boring non-loaded areas (gaps) a minimal amount of motion is possible, but it is limited by the elastic deformability of the neighboring contact zones. As long as there is no destruction of bone in the contact areas, the motion in the gap is small enough to keep interfragmentary strain below 2%. This process allows for the direct formation of bone as well. Within these gaps, granulation tissue appears first, bringing in new blood supply. While loose connective tissue can be observed only briefly in the center of the gap, the deposition of lamellar bone starts early on the surface of the bone fragment ends. This lamellar bone deposition continues until the whole gap is filled. After the complete filling of the gaps with lamellar bone in a direction parallel to the fracture surface, osteons originating in the fragment ends cross the filled gap and enter the other fragment. Thus, the fragments are united by lamellar bone structures that are arranged parallel to the long axes of the bone (**Fig 1.3.3-2a–b**). This process is called primary or direct bone healing, since it does not proceed through the entire tissue differentiation cascade that one observes in the secondary healing process via callus formation. The process of primary healing leads to a gradual disappearing of the fracture line on the x-ray. Since this healing pattern is characterized by an absence of callus formation, it is difficult to judge the progress of healing on x-rays. The intracortical remodeling of a fracture zone is a slow process, and such a fracture needs protection by the plate over a prolonged period. The time span during which a fracture plate needs to be in place is dependent on individual factors like loading patterns, bone quality, fracture patterns, eg, comminuted vs simple fractures, and patient compliance. On average, a period of 6 months is sufficient for all possible scenarios. The pattern of direct healing per se is not a goal to strive for, but the absence of this pattern, ie, the formation of periosteal callus under conditions of plate fixation is an indicator that complete immobilization was not achieved. Too much motion, often combined with a jeopardized circulation and eventually an infection, may result in a delayed healing or even nonunion.

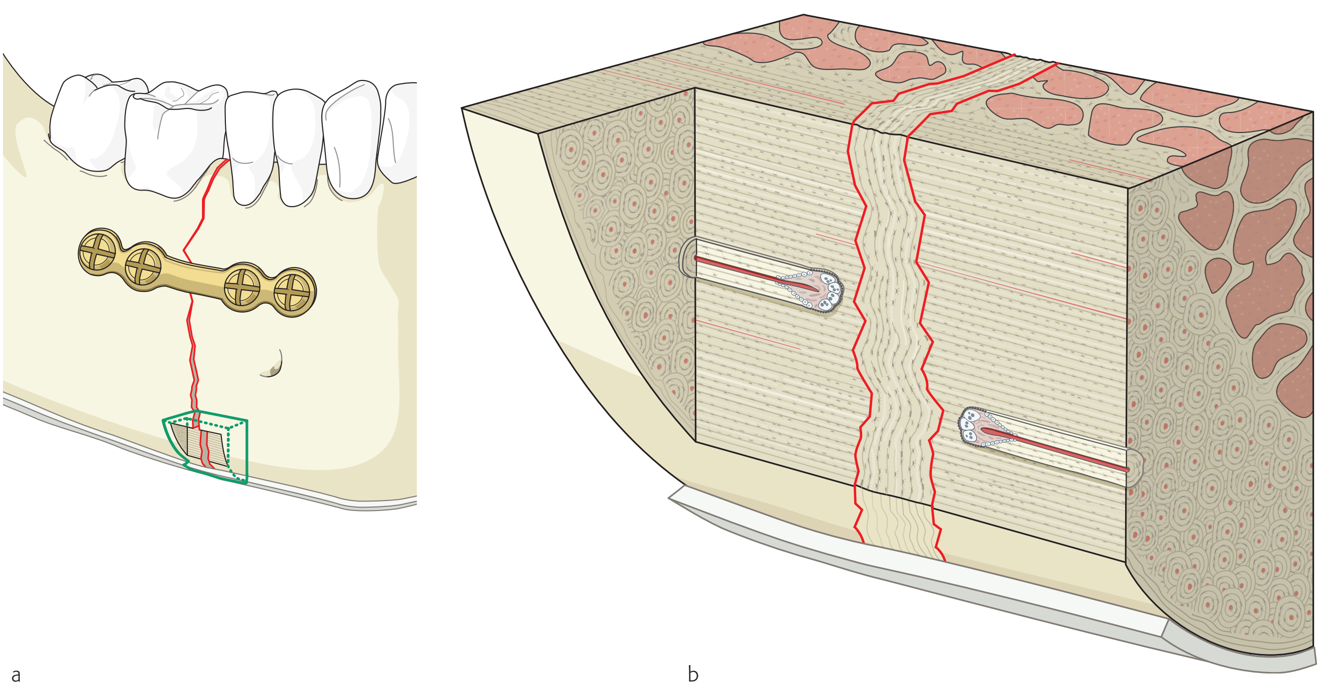


Fig 1.3.3-2a–b
a Stable fixation, load sharing with contact area superiorly and gap area inferiorly.
b Enlarged section of (a): primary healing gap area: complete filling of the fracture gap with lamellar bone in a direction parallel to the fracture surface.

2 Secondary bone healing via callus formation

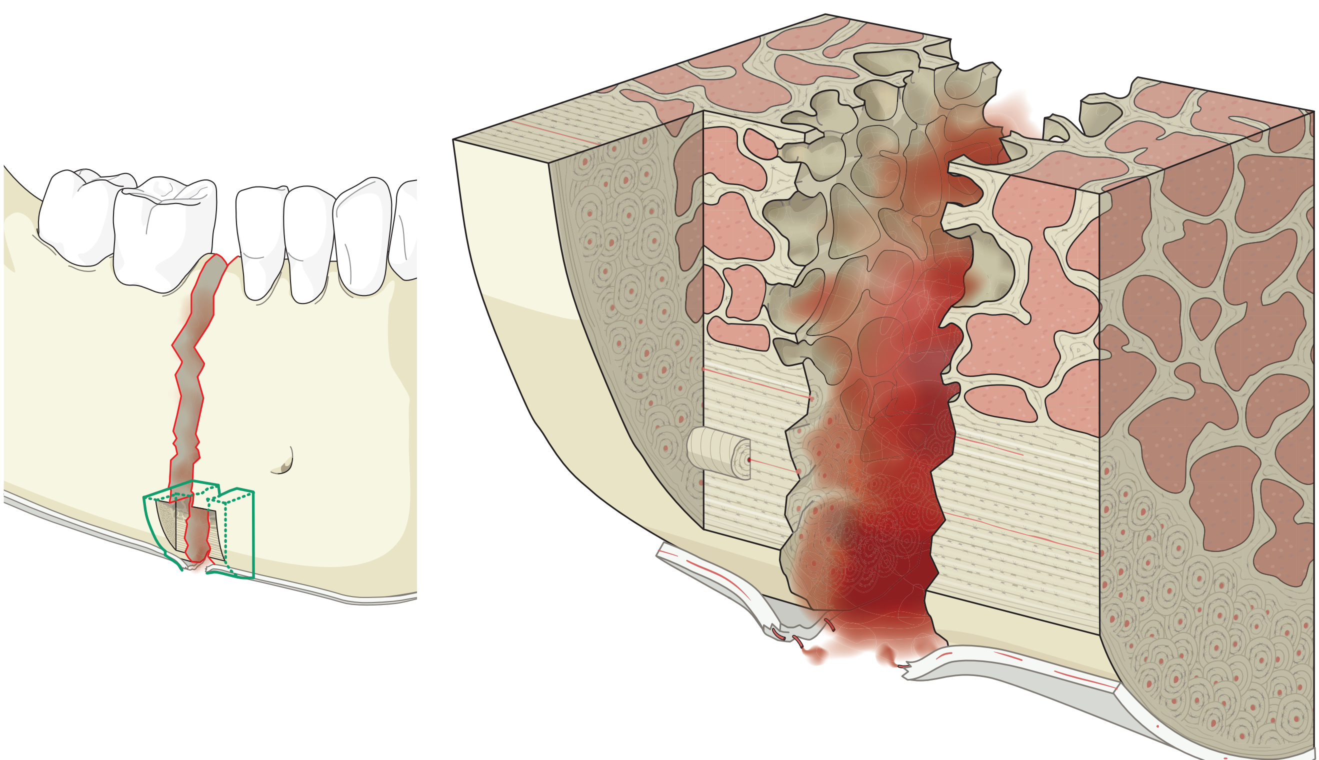
In cases when no fracture fixation or just loose adaptation fixation is done, macromotion between the fragment ends occurs. Under these conditions direct bone healing is not possible. The strain in between the fragments exceeds what bone can tolerate, and new bone developing between the fracture ends would be destroyed before it is formed. The disruption of the bone's integrity is not the only damage caused during a fracture. The trauma also causes an interruption of circulation of larger vessels and especially of microcirculation within the bone. The vessels of the Haversian and Volkman canals are occluded over a distance of a few millimeters from the fragment ends within the first few hours following an injury. This is the cause for resorption at the fracture ends.

During the course of secondary healing, periosteal and endosteal callus is formed. In between the fracture ends a tissue differentiation cascade takes place, during which stiffness and strength increases and strain tolerance gradually decreases. The differentiation cascade starts with a hematoma, thereafter granulation tissue develops which proceeds through connective tissue, fibrocartilage, and mineralized cartilage to woven and finally to compact bone. During this tissue-differentiation process, the stiffness and strength increases until the end when the interfragmentary space is totally reossified.

3 Steps of differentiation cascade

Hematoma

Initially a hematoma is found between the fragment ends (**Fig 1.3.3-3a–b**). The function of the hematoma in the course of fracture healing is still controversial. There is some evidence that the leukocytes within the blood may transform into fibroblasts and other cells of the supporting tissue system. The hematoma might as well act as a guiding structure, which, as a spacer, determines the size and shape of the callus. Then fibroblasts occur within the hematoma.



a b

Fig 1.3.3-3a–b
a Secondary bone healing under the condition of motion between the fracture ends.
b Enlarged section of (a). Secondary bone healing, phase 1: hematoma filling the fracture gap.