ACTA CHIRURGIAE ORTHOPAEDICAE
ET TRAUMATOLOGIAE ČECHOSL., 79, 2012, p. 9–10

Debunking the Myth of the Periosteum

Konec mýtu o periostu

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The foundations of Orthopaedics are a great deal better supported with evidence today than previously. Nonetheless, superstitions and myths have not completely disappeared. One of them is the role of the periosteum in fracture healing.

The anatomy and physiology of the periosteum under normal circumstances have been studied for many a decades, and its role in the growth and development of tubular bones well defined. The evidence regarding its participation in fracture healing in non-rigidly immobilized fractures cannot be questioned at this time. However, extrapolations on its participation in rigidly immobilized fractures are open to question. I submit that under rigid immobilization conditions the periosteum plays a minimal role and that rather than assisting in expediting healing, it retards it (1, 2, 3, 4). (Fig. 1).

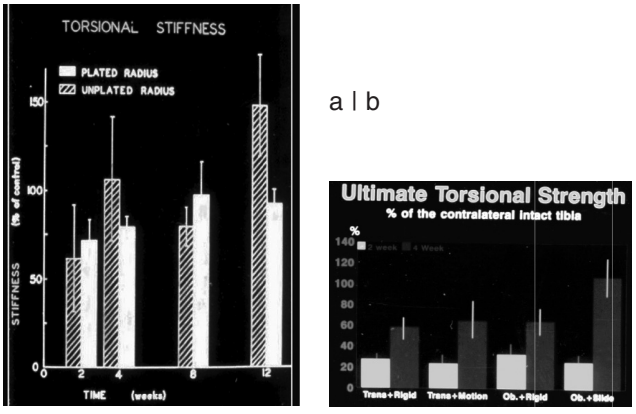

Fig. 1a – Data from animal experimental study demonstrating that the strength of the union in the nonimmobilized fractured bone is significantly greater than that in the immobilized bone. b – The torsional strength of the most vertically unstable fracture is greater than that observed in the more stale ones.

In the same category of "superstitions" is the role of the hematoma in fracture healing. Rather than converting into bone, the hematoma is likely to undergo resorption in order to allow the process of enchondral ossification to take place. Likewise, the importance of coverage of bone with thick muscles being important for osteogenesis suggesting that the myoblast is capable of undergone osteoblastic metaplasia, which is not the case. The theory loses further value as one observes that fractures with minimal or no muscle coverage heal as readily as fractures in other bones with large muscle coverage as exemplified by fractures of the metacarpals, metatarsals, phalanges, the clavicle, the fibula as well as the ribs.

It has become an almost sacred obsession to accept the importance the periosteum has in fracture healing. This is true only if rigid immobilization of the fragments is not created. In the nonimmobilized fracture of a long bone, peripheral callus forms as a result of the intervention of capillaries from the periosteum and the surrounding tissues. Studies that we and others have conducted have demonstrated that in the nonimmobilized diaphyseal fracture this massive invasion of capillaries begins to take almost immediately after the initial insult (Fig. 2).

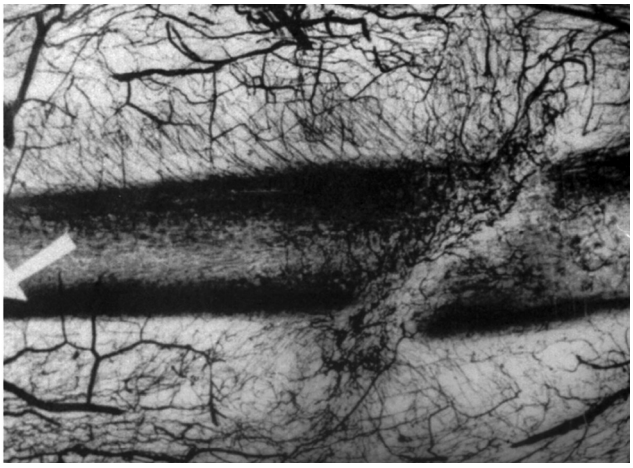

Fig. 2. Motion at the fracture site is the determining feature dictating the presence of peripheral callus. The most desirable motion is the "pistoning" one to be responsible for the inflammatory reaction, manifested by the formation of capillaries. This motion is greater when the fracture is of an oblique or comminuted nature (2). (Fig. 4a and b).

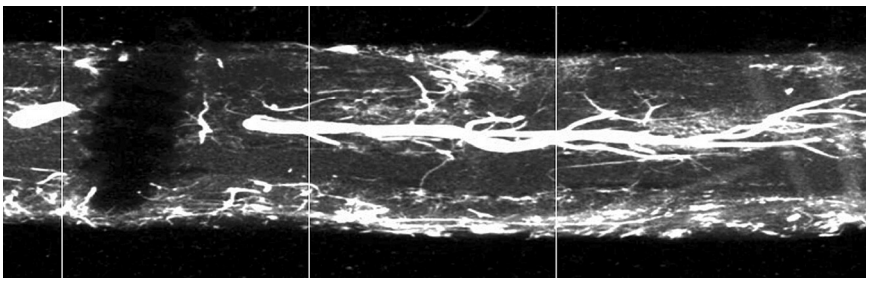

Fig. 3. If the fracture is rigidly immobilized peripheral callus does not form, but the medullary blood supply recovers quickly.

The newly created capillaries are probably a response the irritation that motion between the fragments creates. (3) The perithelial and endothelial cells of the capillaries undergo metaplasia into osteoblasts. Therefore the greater number of capillaries the greater the osteoblastic activity. (Fig. 4a, b and c).

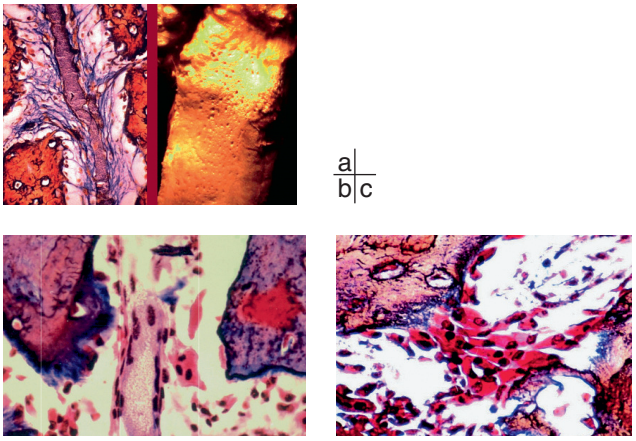

Fig. 4a – (Left) composite photograph of a capillary running in a vertical direction into the newly formed bone. (Right) The surface of the bone at the level of a healed fracture (Right) illustrating the points of entry and exit of the capillaries; b – (Center) closer view of a capillary and its perithelial and epithelial cells undergoing osteoblastic metaplasia; c – illustration of the osteoblastic metaplasia of the capillary cells.

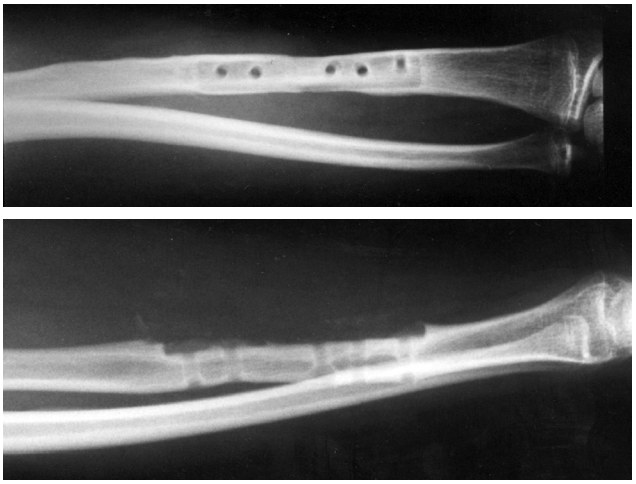

Fig. 5. As long as the fracture is rigidly immobilized the healing is slowed down and not accompanied with peripheral callus. The cortex under the plate, deprived of significant stresses, atrophies.

The idea that plates that have projections on their bone side protect the periosteum is an illusion. With or without projections, peripheral callus does not form. The same applies to long plates applied with the so-called minimally invasive techniques. (Fig. 5)

Based on the above presented evidence one must conclude that the role of the periosteum in fracture healing is dictated by the environment into which the healing process unfolds. What determines the presence or absence of peripheral callus is motion at the fracture site. The rigid immobilization of a fracture silences the periosteum, in such a manner that healing is of an endosteal nature, and mechanically weaker. Since plates, nails and external fixator have very definite indications, their role must be clearly understood. (5) Their mechanical and practical advantages in millions of instances are partially offset by their biological disadvantages. These fractures eventually heal from endosteal activity - not from periosteal callus. The endosteal callus is weak and forms very slowly.

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