Effects of Excimer Laser on Healing of Articular Cartilage in Rabbits

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Summary: This study examined healing of 1.0 mm diameter defects in rabbit knee articular cartilage for as long as 14 weeks after creation of the defects by either laser or drilling. The purpose of the research was to determine the effects of laser debridement of cartilage on the intrinsic biomechanical properties of the repair tissue. We therefore imitated chondral shaving and subchondral abrasion of cartilage by creating partial-thickness and full-thickness cartilage defects of standardized size with both excimer laser and drilling. Light and scanning electron microscopic examinations of the repair tissue showed that healing of osteochondral defects created by laser may be delayed compared with defects created by drilling, for at least 6 weeks postoperatively. Even though there initially was a considerable delay in healing in the laser group, neither laser nor drilling had any appreciable effects on the mechanical properties of the repair tissue, as demonstrated by biomechanical testing at 14 weeks. Specifically, the repair cartilage in the defects in the laser group had the following material properties (mean ± SD): aggregate modulus, 0.40 ± 0.24 MPa; Poisson’s ratio, 0.37 ± 0.08; permeability, 3.72 ± 4.28 × 10⁻¹⁵ m²/N·s; and thickness, 0.20 ± 0.06 mm. The corresponding values for the defects in the drilling group were 0.39 ± 0.23 MPa, 0.34 ± 0.09, 3.82 ± 3.44 × 10⁻¹⁵ m²/N·s, and 0.22 ± 0.09 mm. The repair tissue from both types of defects was pooled, and the values were compared with those for contralateral (control) tissue. The control tissue had a 51% greater aggregate modulus (0.59 ± 0.28 MPa, p = 0.0001), 34% less Poisson’s ratio (0.23 ± 0.25, p = 0.0001), 48% less permeability (1.94 ± 0.96 × 10⁻¹⁵ m²/N·s, p = 0.0001), and was 29% thicker (0.27 ± 0.08 mm, p = 0.0001). Thus, as evidenced by biomechanical testing at 14 weeks, neocartilage in both superficial and osteochondral defects, created by either laser or a drill, exhibited structural integrity inferior to that of normal control tissue.

The treatment of osteoarthrotic cartilage is a problem not yet solved in orthopaedic surgery. Due to fibrillation of the cartilage surface, the friction at the formerly smooth articular surface is increased, and wear particles from the disrupted articular cartilage induce synovial inflammation. In adults, remodeling of the joint surface by formation of new hyaline cartilage usually is not achieved because cartilage cells do not replicate adequately. Since 1941, when Magnuson (19) published results indicating that joint debridement in degenerative arthritis may be beneficial, lavage and shaving of osteoarthrotic cartilage generally have been accepted and widely utilized in orthopaedic surgery. This treatment regimen, which has been discussed again as a treatment modality for osteoarthritis, involves the removal of fibrillated cartilage tissue, results in a smoothing of the cartilage surface, and reduces further wear of the cartilage (10,31). However, mechanical shaver systems that are conventionally used for arthroscopic treatment of osteoarthritis remove biomechanically intact cartilage in addition to layers of damaged tissue. Further disadvantages of the shavers are that they cannot restore a smooth joint surface, they may leave the softened chondromalacic cartilage unaltered, and they do not improve the biomechanical properties of the osteoarthrotic cartilage (9,39). As a result, relief of pain and increase in mobility are only temporary.

In the late 1980s, lasers were introduced as an additional means to achieve debridement of cartilage. Among other parameters, the effect of the laser on tissue depends on the emitted wavelength of its monochromatic coherent light (37,40). Most lasers used in surgery today, like the neodymium or holmium:yttrium aluminum garnet (Nd:YAG or Ho:YAG) laser, have considerable thermal side effects (32-34). For example, due to the short wavelength (308 nm), the energy of the XeCl excimer laser is absorbed in the first few micrometers of the cartilage tissue. Thus, a power density between 10⁸ and 10¹⁰ J/cm² is reached in those tissue layers, leading to an effect called “photodestruction” (37)—the molecular bonds inside the
tissue are broken, and the disrupted tissue fragments are hurled out. Due to this effect, the high temperature at the tissue surface leads to a sort of "welding" of the collagen fibers visible on scanning electron microscopic examination (12,18,25,34). The increase in temperature in deeper layers of tissue is comparably small (8,9).

Low-level noncontact energy from the Nd:YAG laser has been used to stimulate cartilage matrix synthesis in explants of full-thickness articular cartilage from adults (36). The results of this in vivo experiment showed that laser energy can alter the metabolism of articular cartilage. Examinations of human cartilage tissue cultures (8) treated with excimer laser have shown a 0.3-0.7 mm depth of nonvital cartilage next to the treated cartilage. Human cartilage fixed and stained directly after treatment with laser has shown no gross morphological changes next to the ablation crater (12,18,27). Light microscopic examinations have indicated that the morphology of cartilage cells and matrix is altered in a zone ranging from 2 to 20 μm (12,26). Furthermore, light microscopic examinations of chondromalacic cartilage surface treated with excimer laser have shown a smooth joint surface, and the cartilage surfaces have appeared to be "hardened" (8,10). Scanning electron microscopy of cartilage treated with laser has shown a condensed surface matrix layer and welded collagen fibers (10,12,18,25,26,34). Transmission electron micrographs have indicated the presence of three tissue zones that show no distinct boundaries (12): a 6 μm surface layer that has a honeycomb-like structure surrounded by a homogeneous matrix; a second zone, about 22 μm thick, that still contains damaged chondrocytes, but the matrix structures appear to gradually normalize and then transform into a third layer of morphologically intact joint cartilage.

In short-term follow-ups of patients who had gonarthrosis and were treated with either excimer laser or mechanical shaving (10,27), significantly better clinical results were reported in the laser group. These results appear encouraging, but clear proof of the advantages of the use of a laser is lacking. There have been no results of long-term follow-up with control arthroscopy patients. Although the rate of ablation and the working speed of the excimer laser are comparably low (10), its promising effects on the cartilage surface could make it the modality of choice for debridement of cartilage. Of particular interest is whether surface treatment with excimer laser improves the biomechanical properties of the formerly soft osteoarthrotic cartilage. Because the main functions of articular cartilage are mechanical in nature (i.e., it spreads the applied load over a larger area of contact and thus decreases the applied stress, and it allows for effortless movement between the articulating bones by way of a unique lubrication mechanism), understanding its mechanical properties becomes of paramount importance. A mechanically compromised or "inferior" cartilage would be expected to result in abnormal strain fields or aberrant articulation, or both. For example, a disparity in intrinsic mechanical properties between two articulating surfaces will result in dissimilar strain fields, such that the softer side experiences higher principal strains (3,30). Many studies have demonstrated that degeneration of tissue is accompanied by a decrease in stiffness, strength, and other mechanical properties (1,15). Loss of stiffness is represented by a decrease in aggregate modulus compared with normal tissue. Other biomechanical changes may be manifested in terms of changes in permeability, Poisson's ratio, and thickness. To elucidate the effects of debridement modalities, we quantified and compared the intrinsic biomechanical properties of repair knee articular cartilage in full-thickness and partial-thickness defects in response to the excimer laser and mechanical drilling. We also correlated these findings with light microscopic and scanning electron microscopic examinations of repair tissue after surgery. Therefore, this study was undertaken to investigate the reaction of joint cartilage to treatment with excimer laser in comparison with a mechanical drill, with specific focus on the biomechanical properties of the repair tissue formed in the cartilage defects.

METHODS

Forty-eight 6-month-old male Chinchilla rabbits, with an average weight of 3.6 kg, were used as animal models, as they are well accepted as osteoarthrosis models (6,7,13,17,20-22,24). To avoid major intra-individual and interindividual differences expected in osteoarthrotic cartilage, we examined healing of defects in formerly healthy joint cartilage after use of excimer laser and drilling. To keep intra-individual differences as low as possible, both laser and drilling were used to create defects on one knee of the same animal. The animals were supervised for 2 weeks before surgery in single, standard boxes, where they were allowed activity ad libitum. All surgical procedures were performed at the University of Ulm.

Two days before surgery, the right hindlimb was treated with a depilatory cream. The left knee of each rabbit served as the control (no operation). In all rabbits, atropine was given one-half hour before surgery and anesthesia was initiated with ketamine hydrochloride and maintained by halothane. The knee of the right hind limb was operated on through a lateral parapatellar incision. The patella was luxated medially, and the knee was flexed maximally. Care was taken to keep the joint cartilage moist during the entire surgical procedure. Thus the joint was kept under continuous irrigation with sterile normal saline, and the area of untreated cartilage was covered with a wet gauze. Sixteen cylindrical defects, 1 mm in diameter, were created in each animal: eight in the medial condyle and eight in the lateral condyle. The depth of all of the superficial defects was 0.25 mm, and the depth of the osteochondral defects was 0.5 mm. The depths were chosen according to measurements of the thickness of the joint cartilage in the region where the defect was to be made; these measurements ranged from 0.26 to 0.45 mm.

Thus, superficial defects were in the chondral region, and osteochondral defects reached through the subchondral bone. In all cases, the most cranial defect was a superficial defect, followed by an osteochondral defect and another superficial defect (Fig. 1). The most caudal defect was an osteochondral defect. The defects were created in rows, 0.5 cm from the corresponding lateral rims of the joint surface, and the distance between any two adjacent defects was 0.4-0.5 cm. The first defect was located 0.5 cm distal to the cranial rim in the non-weight-bearing area of the joint. Thus, the more caudal defects were located in the weight-bearing region of the articular surface. All defects on one condyle were made with use of excimer laser, and the defects on the corresponding contralateral condyle were made with a mechanical drill. Due to our randomization scheme, in two of the six animals used for biomechanical testing, the defects on the medial condyle were made with the laser and the defects on the lateral condyle were made by drilling. In the other four animals, the defects on the lateral condyle were made with the laser and the defects on the medial condyle were made by drilling.

The excimer laser used in this study was a Max-10 (308 nm; Technolas, Gräfeling, Germany), with an average pulse duration of 60 ns. Laser energy was transmitted to the joint cartilage through a quartz fiber with a 1 mm core diameter (Ceramoptec, Bonn, Germany). The fiber was kept in contact with the surface of the specimen during ablation using slight pressure and application of a vertical beam. The pulse energy of the laser, measured at the beginning and end of each operation with a power densitometer (AR 9080; Technolas), was 20 mJ, with a repetition rate of 10 Hz. From our prior investigations of depth of defects in the rabbit in relation to the energy applied, we chose an application time of 10 seconds for superficial defects and 25 seconds for osteochondral defects. Drilling was done with a micromotor and small (1 mm diameter) drill heads (Huber, Karlsruhe, Germany) used by dentists. The heads were outfitted with plastic sheaths to restrict the depth of drilling to 0.25 or 0.5 mm. In all animals, the medial condyle was operated on first, and all operations were performed by the same surgeon (R.F.). After the defect was created, the joint was lavaged thoroughly with normal saline to wash out all debris. Then, the joint capsule was closed with 4-0 Vicryl (polygactin; Ethicon, Norderstedt, Germany), and the subcutis and skin were closed in layers. Postoperatively, the animals were allowed cage activity ad libitum and the wounds were checked daily.

In 42 animals, examinations by light microscopy and scanning electron microscopy were carried out at 1, 3, and 6 weeks (12 rabbits each) and at 12 weeks (six rabbits); in the remaining six animals, biomechanical testing was done at 14 weeks. The animals were given a lethal overdose of barbiturate intravenously, and the joints were opened through the same lateral approach. Radiographically, the epiphyses of all animals were about to close. In each animal, the distal femora of both hindlimbs were resected and the defects on each joint were documented photographically. The specimens used for histology were dissected at once, and the defects, including surrounding cartilage and subchondral bone, were fixed individually. The specimens used for biomechanical testing were covered with gauze saturated in lactated Ringer’s solution and immediately frozen to –80°C until the time of transport to the United States. During transport, the specimens were maintained frozen in dry ice and arrived frozen at the University of Texas Health Science Center at San Antonio. There, they were immediately placed in a –80°C freezer until the time of dissection.

At the time of sectioning, each distal femur was thawed at room temperature for 1 hour in a solution of normal saline with protease inhibitors (N-ethylmaleimide, 10 mM; benzamidine HCl, 5 mM; EDTA, 2 mM; and phenylmethylsulfonyl fluoride, 1 mM). As it was exceedingly difficult to core out osteochondral samples containing only one test site without affecting some of the other test sites, a specimen containing all 16 defects was removed by use of a v cut, made with a fine band saw under continual irrigation. The osteochondral specimens were tested using an automated creep indentation apparatus to obtain the creep and recovery behavior of articular cartilage on the joint surface in situ (3). At the time of testing, a fiberoptic positioning system was used to align the loading shaft normal to the cartilage surface. Perpendicularity was achieved within 20 seconds. A tare load of 0.01 N then was applied through a rigid, porous, ultrasonically cleaned tip (0.6 mm diameter), and when the slope of the tare creep became smaller than 1 × 10⁻⁸ mm/sec, a test load of 0.04 N was released automatically onto the cartilage. When the slope of the creep curve became smaller than 1 × 10⁻⁸ mm/sec, as measured with a linear variable differential transformer, the load was removed automatically. The tissue was allowed to recover to equilibrium, and data acquisition ceased automatically. The thickness was measured with a needle probe system.

Both knees of six rabbits were biomechanically tested with use of the creep indentation apparatus. In each experimental right knee, testing was performed on 20 femoral sites: repair tissue in 16 defects (eight created by laser and eight created by drilling) and four adjacent sites that were situated approximately 2 mm away from the periphery of the defects (Fig. 1). In each control left knee, we also tested 20 sites (in locations similar to those in the right knee) for a direct comparison. Ten sites were tested in a day: eight defects on one of the condyles plus two adjacent sites—one adjacent to the second superficial defect and one adjacent to the fourth osteochondral defect (Fig. 1). After testing of these first 10 sites, the specimen was frozen again. The following day, the sample was thawed again for 1 hour and indentation testing was
performed on the 10 test sites (eight defects and two adjacent sites) on the other condyle. The sequence of testing was as follows: the most cranially placed superficial defect was tested first, followed sequentially by the rest to the fourth osteochondral defect. The adjacent sites then were tested. As a result of the testing scheme, each specimen had to undergo either two or three freeze-thaw cycles, which are not believed to affect the tissue’s intrinsic material properties (3,16).

The following material properties of the tissue were determined: the aggregate modulus ($H_a$, in MPa), which is the compressive stiffness modulus of the tissue; Poisson’s ratio ($\nu$), which represents the apparent compressibility of the tissue; and permeability ($k$, in m$^4$/N·s), which is indicative of the interstitial fluid flow inside the pores of articular cartilage (23). To determine these properties, a solution scheme (2) based on biphasic finite element analysis (35) and nonlinear optimization techniques (38) was used. A brief description of this solution scheme follows. Each osteochondral specimen was modeled as an axisymmetric cylinder of uniform depth of a linear biphasic material (23), with unknown properties $K_i$, where $K_i = [H_i, \nu, k]^T$. The central region of the cartilage layer was subjected to a step load $P(t) = P_H(t)$ through a frictional, rigid, porous, cylindrical indenter with material properties $K_i = [3 \times 10^6 \text{ MPa}, 0.25, 2.30 \times 10^{-10} \text{ m}^4/\text{N} \cdot \text{s}]^T$. The adjacent articular cartilage was represented by vector $K_o$, obtained by performing creep indentation experiments of cartilage at least 2 mm away from the repair sites. The material properties of that adjacent tissue ($K_o$) were obtained by following a methodology previously described (2), which assumes a continuous layer of amorphous tissue and few fibroblast-like cells.

RESULTS

Two of the 48 animals had secondary wound healing; these animals had removed the stitches and opened the wound to the subcutaneous tissue. The joint capsule was not opened, and the wounds healed under local therapy within 6 days; histological and bacterial examinations showed no signs of joint infection. Four days after surgery, intake of food and fluid reached the preoperative levels. Effusion of the joint developed at 3 weeks in one of the six animals to have biomechanical testing. This rabbit avoided putting weight on the limb that had been operated on but had a normal intake of food and fluid and no sign of infection. At the time of death, severe osteoarthrosis of the experimental knee was found. All other animals applied weight on the experimental limb 2 weeks after operation and had no or only mild effusion of the joint postoperatively. Of the 42 animals used for histological examination, effusion of the joint developed postoperatively in six; the effusion resolved almost completely by the end of the second week. Osteoarthrosis developed in the experimental knee in four animals in the 6-week group and in four animals in the 12-week group; it persisted until the time of death. These animals were excluded from further analysis.

Light microscopic examinations of superficial defects showed some bulging of the crater edges. One week postoperatively, cartilage cells around the crater edges displayed morphological signs of cell death (necrosis, nuclear condensation, or nucleorrhexis) that were more pronounced in the defects in the laser group. Defects from both the laser and the drilling groups showed no ingrowth of cells or filling with amorphous matrix. Three weeks postoperatively, cel-
FIG. 3. Light micrograph (hematoxylin and eosin, ×250) of an osteochondral defect in the laser group at 3 weeks. The defect is partially filled with fibrous tissue, and cell damage is evident in the surrounding cartilage and subchondral bone.

FIG. 4. Light micrograph (hematoxylin and eosin, ×200) of an osteochondral defect in the drilling group at 3 weeks. A large number of fibroblast-like cells fill the defect. Cell damage at the sides of the defect is not as pronounced as in the osteochondral defects in the laser group at the same time.

FIG. 5. Light micrograph (hematoxylin and eosin, ×200) of an osteochondral defect in the drilling group at 6 weeks. The cells appear round and chondrocyte-like and are partly arranged in a "cluster-like" formation.
FIG. 6. Light micrograph (hematoxylin and
eosin, x300) of an osteochondral defect in the
laser group at 6 weeks. The fibrous tissue in the
defect is more cellular, and a larger amount of
amorphous tissue is present.

FIG. 7. Light micrograph (hematoxylin and
eosin, x250) of an osteochondral defect in the
laser group at 12 weeks. The defect is com-
pletely filled with chondrocyte-like cells and
fibrillated tissue.

FIG. 8. Light micrograph (hematoxylin and
eosin, x250) of an osteochondral defect in the
drilling group at 12 weeks. The amount of fi-
brillated tissue is increased, and fewer cells are
in the defect. Chondrocyte-like cells partly
forming “clusters” are still visible.
lular necrosis still was visible, with an increasing number of empty cell lacunae in the cartilage tissue next to the craters. Clusters of cells appeared in the surrounding cartilage tissue, and the bottom of some of the defects was covered with a thin layer of amorphous tissue (Fig. 2). After 6 and 12 weeks postoperatively, the number of cell clusters in the surrounding cartilage tissue increased and fibroblasts partly covered the surface of the crater bottom in both groups.

Osteochondral defects, which penetrated the medullary cavity of the underlying bone, showed the same signs of tissue damage around the crater edges. One week postoperatively, fibrin clots partly filled the osteochondral defects. The clots consisted mostly of amorphous matrix with some fibroblast-like cells and were clearly less pronounced in the defects in the laser group. At 3 weeks postoperatively, the osteochondral defects in the laser group resembled the defects in the drilling group at 1 week (Fig. 3); at 3 weeks, the osteochondral defects in the drilling group had a greater extent of healing, showing more cells per square area (Fig. 4). At 6 weeks, the tissue in half of the defects in the drilling group showed morphological changes—cells resembling chondrocytes partly formed cluster-like configurations (Fig. 5). In contrast, at that same time defects in the laser group contained fibrous tissue with a greater density of fibroblast-like cells and an increased amount of matrix (Fig. 6). Only at 12 weeks postoperatively did both groups have similar healing—the defects were filled with fibrillated tissue, cartilage-like cells, and cluster-like cell formations (Figs. 7 and 8).

At 3 weeks, scanning electron microscopy showed that the defects in the laser group were partially filled (Fig. 9) and had smooth crater edges, with "welding" of collagen bundles on higher magnifications (Fig. 9).

FIG. 9. Scanning electron micrograph (×100) of an osteochondral defect in the laser group at 3 weeks. The boxed area is enlarged on the right. There is only a small amount of amorphous tissue (left), and the crater edges are "welded" (right).

FIG. 10. Scanning electron micrograph (×340) of an osteochondral defect in the drilling group at 3 weeks. There is a greater amount of tissue filling (left), and the crater edges are partly covered by fibrous tissue. On the right is an enlargement of the boxed area. There is a rough surface with collagen fibers extending from the cartilage matrix.
In defects in the drilling group, crater edges had a rough surface with single outstanding collagen fibers (Fig. 10), but overall, there was more filling of the defect (Fig. 10). With increasing time after surgery, the defects in both groups filled with amorphous tissue.

Of the six rabbits used for biomechanical examinations, a total of 239 sites were tested with the creep indentation apparatus and analyzed with use of the finite element/optimization procedure. In 27 defects (11 superficial and 16 osteochondral), articular cartilage was absent. A typical creep-recovery curve of tissue that had formed in an osteochondral defect in the laser group is shown in Fig. 11A. The average creep and recovery times for specimens in this group were 725 ± 427 and 1,033 ± 508 seconds, respectively, with an 80 ± 13% recovery. Figure 11B depicts the curve-fit of the example shown in Fig. 11A. The average creep and recovery times for specimens in the drilling group were 737 ± 427 and 1,058 ± 471 seconds, respectively, with an 81 ± 20% recovery. A typical creep-recovery curve for a contralateral, "normal" articular cartilage site is shown in Fig. 12A. The average creep recovery time for the contralateral control tissue was 1,080 ± 596 seconds, with a 90 ± 9% recovery and an average creep time of 770 ± 422 seconds. The curve-fit of this example is given in Fig. 12B.

For cartilage from defects in the laser group, the aggregate modulus (0.40 ± 0.24 MPa) was significantly less than that in the contralateral (control) tissue (0.59 ± 0.18 MPa; p = 0.0001) or adjacent sites (0.56 ± 0.26 MPa; p = 0.01). The Poisson's ratio (0.37 ± 0.08) was significantly greater than that for the control tissue (0.23 ± 0.25; p = 0.0001) or adjacent sites (0.19 ± 0.11; p = 0.0001), and the permeability (3.72 ± 4.28 × 10^{-15} m^4/N·s) also was significantly greater than that for the control (1.94 ± 0.96 × 10^{-15} m^4/N·s; p = 0.0001) or adjacent (2.08 ± 1.22 × 10^{-15} m^4/N·s; p = 0.08) tissue. The cartilage was significantly thicker (0.20 ± 0.06 mm) than control tissue (0.27 ± 0.08 mm; p = 0.0001) or adjacent sites (0.29 ± 0.09 mm; p = 0.0001).

For cartilage from defects in the drilling group, the
aggregate modulus (0.39 ± 0.23 MPa) was significantly less than that for the control tissue (p = 0.0001) or adjacent sites (p = 0.01). The Poisson’s ratio (0.34 ± 0.09) was significantly greater than that for the control (p = 0.003) or adjacent (p = 0.0001) sites, and the permeability (3.82 ± 3.44 × 10⁻¹⁵ m⁴/Ns) also was significantly greater (control tissue, p = 0.0001; adjacent sites, p = 0.02). The cartilage was significantly thicker (0.22 ± 0.09 mm) than control (p = 0.005) or adjacent (p = 0.008) tissue.

Because the biomechanical properties did not differ significantly between the two groups, the data from both groups were pooled and designated as repair tissue. These pooled data then were compared with the data for all of the adjacent sites (right knees) and the control sites (left knees) (Table 1). The results indicated that the repair tissue had a smaller aggregate modulus (control tissue, p = 0.00001; adjacent sites, p = 0.004), greater Poisson’s ratio (control tissue and adjacent sites, p = 0.0001), greater permeability (control tissue, p = 0.0001; adjacent sites, p = 0.04), and was thinner (control tissue and adjacent sites, p = 0.0001). In contrast, the material properties of the control cartilage were not significantly different from those of adjacent sites (aggregate modulus, p > 0.54; Poisson’s ratio, p > 0.49; permeability, p > 0.56; and thickness, p > 0.36). Table 2 shows specific comparisons of the intrinsic material properties and thickness of repair cartilage between defect group (laser or drilling), location (lateral or medial), depth of defect (chondral or osteochondral), location and depth of defects in the laser group, and location and depth of defects in the drilling group. As seen in Table 2, the statistical results indicated that the only significant difference was in the aggregate modulus of cartilage in the drilling group between lateral and medial locations; specifically, the tissue from the lateral defects was 74% stiffer than tissue from the medial defects.

**DISCUSSION**

Debridement of diseased articular cartilage in arthrotic joints is employed widely as a treatment mo-

### TABLE 1. Comparison of intrinsic material properties

<table>
<thead>
<tr>
<th></th>
<th>Aggregate modulus (MPa)</th>
<th>Poisson’s ratio</th>
<th>Permeability (×10⁻¹⁵ m⁴/Ns)</th>
<th>Thickness (mm)</th>
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<tbody>
<tr>
<td>Repair tissue (n = 69)</td>
<td>0.39 ± 0.24</td>
<td>0.35 ± 0.09</td>
<td>3.76 ± 3.89</td>
<td>0.21 ± 0.08</td>
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<tr>
<td>Adjacent sites (n = 24)</td>
<td>0.56 ± 0.26</td>
<td>0.19 ± 0.11</td>
<td>2.08 ± 1.22</td>
<td>0.29 ± 0.09</td>
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<tr>
<td>Contralateral control tissue (n = 96)</td>
<td>0.59 ± 0.18</td>
<td>0.23 ± 0.25</td>
<td>1.94 ± 0.96</td>
<td>0.27 ± 0.08</td>
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</table>

*Repair tissue includes cartilage pooled from defects in the laser and drilling groups; articular cartilage was absent in 27 defects.

**DISCUSSION**

Debridement of diseased articular cartilage in arthrotic joints is employed widely as a treatment mo-

### TABLE 2. Comparison of intrinsic material properties of repair cartilage

<table>
<thead>
<tr>
<th></th>
<th>Aggregate modulus (MPa)</th>
<th>Poisson’s ratio</th>
<th>Permeability (×10⁻¹⁵ m⁴/Ns)</th>
<th>Thickness (mm)</th>
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</thead>
<tbody>
<tr>
<td>Laser group (n = 38)</td>
<td>0.40 ± 0.24</td>
<td>0.37 ± 0.08</td>
<td>3.72 ± 4.28</td>
<td>0.20 ± 0.06</td>
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<tr>
<td>Drilling group (n = 31)</td>
<td>0.39 ± 0.23</td>
<td>0.34 ± 0.09</td>
<td>3.82 ± 3.44</td>
<td>0.22 ± 0.09</td>
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<tr>
<td>Location</td>
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<td>Pooled repair tissue</td>
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<tr>
<td>Lateral (n = 36)</td>
<td>0.42 ± 0.28</td>
<td>0.36 ± 0.10</td>
<td>4.04 ± 4.44</td>
<td>0.20 ± 0.07</td>
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<tr>
<td>Medial (n = 33)</td>
<td>0.36 ± 0.18</td>
<td>0.35 ± 0.07</td>
<td>3.46 ± 3.24</td>
<td>0.21 ± 0.08</td>
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<tr>
<td>Laser group</td>
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<tr>
<td>Lateral (n = 25)</td>
<td>0.37 ± 0.25</td>
<td>0.37 ± 0.09</td>
<td>3.53 ± 3.86</td>
<td>0.20 ± 0.07</td>
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<tr>
<td>Medial (n = 13)</td>
<td>0.45 ± 0.21</td>
<td>0.36 ± 0.07</td>
<td>4.09 ± 5.13</td>
<td>0.19 ± 0.05</td>
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<tr>
<td>Drilling group</td>
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<tr>
<td>Lateral (n = 11)</td>
<td>0.54 ± 0.30*</td>
<td>0.34 ± 0.13</td>
<td>5.21 ± 5.57</td>
<td>0.20 ± 0.09</td>
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<tr>
<td>Medial (n = 20)</td>
<td>0.31 ± 0.14*</td>
<td>0.34 ± 0.07</td>
<td>3.05 ± 0.76</td>
<td>0.23 ± 0.09</td>
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<td>Depth</td>
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<tr>
<td>Pooled repair tissue</td>
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<tr>
<td>Superficial (n = 37)</td>
<td>0.39 ± 0.23</td>
<td>0.36 ± 0.09</td>
<td>4.05 ± 4.18</td>
<td>0.20 ± 0.07</td>
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<tr>
<td>Osteochondral (n = 32)</td>
<td>0.40 ± 0.25</td>
<td>0.37 ± 0.09</td>
<td>3.42 ± 3.55</td>
<td>0.21 ± 0.09</td>
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<tr>
<td>Laser group</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Superficial (n = 20)</td>
<td>0.38 ± 0.25</td>
<td>0.37 ± 0.07</td>
<td>4.21 ± 4.21</td>
<td>0.19 ± 0.06</td>
</tr>
<tr>
<td>Osteochondral (n = 18)</td>
<td>0.41 ± 0.24</td>
<td>0.36 ± 0.10</td>
<td>3.18 ± 4.40</td>
<td>0.20 ± 0.07</td>
</tr>
<tr>
<td>Drilling group</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Superficial (n = 18)</td>
<td>0.39 ± 0.21</td>
<td>0.33 ± 0.10</td>
<td>3.86 ± 4.25</td>
<td>0.22 ± 0.07</td>
</tr>
<tr>
<td>Osteochondral (n = 13)</td>
<td>0.39 ± 0.27</td>
<td>0.36 ± 0.08</td>
<td>3.75 ± 1.98</td>
<td>0.23 ± 0.11</td>
</tr>
</tbody>
</table>

*The two values were significantly different (p < 0.05) from each other.
dality. It usually is performed with the use of a mechanical shaver or with laser. To examine the effects of excimer laser and mechanical drilling on articular cartilage, the biomechanical properties of repair cartilage were compared in full-thickness and partial-thickness defects in the distal femora of rabbits. Light microscopy and scanning electron microscopy also were performed. The results indicated that the biomechanical properties of neocartilage in defects created by either a drill or excimer laser were similar to each other but significantly inferior to the mechanical properties of normal articular cartilage. There was microscopic evidence that healing in osteochondral defects created by laser may be delayed for at least 6 weeks compared with osteochondral defects created by drilling.

As observed with light microscopy, tissue reaction to either modality was similar to the typical repair process in rabbit joint cartilage, as reported in the literature (17,20,21,24,25). Specifically, we found swelling of cartilage at the crater edges 1 week postoperatively in both groups, similar to that reported by Puhl et al. (24,25). Other investigators have observed cellular necrosis in adjacent tissue layers after debridement of cartilage (4,20,21,31), and we also noted cellular necrosis in defects created by drilling; however, they extended to only a few cartilage layers. Cellular necrosis next to defects created by laser appeared in more tissue layers. This may be explained by additional thermal effects damaging the cartilage during ablation of tissue (8,9). As expected, superficial defects in both groups showed only minor signs of repair between 1 and 12 weeks. This observation is in agreement with findings by other investigators who have reported that only a thin layer of amorphous tissue covered the bottom of partial-thickness defects with only few or no fibroblast-like cells (17,20). In this study, there were no gross differences between the two groups.

The osteochondral defects in the drilling group quickly filled with blood during the operation. As suggested by Campbell (6) and DePalma et al. (7), undifferentiated cells from the marrow enter the defect, modulate into primitive fibroblasts, and form vascular fibroblastic repair tissue. One week postoperatively, we usually found a loose fibrovascular network partly filling these defects, as has been described elsewhere (5,11,14,17,18,20,24,27,29). The repair tissue became more cellular after 3 weeks. In one defect only, we found metaplasia to chondrocyte-like cells after 3 weeks, whereas all other defects were partly or completely filled by granulation tissue. In contrast, Salter et al. (29) reported a higher percentage of cell metaplasia at 3 weeks in their study's group of adolescent rabbits. By 6 weeks, the tissue in most of the defects had converted to a hyaline-like chondroid tissue, as was described by Mitchell and Shepard (22). Although perforation of the osseous endplate with the drill led to bleeding into all of the defects, only a few defects created by laser filled with blood clots during the operation. This may be explained by coagulation-like phenomena caused by thermal effects of the laser. These phenomena could explain the delayed repair that was observed in the laser group.

Lasers have been applied not only for the debridement of fibrillated cartilage but also as an energy source to stimulate metabolic changes of cartilage tissue. Previous reports on the application of low-level Nd:YAG lasers have shown that repair in partial-thickness cartilage defects in guinea pigs was stimulated (32) and that cartilage metabolism in articular cartilage explants was altered for as long as 2 weeks (36). These results, which appear to indicate that cartilage metabolism in defects created by laser may be initially stimulated, are in contrast with those of our study, which showed delayed healing of osteochondral defects for as long as 6 weeks postoperatively.

Scanning electron microscopic examinations of the cartilage surface in defects in the laser group resembled those of human joint cartilage (12,13,19,28); there was a condensed surface and "welding" of the collagen fibers at the crater edge. In comparison, defects in the drilling group showed a disrupted amorphous matrix with single collagen fibers sticking out of the matrix and into the defects, as was described by Kim et al. (17). Until 12 weeks after operation, there were few changes in the defect edges in both groups and fibrous tissue had filled the defects.

The results of biomechanical testing at 14 weeks showed that there were no statistically significant differences between the defects created by laser or drilling; therefore, they were grouped together as repair tissue. Compared with contralateral control cartilage, repair tissue was 34% softer, had a 52% higher apparent compressibility, was 94% more permeable, and was 22% thinner. Thus, although grossly, morphologically, and histologically the repair tissue may resemble hyaline cartilage, biomechanically the repair tissue was inferior to normal articular cartilage. Similarly, the properties of the repair tissue also were significantly different from those of adjacent tissue, which suggests that creation of "small" defects with excimer laser or a drill appears to have negligible effect on cartilage that is situated approximately 2 mm away from the defect. Comparison with the intrinsic material properties of repair cartilage indicated that there were no significant differences between any of the factors, except between the aggregate modulus of cartilage from lateral or medial defects in the drilling group. This suggests that, at 14 weeks postoperatively, the method of creation, the location, and the depth of the defect have no appreciable effect on the structural integrity.
of the tissue from the defect, as manifested by the intrinsic mechanical properties.

Summarizing the results of light microscopy and scanning electron microscopy, it was shown that defects created by laser healed more slowly for the first 6 weeks than did defects created by drilling; however, by 12 weeks, similar healing responses for both types of defects were observed. Biomechanical tests performed at 14 weeks confirmed these observations, as the biomechanical properties of the two types of defects were similar. Although initial differences in the healing responses were significant, these variations vanished with time, as evidenced by both methods of observation.

In conclusion, our biomechanical testing demonstrated that even though repair tissue appeared to be morphologically cartilage-like, its intrinsic mechanical properties suggested that chondroid tissue at 14 weeks has inferior structural integrity compared with normal articular cartilage. Thus, in order to determine the functional abilities of articular cartilage, biomechanical testing may be a more suitable method. Furthermore, there appears to be no statistically relevant differences in biomechanical properties between defects created by excimer laser or by drilling, although there were initially considerable differences in the results of light microscopy and scanning electron microscopy. Of particular interest is that, at 14 weeks, there were no significant differences with respect to the depth of penetration of the defect or its location.

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