FRACTURE HEALING IN COMPRESSION OSTEOSYNTHESIS IN THE DOG

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The healing of fractures varies in pattern within wide limits, depending on several factors. Taking the degree of stabilisation as a parameter, it is usually found that the more rigid the fixation the less the periosteal reaction. This can be observed clinically in the healing of fractures fixed by stable osteosynthesis, in so far as radiologically visible callus seldom appears. If callus does appear it is often an indication of inadequate stability (Weiser and Allgöwer 1962). With adequate stability, direct bone bridging of the fracture gap—the "soudure autogène"—takes place (Danis 1949). Willenegger, Schenk, Straumann, Müller, Allgöwer and Krüger (1962) verified this hypothesis experimentally in sheep and dogs, which Danis had based on clinical experience. Schenk and Willenegger (1963, 1964) studied the healing of fractures histologically after fixation with compression and observed what they called "intracanalicular osteogenesis"—that is, direct formation of secondary osteones across the fracture gap.

After the first phase of bone healing, with exudation, a vascular reaction follows. New vessels branch out from undamaged blood vessels adjacent to the fracture site and invade the fracture site. These vessels have two functions: firstly to break down and remove the inevitable necrotic tissues in the damaged region and, secondly, to create the network of blood vessels and capillaries which is a prerequisite for the construction of new tissue.

The aim of the present investigation was to study by microangiography the vascular reaction and new vessel formation within the fracture ends and in the fracture gap after compression osteosynthesis. By using terramycin* to label the newly formed bone, and by means of current histological methods, it was possible to correlate the bone healing with the vascular reaction.

METHOD

In six dogs both radii were divided with a Gigli saw, with an interval of about a month between the two osteotomies. In another six dogs both tibiae were divided in the same way and with the same time interval. Compression osteosynthesis was done with a compression plate with four or six holes (Müller, Allgöwer and Willenegger 1965).

Under nembutal and nitrous oxide anaesthesia with intubation the skin incisions were made so that the implant lay beneath the skin flap. The incisions were closed with intracutaneous silk sutures with the knots inverted.

In order to label the newly formed bone the dogs were given a single dose of terramycin (50 milligrams per kilogram of body weight) orally seven to ten days before death.

The observation period varied from two to ten weeks with intervals of one to two weeks. Altogether twenty-two observations were made, two cases being excluded because of complications (see "Results").

At the end of the observation period the dog was anaesthetised and a catheter 1.5 millimetres in diameter was introduced into the carotid artery. After the administration of 200 milligrams of heparin an infusion of Indian ink (200 millilitres in 800 millilitres of isotonic sodium chloride) was given through the same catheter at a pressure somewhat higher than arterial pressure. After about 500 millilitres had been infused the blood pressure fell and the animal died. The dog was supported in a harness with the limbs dependent, and a further 4,000 millilitres of Indian ink was infused during the course of two to four hours. The forelimbs and hindlimbs were amputated at the elbow and knee respectively, and were fixed in 96 per cent alcohol. After a few days the bone was stripped of most of the soft tissue and fixation was continued for a further fourteen days.

The implant was then removed and radiographs of the fracture were made. The bone preparation was sawn longitudinally into two halves in the plane of the screw holes. One-half was embedded in methyl methacrylate and sawn into slices 500 μ thick. These slices were ground down to a thickness of about 100 μ and studied microscopically with ultraviolet light with special regard to the vascular structure and fluorescence.

* The terramycin was kindly supplied by Pfizer Ltd.
The other half of the preparation was embedded in paraffin after decalcification. Two slices about 0.5 millimetre thick were sawn from the middle of the specimen for the Spalteholz (1914) procedure, and the remainder, in sections 7 μ thick, was studied histologically after haematoxylin, eosin and azan staining.

RESULTS

During the first few days after operation the dogs appeared to bear no weight on the affected limb. After seven to ten days the limp had usually disappeared, although there was slight swelling in the affected leg. Infection occurred in one radius and there was delayed healing with a tendency to pseudarthrosis in one tibia.

Radiographs of the radii and tibiae showed gradual diminution of the fracture gap as time passed. This is illustrated in Figure 1; the fracture on the right was produced four weeks earlier than the left, which is itself three weeks old; the fracture gap on the right is being filled by densely calcified bone. In neither of these cases is there any radiologically visible callus, which indicates adequate stability.

Most of the preparations using fluorescence and angiography showed a distance of at least 30 μ between the fracture ends. This suggested that the compression was producing contact only at certain points on the fractured surfaces.

In several of the preparations observed after an interval of two to five weeks relatively large areas of cortex without indian ink filled vessels were seen. This was often the case in about half the thickness of the cortex immediately beneath the compression plate. On either side of the fracture gap avascular regions of varying size were always found (Fig. 2). There was less vascularity on the periosteal side of the cortex than on the endosteal side, almost certainly because the periosteal circulation was damaged to a greater extent during the osteotomy and plating than was the endosteal circulation. Also this avascularity appeared to be greater after the operations on the tibia than on the radius.
The fracture gap was vascularised from the endosteal, periosteal and Haversian vascular systems, the vessels anastomosing in the fracture gap. This appeared to take place in the fourth to fifth weeks and was demonstrated most clearly in the Spalteholz preparations (Fig. 4). In certain regions of the avascular cortex large bundles of branching capillaries developed and eroded big areas of the bone but with no simultaneous new bone formation in the immediate vicinity (Figs. 2 and 3); however, in...
other regions revascularisation of bone appeared to take place by the invasion of sinuous vessels into the Haversian canals, the inner layer of which was undergoing progressive reconstruction (Fig. 5). Closely following the vascular proliferation in the fracture gap,

![Fig. 7](image-url)

**FIGS. 7 AND 8**
Figure 7—A plastic-embedded preparation from a tibia at five weeks seen under fluorescent light. Bridging periosteal callus has undergone some peripheral resorption; a vessel loop, originating from the periosteum, is eroding a thin layer from both the fractured bone ends. The fracture site peripheral to the looped vessel is filled with bone which is well attached to the bone ends. (× 48.) Figure 8—A plastic-embedded preparation of a tibia at six weeks seen under fluorescent light. Endosteal callus has been in great part resorbed. The fracture is partially filled with bone and the fracture ends are well vascularised. (× 12.)

![Fig. 8](image-url)

![Fig. 9](image-url)

**FIGS. 9 AND 10**
Figure 9—A plastic-embedded preparation of a tibia at five weeks seen under fluorescent light. The fracture gap is filled with newly formed bone, most of which is oriented along the line of osteotomy. Newly formed secondary osteones traverse the fracture gap. (× 48.) Figure 10—Enlargement of part of Figure 9. Vessel loops from the fracture gap have bored into the fracture ends, and at the apices, osteolysis is taking place, while new bone is deposited at the bases. The secondary osteones formed in this way bridge the fracture gap and show a so-called ‘plug attachment’ at each side. (× 96.)

relatively thick bridges of immature bone were being laid down, which filled the space between the bone ends (Fig. 6). The cortex immediately adjacent to the fracture was progressively invaded by new vessels, which anastomosed with the new vessels in the Haversian systems.

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The ends of the blood vessels acted like drills, boring out small cavities which had a greater width than at the entrance (Fig. 10). Bone lamellae formed along the blood vessels and filled out the spaces and secured the newly formed bone at the fracture site. Figure 7 shows how a vessel loop caused erosion of the surfaces of both fragments simultaneously and, in the posterior part of the newly made cavity, new bone is being laid down. A similar process appears to be taking place in those parts which have a normally vascularised cortex.
At the fracture site periosteal and endosteal reaction occurred at an early stage and gave rise to callus with no cartilaginous or fibrous connective tissue (Fig. 7). The periosteal reaction often produced a cuff of bone which surrounded the fracture, but this was seldom more than 0.5 millimetre thick and was therefore not usually visible on standard radiographs. The endosteal reaction produced bone bridges which filled out the medullary cavity. This periosteal and endosteal callus developed during the course of two to three weeks and after this time usually underwent resorption (Fig. 8).

Six to eight weeks after operation the newly formed bone in the fracture gap was, in the main, oriented along the osteotomy. This is evident in the fluorescent preparations shown in Figures 9 and 10 in which recently formed osteones can be seen, but they are securely attached on either side of the fracture gap.

The orientation of the collagen fibres at the fracture can be seen in the azane-stained histological sections (Figs. 11 and 12). The nuclei of the osteocytes are also oriented in the same direction. In places the fibres curve off into the cortex, with the point of attachment consisting of those cavities which have been bored out by the ends of the blood vessels and then filled by new bone. In those places, however, where the fibres are oriented at right angles to the osteones in the cortex there is attachment to the original bone. This is not surprising because the original bone was probably necrotic.

Gradually, as healing progresses, the vessels in the Haversian canals increasingly take over the nutrition of the fracture zone. The longitudinal orientation of the collagen fibres becomes longitudinal, that is, at right angles to the line of osteotomy, as more points of attachment are obtained within the bone ends. In this way the osseous structure becomes normal, as also does the vascular pattern of the bone, which is evident from the Spalteholz preparations. In Figures 13 and 14, which show an osteotomy in the eighth and tenth week respectively, the vascular pattern of the cortex is almost normal.

DISCUSSION

An unstabilised fracture becomes surrounded by granulation tissue which also encloses the fracture haematoma. By the formation of connective tissue, cartilage and bone in this tissue, stability is produced. Not until this has been established can the fracture gap be bridged by lamellar bone.

A rigid plate takes over the function of the cuff of periosteal callus, which also develops, however, despite stable internal fixation, especially within the first two to three weeks. Regressive changes then begin and the callus undergoes some resorption. No callus is seen radiologically, however, a fact which Danis (1949) demonstrated clinically.

A rigid plate stabilises the fracture from the beginning, thereby obtaining the neutral milieu which Krompecher (1937) considered a prerequisite for the so-called angiogenic bone formation which, in great measure, corresponds to direct bone formation. Wagner (1963) showed similar bone formation in grooves and fissures made in the long bones of the dog. Schenk and Willenegger (1963) found corresponding bone formation under stable conditions after osteotomy—that is, bone formation that may be called primary or direct. A fracture always causes some circulatory disturbance in the adjacent cortex, which is increased by surgical exposure. Our preparations often showed areas of avascular cortical bone in the immediate vicinity of the fracture; they were larger and more obvious in the specimens with shorter observation times. The cortical circulation appeared, however, to have a considerable capacity for restitution, because preparations from animals with longer observation times showed smaller avascular regions. The circulation reappeared so rapidly that perhaps only a few avascular areas become necrotic. Schenk and Willenegger (1963) also found that blood vessels in the Haversian systems could regenerate at a rate of about 50–100 μ per day, which might explain the rapid revascularisation of cortical regions lying close to the fracture.
With rigid fixation, both experimentally and clinically, complete contact of the fracture surfaces is rarely obtained. Thus the fracture surfaces come into contact at several small points, and fairly large areas remain free. In the space between the surfaces a vascular network is formed, originating from the periosteal and endosteal vessels. Around this vascular network bone bridges are laid down which unite the fracture surfaces and vessel loops develop and erode cavities in the necrotic bone ends. These become filled with new bone which, in accordance with the "plugging principle," join the necrotic bone ends together.

Even after rigid fixation there is a continuous breakdown of necrotic bone in the cortex near to the fracture. In the absence of stability, breakdown alone is usual until stability has been produced by the calcification in callus. It is at this stage that a widening fracture gap is seen radiologically. In a stabilised fracture new bone is formed almost immediately to replace resorbed bone tissue. Thus a widening of the fracture gap does not occur.

The areas of contact erode progressively. The residual points of contact are deformed by pressure more easily the fewer they become, which in turn should mean that the pressure gradually decreases. Perron (1967) found that, by and large, the compression pressure decreased linearly over a very long period, in some cases during the course of more than twelve weeks. The progressive reduction of the number of points of contact on the fragment surfaces may well be associated with this pressure decrease.

For a fracture to be considered adequately consolidated, the collagen fibres should be oriented in the longitudinal direction of the bone. The bone which is first formed in the fracture gap usually follows the direction of the blood vessels and consequently the collagen fibres first formed are usually oriented along the fracture gap at right angles to the longitudinal direction of the bone. The erosion cavities formed in the fracture ends are filled with new bone oriented perpendicularly to the fracture gap. By progressive increase in the number of cavities formed, and by the fact that the vessel loops erode farther into the bone to form secondary osteones, the longitudinally oriented fibres increase in number. The bone thus acquires increasing mechanical strength. The fracture gap may well be filled in the early stages by bone with more or less equal density to that of the fracture ends themselves, without there necessarily being adequate mechanical stability. In other words, a fracture treated with compression plating may be radiologically healed at an early stage, but a further period of reconstruction is needed before the fracture is sufficiently strong to withstand great stress.

Schenk and Willenegger (1964) have described the healing of bone under compression in ideal conditions. They chose a highly vascularised bone and a very small fracture gap. Under these conditions the blood vessels in the Haversian systems can play an important role in the formation of new bone across the fracture gap. With less vascularised bone, such as the dog's tibia, and with considerable periosteal stripping—which is common clinically—during the very first stage of the bone healing process vascularisation of the fracture is from the endosteal more than the periosteal vessels. The amount of cortical necrosis can probably vary considerably, depending on the way in which the osteotomy has been produced (with a saw blade or a Gigli saw). In certain "naturally" produced fractures such as torsion fracture, the degree of cortical necrosis at the fracture site is probably very small, and in these it is possible that the cortical vessels may even precede those of the periosteum and endosteum in their contribution to vascularisation of the fracture gap.

In practice the origin of the vessels entering the fracture healing zone is of slight importance. Under stable conditions bone appears to form immediately around the vessels in the fracture gap, whether these vessels arise from periosteal or endosteal regions or from vessels in Haversian canals in undamaged cortex.

CONCLUSIONS

Rigid fixation provides certain advantages in the healing of fractures. Firstly it reduces the fracture gap to be bridged, and secondly it increases the stability of the fracture.
Radiologically the healing is characterised by progressive diminution of the fracture gap and of the presence of very little periosteal callus reaction.

In an experimental osteotomy fixed by compression osteosynthesis, avascular cortical regions of varying extent appear in the immediate vicinity of the fracture. The revascularisation of these regions is derived mainly from vessels in intact Haversian systems. Under the experimental conditions of the present study the revascularisation took place mainly from endosteal and periosteal vessels, which invaded the avascular regions even via the fracture gap. In other avascular cortical regions, especially those situated beneath the compression plate, an osteolytic process, with a specific vascular reaction, occurs.

In the vicinity of vessels which invade the fracture, bone bridges are formed which, after becoming securely attached to each bone end, become joined together. Initially transverse, the orientation of the new bone becomes longitudinal after the formation of secondary osteones across the fracture site.

**SUMMARY**

1. The healing of the radius and tibia in dogs after compression plating of osteotomies made by a Gigli saw was studied.
2. The methods used were Indian ink microangiography and terramycin labelling. The Spalteholz technique and azane colouring were used.
3. Revascularisation of the fracture region took place both from newly formed vessels in the Haversian systems and from periosteal and endosteal vessels.
4. The fracture gap was filled at an early stage by a vascular network. Under stable conditions direct angiogenic bone formation took place around this network.
5. Rebuilding of the cortical bone in the fracture region occurred by osteoclastic activity. Groups of osteoclasts made cavities in the necrotic bone and were immediately followed by loops of vessels; behind and around the loop new bone was formed. Another form of bone absorption consisted of bundles of vessels which eroded necrotic cortical bone without new bone formation.
6. The new bone was initially oriented along the fracture gap but, by conversion into secondary osteones, it became progressively oriented longitudinally in the direction of the original bone.
7. Under stable conditions some periosteal and endosteal callus formation occurred although it was of slight importance. It regressed very soon and was seldom seen in the radiographs.

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**REFERENCES**


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