

Invited review article

Internal plate fixation of fractures: short history and recent developments

HANS K. UHTHOFF¹, PHILIPPE POITRAS², and DAVID S. BACKMAN²

¹Bone and Joint Research Laboratory, Faculty of Medicine, Division of Orthopaedic Surgery, University of Ottawa, 1321-451 Smyth Road, Ottawa, Ontario K1H 8M5, Canada

²Orthopaedic Biomechanics Laboratory, Faculty of Medicine, Division of Orthopaedic Surgery, University of Ottawa, Ottawa, Ontario, Canada

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 polylactide (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 fragment 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

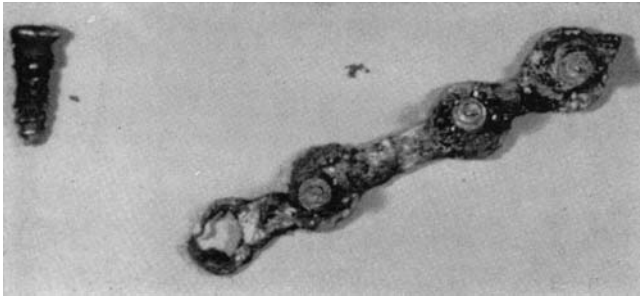


Fig. 1. Lane's plate abandoned because of corrosion (1895). (From Bechtol CO, Fergusson AB, Laing PE. *Metals and Engineering in Bone and Joint Surgery*. Williams & Wilkins; Baltimore: 1959. p. 20, with permission)



Fig. 2. Lambotte's plate (1909) is thin, round, and tapered at both ends

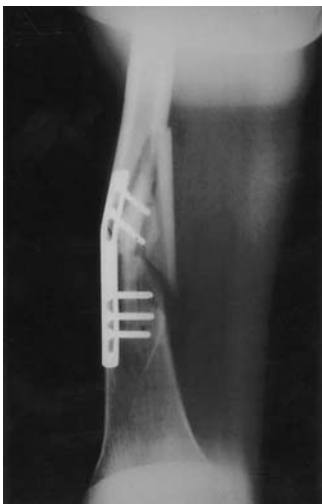


Fig. 3. Structural instability of Eggers' plate

(Fig. 7). The plate was heavier and thicker (4.5mm) 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



Fig. 4. Danis' plate (1949) called "coaptateur" suppresses interfragmentary motion and increases stability of fixation through interfragmentary compression achieved by tightening the side screw

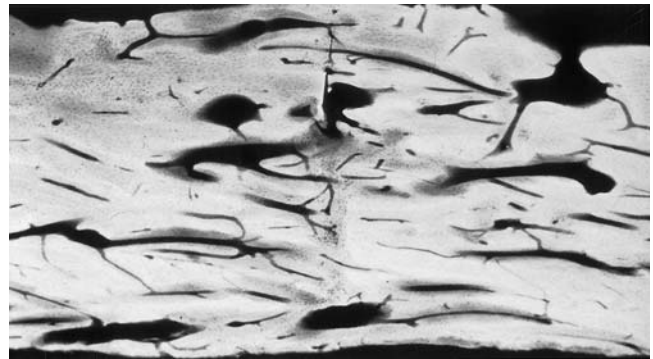


Fig. 5. Primary bone healing

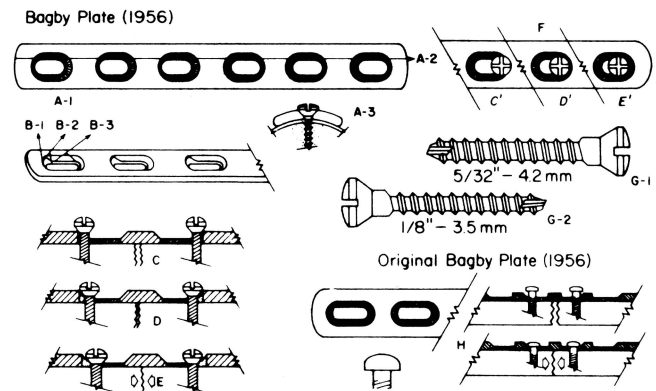


Fig. 6. Bagby and Janes' (1956) oval holes designed for interfragmentary compression during screw tightening. (From Uthoff HK. *Current Concepts of Internal Fixation of Fractures*. Heidelberg: Springer-Verlag; 1980. p. 175, with permission of Springer Science and Business Media)

interpreted as a sign of instability. "The appearance of callus after plate fixation may be an indicator for an unknown degree of instability."⁸

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),⁹ was claimed to have been developed without the knowledge of Bagby and Jane's invention. Yet in 1967 Schenk and Willenegger,¹⁰ both members of a Swiss group of investigators, made

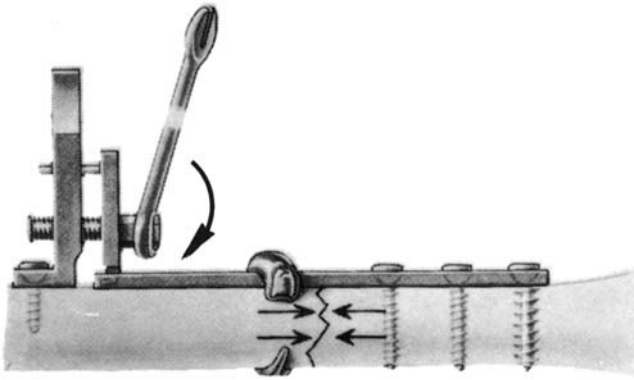


Fig. 7. Müller's plate design (1965) achieves interfragmentary compression by tightening a tensioner that is temporarily anchored to the bone and the plate. (From Sequin F, Texhammar R. *AO/ASIF Instrumentation*. Heidelberg: Springer-Verlag; 1981. p. 72, with permission of Springer Science and Business Media)

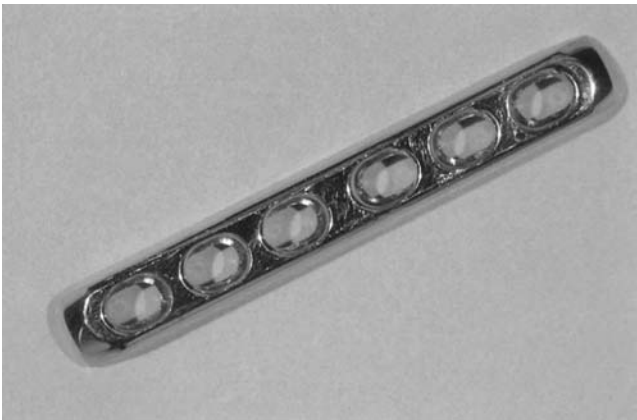


Fig. 8. Dynamic compression plate (DCP) incorporates specially designed oval holes similar to Bagby and Janes' invention to compress bony fragments during screw tightening

reference to the compression technique advocated by Bagby and Janes.⁶ Although this plate was called a dynamic compression plate (DCP) only one-time static compression could be obtained.

The advantages of the DCP included low incidence of malunion, stable internal fixation, and no need for external immobilization, thus allowing immediate movement of neighboring joints. Meticulous surgical technique and an excellent teaching program further contributed to the advantages and success of this plating system.

Despite the obvious advantages, the developers of the DCP still looked for improvement in the design. This was probably because of certain disadvantages with the DCP that included delayed union as well as persistence of a microscopically detectable fracture gap that acted as a stress riser after plate removal. Cortical



Fig. 9. Cortical bone loss under rigid plating

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.⁷ A study by Kessler and colleagues showed that plate removal at an average of 20.1 months still resulted in refracture.¹¹ 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.¹² 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.⁸ 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.⁸

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%.¹³ The subsequent development of the point contact fixator¹⁴ 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.¹⁵ 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%.¹³

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.¹⁶ 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.”¹⁶

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 associates⁸ denied the role of stress shielding, refuting the results of many researchers extending over 25 years.^{17–22} 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 collaborators²³ 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.²⁴ 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 data²⁴ assessing necrosis in the periosteal and endosteal halves of the cortex when using both contact and railed



Fig. 10. Railed plates (*left*) have limited bone–plate contact area compared to contact plates (*right*)

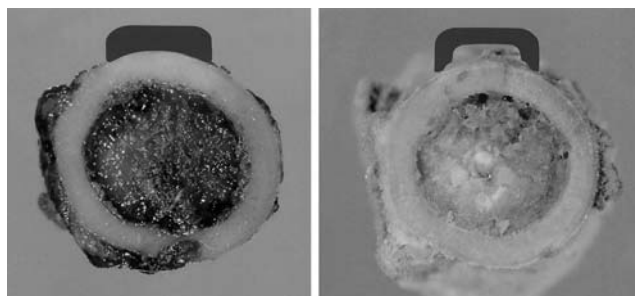


Fig. 11. Decreased cortical perfusion is more pronounced with increased bone–plate contact with contact plates (*left*) versus railed plates (*right*)

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.²⁴

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.⁸ 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 less²⁴ (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.

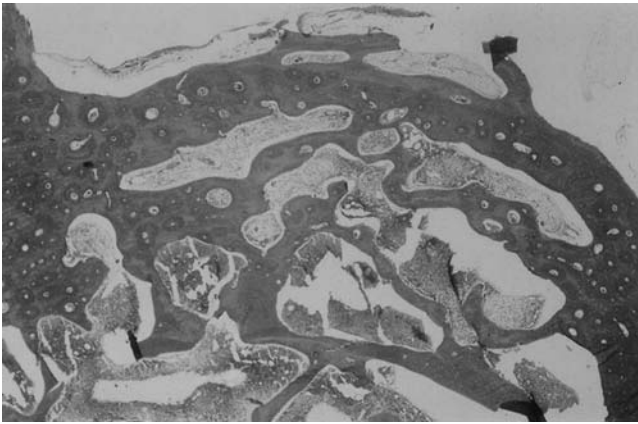


Fig. 12. Porosis is more pronounced in the endosteal half of the cortex of a beagle femur plated with six-hole stainless steel rigid plates. HPS $\times 25$

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

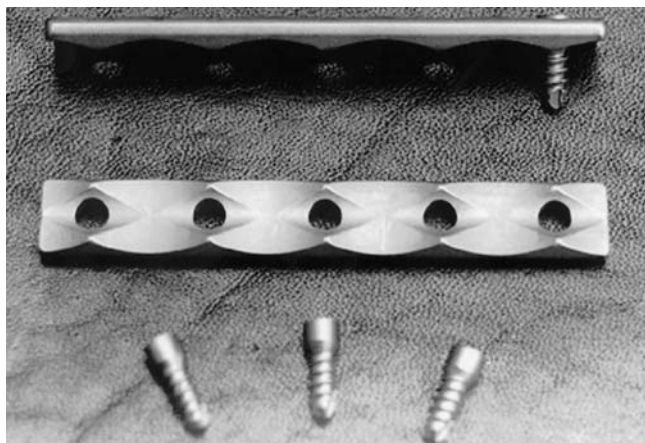


Fig. 13. Point-contact fixator (PC-Fix) acts like a fixator as the monocortical screws lock into the plate achieving angular stability and preventing the bone to be pulled toward the plate. (From Fernandez Dell'Oca AA, Tepic S, Frigg R, Meisser A, Haas N, Perren SM. Treating forearm fractures using an internal fixator: a prospective study. *Clin Orthop* 2001;389:196–205, with permission of Lippincott Williams & Wilkins)

an assessment. 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.³⁹ 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.⁴⁰ 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 often-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.⁴¹

PC-Fix plates are no longer available, with no reason having been given for discontinuing the product. Also



Fig. 14. Radiograph of mongrel canine femur plated with six-hole limited-contact dynamic compression plate (LC-DCP) at 16 weeks following harvest. Endosteal buildup at both ends of the plate is indicative of a high percentage load transferred through the plate

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^{17–19} and experimental studies²⁴ 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.⁴²

Based on the original research done by Goodship and Kenwright⁴³ 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.⁴⁴ 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

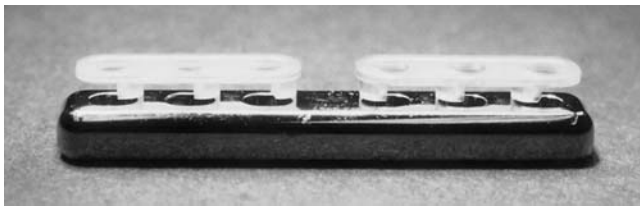


Fig. 15. Axially flexible plate (AFP) for beagle femors incorporates polymethylmethacrylate (PMMA) cushions between plate and screw head to allow increased micromotion in the axial direction only

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.⁴⁴ Periosteal callus helped in the radiologic assessment of progression of consolidation, and histologic examinations showed early bridging between the fragments.⁴⁴

We then asked whether the use of a bioresorbable 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.⁴⁵

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

The PLA inserts failed prematurely, leading to unstable fixation. The premature failure was due to poor mechanical properties of the insert or early degradation (or both). We first investigated the degradation properties of the PLA inserts by immersing the inserts in a phosphate-buffered saline solution at body temperature for 2 weeks. Significant deformation of the inserts was observed after only 5 days. This may be due to internal stresses introduced during the injection molding process or to early degradation.

Further creep testing of the inserts confirmed the poor compression creep properties of the PLA insert.⁴⁶ 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

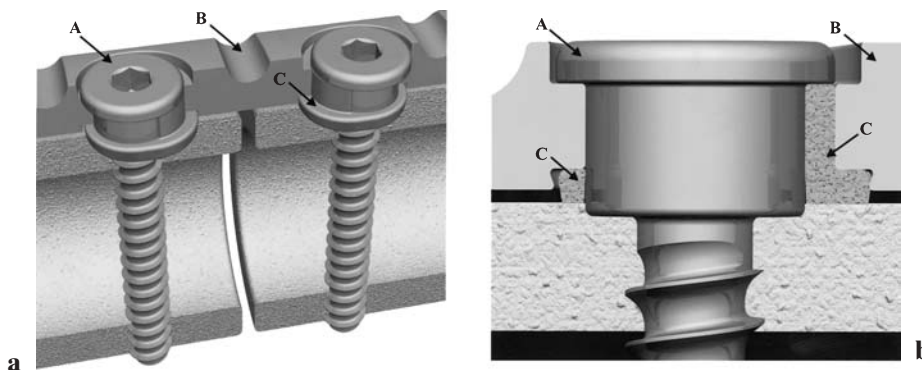


Fig. 16. **a** Axially compressible plate (ACP). Screws (A) and outer shell (B) are titanium, and inserts (C) are polylactic acid (PLA). Inserts allow increased micromotion in the axial plane during union and reduced stress shielding during remodeling as the inserts degrade. **b** Cross section of ACP screw (A), outer shell (B), and insert (C). Design allows screw head to subside slightly in bone to reduce plate–bone contact

reduces stress shielding and allows micromotion at the fracture site to more closely mimic biologic healing.

References

- Lane WA. Some remarks on the treatment of fractures. *BMJ* 1895;1:861–3.
- Lambotte A. Technique et indication des prothèses dans le traitement des fractures. *Presse Med* 1909;17:321.
- Sherman WO. Vanadium steel bone plates and screws. *Surg Gynecol Obstet* 1912;14:629–34.
- Eggers GWN. Internal contact splint. *J Bone Joint Surg Am* 1948;30:40–52.
- Danis R. *Théorie et pratique de l'ostéosynthèse*. Paris: Masson; 1949.
- Bagby GW, Janes JM. The effect of compression on the rate of fracture healing using a special plate. *Am J Surg* 1958;95:761–71.
- Müller ME, Allgöwer M, Willenegger H. Compression fixation with plates. In: *Technique of internal fixation of fractures*. Berlin: Springer; 1965. p. 47–51.
- Perren SM, Cordey J, Rahn BA, Gautier E, Schneider E. Early temporary porosis of bone induced by internal fixation implants: a reaction to necrosis, not to stress protection? *Clin Orthop* 1988;232:139–51.
- Perren SM, Russenberger M, Steinemann S, Müller ME, Allgöwer M. A dynamic compression plate. *Acta Orthop Scand* 1969;Suppl 125:31–41.
- Schenk R, Willenegger H. Morphological findings in primary fracture healing. In: Krompecher S, Kerner E, editors. *Callus formation symposium on the biology of fracture healing*. Budapest: Akadémiai Kiadó; 1967. p. 75–86.
- Kessler SB, Deiler S, Schiffl-Deiler M, Uthhoff HK, Schweiberer L. Refractures: a consequence of impaired local bone viability. *Arch Orthop Trauma Surg* 1992;111:96–101.
- Berkin CR, Marshall DV. Three-sided plate fixation for fractures of the tibial and femoral shafts: a follow-up note. *J Bone Joint Surg Am* 1972;54:1105–13.
- Gautier E, Perren SM. Die limited contact dynamic compression plate (LC-DCP): Biomechanische Forschung als Grundlage des neuen Plattendesigns. *Orthopade* 1992;21:11–23 (in German).
- Tepic S, Perren SM. The biomechanics of the PC-Fix internal fixator. *Injury* 1995;26(Suppl 2):S-B5–10.
- Field JR, Hearn TC, Caldwell CB. Bone plate fixation: an evaluation of interface contact area and force of the dynamic compression plate (DCP) and the limited contact-dynamic compression plate (LC-DCP) applied to cadaveric bone. *J Orthop Trauma* 1997;11:368–73.
- Jain R, Podworny N, Hupel TM, Weinberg J, Schemitsch EH. Influence of plate design on cortical bone perfusion and fracture healing in canine segmental tibial fractures. *J Orthop Trauma* 1999;13:178–86.
- Akeson WH, Woo SLY, Rutherford L, Coutts RD, Gonsalves M, Amiel D. The effects of rigidity of internal fixation plates on long bone remodeling. *Acta Orthop Scand* 1976;47:241–9.
- Slätis P, Paavolainen P, Karaharju E, Holström T. Structural and biomechanical changes in bone after rigid plate fixation. In: Uthhoff HK, editor. *Current concepts of internal fixation of fractures*. Berlin: Springer; 1980. p. 291.
- Strömberg L, Dalén N, Låftman P, Sigurdsson F. Atrophy of cortical bone caused by rigid plates and its recovery. In: Uthhoff HK, editor. *Current concepts of internal fixation of fractures*. Berlin: Springer; 1980. p. 289–90.
- Uthhoff HK, Bardos DI, Liskova-Kiar M. The advantages of titanium alloy over stainless steel plates for the internal fixation of fractures: an experimental study in dogs. *J Bone Joint Surg Br* 1981;63:427–34.
- Uthhoff HK, Dubuc FL. Bone structure changes in the dog under rigid internal fixation. *Clin Orthop* 1971;81:165–70.
- Uthhoff HK, Finnegan M. The effects of metal plates on post-traumatic remodelling and bone mass. *J Bone Joint Surg Br* 1983;65:66–71.
- Gunst MA, Suter C, Rahn BA. Bone perfusion after plate osteosynthesis: a study of the intact rabbit tibia with disulfon blue vital staining. *Helv Chir Acta* 1979;46:171–5 (in German).
- Uthhoff HK, Boisvert D, Finnegan M. Cortical porosis under plates: reaction to unloading or to necrosis? *J Bone Joint Surg Am* 1994;76:1507–12.
- Jaworski ZFG, Liskova-Kiar M, Uthhoff HK. Regional disuse osteoporosis and factors influencing its reversal. In: Uthhoff HK, editor. *Current concepts of internal fixation of fractures*. Berlin: Springer; 1980. p. 17–26.
- Uthhoff HK, Jaworski ZFG. Bone loss in response to long-term immobilization. *J Bone Joint Surg Br* 1978;60:420–9.
- Korvick DL, Newbrey JW, Bagby GW, Pettit GD, Lincoln JD. Stress shielding reduced by a silicon plate-bone interface. *Acta Orthop Scand* 1989;60:611–16.
- Perren SM. Evolution of the internal fixation of long bone fractures: the scientific basis of biological internal fixation; choosing a new balance between stability and biology. *J Bone Joint Surg Br* 2002;84:1093–110.
- Andersen LD, Sisk D, Tooms RE, Park WI III. Compression-plate fixation in acute diaphyseal fractures of the radius and ulna. *J Bone Joint Surg Am* 1975;57:287–97.
- Chapman MW, Gordon JE, Zissimos AG. Compression-plate fixation of acute fractures of the diaphyses of the radius and ulna. *J Bone Joint Surg Am* 1989;71:159–69.
- Hertel R, Pisan M, Lambert S, Ballmer FT. Plate osteosynthesis of diaphyseal fractures of the radius and ulna. *Injury* 1996;27:545–8.
- McKee MD, Seiler JG, Jupiter JB. The application of the limited contact dynamic compression plate in the upper extremity: an analysis of 114 consecutive cases. *Injury* 1995;26:661–6.
- Gupta R, Raheja A, Sharma V. Limited contact dynamic compression in diaphyseal fractures of the humerus: good outcome in 51 patients. *Acta Orthop Scand* 2000;71:471–4.
- Gerber C, Mast JW, Ganz R. Biological internal fixation of fractures. *Arch Orthop Trauma Surg* 1990;109:295–303.
- Van Frank Haasnoot E, Münch TWH, Matter P, Perren SM. Radiological sequences of healing in internal plates and splints of different contact surface to bone (DCP, LC-DCP and PC-Fix). *Injury* 1995;26(Suppl 2):S-B28–36.
- Haas N, Hauke C, Schütz M, Kääh M, Perren SM. Treatment of diaphyseal fractures of the forearm using the point contact fixator (PC-Fix): results of 387 fractures of a prospective multicentric study (PC-Fix II). *Injury* 2001;32:S-B-51–62.
- Hertel R, Eijer H, Meisser A, Hauke C, Perren SM. Biomechanical and biological considerations relating to the clinical use of the point contact-fixator: evaluation of the device handling test in the treatment of diaphyseal fractures of the radius and/or ulna. *Injury* 2001;32:S-B-10–14.
- Fernandez Dell'Oca AA, Masliah Galante R. Osteosynthesis of diaphyseal fractures of the radius and ulna using an internal fixator (PC-Fix): a prospective study. *Injury* 2001;32:S-B-44–50.
- Perren SM, Buchanan JS. Basic concepts relevant to the design and development of the point contact fixator (PC-Fix). *Injury* 1995;26(Suppl 2):S-B-1–4.
- Leung F, Chow SP. A prospective, randomized trial comparing the limited contact dynamic compression plate with the point contact fixator for forearm fractures. *J Bone Joint Surg Am* 2003;85:2343–8.
- Schütz M, Südkamp NP. Revolution in plate osteosynthesis: new internal fixator systems. *J Orthop Sci* 2003;8:252–8.
- Ilizarov GA. Clinical application of the tension-stress effect for limb lengthening. *Clin Orthop* 1990;250:8–26.

43. Goodship AE, Kenwright J. The influence of induced micro-movement upon the healing of experimental tibial fractures. *J Bone Joint Surg Br* 1985;67:650–5.
44. Foux A, Yeadon AJ, Uthhoff HK. Improved fracture healing with less rigid plates: a biomechanical study in dogs. *Clin Orthop* 1997;339:232–45.
45. Backman D, Uthhoff H, Poitras P, Schwamberger A. Mechanical performance of a fracture plate incorporating bioresorbable inserts. *J Bone Joint Surg Proceed Br* 2004;86(Suppl III): 300.
46. Poitras P, Backman D, Uthhoff HK, Schwamberger A. Creep properties of a bioresorbable polymer for an axially compressible fracture plate. Presented at the 5th Combined Meeting of the Orthopaedic Research Societies of Canada, USA, Japan and Europe. Banff, AB. October 2004.