BIOLOGICAL INTERNAL FIXATION INTERFACE BETWEEN BIOLOGY AND BIOMECHANICS

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INTRODUCTION: Biology and biomechanics of treatment and healing of fractures interact and are closely linked. The model of biological internal fixation serves to highlight some of these links.

The fracture disrupts the stiffness of the bone and results in painful loss of limb function. Soft tissue complications such as reflex dystrophy are attributed to spontaneous or iatrogenic immobilization of articulations with consecutive dysfunction of blood supply, patchy bone loss and pain. Internal fixation (surgical stabilization using implants) restores continuous stiffness, abolishes pain and allows early mobilization. Mobilization of the articulations prevents soft tissue complications that were the rule after extensive external splinting of articulations by plaster cast. Still, internal fixation, especially when carried out without care, produces damage to the vascular support of bone and soft tissues.

Internal fixation has recently undergone a basic evolution, which sets a new balance between requirements of stabilization and of damage to blood supply. On the one hand damage to the blood supply is minimized, on the other hand induction as well as tolerance of a certain degree of instability as a result of a more flexible type of fixation is an objective. In the following some aspects of biological internal fixation are discussed based on today’s understanding of biology, biomechanics and mechanics. The design of implants and even more so their surgical application are important players with a potential to support striving for a better and safer fracture treatment.

EVOLUTION OF INTERNAL FIXATION: The means of early internal fixation were selected to provide absolute immobilization of the fracture with the goal of immediate restoration of mobility of the articulations in mind[1]. “To allow the fracture to heal in a mechanically neutral environment” precise reduction and application of compression across the fracture plane were the rule. A new type of fracture healing consisting of direct internal remodeling without scar (callus) tissue formation has been observed. It was called “primary bone healing[2]. This type of healing was seen after absolutely stable fixation. It consists, in respect to timing and pattern, of only the last phase of natural healing i.e. the final internal remodeling restoring the original integrity of bone. A prerequisite of primary healing is close and stable adaptation of the fracture planes maintained throughout the period to solid bridging. As long as the applied compression locally exceeds the traction produced e.g. by bending, the fracture plane remains in uninterrupted contact. To achieve such contact the fracture planes were stabilized using the technique of interfragmentary compression[1]. Plates applied as splints across the fracture protected the screw fixation from excessive functional load. Combinations of the two techniques were the rule, pure splinting by plate without compression was considered to be inappropriate.

The observation of callus after internal fixation, which aimed at absolute stability of fixation, was considered to indicate that the goal of stabilization was not achieved. Callus fell into disrepute though it was often callus that ensured the healing in difficult situations.

Beside screw and plate fixation alternative methods of fracture fixation consist in application of either an intramedullary rod[3] or of an external rod connected to the bone by means of transcutaneous pins. Nail or external fixator result in pure splinting that is they reduce but do not abolish mobility at the fracture. The usual pattern of healing after splinting is indirect healing with formation of callus followed by internal remodeling.

Any surgical intervention results in either periosteal (with plate fixation) or endosteal (with nail fixation) damage to the blood supply to bone. The damage to blood supply is in part the result of the surgical preparation (periosteal stripping, medullary reaming etc.) in part the damage is produced by the contact of the implant, a contact, which impedes the inflow and/or outflow of blood to and from bone. Beside the advantages of restoring immediate function of the articulations the shortcoming of absolute stability of fixation is that no relevant callus is induced. Callus appears earlier than internal remodeling and its location outside the bone provides better leverage than the direct connection from bone to bone.

BIOLOGICAL INTERNAL FIXATION: is a kind of surgical stabilization of the fracture that addresses the two shortcomings mentioned[4].The surgical procedure is geared towards minimizing damage to blood supply by avoiding excessive surgical approach abstaining from perfect reduction and absolute stability of fixation. Flexible splinting without interfragmentary compression then provides the natural stimulus for callus production.

The biological internal fixation using the internal fixator principle demonstrates an indirect healing pattern and a low infection rate, which was solidly proven with a very high follow up rate of 97%[5]. The fact that the ends of fracture fragments are not closely adapted allows observing the progress of union. The radiological pattern of healing is similar to the one seen after external fixator treatment. The rate of mechanical failure of screws and plates is exceptionally low in spite of the fact that pure titanium was used as implant material. Pure titanium is known for its low corrosion rate and exceptionally good biological tolerance, but its limited ductility has surprised surgeons that were accustomed to steel, which deforms markedly before breaking occurs. The use of internal fixators with screws that lock within the "plate" alleviates the screw thread from torque and axial tension. The reason for this behavior is that the Morse cone seat locks tightly upon minimal axial traction applied.

The low infection rate observed in clinical studies is backed by animal research testing the local resistance to infection with different designs, materials and procedures of internal plate fixation[6]. The amount of colony forming human pathogenous staphylococci aureus to produce clinical infection was assessed. Between conventional steel DCP (dynamic compression plate) and PC-Fix (point contact fixator) made of pure titanium the latter resisted a 450 times higher load of staph. aureus.

The radiological follow up of adapted and compressed fractures does not allow detecting the process of healing within the fracture but is based on the absence of signs of disturbed healing such as cloudy callus indicating that absolute stability was not achieved[7]. The radiological pattern of healing after non adapted and non compressed splinting appears to be slow at first glance. Still, the successful very early removal of the internal fixator in clinical animal studies seems to prove the quick recovery of load bearing capacity of the fracture after flexible internal fixator splinting[8].

In conclusion biological internal fixation demonstrates a fascinating link between biology and biomechanics after surgical fracture stabilization. It seems to open a promising effect of a newly adapted balance between requirements of stability for painless function, those of minimizing biological damage and taking advantage of callus induction by a small degree of elastic instability.

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