Introduction

Internal fixation of fractures using plates was developed more than a hundred years ago following the widespread use of radiographs. Hansmann reported the technique of plating lower limb shaft fractures as early as 1886 and presented a plate design allowing subcutaneous insertion of the plate and percutaneous insertion of the plate screws [1]. Lambotte, Lane, and Sherman experimented in the first decades of the twentieth century with new implant materials and improved the plate and screw designs to decrease the risk of corrosion and mechanical failure of the implants [2-4]. Danis, Bagby, and Müller introduced the concept of compression plating to improve the stability of fixation and to protect the implant from mechanical overload [5-7].

Since then, conventional plating techniques and plate designs have evolved constantly. This evolution is based on an improved understanding of the biology of fracture healing, of the biomechanics of fracture fixation, and on experience analyzing previous failures – such as fatigue failure of the implants, deep infection, and delayed union or non-union. It has involved the implants, the technique of its application, and the surgical technique with bone and soft tissue care or reconstruction [8-32].

Biological Aspects of Plate Fixation

Blood Supply of Cortical Bone

The three primary components of the afferent vascular system to bone tissue are the principal nutrient artery, the metaphyseal arteries, and the periosteal arterioles. The nutrient and metaphyseal arteries together compose the medullary arterial system, which is the major afferent supply nourishing about the inner two thirds of the bone cortex. The periosteal vessels enter the cortex mainly at sites of fascial and muscle attachment and appear to supply the outer third of the bone diaphysis [33-38]. Cortical circulation usually flows in a centrifugal direction. In the diaphysis, the inner cortical layers are drained through venous channels, the periosteal layers directly by periosteal capillaries. In case of damage to the medullary system following trauma or operation a compensatory flow reversal occurs to some extent [33,38-42].

Vascular Disturbance Due to Trauma, Surgery, and Implant

As a result of bone fragmentation and displacement of fragments, periosteal, intracortical, and endosteal vessels are ruptured [13,35-37,41,43]. At each fracture line all intracortical vessels are disrupted due to the direct damaging of its surrounding osteons. Major displacement of the fracture fragments may disrupt larger vessels like the nutrient artery, the central artery, or its intramedullary branches. This disruption of the medullary blood supply in turn leads to avascu-
larity and devitalization of a large amount of the bone cortex. The stripping of the periostium with its vascular network during injury is of particular importance, because disruption of the periostium may be severe or total between fragments, leaving smaller fragments completely devascularized.

The surgical approach to the fracture leads to an additional considerable vascular damage to the bone tissue by the soft tissue retraction. Additional damage is added by subperiosteal exposure that leads to more damage compared to careful epiperiosteal exposure [44]. Fragment manipulation by reduction clamps and the plates itself result in further damage to the blood supply of bone. Complete visualization of the fracture area is needed neither for fracture reduction nor for positioning of the plate and insertion of the screws [15,45,46]. In conventional plating techniques, some amount of contact between plate and bone is needed for stability reasons to allow load transmission by friction at the interface. The axial screw force generated by tightening the screws and the compressive strength of cortical bone gives the minimum area required for load transfer. Shaping the plate to the bone surface as exactly as possible is mandatory so as not to be faced with the problem of secondary fracture dislocation when the fragment is pulled towards the plate by tightening the screws. The biological disadvantage of the conventional contact plating concept is the appearance of a relatively large zone of blood supply disturbance directly underneath the implant (Figures 34.1a, b, plate section). This deficiency of perfusion is caused by direct compression of the periosteal vascular network under the plate and leads to necrosis of cortex adjacent to the plate [47–55]. Dead bone can only be revitalized by removal and replacement (creeping substitution), a biological process which takes a long time to be completed. During the recovery of the blood supply, a temporary porosis of the bone is observed as a result of the tremendous intracortical remodeling. The remodeling activity starts at the boundary between vital and initially devascularized bone and is usually directed towards the implant (Figures 34.2a, b, plate section). It is accepted that necrotic tissue disposes to and sustains infection [56]. The recovery of the original bone structure and vitality generally takes more than one year. In the past, many authors have tried to explain this temporary bone porosity as a functional adaptation of the bone structure to the unloading effect of the plate according to Wolff’s law [57–69]. The newer generations of plates (limited contact, no contact implants) decrease the amount of devascularization of cortical bone due to a reduction or the complete absence of implant–bone contact.

**Fracture Healing and Stability of Fixation**

Fracture healing is the recovery of the biological and mechanical integrity of the osseous tissue, i.e., return of the prefracture tissue vitality and structure as well as the prefracture stiffness and strength of the injured bone segment [70]. The amount of stability achieved by implants is the mechanical input for the biological response of bone healing. Beside the injury itself, the healing process additionally is modulated by the additional surgical damage to the bone and surrounding soft tissue envelope during the process of reduction and fixation [25,71–73]. In plate osteosynthesis the importance of the amount of mechanical stability to achieve direct bone healing was overestimated for a long time. Forcing precise reduction to improve the postoperative radiological appearance was likely to be linked to additional and sometimes extensive surgical trauma with stripping and denuding of bone fragments. Because dead bone is unable to heal, some of the possible complications such as deep infection, nonunion, delayed union, and refracture have to be attributed to the iatrogenic surgical tissue damage during the operative procedure. Radiographically and histologically, different healing patterns can be differentiated depending on the local mechanical environment [3,6,74].

Absolute stability is present when the fracture is stabilized by a stiff implant which maintains the fracture reduction with no or minimal displacements occurring under functional loading. As a biological consequence, primary bone