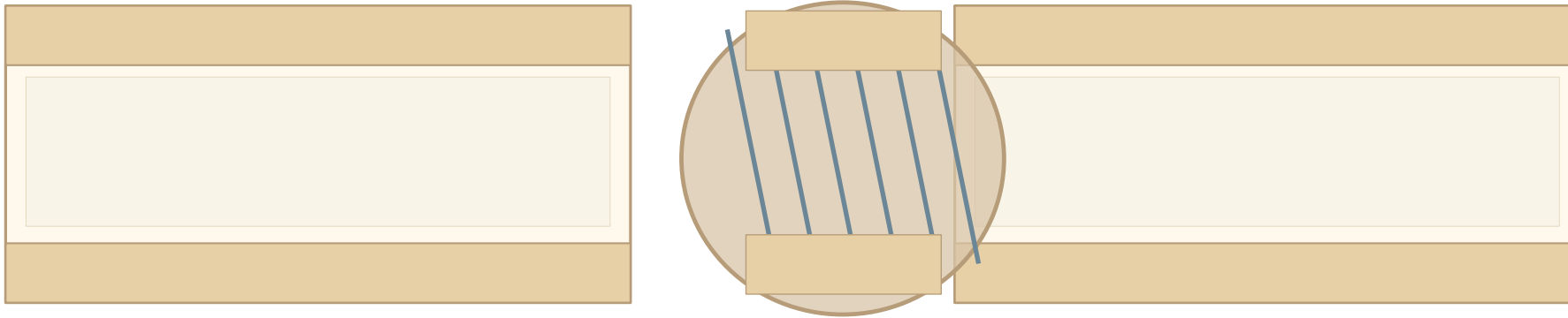
- Produce fibrous tissue, cartilage, and woven bone
- Create a broad provisional bridge
- Prioritize union across the fracture

Mature repair

- Resorb mechanically unnecessary tissue
- Replace irregular matrix with lamellar bone
- Restore cortical outline and medullary space

The mature callus regains a narrower profile

Excess callus is resorbed as the original bone contours return.



Schematic

Lamellar matrix progressively replaces woven bone.

The medullary cavity is restored as repair completes.

Lamellar bone provides mature architecture

- The disordered collagen arrangement of woven bone is replaced by parallel lamellar organization.
- Maturation restores the shape and outline of the fractured bone.
- The late process is architectural as well as cellular.

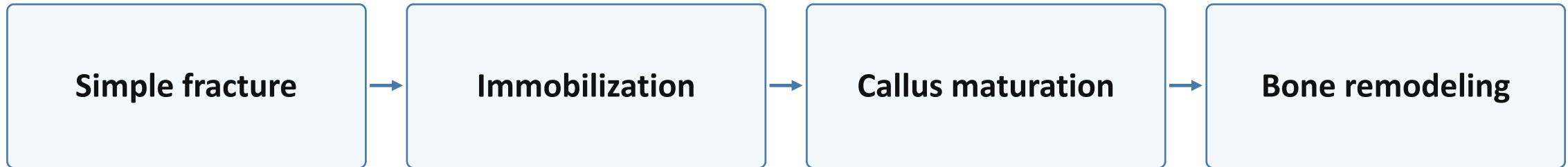
Restoration of the medullary cavity completes repair

- Early bony callus can occupy medullary regions of the injury.
- Remodeling removes unneeded tissue and restores the central cavity.
- The healed bone more closely approximates the prefracture cortex and marrow arrangement.

Repair stages: Predominant process and consequence

Stage	Principal substrate / process	Functional implication
Immediate	Hematoma + fibrin scaffold	Sealed injury; Cell recruitment
First week	Soft callus / procallus	Anchorage, not load-bearing rigidity
Around 2 weeks	Woven-bone callus	Increasing stiffness as bone mineralizes
Late	Resorption and lamellar remodeling	Restored contour and medullary cavity

What allows nearly complete union?



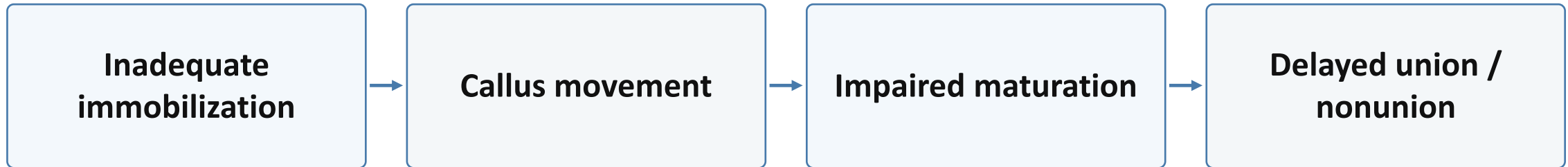
The absence of interfering comorbidities.

Near-perfect union is the expected outcome when conditions are favorable.

Complications arise when a necessary step fails

- Unstable callus cannot mature normally.
- Malaligned fragments can be united but remain deformed.
- Infection, neoplastic bone disease, and systemic comorbidity hinder the repair response.
- Delayed union, nonunion, and pseudoarthrosis are distinct possible outcomes.

Instability can progress to nonunion



Persistent movement disrupts conversion of the provisional bridge.

The mechanical environment influences biological progression.

Delayed union versus nonunion

Delayed union

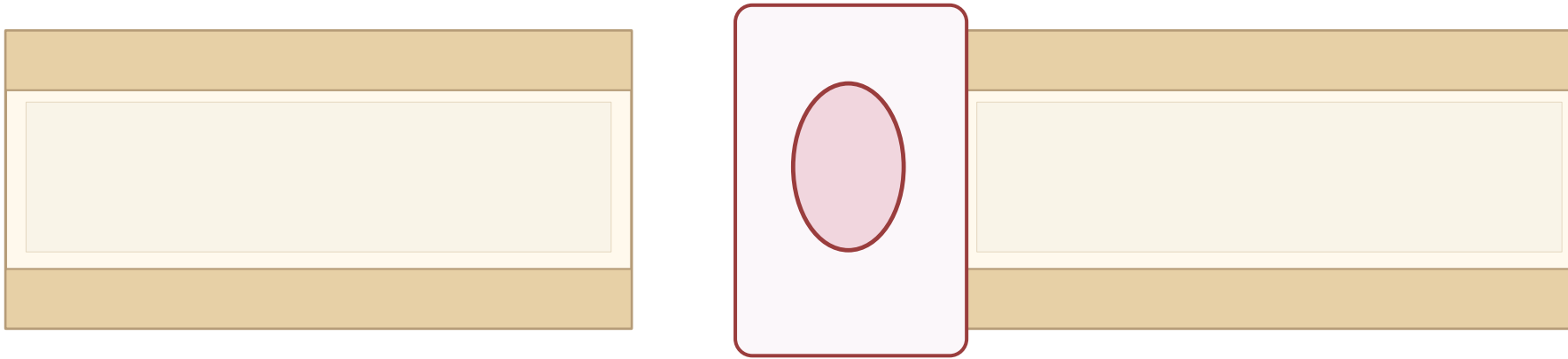
- Healing proceeds more slowly than expected
- Insufficient immobilization as a possible cause
- Progress may remain incomplete

Nonunion

- The fractured ends fail to unite
- A malformed callus may persist
- Long-standing cases may form pseudoarthrosis

Pseudoarthrosis: A false joint in persistent nonunion

Persistent nonunion may form a cavity with a synovial-like lining.



Schematic

Malformed callus can undergo cystic degeneration.

The luminal surface may acquire synovial-like cells.

This is a pathologic false joint, not a normal articular joint.

The false-joint sequence

Persistent nonunion



Malformed callus



Cystic degeneration



Synovial-like lining

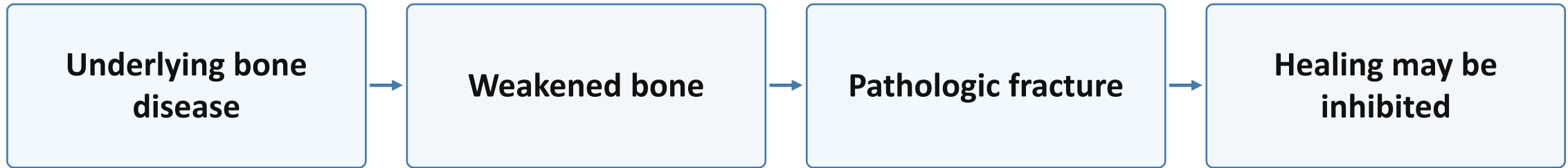
The final structure is called pseudoarthrosis.

It reflects failed fracture bridging and maturation.

Displacement and comminution can leave deformity

- Fragment alignment is central to the final shape of the repaired bone.
- Displaced fractures present nonaligned ends.
- Comminuted fractures contain fragmented bone.
- Both categories frequently result in some deformity.

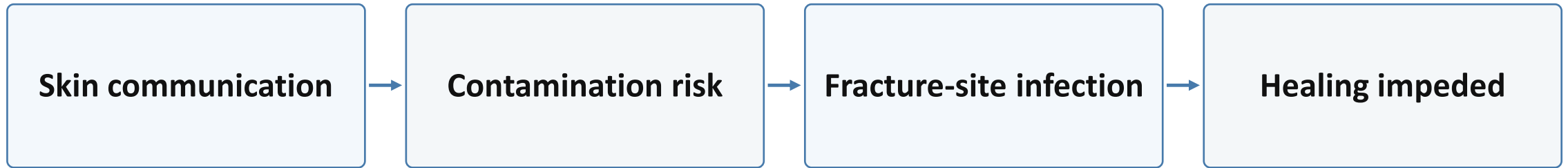
Pathologic bone changes the repair substrate



Primary or metastatic bone neoplasms are highlighted inhibitors.

Union problems may reflect both the break and the underlying lesion.

Open fractures expose the repair site to infection



Compound/open fractures are particularly associated with infection.

Infection is described as a serious obstacle to healing.

Systemic modifiers of the repair response

Malnutrition

Insufficient nutritional support

Impedes effective healing

Diabetes

Comorbid metabolic disorder

Hinders fracture repair

Skeletal dysplasia

Abnormal skeletal substrate

May compromise repair

Robbins specifically includes osteoporosis as an example in this context.

Osteoporosis affects fracture vulnerability and healing

- The source lists osteoporosis among skeletal disorders that can hinder healing.
- Abnormal bone substrate complicates the biological and mechanical context of repair.
- Do not equate osteoporosis with a neoplastic pathologic fracture; The mechanisms differ.

Barriers to union: A mechanism-based comparison

Barrier	Main disturbance	Potential consequence
Instability	Repetitive callus movement	Delayed union / nonunion
Malalignment	Fragment position abnormal	Residual deformity
Infection	Infected fracture site	Impaired healing
Bone neoplasm	Diseased osseous substrate	Inhibited normal repair
Systemic comorbidity	Malnutrition, diabetes, skeletal disease	Impaired healing capacity

Applied case: Favorable repair

A simple, aligned fracture is adequately immobilized; There is no major accompanying illness.

What sequence should follow?

Organized hematoma → soft callus during the first week → woven-bone callus around two weeks → mineralization and remodeling toward lamellar bone.

Applied case: Persistent movement

A fracture remains mobile within its callus, and bridging fails to mature normally.

What mechanism best explains delayed union?

Inadequate immobilization permits repeated callus movement, interfering with maturation. Persistent nonunion may eventually form a pseudoarthrosis.

Applied case: Infected open fracture

An open fracture develops local infection during the repair period.

Why is union at risk?

Infection of the fracture site is a major impediment to repair and is particularly associated with compound fractures.

Applied case: Fracture through tumor

A break occurs through bone involved by a primary or metastatic neoplasm.

What makes this more than an ordinary fracture?

The underlying neoplastic lesion weakens bone and may inhibit the normal repair response. The bone substrate must be considered alongside the fracture.

Applied case: Weak callus at one week

A fractured bone has developed a soft, predominantly uncalcified bridge by the end of week one.

Can bridging be equated with safe weight bearing?

No. Soft callus provides some anchorage but lacks load-bearing rigidity. Woven bone deposition and mineralization increase stiffness later.

Recognize structures by the process they indicate

Hematoma + fibrin

Vascular injury and scaffold

Early organization

Cartilage + woven bone

Temporary callus matrices

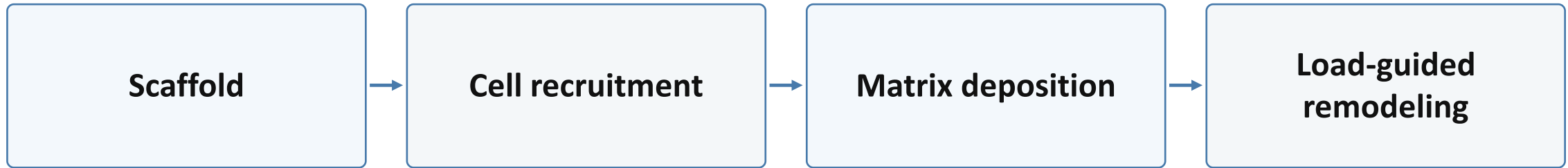
Active osseous repair

Lamellar architecture

Remodeled organized bone

Later maturation

Integrating biology with mechanics



Cells and signals construct a provisional bridge.

Adequate immobilization permits maturation of that bridge.

Mechanical forces help select the final architecture.

Essential clinicopathologic synthesis

- Clot and fibrin initiate organization; They are not the final bone repair product.
- Soft callus anchors before bony callus can bear load.
- Woven bone and optional cartilage-mediated ossification build the bridge.
- Lamellar remodeling restores contours and the medullary space.
- Stability, alignment, infection, bone disease, and nutrition modify the outcome.

Primary source and scope

- Robbins, Cotran & Kumar. Pathologic Basis of Disease. 11th edition (2026).
- Chapter 26: Bones, Joints, and Soft Tissue Tumors — “Healing of Fractures,” pp. 1075–1076.
- Relevant parent context: “Basic Structure and Function of Bone,” pp. 1064–1065; “Fractures,” p. 1075.
- All fracture-stage and complication claims in this lecture are based on those supplied excerpts.