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EFFECT OF INTRAMEDULLARY BONE CEMENT FIXATION ON FRACTURE HEALING

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INDEXING WORDS

fracture healing; bone cement; intramedullary fixation

SYNOPSIS

The effect of bone cement implanted in the marrow cavity on the fracture healing process was assessed in rabbits with an experimentally induced transverse fracture of the tibia. In the cement treated animals external callus formation occurred slowly, due to poor bone-forming capacity of the endosteum and a failure to form a bridge at the fracture site. Numerous resorption cavities then occurred in the cortical bone. The microangiogram revealed a lack of blood supply from the marrow in the cement treated group. Thus, the histological changes were probably due to obstruction of the marrow blood supply to cortical and endosteal bone.

INTRODUCTION

Bone cement, by virtue of its excellent properties as a grouting material, has been in wide use for prosthetic replacement arthroplasty. More recently it has also been used for immobilization of bone in cases of pathological fracture or fracture of

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bone in the elderly. Not much is known, however, as to the effect of bone cement implanted into the marrow cavity for these purposes on fracture healing.

With this question in mind a study was made in rabbits with an experimentally induced transverse fracture of the tibia with bone cement implanted into the medullary canal. Microangiographic as well as histologic changes in undecalcified hard tissues were used to evaluate its effect on fracture healing.

MATERIALS AND METHODS

Twenty eight Japanese albino rabbits at 15 weeks of age (weighing approximately 2.5 kg each) were used. Half of these animals were treated with bone cement and the others served as controls.

1) The procedure of experimental fracture

Under anesthesia with Nembutal (Pentobarbital) 1.5 ml i.v. and Ketalar (Ketamine) 2 ml i.m. each animal underwent surgical exposure of the right tibia. Using an electric-powered drill 2 small holes (about 4 mm in diameter) were made about 2 cm apart in the middle of the anterior aspect of the tibia. A transverse fracture through the bone was then produced by means of a dental drill. The marrow tissue was removed and intramedullary fixation was provided by a Kirschner's wire.

Bone cement (Surgical Simplex) was inserted through a syringe into the marrow cavity at the fracture site through the proximal hole. The bone cement was made by mixing a polymerized methyl-methacrylate powder with a PMM monomer liquid in the same proportion as for ordinary surgery, and rapidly implanted before complete polymerization. The distal hole served the purpose of keeping the intramedullary pressure from rising by cement implantation. The heated bone cement was cooled with saline irrigation. The wound was closed after the cement had hardened.

Control animals were operated essentially with the same procedure except that no bone cement was inserted; the Kirschner's wire was of as large a diameter as possible for intramedullary fixation (Fig. 1).

Animals were sacrificed weekly over a 5-week period post-

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operatively by intravenous injection of Nembutal. Two and nine days before sacrifice each animal was given tetracycline 20 mg per kg body weight intramuscularly for bone labeling. The treated bones were prepared for the purpose of the following studies.

2) The procedure of microangiography

At 5 weeks postoperatively 2 animals from each group were subjected to this study.²⁾

A median abdominal incision was made under Nembutal anesthesia (1.5 ml i.v.). The intestine was displaced to one side to expose the inferior vena cava and abdominal aorta. A cannula was then inserted into the vessels about 2 cm proximal to the junction of the iliac arteries. After preparatory perfusion with heparinized saline (10 mg/500 ml) at about 40C a mixture of 30 grams contrast medium (Baritop 120) and 5 grams gelatin in 100 ml saline, kept at 45C, was infused by means of continuous infusion pump at a pressure of approximately 180 mmHg. Infusion was maintained until an adequate blood level and outflow of the contrast material was attained. At the termination of infusion the vessels were ligated and the animals were isolated in an ice-chamber. Once the contrast medium had set the animals were taken out for resection of the right leg bone.

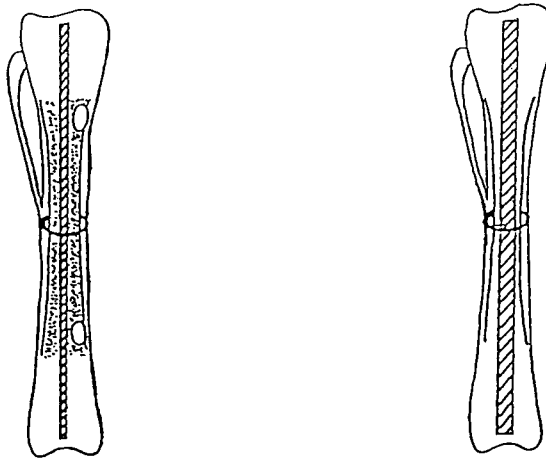


Fig. 1 Experimental methods
left : cement treated group
right : control group

3) Method of radiographic and histologic observations

- i) Soft x-rays of excised specimens: 3 min' irradiation at 30kV and 10 mA.
- ii) Microscopy of undecalcified tissue: The tissue block, after fixation in 70% ethanol and methylmethacrylate embedding, was cut into thin sections, 150-200 μ in thickness, along the long axis of the bone using a Velnus Cut Type VA-221 (Komatsu Trading Co.) and ground in Maruto Speed Wrap to the thickness of approximately 30 μ . The sections were then stained with paragon multiple stain with warming for light and fluorescence microscopy.
- iii) Microangiographic observation: The resected bone was fixed in formalin, decalcified and cut into sections about 2 mm in thickness. These were then subjected to soft x-ray examination as well as light microscopy following Hematoxylin-Eosin staining.

RESULTS

1) Radiographic findings (Fig. 2)

At 1 and 2 weeks after treatment, external callus was seen developing at a short distance from the fracture site in both groups. The callus appeared to enlarge as it approached the fracture line.

At 3 weeks external callus, developing around the opposing ends of the bone fragments, was closer. In the control group the callus had joined in most animals while a noticeable gap was present between the bone ends. A considerable fracture gap was still present in the treated group.

At 4 weeks the gap between the bone ends was somewhat less distinct in the control group whereas it was still clear-cut; no bridging of external callus had yet occurred in the treated group.

At 5 weeks the fracture gap was indistinct enough in the control group to indicate that the bone union had been achieved. In the treated group, in contrast, the gap was distinct. Callus still failed to form a bridge.

2) Histologic findings (Fig. 3)

At 1 week after treatment fibrous tissue enveloped the frac-

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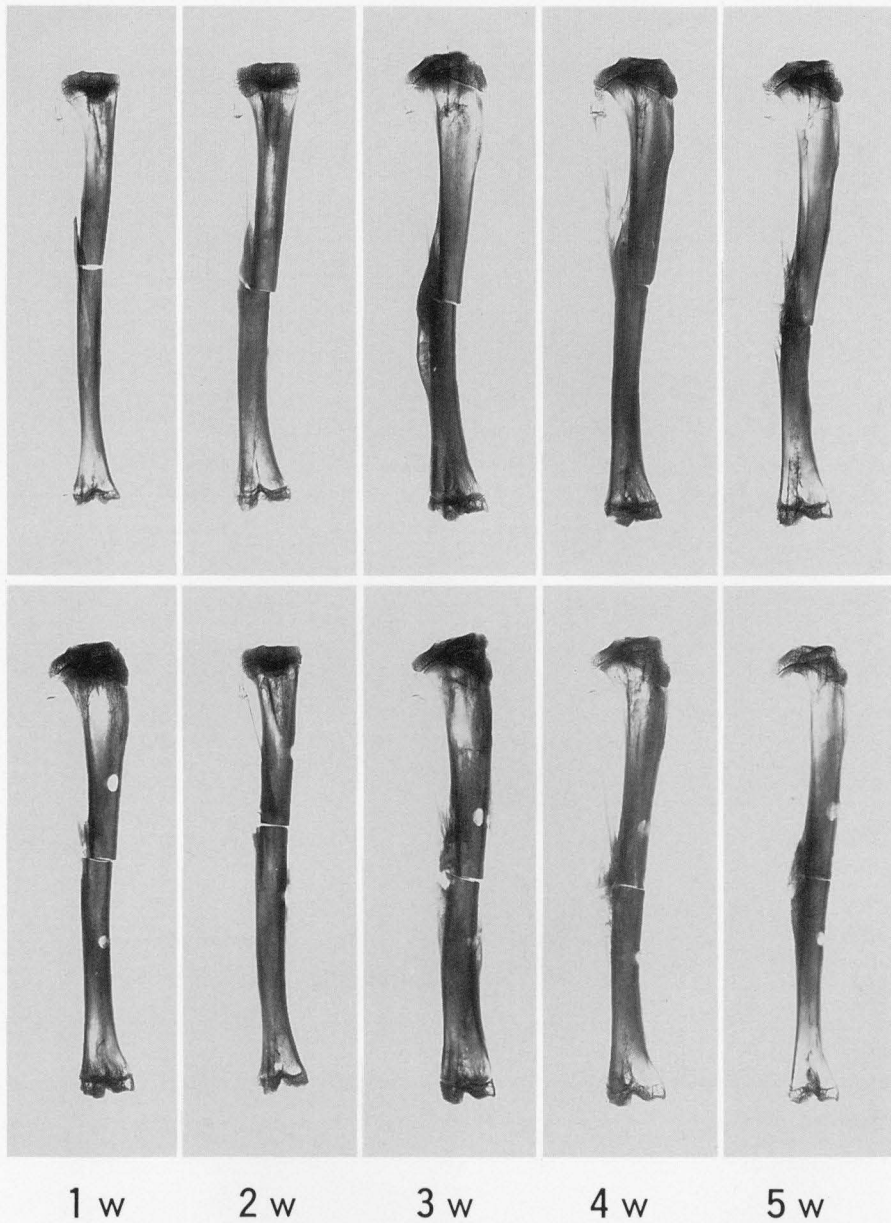
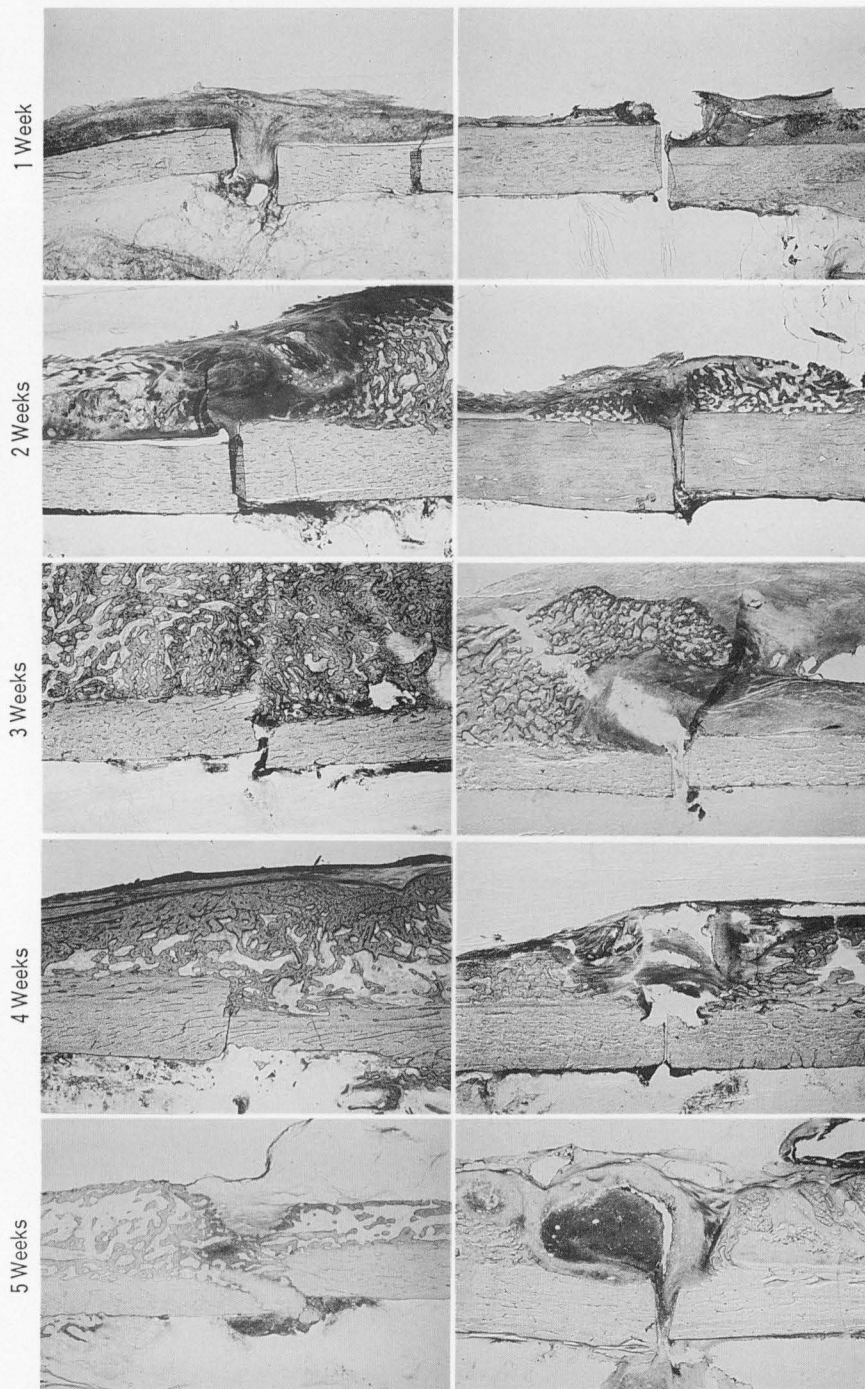


Fig. 2 Soft x-ray findings on fracture healing
top : control group
bottom : cement treated group



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ture site in both groups. The fibrous layer was less pronounced in the cement treated animals but in both groups external callus was seen at a distance from the fracture site. Thus no substantial difference was present between the groups with regard to external callus formation (Fig. 4).

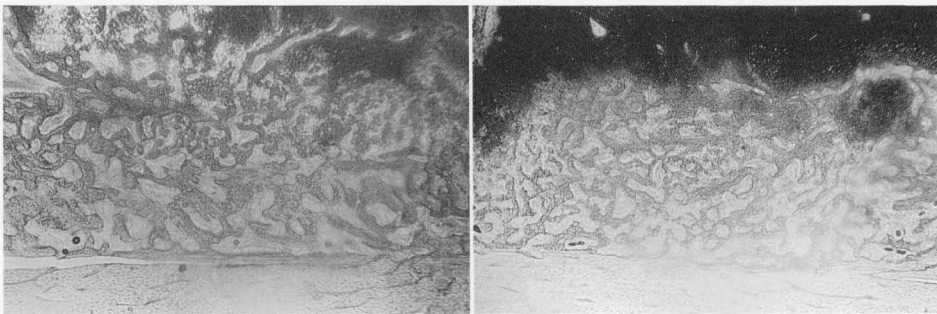


Fig. 4 External callus formation at a week after fracture (x9)
left : control group
right : cement treated group

At 2 weeks external callus formation progressed further in both groups, with the callus closer to the fracture site than at 1 week but still short of union.

At 3 weeks there was fusion of external callus with evidence of remodeling of the cortex at the bone ends in the control group. In the treated group the callus was approaching but not bridging; separation being continued by fibrous tissue.

At 4 weeks the bridging process had progressed further with remodeling of the cortex in the control group. In the treated group external callus had still not joined. No evidence was seen for remodeling of the cortices at either bone end.

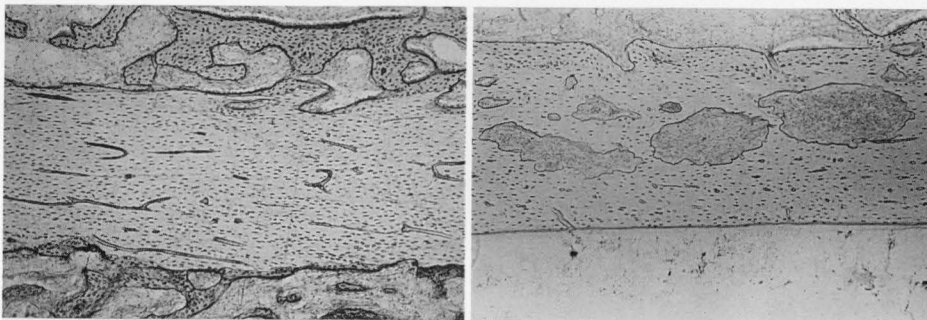
At 5 weeks remodeling of the bone was under way in the controls. In the treated group no bridging of external callus had yet occurred at the fracture site and there was little changes noted in the cortices at the bone ends.

This time after onset of the fracture revealed the maximal

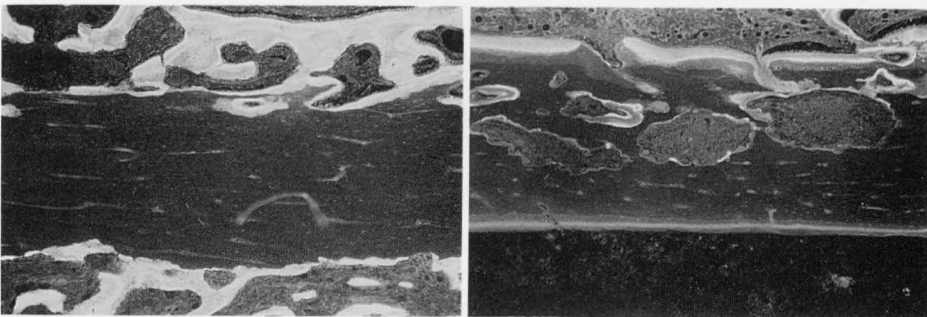
Fig. 3 Microscopic findings of undecalcified sections on fracture healing (x3)
left : control group
right : cement treated group

differences between the groups with respect to the histologic features. These features are illustrated in Fig. 5. As seen, there was satisfactory formation of internal as well as external callus in the control group. In the treated group, in contrast, internal callus formation was absent with a number of large resorption cavities within the cortices of the bone. In the controls bands of tetracycline fluorescence were detected in the endosteum even at sites where internal callus was absent whereas in the treated group the endosteum exhibited no appreciable tetracycline fluorescence. This suggested a diminished capacity for bone formation.

In short, there was no radiological or histological evidence of internal callus developing in the treated group, where tetracycline fluorescence study of the endosteum also suggested depressed bone formation within the marrow. External callus did develop but to a lesser extent than in the control group, failing



Paragon's staining



Tetracycline labeling

Fig. 5 Changes of cortical bone at five weeks after fracture
(x20)
left : control group
right : cement treated group

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to form a bridge even at 5 weeks, when it must be added that larger resorption cavities were also observed in the cortex.

3) Microangiographic findings

In search of a cause for the depressed bone formation in the treated group an evaluation was made of the microangiograms taken at 5 weeks when the difference between the groups was most pronounced in radiographic as well as histologic respects.

Normal animals undergoing no surgical procedures had the bone marrow profusely vascularized with the cortex of the bone receiving a blood supply via the outer layer of periosteum as well (Fig. 6). In the control group the blood supply of the cortex was both via the periosteal vessels and from the marrow. In the treated group, in contrast, a blood supply was evident externally but little from the marrow.

Decalcified tissue specimens in the control group revealed contrast medium in the medullary tissue and at some parts in the cortex via the marrow (Fig. 7). External callus developed from the outer aspects of the bone, suggesting an intact cortical blood supply. In the treated group the contrast medium entered the cortex not via the marrow; rather from periosteal sites.

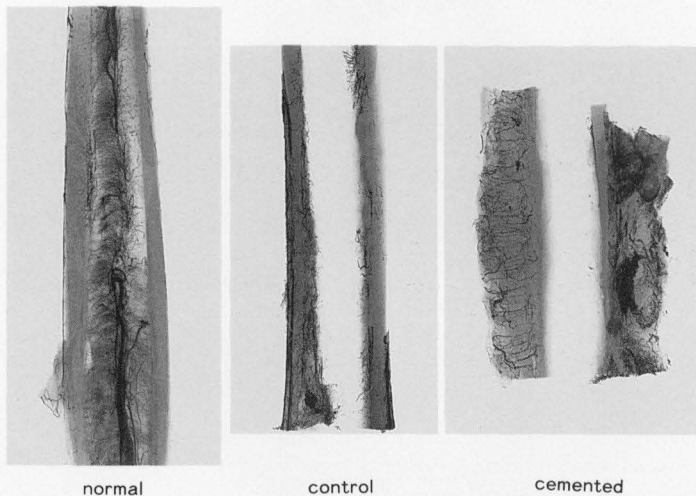


Fig. 6a Microangiographic findings (longitudinal section) (x2)

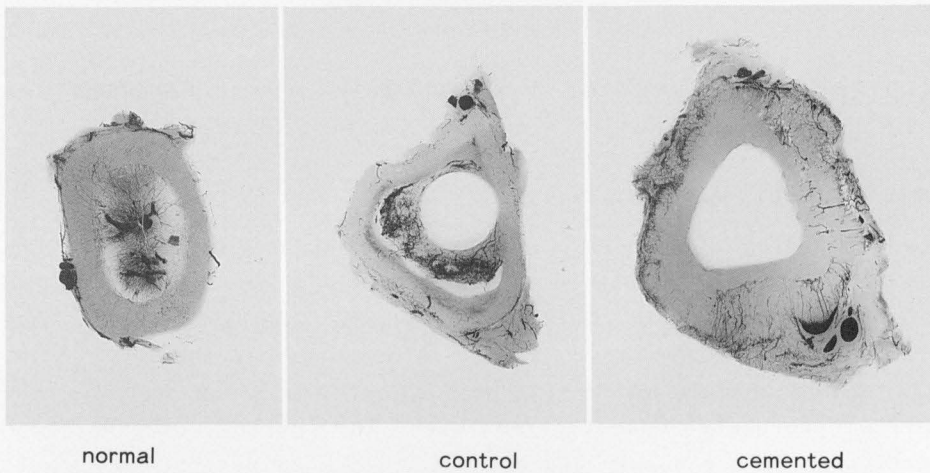


Fig. 6b Microangiographic findings (transverse section) (x4)

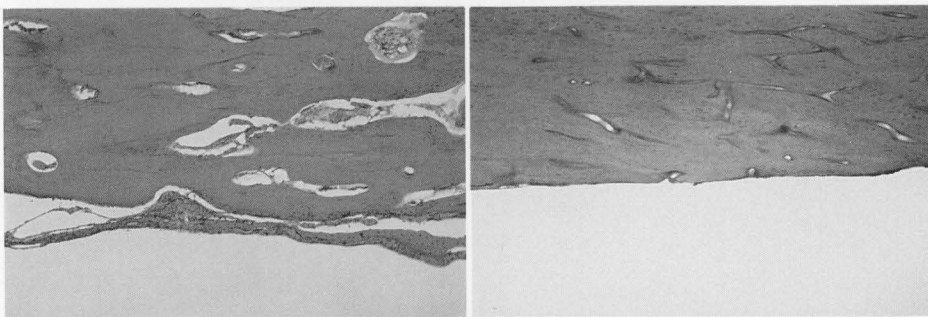
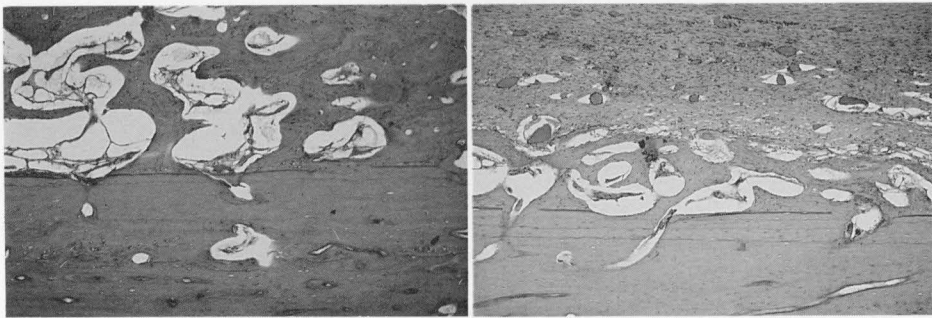


Fig. 7 Microangiographic findings of decalcified sections at five weeks after fracture (x21)
left : control group
right : cement treated group

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DISCUSSION

Bone cement (polymethylmethacrylate) has been most commonly used in prosthetic replacement arthroplasty as a material for fixing the prosthesis. Bone cement, which is formed from a polymerized methylmethacrylate powder mixed with a PMM monomer liquid, is endowed with versatile plasticity and can firmly fix the prosthesis to bone.

The history of use for bone cement dates back to 1890 when Gluck in Germany advanced the concept of using resin cement to fix a spheroid joint prosthesis made of ivory to replace a degenerated hip joint. Bone cement at orthopaedic surgery was initiated by Haboush,³⁾ who for the first time applied it in 1951 to a femoral head prosthesis. The use of bone cement for fixation of joint prosthesis, which began with Charnley in 1960, has since been widespread and in Japan bone cement has been commonly used since the 1970s. Because of properties as a bonding material and compatibility, bone cement has found applications in other areas as well; e.g., in the treatment of fractures,^{4,9)} especially pathological fractures.^{5,13)}

While studies have been made on the systemic and local effects of bone cement,^{1,7,12,15)} much remains to be investigated in respect to fracture healing.

The first experimental work with bone cement implanted in long bones of laboratory animals was done by Henrichsen⁶⁾ in 1951. Subsequent specific problems with the use of bone cement were identified, including the heat of polymerization, damage to blood supply and release of some chemical substances from cement. Of these, the former 2 were reported by Wiltse,¹⁸⁾ Rietz¹¹⁾ and Slooff¹⁴⁾ and the last by Lindwer.⁸⁾ Tsuji,¹⁷⁾ on the other hand, found that callus formation was not inhibited by bone cement packed into bone defects. The influence on fracture healing in a long bone when cement was implanted in the marrow cavity is not well understood.

The normal fracture healing may be identified 5 histologic stages.¹⁶⁾

In this study we showed that the fractured tibia of the cement treated animals had little development of callus from the endosteum; nor any appreciable endochondral callus formation. Callus did emerge from the outer aspects of the periosteum but it

failed to form a bridge.

These observations correlate with the delayed union evident in the cement treated group. It appears that cement implantation in the marrow resulted in diminished bone-forming capacity locally. This may be due to interruption of intramedullary circulation that limited blood supply to the inner layer of the cortex as demonstrated by microangiography. Ultimately, union of the bone will be occurred with revascularization through periosteal blood vessels, as suggested by the microangiogram.

Deprivation of blood supply may also have been the main factor responsible for the changes in the bone cortex observed in the treated group. One-third of the blood supply to the cortex of a long bone is provided from periosteal blood vessels and the other two-thirds from nutrient vessels distributed throughout the marrow and endosteum.¹⁰⁾ Consequently, if intramedullary circulation was completely abolished, ischemic necrosis will occur in the areas supplied by these nutrient vessels. Later, resorption of the necrotic bone will take place in conjunction with compensatory revascularization of periosteum; this may explain the porotic changes in the cortex that were observed in the treated group.

It may be concluded that deprivation of blood supply of the marrow as a result of bone cement implantation was responsible for the histologic and microangiographic changes that were observed in the fractured tibia in this study; these lesions resulting in delayed healing of the fracture.

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