

Degenerative Lesions Of The Patellofemoral Joint: An Autopsy Study

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Abstract: 30 knee joints were collected from individuals who were between 55 and 85 at the time of death. Three distinct types of lesions were observed on the patellofemoral joint. Type I (fibrillation) occurred on 11 of the 30 patellofemoral surfaces. Cracks (Type II) through the cartilage from the surface to the lower mid- or deep zone occurred in seven specimens, and Type III (erosion), a localized depression of the cartilage with well defined boundaries, was present in 6 of the joints. Five of the patellofemorals were relatively undamaged, and one was eburnated.

The lesions types appeared on different sections of the joints, and had distinct microscopic appearances in various grades of severity. The lesion types do not progress from one type to another. Rather, the lesion types differ from the earliest moments of their formation and resulted from the varying mechanical and biochemical environments.

Key Words: Knee, Knee Joint, Degenerative Joint Disease, Osteoarthritis

I. Introduction

The articular cartilage lesions associated with degenerative joint disease were first clearly delineated by Weischelbaum in 1877 (1), who also correlated the microscopic features of the lesions with their gross appearances. Weischelbaum was also one of the first workers to note the regular as opposed to random appearance of lesions on joint surfaces.

In 1934, Parker, Keefer, Myers & Irwin (2) concluded that the lesions of advancing age were of exclusively one type and progressive, from fibrillation and subchondral thickening, to total cartilage loss, or eburnation. They explained the development of these lesions by invoking the theories of Pommer (3), who proposed that with an initial loss of elasticity in the articular cartilage, the subchondral bone would no longer be protected from the localized effects of pressure, and impact. Further, this lack of protection and the subsequent trauma caused increased vascularity and ultimately bony sclerosis.

In their systematic review of the age changes in the knee joint, Bennet, Waine and Bauer (4) noted that all of the joints obtained from individuals beyond the second decade of life exhibited, to some degree, alterations similar to those observed in old age. Their conclusion that age related joint changes were primarily a degenerative process of hyaline cartilage was influenced by their belief that articular cartilage had little or no ability to repair itself.

The first researchers to suggest an etiology for osteoarthritis more complex than degeneration due to weight bearing were Harrison, Schajowicz & Trueta in 1953 (5). They found that daily use ensures adequate cartilage nutrition and therefore preserves rather than destroys articular cartilage. Further, they proposed that the lesions resulting from too much or too little pressure were different from one another.

The establishment of osteoarthritis as a disturbance of joint remodeling was forwarded by Lent C. Johnson first in 1959 (6) and again in 1962 (7). In the normal joint, Johnson proposed a balance between progressive and regressive remodeling. Progressive remodeling is identified by an increased thickness of the articular cartilage, a thickened subchondral plate due to the turnover of calcified cartilage into bone, and the deposition of new bone by osteoblasts. Regressive remodeling, on the other hand, was due largely to

the wearing away of the articular cartilage due to the joint's load bearing, and osteoclastic resorption of the underlying bone. Johnson suggested that osteoarthritis resulted from decompensated or unbalanced remodeling.

The details of cartilage and bone remodeling in relation to advancing age began to be worked out by Lane, Villancin & Bullough in 1977 (8). These authors found that both the number of blood vessels entering the calcified cartilage and the rate of endochondral ossification change with age. These changes in modeling activity of the joint may meet an increased need for compensating remodeling due to a loss of joint stability in older individuals due to neuromuscular degeneration.

Although many workers in the past have blurred the distinctions between the various types of joint disease, the current scientific consensus is that the joint changes in osteoarthritis are markedly different from those of age. The relationship between age-related degeneration and osteoarthritis remains, however, unclear. It is not known whether the changes of age predispose the joint to the development of osteoarthritis, or whether osteoarthritis develops independent of aging (9). It is the purpose of this paper to report our observations on degenerative lesions in the patellofemoral joint.

II. Materials and Methods

40 human knee joints were collected at either autopsy or from Cornell University Medical College anatomy lab cadavers. Ten knee joints from children and young adults were used for comparison, and only 30 of the original 40 knees from individuals between the ages of 55 and 85 were selected for study. The knee joints were preserved in a formalin and glycerine solution. Each knee was then disarticulated, described, sketched and photographed using ultraviolet illumination to depict maximum lesion detail (10). Parameters of gross description were: observation of chondromalacia and cartilage lesions, and position of cartilage lesions. Lesion types were then grouped and arranged in a series from the least damaged to the most severely damaged. Progressive pathways of lesion development were thus observed.

Twenty-one of the patellae and patellofemora were completely cut on the band saw in 2 millimeter serial sections, and representative sections from the lesioned areas of the remaining knees were cut. These sections were

decalcified in 5% nitric acid, run through a series of alcohol washes, and embedded in paraffin for histological sectioning. At least two, 4 micrometer sections were taken from each block using a Reichert microtome, and stained with hematoxylin and eosin. Each slide was examined using transmitted light microscopy with both blue and polarized filters. Parameters for microscopic description were: articular cartilage cellularity and matrix proteoglycan levels, levels of cloning, thickness, regularity and duplication of the tidemark, thickness and regularity of calcified cartilage, levels of bony sclerosis and levels of subchondral lamellar and woven bone. In all, 500 histological sections were cut and examined.

III. Results: Description of the Three Lesion Types

Type I: Fibrillation

Fibrillation was the most common lesion of the patellofemoral articulation. 11 of the 30 femora were fibrillated (Specimens: 2, 3, 4, 6, 7, 8, 10, 11, 16, 20 & 22) (Table 1). Fibrillation occurred either mildly in which only a few fibrils were pulled off the cartilage surface, or severely, in which the entire

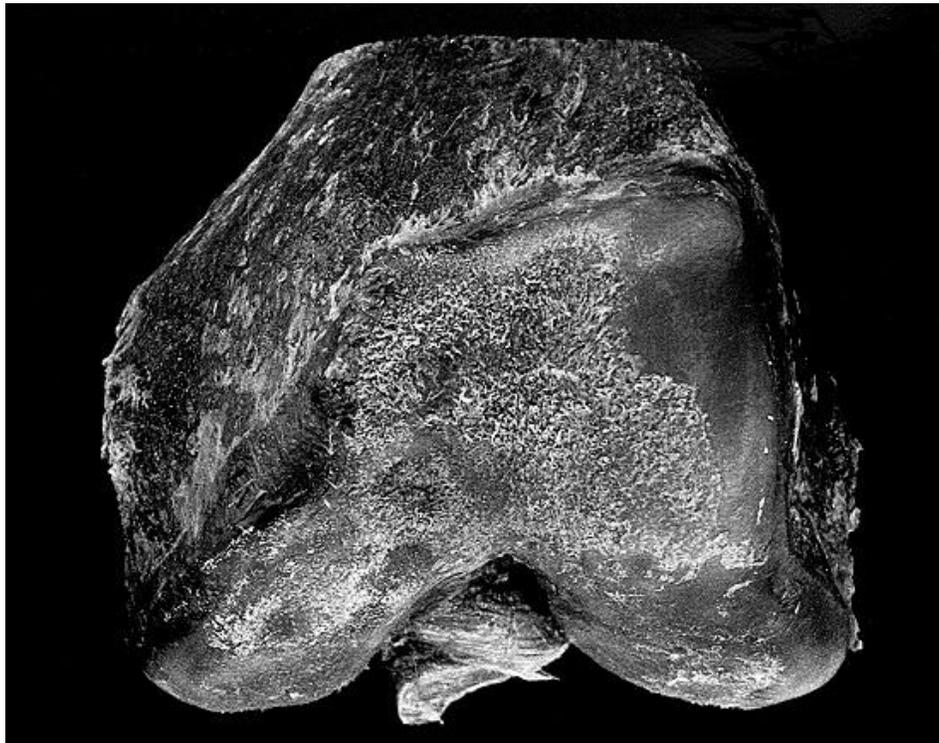


Figure 1. An ultraviolet photograph of a Type I lesioned (fibrillated) patellofemoral (specimen 7). This specimen shows widespread and severe fibrillation. Medial is to the left.

surface of the joint had the appearance of a shag rug (See Figure 1). In its earliest stages it appeared as dimpling and scratching in the intercondylar region of the patellofemoral joint. From this initial focus, fibrillation next appeared on the medial condyle and then distally on the lateral patellofemoral condyle. The first appearance of fibrillation on the patella was around the periphery of the joint surface, then the lesion also appeared on the upper lateral and lower central facet.

	Specimen Number	Femoral Lesion	Patellar Lesion
Table 1. Lesion Types in Study Sample	2	Fibrillation	Fibrillation
	3	Fibrillation	Cracking/Erosion
	4	Fibrillation	Fibrillation/Erosion
	6	Fibrillation	Fibrillation
	7	Fibrillation	Fibrillation/Erosion
	8	Fibrillation	Fibrillation
	9	Cracking	Cracking
	10	Fibrillation	Fibrillation/Cracking
	11	Fibrillation	Fibrillation/Cracking
	12	Undamaged	Undamaged
	16	Fibrillation	Fibrillation/Erosion
	17	Cracking	Fibrillation
	18	Cracking	Fibrillation/Cracking
	19	Cracking	Fibrillation/Cracking
	20	Fibrillation	Eroded
	21	Cracking	Fibrillation/Erosion
	22	Fibrillation	Fibrillation
	23	Cracking	Fibrillation/Cracking
	24	Erosion	Fibrillation/Eroded
	25	Erosion	Fibrillation/Cracking
	26	Erosion	Fibrillation/Cracking
	28	Cracking	Fibrillation/Cracking
	29	Erosion	Fibrillation/Cracking
	30	Eburnation	Eburnation
	31	Undamaged	Fibrillation
	32	Undamaged	Fibrillation
	33	Undamaged	Fibrillation/Erosion
	34	Erosion	Fibrillation/Cracking
	35	Erosion	Fibrillation/Cracking
	36	Undamaged	Fibrillation

The patellae paired with the 11 fibrillated patellofemora were either exclusively fibrillated (Specimens: 2, 6, 8 & 22), or eroded (# 20), or showed mixed lesions on different joint facets. Three specimens showed a mixture of fibrillation and erosion (4, 7 & 16), two specimens had a mixture of fibrillation and cracking (10 & 11), and one specimen (# 3) was both cracked and eroded. Of these



Figure 2. A photomicrograph of a fibrillated patellofemoral (specimen 16). Note the very thick and rippled tidemark, thickened calcified cartilage, and moderately sclerotic subchondral bone. (H&E x2 obj).

eleven knee joints, seven of them were more severely damaged on the patella than patellofemoral.

Microscopic examination of fibrillated joints showed a widespread lesion with little or no noticeable cartilage loss in the lesion areas (See. Figure 2). The upper zone cartilage was lightly stained, from which we can infer high proteoglycan content. There was a mild to moderate degree of chondrocyte cloning, especially near the lesions, and a surface ghosting of cells. The layer of calcified cartilage

was mildly to moderately thickened, especially in the patellofemoral intercondylar region, and the tidemark in fibrillated joints was thicker, darker, more smeary, and more locally rippled than in other lesion types. Effected tidemarks tend to appear medial to a lesion on the medial condyle or facet, and lateral to a lesion on the lateral condyle or

facet. The appearance of the tidemark on fibrillated patellae and patellofemora is nearly identical. There was also a mild to moderate deposition of sclerotic bone, again prominent in the intercondylar region. A large percentage of this bone was woven bone, existing in small islands. The trabeculae immediately beneath the subchondral layer tended to be thickened and also had woven bone cores. Another microscopic feature unique to fibrillated joints is a large amount of calcified cartilage left behind deep in the bone, and not modeled out.

Type II: Cracking

Cracking was the second most common lesion in the patellofemoral sample. It occurred in 7 of 30 specimens examined (Specimens: 9, 17, 18, 19, 21, 23 & 28). Cracking began in the intercondylar region of the patellofemoral joint, and seemed to follow one of two progressive pathways. Either the cracks spread across the surface of the joint, causing chunks of articular cartilage to break off, or the cracks extended deeper into the cartilage. (See Figure 3).

Cracking on the patella was always accompanied by fibrillation across all facets. This fibrillation is thought to be secondary to the original cracking for three reasons: 1.) Fibrillation was seen alone on 11 femoral specimens. 2.) These fibrillated specimens could be arranged in a series ranging from little damage to extreme damage 3.) Exclusively fibrillated specimens of all degrees of severity **always** had a histological pattern that was different from the cracked and fibrillated specimens. Cracks on the patella first occurred either in the central or lower central regions, and then also appeared on the upper lateral and medial facets.

The patellae paired with the cracked patellofemora most frequently showed a mixed lesion of cracking and fibrillation. This occurred in four of the seven specimens (Specimens: 18, 19, 23 & 28). The patellar cracking was usually on the central and medial facets. Other lesions of patellae paired with cracked patellofemora were fibrillation (# 17), cracking (# 9) and a mixed lesion of fibrillation and erosion (# 21). Of these 7 cracked specimens, 5 of them had a much more severely damaged patella than patellofemoral.

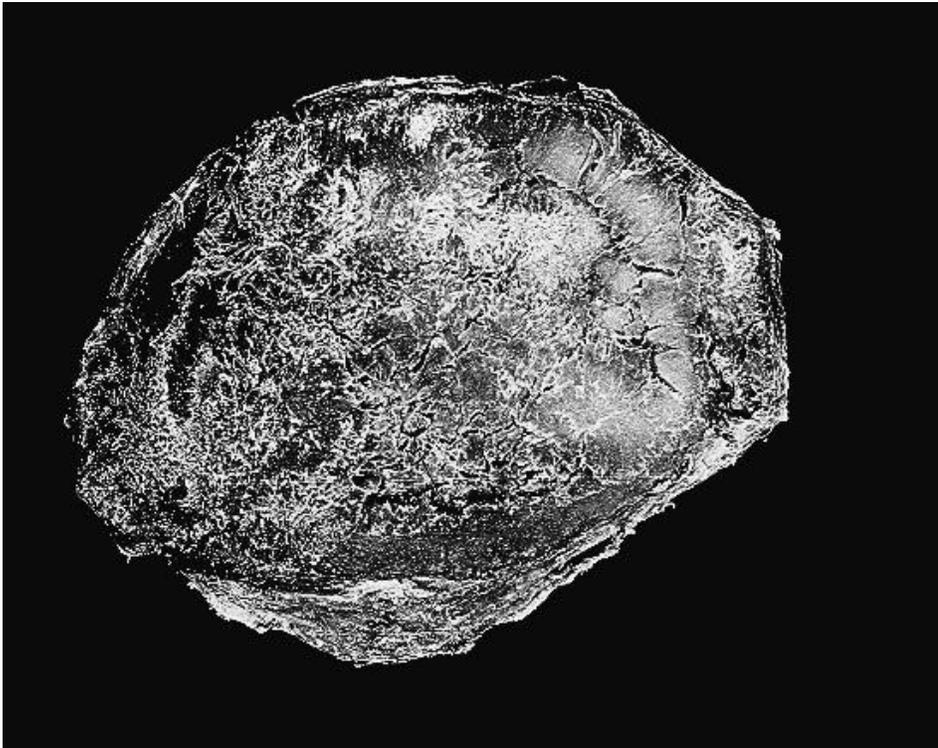


Figure 3. An ultraviolet photograph of a severely Type II lesioned (cracked) patella (specimen 28). Arrangement of all cracked specimens in a graded series from least to the most damaged suggests that the fibrillation in this specimen appeared secondary to the cracking. The histological sections of this patella show only characteristics of cracking and none of fibrillation. Medial is to the left.

The microscopic features of cracking included very thick articular cartilage, with cracking as a localized lesion (Figure 4c). The lesions usually ran from the surface to the lower mid-zone or deep cells. Also, unique to cracked joint are horizontal lesions in the middle of the cartilage which do not appear to communicate with the surface. Cells around the lesion stained very darkly extra-territorially, from which we infer very low proteoglycan content. There was some cloning, and subsequent bundling of collagen in the lesion areas, probably to increased proteoglycan production by the clones.

Due to the thick articular cartilage in cracked specimens, the calcified cartilage appeared very thin. It was, however, relatively thicker than the calcified cartilage in a normal joint (compare Figure 4a with Figure 4c). The calcified cartilage layer was also very irregular. Reactive tidemarks in cracked joints usually occurred in the presence of the mid-zone lesions, but also were seen when a surface lesion penetrated to the mid-zone articular

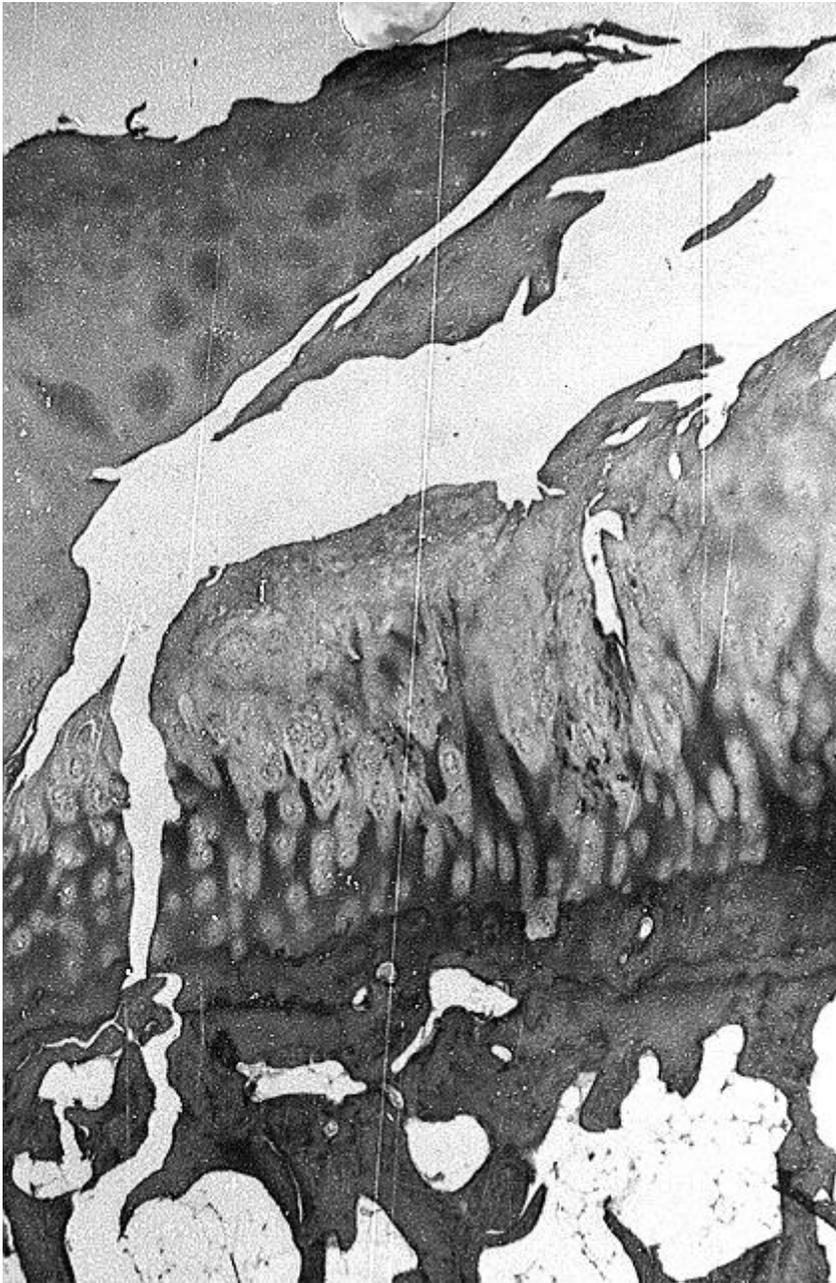


Figure 4. A photomicrograph of a cracked patellofemoral (specimen 28). Note the very thick articular cartilage, thin and distinct tidemark, thin calcified cartilage, and only mildly sclerotic subchondral bone. (H&E x2 obj).

cartilage. The disturbed tidemark appeared as an irregular flame pattern with local smeariness, and tended to be more reactive on cracked patellofemora than on cracked patellae. Further, lesioned patellae showed more dystrophic calcification of lower-zone chondrocytes than did the patellofemora. The bony arcade in cracked specimens was mildly sclerotic, with only small amounts of woven bone.

Type III: Erosion

Erosion was the least common lesion type in the patellofemoral sample, and was present in 6 of the 30 femora (Specimens: 24, 25, 26, 29, 34 & 35). This type of lesion appeared as a localized depression in the cartilage with very well defined boundaries (See Figure 5). Eroded cartilage first appeared in the intercondylar region of the patellofemoral as a canalated area, stripped of surface cartilage. From this focus, the lesion appeared most commonly on the lateral condyle. Erosions on the patella occurred either horizontally across all of the joint facets, or vertically on the medial facet.

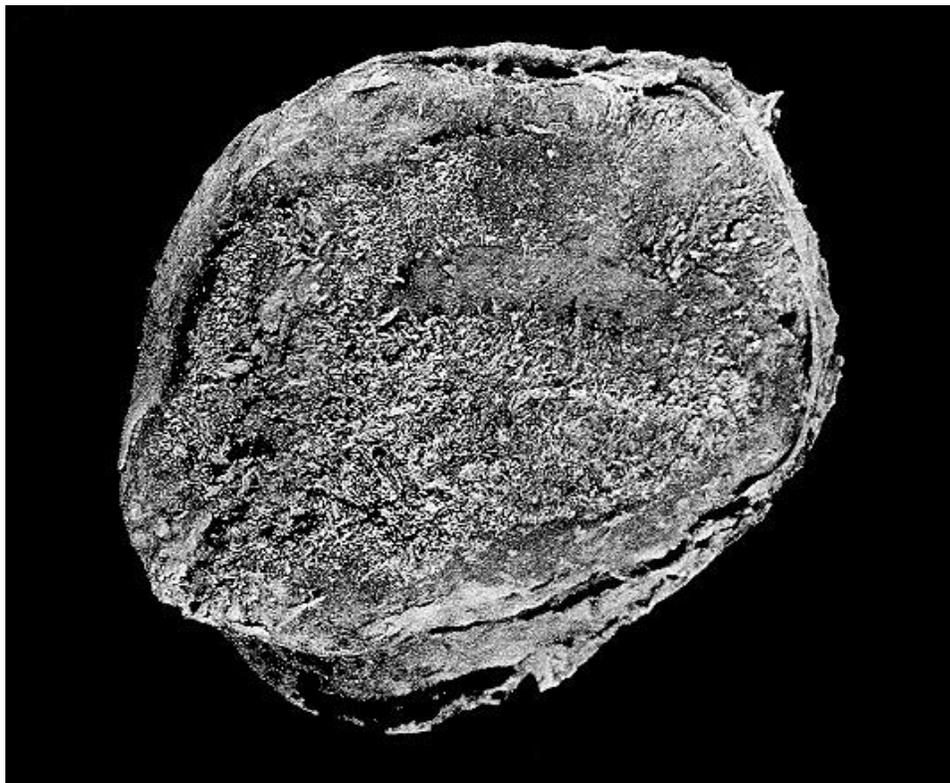


Figure 5. An ultraviolet photograph of a Type III lesioned (eroded) patella (specimen 4). The erosion appears as a stripe across all joint facets. Medial is to the left.

Of the six eroded patellofemora, five of them had a mixed lesion of fibrillation and cracking in the matched patellae (Specimens: 25, 29, 34 & 35). These cracked patellae were similar to those matched with cracked patellofemora, and were fibrillated in a stripe across all of the joint facets, and tended to be cracked centrally, or on the medial facet. One patella matched with an eroded patellofemoral (# 24), and showed a mixed lesion of fibrillation and erosion. Five of these six matched pairs were moderately

to severely and equally damaged (specimens: 24, 25, 29, 34 & 35). Specimen # 26 was more severely lesioned patellofemoral than patella.



Figure 6. A photomicrograph of an eroded patellofemoral (specimen 24). Note the very thick articular cartilage, thin and distinct tidemark, thin calcified cartilage, and only mildly sclerotic subchondral bone. (H&E x2 obj).

The microscopic features of eroded joints were moderate to severe cartilage loss in the localized lesion areas (Figure 4d). The territorial matrices of the chondrocytes were visible to the lower upper-zone cells, and the joint surface was lightly stained, from which we inferred high proteoglycan content. There was some cloning of cells near the lesion. The most striking feature of eroded joints was the extremely thick layer of calcified cartilage, especially prominent in the intercondylar

region of the patellofemoral. The tidemark in eroded joints was thin and distinct with very little irregularity, and was most often doubled. Further, the reactive tidemarks on eroded patellae were more irregular than on patellofemora. The bony arcade in eroded specimens was highly sclerotic,

especially in the intercondylar region of the patellofemoral. The sclerotic bone in eroded specimens was mostly lamellar, with scarce amounts of woven bone.

One of the 30 patellofemoral joints (# 30) examined showed a lesion pattern that did not fit into one of the established categories. Part of the joint surface was completely devoid of cartilage, or eburnated, leaving only bone or calcified cartilage exposed. Further, its matched patellae was eburnated in exactly the same area. The microscopic features of eburnation were cartilage only at the edges of the lesion, with a darkly stained surface, from which we inferred very low proteoglycan content. Also present in the eburnated joint was a large amount of replacement fibrocartilage, of subchondral origin, spreading over the lesioned areas. The bony arcade was thick and sclerotic in lesioned areas, and tended to thin a bit towards the edges of the lesion. Large tracts of lamellar bone alternated with areas of almost exclusively woven bone.

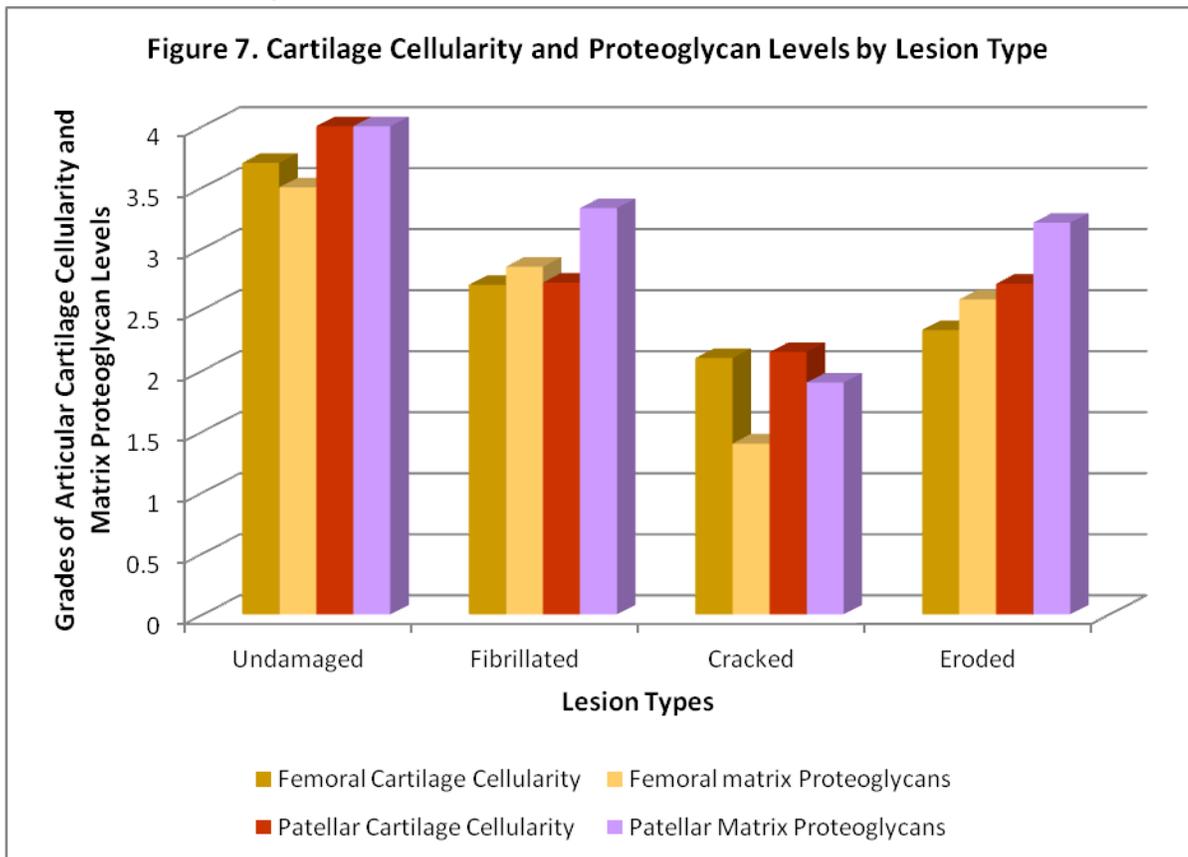


Figure 7: Articular cartilage cellularity and matrix proteoglycan grades in the relatively undamaged, fibrillated, cracked and eroded patellae and patellofemora. All characters were graded on a scale from 0 to 4 (0,1,1.5,2,2.5,3,3.5,4) with 0 representing the absence of a character and 4 indicating a high degree.

IV. Discussion

Conventional views of the pathogenesis of degenerative lesions have focused primarily on the dissolution of the articular cartilage. In fact, Meachim (11 & 12) thought it possible to, "arrange a suitable collection of histologic sections to demonstrate a continuous series of changes, with superficial fibrillation of cartilage at one end of the series, and at its other end, deep erosion of cartilage, bone exposure, subchondral sclerosis and osteophyte formation." Other workers (Radin (13) and Radin & Rose (14)), have stressed the importance of subchondral bony sclerosis as the initiator of articular cartilage damage. The present study has found anatomic and inferred biochemical changes concurrently in all of the joints materials: articular cartilage, calcified cartilage, and subchondral bone. We never observed subchondral sclerosis in the presence of completely intact cartilage.

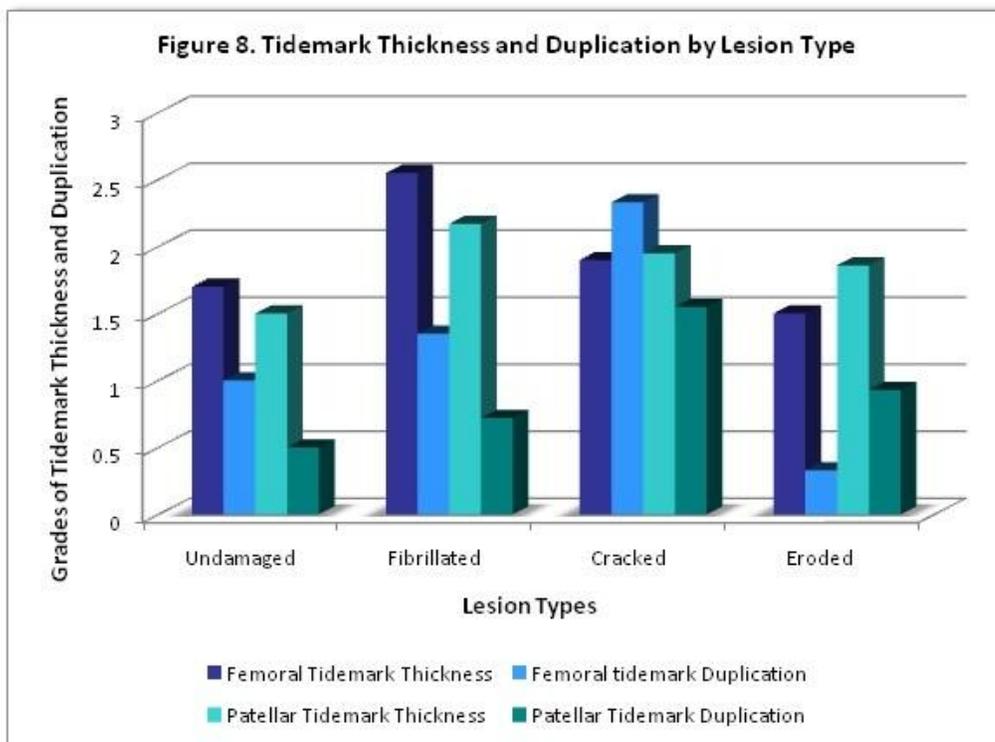


Figure 8: Tidemark thickness and duplication in the relatively undamaged, fibrillated, cracked and eroded patellae and patellofemora.

The microscopic features of Type I (fibrillated) joints: thick, rippled tidemark, moderately thickened calcified cartilage, large amounts of subchondral woven bone, and islands of calcified cartilage left in the bone and not modeled out, all suggest very rapid joint remodeling (See Figures 7

- 10). Additionally, the initial foci of fibrillation (in the patellofemoral intercondylar groove, and around the edges of the patella) tend to be areas of low load at most joint flexion angles (Goodfellow (15); Ahmed & Burke (16)). The spread of fibrillation across the surface of the joint (onto adjacent areas of the medial and lateral patellofemoral condyles, and on to the upper lateral and lower central facets of the patella) suggests that the normal sliding motion of the patella through the patellofemoral groove may be responsible for the spread of the lesion.

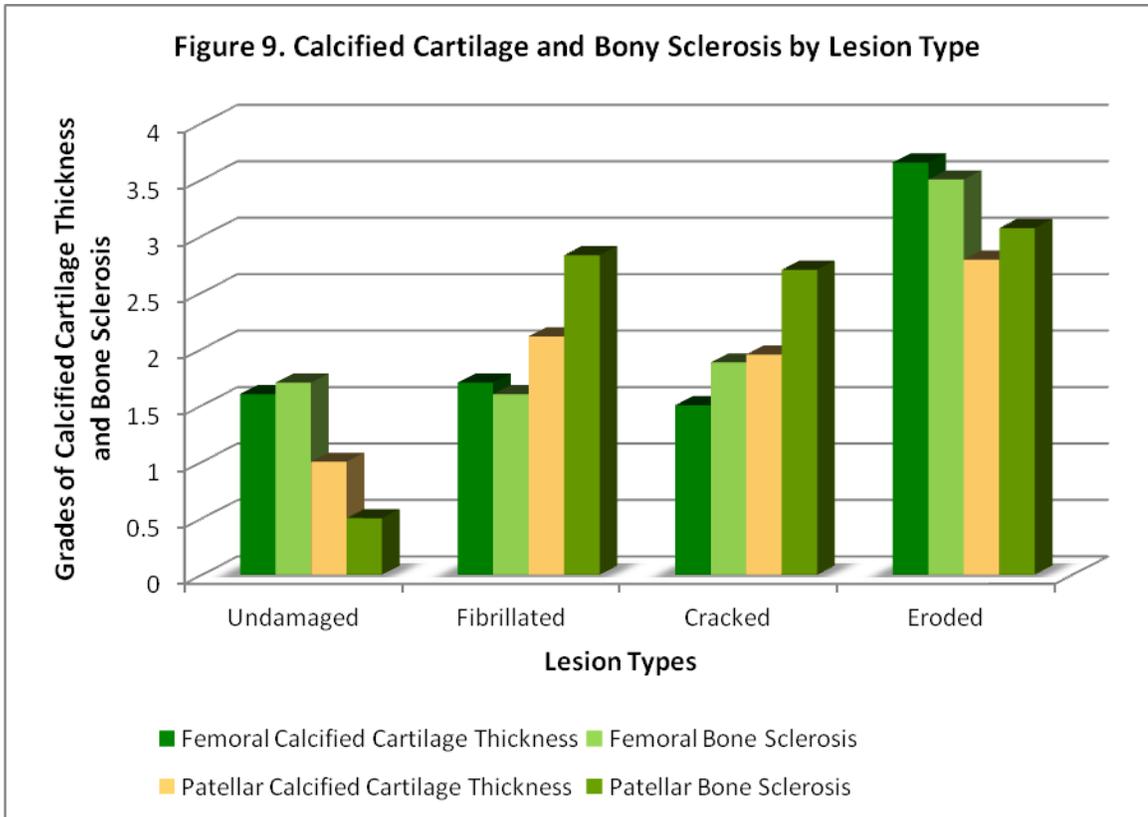


Figure 9: Calcified cartilage thickness and bony sclerosis in the relatively undamaged, fibrillated, cracked and eroded patellae and patellofemora.

On the other hand, the microscopic features of Type III lesioned (eroded) joints: thin, regular tidemark, thick calcified cartilage, and sclerotic bony arcade composed mostly of lamellar bone, all point to very slow joint remodeling. Because both the calcified cartilage and bone are thickened, it is likely that articular cartilage calcification is proceeding at the normal rate, but that the turnover of calcified cartilage into bone and/or the resorption of bone are slowed down. Further, the position of erosions on the patellofemoral joint are in those areas of habitual contact at high flexion angles (Goodfellow (15)), and tend to occur across the entire load gradient (Ahmed & Burke (16)), from low loads to very high compressive loads.

Differing dramatically from these two lesion types, are Type II lesioned (cracked) joints. The microscopic features of cracked joints: proteoglycan depletion, and horizontal lesions in the mid-zone cartilage, irregular flame-pattern tidemark, thin calcified cartilage, and only a small amount of sclerosis and woven bone in the bony arcade, suggest a primary disturbance in articular cartilage biochemistry, rather than a set of mechanical conditions for an etiology of cracking. The depleted proteoglycans in the articular cartilage outside of the clones in the surface lesion areas suggests that either the proteoglycans are lost through the surface, degraded by incoming enzymes, and/or that chondrocyte matrix production is very low. The initial foci and spread of cracking across the joint components is nearly identical to the origination and spread of erosions, especially in regards to patellar position. Also, like eroded patellofemora, cracked patellofemora are matched with patellae showing a mixed lesion of primary cracking and secondary fibrillation (Table 1).

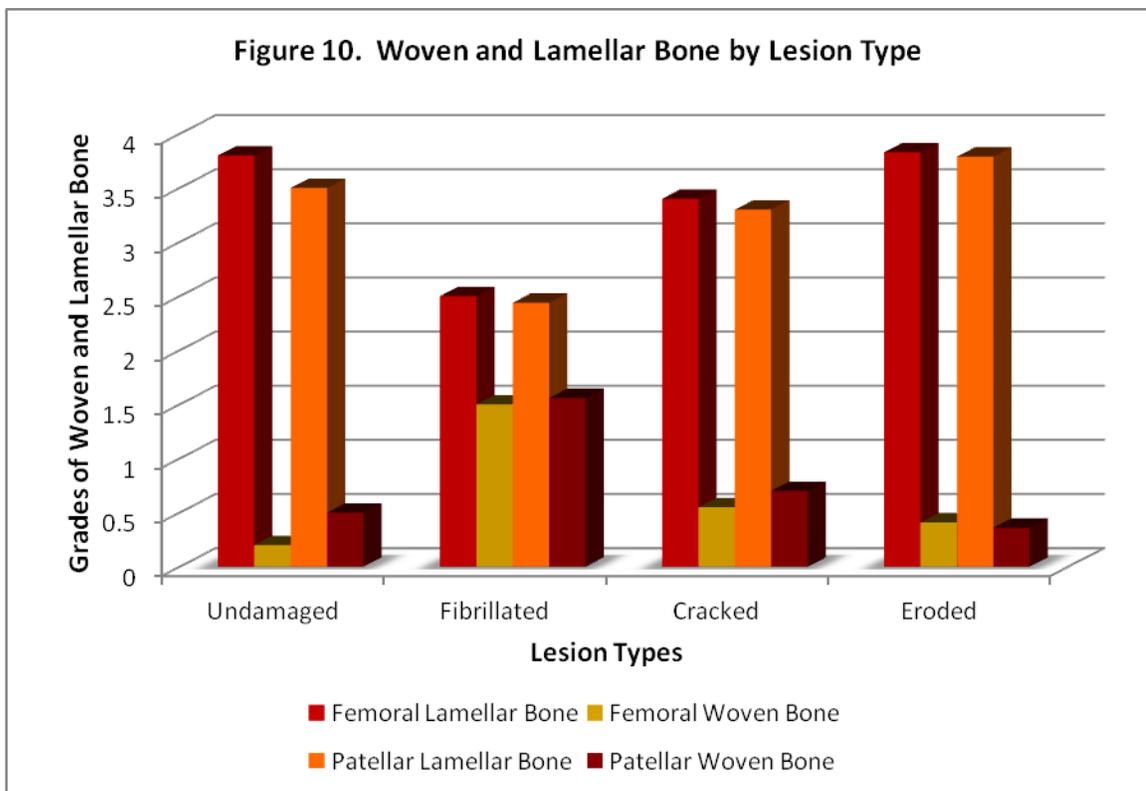


Figure 10: Lamellar and woven bone grades in the relatively undamaged, fibrillated, cracked and eroded patellae and patellofemora.

The presence of woven bone, to some degree, in the subchondral region and trabeculae of all of the lesioned specimens deserves special consideration, as it is not commonly associated with the lesions of advancing

age or osteoarthritis. Woven bone is laid down very quickly, its collagen is fine fibered, 0.1 μm or so in diameter, and it is oriented almost randomly, (Currey(17)). Also, the final degree of mineralization of woven bone is also much greater than that of lamellar bone (Boyde (18)). Woven bone is most notable in fibrillated joints, but is also present in cracked and eroded specimens and is probably due to an ongoing process of microfracture and repair of the subchondral bony end plate and trabeculae. If this highly mineralized woven bone is not quickly modeled out and replaced by lamellar bone, its presence would greatly increase the materials stiffness of the bony end plate, causing the likelihood of future fracturing to rise. Further, a stiff bony end plate would also promote further articular cartilage damage.

Conclusion

Finally, although we believe that we have identified three distinct degenerative changes in the patellofemoral joint, much work remains to be done. Larger sample sizes with more rigorous histochemical techniques are needed to confirm our inferences based on hemotoxylin and eosin staining. With cadaver specimens, we have developed a method of imaging the three lesion types in an undissected knee joint. Using this technique, it may be possible in the future to mount a prospective study of degenerative joint changes.

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