Bone Vascularization and Its Effect on Fracture Healing

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INTRODUCTION

"In operative procedures the surgeon tries to find a convenient and favourable way to reach the organ to be treated. Especially in the extremities he has to use planes of cleavage chosen in such a way, that the tissues are left intact as a unit as much as possible. Only here and there on this way—which is sometimes called the physiological approach—impediments are to be found, mainly formed by neuro-vascular bundles running between the muscles and entering them. Each muscle itself has a vascular system existing more or less on its own. This fact makes it possible for this moving organ to change form and place with respect to its surroundings. . .

Approaching the bone the surgeon may notice that the vessels of muscle and bone are a vascular unit. This is most clearly observed at the origin or insertion of the muscles. It is the first indication of a vascular supply entirely typical of and peculiar to bone. As a special manifestation of the mesoderm bone appears to have its vascular system in common with the suraounding mesodermal structures. This does not alter the fact that big arteries enter the bone and form an arterial pattern, which is limited to it. . . During the period of growth the periosteum is the organ which builds up the cortex layer by layer and causes its growth in thickness. Accordingly, this organ has an extensive vascular supply originating direct from the surrounding mesodermal tissue. The periosteum gives again and again vessels to the structure it builds up. The vessels are incorporated in the newly formed bone tissue and consequently keep running almost parallel to the periosteum. These are the Haversian vessels which form an anastomosing network in the cortex. This network is the continuation of the periosteal vascularzation, which in its turn forms again a unit with the vessels of the surrounding tissues. Therefore the vascular supply of the bone cannot be simply isolated from its surroundings.

For the surgeon an important question is how to handle the bone without seriously disturbing this physiological vascular unit. In order to find an answer to this question, he must have a clear notion of the entire vascular pattern of the bone." [6]

The healing of fractures is determined to a large extent by three factors: the quality of reduction, the degree of fixation, and the regional blood supply. Each has a bearing on bone healing. Poor reduction increases the gap between fragments

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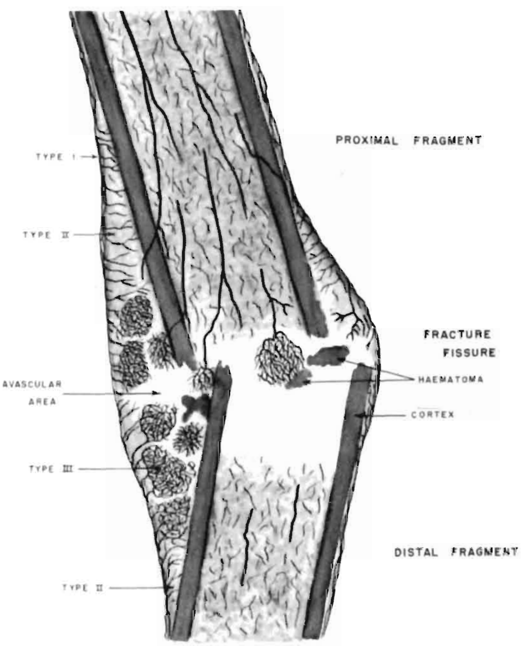


Figure 1. Diagram showing vascularization types I, II, and II. (From Vascularization in the Heating of Fracture by Dr. L. J. L. Koekenbery, used by permission.)

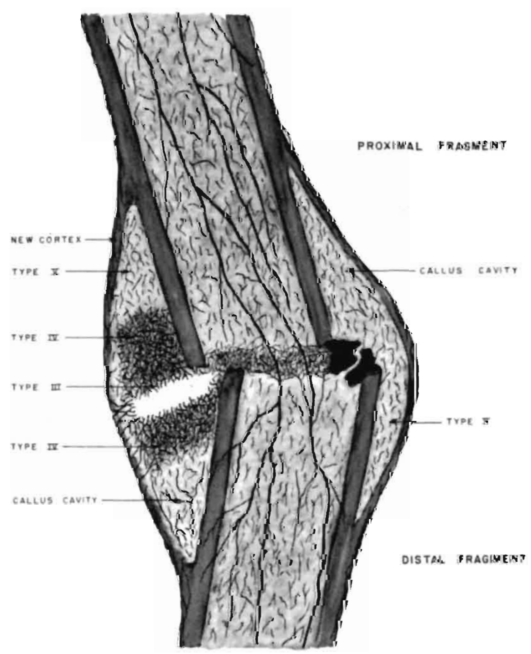


Figure 2. Diagram showing vascularization types III, IV, and V. (From Vascularisation in the Heating of Fracture by Dr. L. J. L. Koekenbery, used by permission.)

which increases the callus requirement and prolongs healing. Poor or incomplete fixation of the fragments may convert the specialized cells of the callus into scar tissue and thereby cause non-union. A poor blood supply may delay the healing process or lead to non-union.[3]

NORMAL BONE VASCULARIZATION

Before discussing the vascularization of the fracture callus, it is important to first understand the normal bone vascularization . The work of Johnson has shown that the blood supply to long bones comes from two major sources: (1) the nutrient arteries which enter the diaphyseal cortex through foramina and (2) the periosteal arteries.

The nutrient artery is the main vessel to supply the blood to the shaft of long bones. It is responsible for irrigating all of the marrow cavity and the inner two-thirds to three-quarters of the cortex. The periosteal vessels supply the outer cortex which is not supplied by the medullary vessels.[9]

This illustrates that the normal arterial blood supply of the medulla and of the major thickness of the cortex is supplied by the medullary circulation, but at the same time there is an anastomosis between existing arterioles of both the medullary and the periosteal systems. In the event of injury blood can be shunted quickly from one system to another with marked hypertrophy of these vessels occurring. If the medullary vessels are destroyed, they quickly regenerate to a degree greater than normal.[8]

GENERAL CONCEPTS OF CALLUS FORMATION

Before discussing fracture healing in more detail, it is important to know the general concepts of callus formation. Callus formation is thought to occur in six stages:

1. Formation of a clot in the hematoma around the ends of the fragments. This is the result of the original insult.
2. Invasion of the clot by fibroblasts and capillaries with gradual transformation into a mass of immature connective tissue. This stage starts

within 48 hours.
3. Cellular differentiation of osteoblasts with formation of osteoid and proliferation of chondroblasts, fibroblasts, and varying amounts of chondroid matrix. This is the stage of callus formation.
4. Replacement of devitalized bones from the ends of the fracture fragment. The devitalized bone ends are absorbed and replaced by new bone.
5. Maturation of the callus and osteoid tissue into calcified bone trabeculae.
6. Consolidation and remodeling of the newly formed bone with concominant decrease in size of the bony mass. Complete return to normal requires about a year.[4]

VASCULARIZATION OF THE FRACTURE CALLUS

Vascularization of the fracture callus is one of the important factors that controls fracture healing. Certain investigators describe the vascularization of the fracture callus occuring in five different stages or types:

The first type might be called the stage of hyperemia. The hyperemia is not limited to the vasculature of the fracture callus, but includes all tissues of the entire extermity. This hyperemia lasts about two weeks and accounts for the original swelling associated with a fracture. (See Fig. 1)

Type II vascularization is characterized by very small vessels running parallel to each other and growing perpendicular to the cortex. This type of vascularization is found at the periphery of the hematoma and at the level of the fracture fissure. During the first two weeks of fracture repair, these vessels are found only sporadically, but they are found more abundantly during the later stages. (See Fig. 1)

Type II vascularization gives rise to type III vascularization. Type III vascularization is characterized by abundantly ramified vessels which appear like thick "bushes." Each "bush" has only one afferent vessel which is usually a type II vessel. After about four weeks, type III vascularization dominates the callus and continues so for a greater part of the healing time. (See Fig. 1 & 2)

After about five weeks type IV vascularization is seen consisting of a network with small meshes of very thin walled sinuses. Type IV vascularization is actually a smaller gradation of type III vascularization. Vascularization of this type is

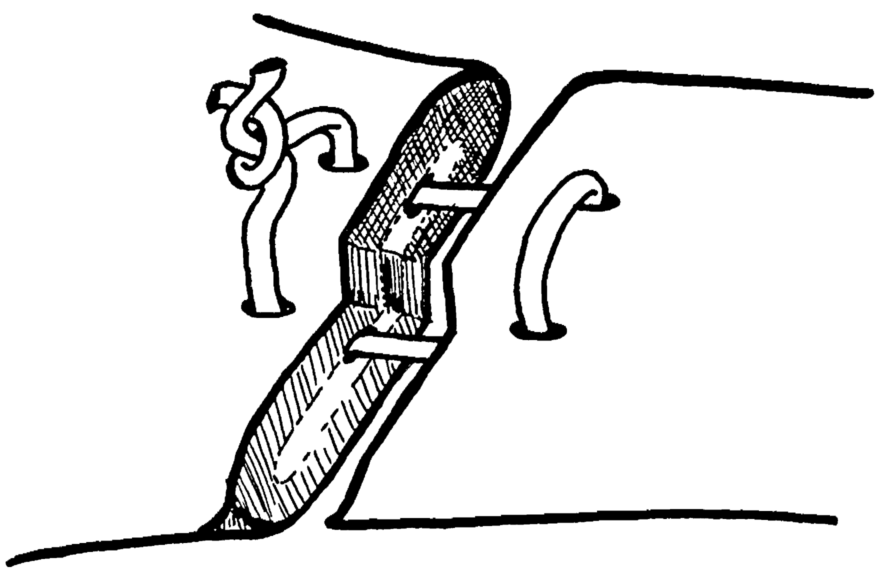


Figure 3. If wire must be used, it should be placed in a suture pattern similar to that in the diagram.

so dense that the spaces between the vessels are only a little wider than the lumen of the vessels. (See Fig. 2)

Type IV vascularization gradually changes; the meshes become wider and more elongated. This is called type V vascularization and resembles the normal medullary vascularization.[6] (See Fig. 2)

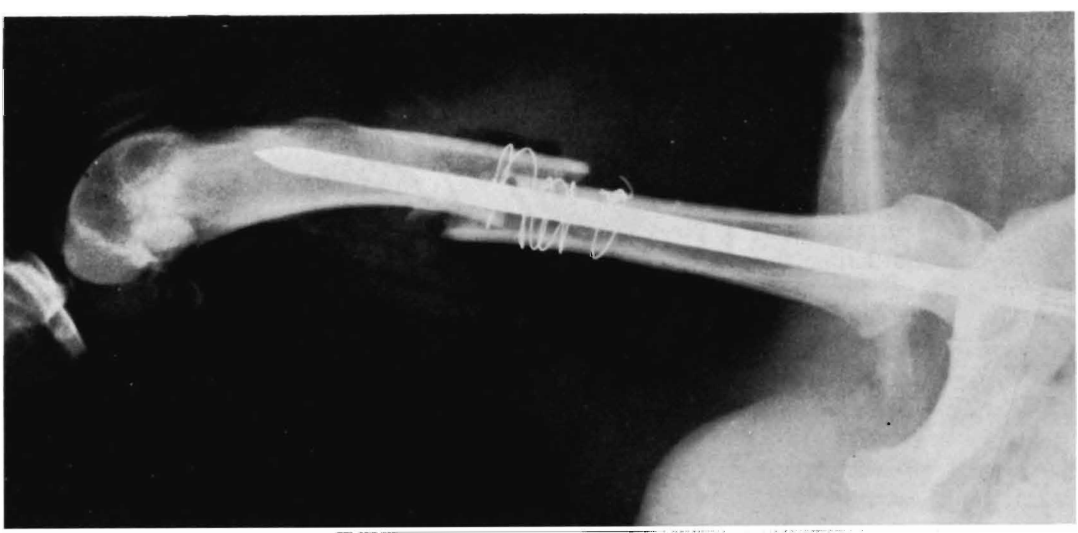
Successful organization as well as further development of the preliminary callus are in direct relation to the vascular activity elicited by interruption of bone continuity.[2] New vessels arising in soft tissue surrounding the callus are primarily responsible for the vascularization of the callus. In the early stages of fracture healing vascular regeneration lags behind cellular proliferation. Bone formation does not occur in the absence of vessels. On the oter hand, as the rich vascularity of the bony callus begins to recede, bone formation becomes more dense.[10]

Fractures are frequently complicated by bone fragments. If these bone fragments are carefully repositioned and placed under compression, they will become incorporated in the fracture callus and promote healing. Circulation can be impaired by periosteal stripping; therefore, under no circumstances should one separate a loose fragment from its adhering soft tissue. Heat can destroy blood vessels and cause avascular necrosis of bone. While reaming a medullary canal, drilling through bone, or cutting bone, one should take care that too much heat is not generated.[7]

FRACTURE MANAGEMENT

Various osteosyntheses affect the vascularization of the fracture callus. There are many methods and devices for handling a fracture. Only three different methods will be considered here. They are: conservative treatment, intramedullary nail, and the bone plate.

Conservative treatment consists of cage rest or external splintage. The main advantage to this type of treatment is that the blood supply is not altered by surgery. Fractures of the pelvis respond well to cage rest, mainly because the pelvis has such a good blood supply. Healing takes place first by cartilage formation and then bone deposition. Fracture of the long bones below the stifle and elbow in young dogs lend themselves well to external splintage. The skeletal system of young dogs has a high metabolic rate, i.e., there is a rapid turnover of calcium. To do this, it must have a good blood supply. This is the rea-



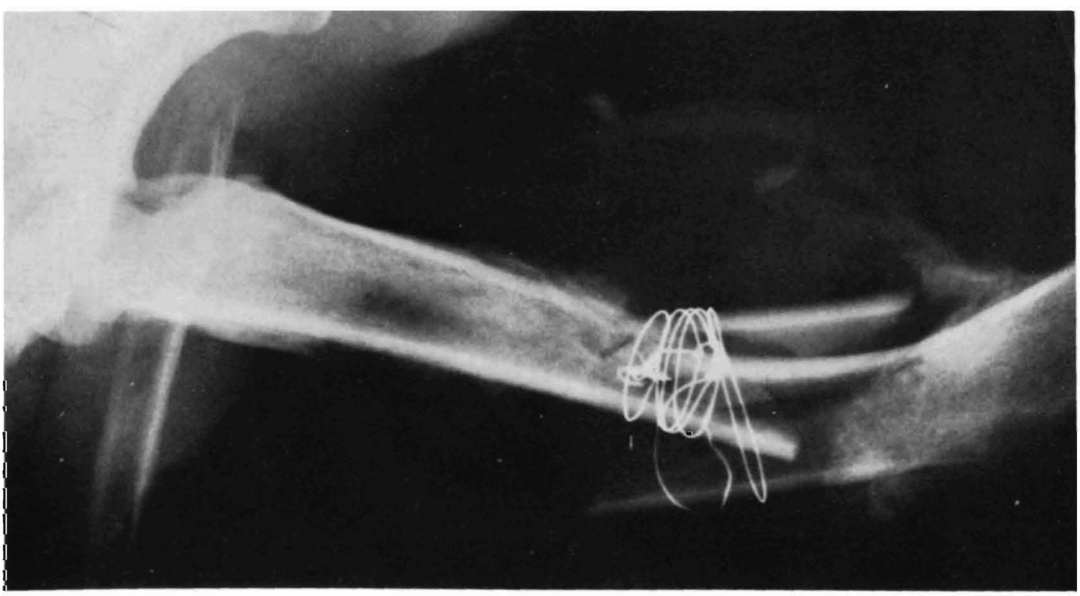
Rad No. 1-A. Radiograph taken immediately post-operatively showing the fracture affixed with an intramedullary pin and wire wrapped around the cortex.

son why young dogs respond better to external splintage than older dogs. However, if the external splint fails to rigidly fix the fracture fragments, the healing time is generally longer, and the fracture callus is larger. With poor immobilization, nonunion is a frequent sequela due to the destruction of blood vessels.

When a fracture is affixed with an intramedullary nail, the fracture is united by a peripheral callus only. The endosteal callus is prevented by the nail. There is little doubt that insertion of an intramedullary nail is unphysiological, since it disrupts the medullary blood supply and prevents the endosteal callus from forming. If the nail is tight, formation of the peripheral callus is more rapid. It has been said that use of the intramedullary nail is justfied only when the stability which it provides outweighs the biological disadvantage and the rotation can be controlled. Many nonunion fractures which have been treated with an intramedullary nail are the result of using too small a nail or because rotation is not controlled.

In certain cases after placing an intramedullary nail in place, the surgeon feels that additional fixation is needed. Stainless steel wire is commonly employed to supply this additional fixation. This wire should be placed in a suture pattern across the fracture sight (See figure 3) and not wrapped around the fracture sight. Radiographs numbered one and two illustrate this fact. The intramedullary nail disrupts the endosteal blood supply, and the wire wrapped around the cortex acts as a turniquet and disrupts the periosteal blood supply. Notice the periosteal reaction and how abruptly it stops at the level of the wire. A healing callus cannot be expected to form in this area since its endosteal and periosteal blood supplies have been damaged.

In contrast to the intramedullary nail, bone plate and screw fixation does much less damage to the medullary and cortical blood supplies, and therefore, allows early callus formation. Care must be taken while placing the screws so as not to generate too much heat. If this occurs the



Rad No. 1-B. Radiograph taken two weeks post-operatively following the removal of the intramedullary pin. Notice how the periosteal reaction stops at the level of the wire. Wrapping the cortex with wire increases the callus requirement and healing time.

screws will become loose due to the necrosis of the bone and the fixation of the device will be lost.[1, 6]

To summarize, vascularization of the fracture callus is one of the most important factors of fracture healing. Alterations of bone vascularity cause such problems as avascular necrosis, non-union fractures, and similar problems. In such cases where bone vascularity has been disrupted, a more direct method of increasing blood flow into a given area has been developed. An expendable artery with maintainence of its blood flow is transferred directly into the medullary space. This method helps maintain the viability of the bone until vascular regeneration can occur. Although not practical in many cases, in the authors opinion, this exemplifies the importance some investigators place on the vascularity of bone. It was also pointed out that the choice of osteosynthesis affects the vascularization of the fracture callus. Conservative treatment spares damage to vessels due to surgery, but if the mobility of the fractured fragments is not controlled, the trauma to vessels of the fractured parts causes a more deliterious effect. The intramedullary nail destroys the medullary circulation, but compensates for this fact due to the rigid immobilization it offers. Considering vascularization only, bone plating does less damage to the blood vessels and also offers rigid immobilization.

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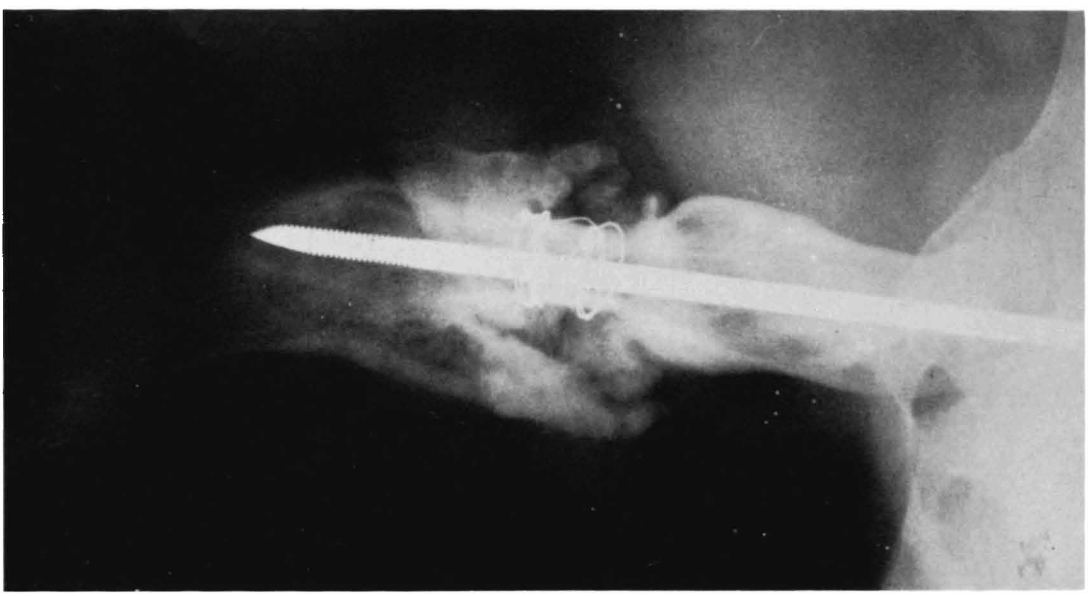

Rad No. 2. This radiograph shows the result of intramedullary pinning and wrapping wire around the cortex. Notice how the periosteal reaction stops at the level of the wire and how the callus is attempting to bridge this space.