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EFFECTS OF JOINT MOTION ON THE REPAIR OF ARTICULAR CARTILAGE WITH FREE PERIOSTEAL GRAFTS

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The effects of joint motion on the chondrogenic potential of free autogenous periosteal grafts were studied histologically in 6-month-old rabbits. The grafts were taken from the tibia and transplanted to artificial defects of the femoral articular cartilage. The joint was postoperatively first immobilized and then remobilized for various periods.

The results revealed that immobilization for 3 weeks had an inhibitory effect on the chondrogenesis, which was even more pronounced after 6 weeks of immobilization. After remobilization the chondrogenesis partially recovered. This recovery of chondrogenesis was more pronounced after 3 weeks' immobilization than after 6 weeks' immobilization. However, degenerative changes were observed in both series.

Key words: articular cartilage repair; chondrogenesis; immobilization; periosteal graft; remobilization

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It is known that factors which interfere with the normal function of the joints lead to degenerative changes in the articular cartilage, probably because of nutritional deficiency (Fisher 1922, Sood 1971). Experimental degenerative arthritis of joints has been provoked by many investigators (Menzel 1871, Müller 1924, Ely & Mensor 1933, Salter & Field 1960, Langenskiöld et al. 1979) using immobilization methods.

In a previous experimental study (Rubak 1982) the chondrogenic capacity of free periosteal grafts in articular cartilage defects has been confirmed. The purpose of the present study is to evaluate the effects of immobilization and remobilization on the chondrogenesis and the repair of the articular cartilage defects with free periosteal grafts.

MATERIAL AND METHODS

The experimental material comprised 40 rabbits weighing between 2500 and 3500 g. The operations were performed as described in a previous study (Rubak 1982). After transplantation of the periosteal graft from the tibia to the artificial defect in the articular cartilage, the knee joint was immobilized using a method introduced by Langenskiöld et al. (1979). A straight and somewhat elastic splint of PVC plastic was applied on the dorsal aspect of the right leg from the proximal end of the thigh to the distal end of the limb. The splint was fixed to the limb with a Tensoplast® bandage (Figure 1). Ten animals were killed after 3 weeks and ten after 6 weeks. In ten animals the splint was removed after 3 weeks and these animals were killed 5 weeks later. In ten animals the splint was removed after 6 weeks and these animals were killed 6 weeks later. The histological specimens were prepared using the same method as in a previous study (Rubak 1982).

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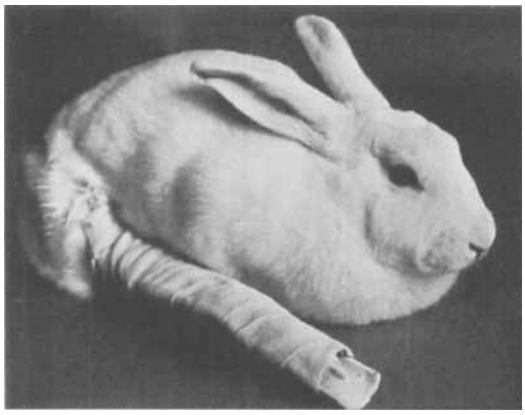

Figure 1. The immobilization method. A rabbit with the right hind limb immobilized with a PVC plastic splint and Tensoplast®.

RESULTS

Findings after 3 weeks' immobilization

Macroscopic. The joint was found to be rather stiff with only a few degrees of passive motion. Intra-articular adhesions were seen in the transplantation area. Most of the defects were still visible and partially filled with a grey soft tissue.

Microscopic. The defects were partially covered with connective tissue. In some defects chondrous tissue had begun to appear in the deeper part of the transplant facing the subchondral bone (Figure 2).

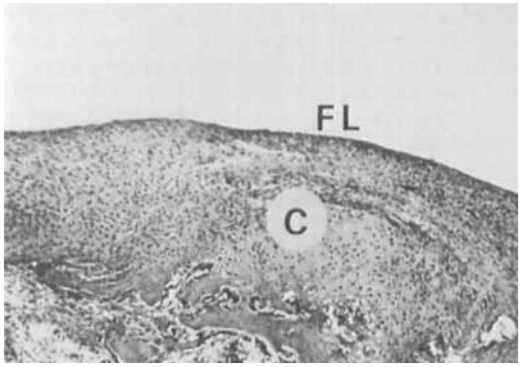

Figure 2. Histological picture from the transplantation area after 3 weeks' immobilization. There is a fibrotic layer (FL) facing the joint cavity, beneath which a focus of immature cartilage (C) is observed. (v–G stain, × 125).

Findings after 6 weeks' immobilization

Macroscopic. The joint was stiff, muscular atrophy had occurred and thickening of the distal end of the femur was observed. In a few animals hyperextension occurred. Intra-articular adhesions were present both in the grafted area and also between the articular surfaces. Furthermore, a smaller amount of synovial fluid was noted, compared with that found at the opening of the joint. The defect was in most experiments covered with a grey soft tissue.

Microscopic. The tissue filling the defect consisted of connective tissue with fibroblasts, inter-

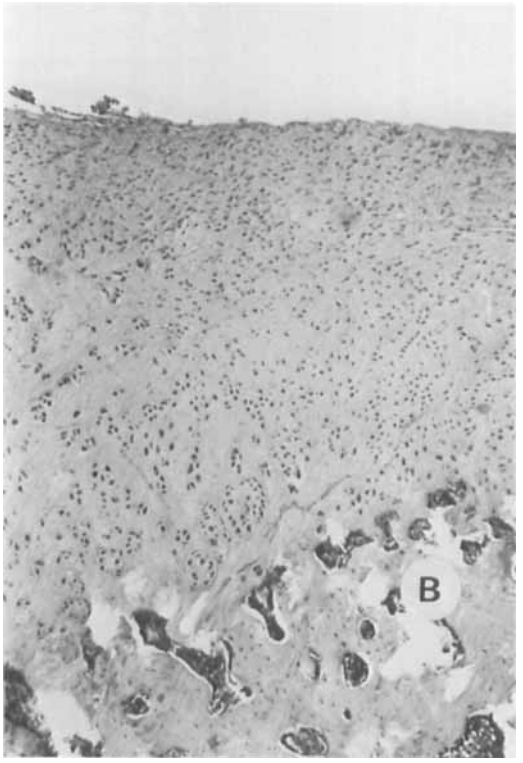

Figure 3. Histological picture from the transplantation area after 6 weeks' immobilization. The tissue covering the cancellous bone (B) is made up of fibrotic cells in the superficial part; in the deeper part the cell packings have the appearance of fibrocartilage rather than hyaline cartilage. (H–E stain, × 125).

stitial stroma and some fibrocartilage-like tissue. In the deeper layers of the proliferating tissue chondrous tissue could be found. No bone formation was observed (Figure 3).

Findings after 3 weeks' immobilization and 5 weeks' remobilization

Macroscopic. Eight rabbits achieved almost normal mobility of the previously immobilized joint. The defect was covered with a grey, glistening tissue.

Microscopic. The surface consisted of connective tissue with fibroblasts. In the deeper part of the generated tissue chondrocytes were found. Generally better chondrogenesis was observed in these animals than in animals killed immediately after 3 weeks of immobilization. However, in those experiments where the chondrous tissue nearly reached the original level of the articular surface, the surface was characterized by fibrillation and erosion as a sign of incipient degeneration (Figure 4).

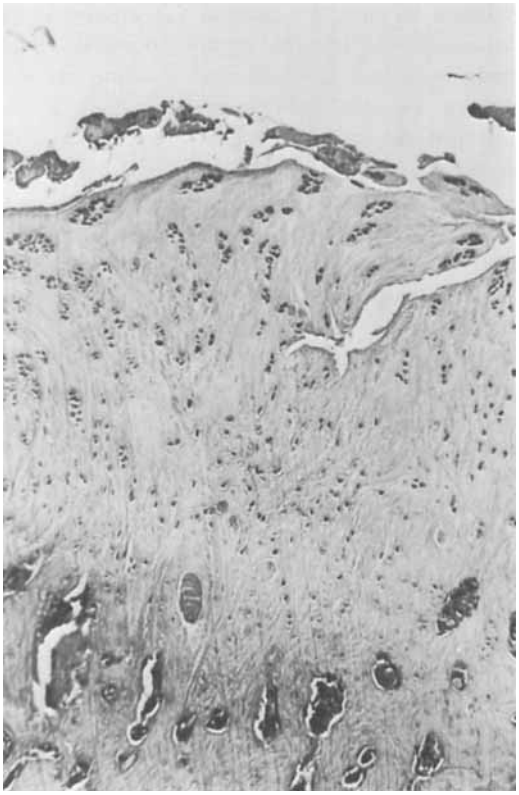

Figure 4. Histological picture from the transplantation area after 3 weeks' immobilization followed by 5 weeks' remobilization. The generated tissue comprises chondrocytes; on the surface degenerative features are seen. (H–E stain, × 125).

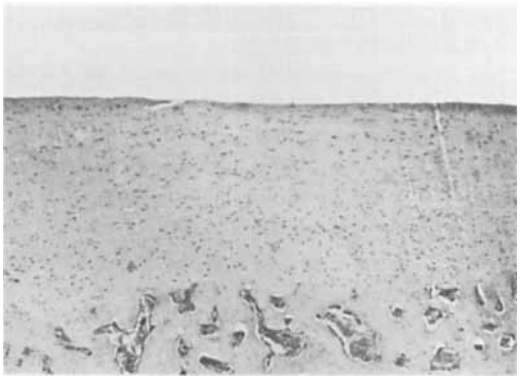

Figure 5. Histological picture from the transplantation area after 6 weeks' immobilization followed by 6 weeks' remobilization. A fibrocartilage-like layer covers the cancellous bone. The surface looks even and no degenerative changes are observed in this cartilage. (H–E stain, × 125).

Findings after 6 weeks' immobilization and 6 weeks' remobilization

Macroscopic. None of the rabbits achieved normal mobility of the joint. The joint was thickened and a sparse amount of synovial fluid was noted. The defect was filled with a grey, glistening tissue.

Microscopic. The generated tissue in the defect consisted of fibrocartilage tissue. In the deeper layer of the proliferated tissue chondrocytes were observed, but there were fewer than in the animals remobilized after only 3 weeks of immobilization. There was no osteogenesis in the defects (Figure 5).

DISCUSSION

Immobilization of a joint with or without compression causes degenerative changes in the articular cartilage, as shown by Salter & Field

(1960), Evans et al. (1960), Hall (1963), Langenskiöld et al. (1979).

Most writers consider the synovial fluid to be the chief source of nutrition in articular cartilage (Strangeways 1920, Ito 1924, Maroudas et al. 1968, Zahir & Freeman 1972, Ogata & Whiteside 1979). Physiological exercise increases the volume of synovial fluid (Ekholm & Nordbäck 1951). If it is assumed that the converse is true, there will be a decrease in the volume of synovial fluid in an immobilized joint with the diminished possibility of nutrition as a result.

In the embryonic stage mechanical stimulation is found to be of great importance among the factors determining the differentiation of mesenchymal cells (Fell 1932, Basset 1962, Hall 1969). Thorogood (1979) found by *in vitro* studies that intermittent pressure and/or shearing forces in some way affect the development of the periosteal cells.

In a previous study (Rubak 1982) articular cartilage defects were completely filled with hyaline-like cartilage after 3–4 weeks. In the present study with postoperative immobilization of the joint for 3 weeks, the same defect was filled with connective tissue containing only a few chondrocytes. After 6 weeks' immobilization the defect was filled only with connective tissue. In some animals chondrous tissue was found in the transition area between the transplant and the subchondral bone.

The development of connective tissue in the defects after immobilization is in accordance with the studies of Salter et al. (1975). They studied the effect of movement on the healing of small drill-holes in articular cartilage in rabbits. After immobilization, the defects were filled with fibrous tissue. With continuous passive motion healing with hyaline cartilage occurred in most defects. DePalma et al. (1966) and Furakawa et al. (1980) observed the most active repair of articular cartilage defects, with cartilage-like tissue, in the weight-bearing areas of the joint. Mooney & Ferguson (1966) found evidence that the primitive mesenchymal cell can be influenced to produce fibrocartilage according to the timing and duration of immobilization and the subsequent motion after operation.

In animals remobilized after 3 weeks' immobilization, cartilage of poorly-differentiated type appeared as islands in the connective tissue, mostly in the deepest part of the generated tissue. The cartilage was characterized by fibrillation and erosion. In animals remobilized after 6 weeks' immobilization, there were some signs of differentiation towards chondrous tissue, but to a much lesser degree than in animals with 3 weeks' immobilization. These findings are in accordance with studies of Evans et al. (1960), Finsterbush & Friedman (1975), Refior & Hübner (1978), who observed that the reversibility of the structural degenerative changes in normal articular cartilage depended on the length of immobilization. Glücksmann (1939) found by *in vitro* studies that the first effect of pressure on periosteal tissue was to flatten and compress the cells and fibres. Later the fibres surrounding the cells fused, thus forming an irregular network in which an increased amount of ground substance was observed. He concluded that pressure seemed to initiate chondrogenesis mechanically, and in some yet unknown way established conditions which led to the formation of ground substance with a hyaline appearance.

In a previous study (Rubak et al. 1982) it was demonstrated that the cartilage tissue in the defects develops from the periosteal graft and not from the subchondral bone. How chondrogenesis is regulated is not yet clear, but from the present study it can be concluded that among the chondrogenesis promoting factors, motion seems to be very important. This fact may be of importance in clinical work.

REFERENCES

Basset, C. A. L. (1962) Current concepts of bone formation. *J. Bone Joint Surg.* **44**-A, 1217–1244.

DePalma, A., McKeever, D. & Subin, D. K. (1966) Process of repair of articular cartilage demonstrated by histology and autoradiography with tritiated thymidine. *Clin. Orthop.* **48**, 229–242.

Ekholm, R. & Norbäck, B. (1951) On the relationship between articular changes and function. *Acta Orthop. Scand.* **21**, 81–88.

Ely, L. W. & Mensor, M. C. (1933) Studies on the immobilization of the normal joints. *Surg. Gynecol. Obstet.* **57**, 212–215.

Evans, E. B., Eggers, G. W. N., Butler, J. K. & Blumel, J. (1960) Experimental immobilization and remobilization of the rat knee joints. *J. Bone Joint Surg.* **42**-A, 737–758.

Fell, H. B. (1932) Chondrogenesis in cultures of endosteum. *Proc. Roy. Soc. Lond.* [*Biol.*] **112**, 417–427.

Finsterbush, A. & Friedmann, B. (1975) Reversibility of joint changes produced by immobilization in rabbits. *Clin. Orthop.* **111**, 290–298.

Fisher, A. G. T. (1922) A contribution to the pathology and aetiology of osteoarthritis. *Brit. J. Surg.* **10**, 52–80.

Furakawa, T., Eyre, D., Koide, S. & Glimcher, M. (1980) Biochemical studies on repair cartilage resurfacing experimental defects in the rabbit knee. *J. Bone Joint Surg.* **62**-A, 79–89.

Glücksmann, A. (1939) Studies on bone mechanics in vitro. *Anat. Rec.* **73**, 39–55.

Hall, M. C. (1963) Cartilage changes after experimental immobilization of the knee joint of the young rat. *J. Bone Joint Surg.* **45**-A, 36–44.

Hall, B. K. (1969) Hypoxia and differentiation of cartilage and bone from common germinal cells in vitro. *Life Sci.* **8**, 553–558.

Ito, L. K. (1924) The nutrition of articular cartilage and its method of repair. *Br. J. Surg.* **12**, 31–42.

Langenskiöld, A., Michelsson, J-E. & Videman, T. (1979) Osteoarthritis of the knee in the rabbit produced by immobilization. *Acta Orthop. Scand.* **50**, 1–14.

Maroudas, A., Bullough, P., Swanson, S. A. V. & Freeman, M. A. R. (1968) The permeability of articular cartilage. *J. Bone Joint Surg.* **50**-B, 166–177.

Menzel, A. (1871) Ueber die Erkrankung der Gelenke bei dauernder Ruhe derselben. *Arch. Klin. Chir.* **12**, 990–1009.

Mooney, U. & Ferguson, A. B. (1966) The influence of immobilization and motion on the formation of fibrocartilage in the repair granuloma after joint resection in the rabbit. *J. Bone Joint Surg.* **48**-A, 1145–1155.

Müller, W. (1924) Experimentelle Unterzuchungen über die Wirkung langdauernder Immobilisierung auf die Gelenke. *Z. Orthop. Chir.* **44**, 478–488.

Ogata, K. & Whiteside, L. (1979) Barrier to material transfer at the bone cartilage interface. *Clin. Orthop.* **145**, 273–276.

Refior, H. J. & Hübner, G. (1978) Zur Morphologie des hyalinen Gelenkknorpels unter Immobilization und Remobilization. *Arch. Orthop. Traumat. Surg.* **91**, 305–314.

Rubak, J. M. (1982) Reconstruction of articular cartilage defects with free periosteal grafts. *Acta Orthop. Scand.* **53**, 175–180.

Rubak, J. M., Poussa, M. & Ritsilä, V. (1982) Chondrogenesis in repair of articular cartilage defects by free periosteal grafts in rabbits. *Acta Orthop. Scand.* **53**, 181–186.

Salter, R. B. & Field, P. (1960) The effects of continuous compression on living articular cartilage. *J. Bone Joint Surg.* **42**-A, 31–49.

Salter, R. B., Simmonds, D. F., Malcolm, B. W., Rumble, E. J. & MacMichael, D. (1975) The effects of continuous passive motion on the healing of articular cartilage defects. *J. Bone Joint Surg.* **57**-A, 570–571.

Sood, S. C. (1971) A study of the effects of experimental immobilization on rabbit articular cartilage. *J. Anat.* **108**, 497–507.

Strangeways, T. S. P. (1920) The nutrition of articular cartilage. *Br. Med. J.* **1**, 661–665.

Thorogood, P. (1979) In vitro studies on skeletogenic potential of membrane bone periosteal cells. *J. Embryol. Exp. Morphol.* **54**, 185–207.

Zahir, A. & Freeman, M. A. R. (1972) Cartilage changes following a single episode of infarction of the capital femoral epiphysis in the dog. *J. Bone Joint Surg.* **54**-A, 125–136.

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