Invited review article

Internal plate fixation of fractures: short history and recent developments

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Abstract

Metal plates for internal fixation of fractures have been used for more than 100 years. Although initial shortcomings such as corrosion and insufficient strength have been overcome, more recent designs have not solved all problems. Further research is needed to develop a plate that accelerates fracture healing while not interfering with bone physiology.

The introduction of rigid plates had by far the greatest impact on plate fixation of fractures. However, it led to cortical porosis, delayed bridging, and refractures after plate removal. These unwarranted effects were said to be caused by bone–plate contact interfering with cortical perfusion. Consequently, further plate modifications aimed to reduce this contact area to minimize necrosis and subsequent porosis.

The advocates of limited-contact plates have not published measurements of the contact area or proof of the temporary nature of the porosis. Moreover, clinical studies of newer plate types have failed to show a superior outcome. Histomorphometric measurements of the cortex showed no difference in the extent of necrosis under plates having different contact areas. Necrosis was predominant in the periosteal cortical half, whereas porosis occurred mostly in the endosteal cortical half. No positive correlation was found between either.

The scientific evidence to date strongly suggests that bone loss is caused by stress shielding and not interference with cortical perfusion secondary to bone–plate contact. Consequently, an axially compressible plate (ACP) incorporating polyactic acid (PLA) inserts press-fit around screw holes was designed. The bioresorbable inserts should allow for (1) increased micromotion in the axial plane to promote healing during the union phase and (2) gradual degradation over time to decrease stress shielding during the remodeling phase.

Results of ongoing experimental results are encouraging. Only plates allowing dynamic compression in the axial plane can lead to a revolution in fracture fixation.

Introduction

Plates for internal fixation of fractures have been used for more than 100 years. Plating of fractures began in 1895 when Lane first introduced a metal plate for use in internal fixation.¹ Lane’s plate was eventually abandoned owing to problems with corrosion (Fig. 1). Subsequently, Lambotte in 1909² (Fig. 2) and then Sherman in 1912³ introduced their versions of the internal fracture fixation plate. Improvements in the metallurgical formulation of the plate increased their corrosion resistance; however, both designs were eventually abandoned as a result of their insufficient strength.

The next important development in fracture plate design was initiated in 1948 by Eggers.⁴ The Eggers plate had two long slots that allowed the screw heads to slide and thus compensate for resorption of the fracture ends. Widespread use of his plate was limited by its structural weakness and the resulting instability of the fixation (Fig. 3).

Compression plating

Danis in 1949 recognized the need for compression between the fracture fragments. He achieved this goal using a plate he called the coapteur, which suppressed interfragmentary motion and increased the stability of the fixation (Fig. 4).⁵ It led to a mode of healing he called soudure autogène (autogenous welding), a process now known as primary bone healing (Fig. 5). His revolutionary concept influenced all subsequent plate designs.

In 1958 Bagby and Janes⁶ described a plate with specially designed oval holes to provide interfragmentary compression during screw tightening (Fig. 6). Müller et al. in 1965⁷ presented another design that permitted interfragmentary compression by tightening a tensioner that was temporarily anchored to the bone and the plate.
The plate was heavier and thicker (4.5 mm) than those designed by Eggers and Danis. With this design, Müller and his group set the stage for the rigid plating of fractures that resulted in a mode of bone healing characterized by the absence of periosteal callus formation. The appearance of any periosteal callus was interpreted as a sign of instability. “The appearance of callus after plate fixation may be an indicator for an unknown degree of instability.”

The use of the tensioner was eventually abandoned in favor of oval holes with a design similar to that of the Bagby plate (Fig. 8). This new design, known as a dynamic compression plate (DCP), was claimed to have been developed without the knowledge of Bagby and Jane’s invention. Yet in 1967 Schenk and Willenegger, both members of a Swiss group of investigators, made...
bone loss under the plate was another disadvantage (Fig. 9). Moreover, clinicians deplored the fact that it was impossible to assess the state of healing of the fracture radiologically. During physiological healing, disappearance of the fracture gap and development of an external bridging callus are criteria for assessing the state of healing of the fracture; they are not present after rigid internal fixation.

In light of possible refracture after plate removal, it was recommended that the plate not be removed for at least 15–18 months. A study by Kessler and colleagues showed that plate removal at an average of 20.1 months still resulted in refracture. Histologic examinations of 28 refractures confirmed that failure occurred at sites of absent gap bridging.

Two causes were thought to be responsible for refracture after plate removal. The first hypothesis attributed refracture to inadequate rigidity of the fixation. As a consequence, double plating was recommended. This approach did not solve the problem of refracture. The second hypothesis, advanced by Perren’s group, was that porosis and refractures are due to cortical necrosis that is secondary to excessive plate–bone contact interfering with cortical perfusion. The natural process of removal of the necrotic bone and its replacement by living bone was suspected to be the cause of transient porosis of the cortex. The duration of the transient porosis was not specified, but Perren and collaborators claimed that the temporary reduction of bone strength did not allow early plate removal owing to the possibility of refracture.

The Swiss group developed a new plate design intended to reduce the plate’s interference with cortical perfusion and thus decrease cortical porosis. The design was called the limited contact-dynamic compression plate (LC-DCP), which was claimed to reduce bone-
plate contact by approximately 50%. The subsequent development of the point contact fixator reduced bone–plate contact to the point where it was essentially negligible.

Only one published study has reported on actual measurements of the plate–bone contact area of the DCP and LC-DCP. Field et al. measured the bone–plate contact area for both DCPs and LC-DCPs fixed to cadaveric bone and found “no apparent differences in interface contact area attributed to bone plate design.” This contradicts the assertion by Gautier and Perren that the LC-DCP reduces the contact area by 50%. Jain et al. measured cortical blood flow with laser Doppler flowmetry of canine tibias fixed with a DCP or LC-DCP. They found no difference in cortical blood flow between the two groups, supporting the findings of Field et al. They also reported on the biomechanical properties of the tibia and found no difference between the two groups. The authors concluded that “the LC-DCP is not advantageous in fracture healing or restoration of cortical bone perfusion to devascularized cortex.”

**Bone loss under plates**

Is the porosis really transient and due to remodeling of the necrotic bone area under the plate, or is it permanent due to stress shielding induced by rigid plates? Perren and associates denied the role of stress shielding, refuting the results of many researchers extending over 25 years. These published results documented that any unloading of bone leads to bone loss. The weight of the scientific evidence strongly supports the idea that stress shielding plays a greater role than the size of the area of plate–bone contact.

Before presenting our hypothesis, we first discuss the outcome of rigid plating based on pertinent publications. Gunst and collaborators used intravital disulfine injections administered 15 min prior to killing of sheep and found that an absence of cortical perfusion existed under contact plates but not under plates with a reduced contact area. We were able to confirm their findings when comparing contact plates with railed plates fixed to the lateral aspect of intact beagle femors. Figure 10 shows cross sections of both plates. Intravital disulfine blue injections helped to show that there is better cortical perfusion under plates having contact with bone limited to two longitudinal rails (Fig. 11).

Neither the exact location nor the extent or changes of the necrosis over time have been determined by advocates of the DCP or the LC-DCP. In an effort to quantify necrosis, we published histomorphometric data assessing necrosis in the periosteal and endosteal halves of the cortex when using both contact and railed plates. We found that the degree of necrosis was more pronounced in the periosteal half. Furthermore, the degree of necrosis was independent of the plate–bone contact area and did not change appreciably between 8 and 24 months.

We were unable to find a pertinent study published by advocates of compression plates proving that the porosis was transient. Nevertheless, Perren and colleagues came to the conclusion that the induction of early porosis is not caused by stress shielding. We also measured the porosis and differentiated again between the periosteal and the endosteal halves of the cortex in dogs. If the hypothesis of Perren’s group holds true, one would expect the porosis to be at the site of necrosis (i.e., the periosteal half of the cortex). To our surprise we found that the porosis was much more pronounced in the endosteal half of the cortex where the amount of necrosis was less (Fig. 12).

In the course of our experimental studies in dogs we did not find a difference in necrosis between the railed (i.e., limited contact) and the contact fracture plates.
For both types of plate the amount of necrosis was greater in the periosteal half than in the endosteal half of the cortex. Moreover, the amount of necrosis did not decrease between 8 and 24 weeks. On the other hand, the amount of porosis was greater in the endosteal half of the cortex than in the periosteal half and more so under the railed plates than the contact plates. Consequently, we failed to find a positive correlation between necrosis and porosis.24

In another canine study, we showed that rigid plating of osteotomized femoral diaphyses led to widening of the femoral canal and to thinning of the cortices at 60 weeks.22 Akeson and collaborators also reported cortical thinning under rigid plates due to endosteal resorption.17 Moreover, we showed that immobilization of a canine limb in a plaster cast led to porosis of the inner layer of the cortex after 8 weeks and to widening of the medullary canal after 40 weeks, a finding that supports the role of stress shielding in bone loss.25,26

The contention of the Swiss group that intimate plate–bone contact increases necrosis and leads to transient porosis is also contradicted by the study of Korvick and colleagues.27 Despite almost total plate–bone contact, they found a decrease in porosity. In conclusion, we can state that there is no proof that limited plate–bone contact reduces porosity or that porosis is reversible.

Bone loss induced by stress shielding results from a resorptive process characterized by porosis of the endosteal half of the cortex during the early phases of plate fixation and culminating in the loss of porotic endosteal bone during the late phases. As a result of the loss of endosteal bone, the medullary canal widens and the cortices narrow. If the porosis was indeed transient, as suggested by Perren28 and his group, one would expect filling of the pores without affecting the thickness of the cortex. However, our findings clearly prove that the early porosis, mostly affecting the endosteal half of the cortex, led to loss of the porotic bone resulting in definitive cortical thinning. This fact can only be explained by stress shielding.

Clinical studies

Did the reduction of plate–bone contact improve the clinical outcome? Andersen and colleagues reported on 244 patients with forearm fractures treated with dynamic compression (DC) plates and found an incidence of union of 97.9% (radius) and 96.3% (ulna) respectively.29 Time to union was 6.0–8.8 weeks. Chapman et al. treated forearm fractures with 3.5-mm DC plates. In 117 patients they observed a 97% union rate at an average of 12 weeks.30 Hertel and collaborators treated 133 patients with forearm fractures and reported a 96.2% union rate before 6 months.31 They observed three refractures (4.3%) after plate removal at an average of 33.1 months.

McKee and colleagues treated upper limb fractures with LC-DC plates in 114 patients and reported an overall union rate of 97.3%. Union was achieved at an average of 10.7 weeks.32 Gupta and colleagues treated humeral diaphyseal fractures with LC-DC plates in 51 patients and reported a union rate of 94.1%. Union was achieved at a median of 2.5 months.33

Up to the time of these publications, the stabilization of fragments under compression was one of the mainstays of internal fixation of fractures taught by the Arbeitsgemeinschaft für Osteosynthesefragen/Association for the Study of Internal Fixation (AO/ASIF), but this concept was abandoned with the introduction of biologic osteosynthesis.34 Van Frank Haasnot and coworkers35 noted that the “appearance of callus is a welcome sign, a sign of a prompt and positive reaction,” thus refuting earlier statements of the advocates of rigid internal fixation.

The concept of biologic osteosynthesis led to the development of the point-contact fixator (PC-Fix), which abandoned interfragmentary compression and bicortical fixation (Fig. 13). Haas and coworkers treated 387 forearm fractures with the PC-Fix and reported a 91.7% union rate within 4 months.36 They reported 7 refractures after 150 (4.7%) plate removals at an average of 13 months. Hertel and colleagues, reporting on results of 83 forearm fractures, found a 91% union rate without stating the time to union.37 They encountered two refractures after plate removal at an average of 10.3 months. Fernandez Dell’Oca and Masliah Galante internally fixed 119 forearm fractures in 80 patients.38 Fractures in 33 patients healed before the 17th week and in 23 after the 17th week. Inadequate radiologic documentation in the remaining 24 patients precluded
There was no mention of refractures. Perren and Buchanan recommended plate removal with the PC-Fix only after 1.5–2.0 years, as the persisting fracture gap may act as a stress riser. Obviously, the switch to plates with almost no plate–bone contact failed to improve the clinical results. Moreover, the timing of plate removal was postponed from 15 months to 18 months for DCP to 18–24 months for the PC-Fix.

Recently, a study has been published that compared the clinical outcome of LC-DC plates to that of PC-Fix in 125 forearm fractures. Leung and Chow found the implants equally effective: 62% of fractures healed by 16 weeks, 82% by 20 weeks, and 93% by 24 weeks. Thus, the reduced contact area, limited unicortical fixation, and abandonment of fixation under compression failed to improve the clinical outcome. Moreover, the reduction in the bone–plate contact area did not solve the problem of delayed healing. To the contrary, it seems that clinically union occurred faster with the DC plates than with the PC-Fix.

In summary, to our knowledge no clinical study has been published proving the superiority of the LC-DCP and PC-Fix plates. It is surprising that despite the oft-cited AO Clinical Investigation and Documentation (AOCID) no clinical study of plated diaphyseal fractures based on hard data has been published in journals with an acceptable impact factor. The use of the less invasive stabilization system (LISS) and the locking compression plate (LCP) seems limited to metaphyseal and epiphyseal fractures.

PC-Fix plates are no longer available, with no reason having been given for discontinuing the product. Also missing is any recommendation regarding which plate to use for internal fixation of diaphyseal forearm fractures.

LC-DC plates and PC-Fix plates, despite their reduction in the plate–bone contact area, have not altered the clinical outcome. This sheds doubt on the theory that interference with the blood supply is the culprit. Results of clinical studies and experimental studies using histomorphometry support our view that stress shielding is the real cause.

**Recent developments**

It is highly improbable that an overall decrease in modulus to reduce stress shielding is the right answer to the current disadvantages of internal fixation. Elastic fixation allowing motion for bending, torsion, and shear as well as compression/distraction is not desirable as it leads to delayed union or non-union.

A fundamentally new approach to remedy bone loss under plates and delayed union must be sought. Rigid plates carry a great percentage of loads relieving the plated bony segment of stimuli needed to maintain bone mass and for posttraumatic osteogenesis (i.e., formation of new bone for bridging the fracture gap). Endosteal bone buildup at both ends of the fracture plate where load is shunted from bone to plate proximally and from plate to bone distally is proof that rigid plates carry a higher percentage of load than bone (Fig. 14).
To remain strong, bone must be dynamically loaded. Moreover, to heal a fracture, nature must sense the absence of bony continuity. Primary bone healing observed under rigid fixation resembles physiologic remodeling. It is a slow process, definitively slower than the process of fracture healing under less rigid plates. The only possible solution to improving fracture healing under plates is, in our opinion, a construct that allows micromotion through the fracture site. Moreover, micromotion must be limited to the axial direction meaning, that the construct must be designed in such a way that it resists bending, torsional, and shear moments. From the clinical successes of the Ilizarov method we know the importance for posttraumatic osteogenesis of dynamic loads transmitted in the axial direction.

Based on the original research done by Goodship and Kenwright that documented the beneficial influence of micromotion in the axial direction on posttraumatic osteogenesis, we developed plates that allow increased micromotion in the axial direction only while maintaining adequate shear, bending, and rotational rigidity. Insertion of elastic cushions made from polymethylmethacrylate (PMMA) between the screw shank and the wall of the oval screw holes in the plate allow compression of the fracture gap under loading and rebound after unloading (Fig. 15). In essence, these plates allow true dynamic compression at the fracture site. Polar flexural rigidity profile (PFRP) testing of beagle femors fixed with the axially flexible plates (AFPs) showed more rapid healing than conventional plates. Periosteal callus helped in the radiologic assessment of progression of consolidation, and histologic examinations showed early bridging between the fragments.

We then asked whether the use of a biodegradable material for manufacturing the cushions would further improve our results. Based on the design principles of the beagle AFP, a new axially compressible plate (ACP) for use in humans was designed and manufactured (Fig. 16). Polylactic acid (PLA) was recommended by the manufacturer for the inserts, claiming that a change in physical characteristics would not occur before 4–6 months. We hypothesized that as the PLA inserts degrade the load transmission through the healing bone would increase to a point of completely eliminating load sharing by the plate.

Unfortunately, in canine pilot studies, the biodegradable inserts did not perform up to our expectations. Nevertheless, the plates did show a decrease in stress shielding when compared to LC-DC plates.

Further creep testing of the inserts confirmed the poor compression creep properties of the PLA insert. At this time, it appears that the current PLA inserts do not have adequate (compressive) creep properties, thereby allowing excessive micromotion at the fracture site during the union phase with subsequent delayed union and excessive callus formation. The successful outcome of this research would be a plate design that...
reduces stress shielding and allows micromotion at the fracture site to more closely mimic biologic healing.

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