IS CHONDROMALACIA PATELLAE A SEPARATE CLINICAL ENTITY?

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The articular cartilage of the patella was studied in 100 knees at necropsy. In twenty-one of these knees the cartilage changes were related to the trabecular architecture of the underlying bone. It would appear that the initiation and location of cartilage damage and its rate and degree of progression are related to the relative local stiffness of the underlying cancellous bone. On the basis of our observations we suggest that the diagnosis “chondromalacia of the patella” should be reserved for patients with asymptomatic or transiently symptomatic fibrillation of the articular cartilage of the central medial patellar facet. Those patients with persistent patellofemoral pain should be considered to have some other syndrome.

In clinical terms most orthopaedic surgeons consider chondromalacia of the patella to be a syndrome characterised by retropatellar discomfort exacerbated by certain activities and which may be associated with patellofemoral crepitus. The cause of these symptoms is still uncertain, since conflicting conclusions have resulted from studies of symptomatic chondromalacia.

Wiles, Andrews and Devas (1965) reported that the earliest change was swelling and softening of the cartilage present in at least five out of six people by the age of thirty. The next stage of fissuring and flaking could be found in more than half the population at the same age. They also reported that it was difficult to estimate how often chondromalacia gave rise to symptoms.

Outerbridge (1961) described a medial femoral condylar ridge that could impinge on the medial patellar surface. This ridge has not been found to correlate with the presence of chondromalacia patellae (Meachim 1977).

Insall, Falwo and Wise (1976) reported that the cartilage damage started in the crest region and that malalignment of the patella was the usual cause.

Goodfellow, Hungerford and Woods (1976) have found a region of softening and eventual blistering of the cartilage of the medial facet. They associate this with the existence of an “odd”, or third, facet at the extreme medial border of the patella.

Darracott and Vernon-Roberts (1971) reported little or no obvious cartilage damage in early symptomatic chondromalacia. They did find a general decrease in subchondral bone density in patellae excised for patellar pain.

Stougård (1975) studied patellar bone changes in pathological chondromalacia. Although his results were not consistent, there appeared to be an eventual increased density of bone confined to the lateral facet of the patella.

In their study of Liverpool necropsy material, Meachim and Emery (1974) found that age-related changes in the cartilage started on the periphery of the patella and then appeared on the central medial facet; with increasing age these changes tended to spread to involve the remainder of the patellar surface. According to these authors, it was unusual for the cartilage changes on the medial facet to progress to an erosion of bone. The cartilage changes which had progressed to bony erosion first appeared on the central part of the lateral facet.

Byers, Contepomi and Farkas (1970) have pointed out that limited non-progressive cartilage damage usually occurs in those areas of the hip which are habitually non-loaded, whereas progressive cartilage changes start in the habitually loaded areas. A similar explanation for the characteristically limited chondromalacic changes in the central area of the medial facet of the patella is inconsistent with studies of the contact surface areas of the patellofemoral joint, which show the medial facet to be an habitual area of joint contact (Goodfellow, Hungerford and Woods 1976; Goodfellow, Hungerford and Zindel 1976; Townsend et al. 1976).

Previous work has shown that cartilage damage can be related to changes in the structural properties of the subchondral bone. It has been proposed that stiffening
(the resistance to deformation in compression) of the subchondral bone plays a central role in the development of progressive cartilage destruction ultimately leading to osteoarthritis. Although the exact sequence of these changes in cartilage and bone has not been definitely established, it appears that some relationship between them does exist. General or localised variations in subchondral bone stiffness may alter the stresses on the overlying cartilage. Considerable information on the structure and properties of the subchondral bone of the human patella exists in the literature. The three-dimensional trabecular architecture of the normal patella has been described (Raux et al. 1975), and the functional relationship between the trabecular architecture and the properties of cancellous bone has been reported (Townsend et al. 1975). Figure 1 is a contour map based on this earlier work which shows the local stiffness of the patella when viewed en face. Using this information, a systematic study was undertaken on a series of human patellae obtained at necropsy in which the architecture of the trabecular bone was related to the observed changes in the overlying articular cartilage.

Fig. 1
Isostiffness lines for the cancellous bone of the patella as viewed from the articular surface. All values \( \times 10^4 \) p.s.i. \( (6.9 \times 10^4 \) kPa).

Fig. 2
Grading of articular cartilage changes after staining with Indian ink. Figure 2—Shows normal (0); superficial streaking against pale grey background with new areas of earliest superficial fibrillation (1); and confluent staining where the fibrillation is still localised to the superficial zone (2). Figure 3—Shows fibrillation (3) extending to bone; and exposure of bare bone (4).

Fig. 3

Fig. 4
Chronological sequence of the location and degree of cartilage damage.
METHODS
One hundred knees were studied. These were obtained at necropsy from fifty-six Edinburgh patients in the age range of thirty to eighty-six years who had died from non-malignant conditions. The joints were opened through medial parapatellar incisions, and the patellar surface was viewed en face after staining with Pelikan Indian ink and washing with normal saline for a fixed period to delineate the cartilage changes as described by Goodfellow et al. (1976) and Meachim (1972). The surfaces were inspected through a Stereomaster microscope at × 2 magnification. The cartilage changes were graded using Meachim's modification of Collins's classification. Grade 0 was normal. Grade 1 was an early superficial fibrillation characterised by streaking of the Indian ink against a pale grey background. Grade 2 change represented a more diffuse form of superficial fibrillation characterised by coalescence of Indian ink on the surface of the cartilage. Grade 3 change was the presence of fibrillation within the cartilage which extended down to, but did not expose, the bone. Grade 4 changes were associated with exposure of bare bone (Figs. 2 and 3).

The presence or absence of an extreme medial ("odd") facet was noted. This was subjectively defined as a facet with its plane lying in a significantly different plane from the medial articular surface and separated from it by a definite ridge. The relationship between the presence or absence of such a facet and a synovial fringe overlying its medial aspect was also noted.

![Image](https://via.placeholder.com/150)

Fig. 5
Orientation of patellar bone sections.

![Image](https://via.placeholder.com/150)

Fig. 6
Grid positions for quantitation of bone across both surfaces of the three patellar slices.

Analysis of the cartilage changes in the 100 knees studied allowed a chronological assessment to be made of both the location and degree of cartilage damage (Fig. 4). A patella with Grade 0 changes on both the lateral and medial facets was regarded as normal. The earliest pattern of change was found to be Grade 1 medial cartilage change in association with Grade 0 change in the lateral facet. This was followed, with increasing age, by Grades 1 and 2 changes in the medial facet associated with Grade 1 change in the lateral facet. The next sequential changes were Grades 1 and 2 in the medial facet associated with Grades 2 and 3 in the lateral facet, followed by Grade 3 change in the central third of the whole width of each patella to include the medial, lateral and crest regions. The initial section was embedded in methacrylate and divided into three equal horizontal sections with the saw (Fig. 5). The bony surfaces were polished with a diamond lapping wheel and made parallel. Both surfaces of the resulting three sections were stained with a 10 per cent solution of silver nitrate to blacken the bone trabeculae and improve bone contrast, then photographed and developed on a 12 × 12 centimetres negative. The negatives corresponding to each surface of the three patellar slices were projected (at × 21 magnification) onto a grid made up of a random placement of dots. (It has been demonstrated that the frequency with which the dots overlap bony tissue is numerically equal to the fraction of the total volume occupied by the bone tissue as opposed to the marrow space—de Hoff and Rhines (1968). This is referred to as the "volume fraction" of the sample.) The whole width of the bone was quantitated (Fig. 6). Care was taken not to include the subchondral plate nor the dense anterior region of the patella where the effects of tendon attachment prevail.

RESULTS
Cartilage damage on both the medial and lateral facets increased with age, as shown in Figures 7 and 8. Isolated cartilage damage was never observed in the region of the crest. When such changes appeared they always occurred in association with more widespread cartilage changes. Early limited cartilage damage (Grades 1 and 2) was almost always observed along the periphery of the patella, but this damage was never more advanced than Collins's Grade 2 except when the whole patellar surface was affected by more severe changes. Advanced cartilage fibrillation (up to Grade 3) was seen in the central medial area but only rarely did these changes progress to expose bare bone (4 per cent). Severe (Grade 4) cartilage changes affected the central area of the lateral facet in twelve cases. In only four cases did Grade 4 lateral facet cartilage change become confluent with Grade 4 cartilage changes on the medial facet. When Grade 3 or Grade 4 cartilage changes were
observed on the lateral facet they were usually more advanced than those seen on the medial facet of the same patellae (70 per cent). The severe (Grade 4) cartilage changes, when they occurred, appeared to begin laterally and progress medially across the patellar surface (Fig. 4). The chronological relationship between location, degree of damage and age is seen in Figure 4. For example, when the medial facet tends to show early damage (Grade 1) the lateral facet remains normal (Grade 0).

The bone underlying the medial facet remains relatively less stiff than that of the rest of the contact area of the patella, even in the face of significant cartilage damage. Below the lateral facet there is an apparent initial decrease and then a statistically significant increase in stiffness beyond normal between cartilage Grades 1 and 2 on the medial facet plus cartilage Grade 1 on the lateral facet and the group showing Grade 3 cartilage on the medial facet plus Grade 4 on the lateral facet. The character of the bony changes under the crest fell

The volume fraction has been shown to be directly related to the stiffness of bone (Townsend et al. 1975). With the magnifications used, these dot-on-grid counts provide a sensitive assay of the stiffness of the trabecular bone. The relative stiffness of the underlying cancellous bone as a function of cartilage damage for both the medial and lateral facets is represented in Figure 9. Below the medial facet there is a statistically significant decrease in the stiffness associated with Grade 1 cartilage change combined with Grade 0 on the lateral facet, when this group is compared with the normal. This is followed by an increase in stiffness with Grade 1 change on the medial facet, associated with Grade 1 on the lateral facet. Then follows a levelling off in bone stiffness with no further increase in the remaining groups. roughly in between the results for the medial and lateral regions. The bone under the lateral facet was always more stiff than the bone of the remainder of the patella.

Table 1 lists the relative stiffness gradients as a function of gross grid position (Fig. 6). This information was obtained by subtracting the stiffness values at two grid points and dividing by the distance between these points. It is therefore a measure of the steepness of the stiffness contour.

Some degree of cartilage damage was found in 94 per cent of the patellae studied. A separate extreme medial ("odd") facet was observed in twenty-nine patellae. No relationship could be found between the damage which occurred on this odd facet and changes observed more generally on the medial and lateral facets.
of the same patella. No relationship was found between the existence of an odd facet and the presence of an overlying synovial fringe: in the twenty-nine patellae with an extreme medial facet twelve had an overlying fringe; the fringe was also seen in an additional eighteen patellae in which no extreme medial facet was present.

Table 1. Stiffness gradients of the subchondral bone

<table>
<thead>
<tr>
<th>Collins's grade of cartilage damage</th>
<th>Lateral facet</th>
<th>Medial facet</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Lateral</td>
<td>Medial</td>
</tr>
<tr>
<td>Medial 0 + lateral 0</td>
<td>0.414</td>
<td>0.203</td>
</tr>
<tr>
<td>Medial 1 + lateral 0</td>
<td>0.597</td>
<td>0.390</td>
</tr>
<tr>
<td>Medial 1-2 + lateral 1-2</td>
<td>1.143</td>
<td>0.061</td>
</tr>
<tr>
<td>Medial 3 + lateral 4</td>
<td>0.691</td>
<td>0.211</td>
</tr>
<tr>
<td>Medial 4 + lateral 4</td>
<td>0.643</td>
<td>0.217</td>
</tr>
</tbody>
</table>

DISCUSSION

There are changes in the stiffness of the cancellous bone with time which accompany the progression of the cartilage degeneration. No consistent association appears between a particular relative bone stiffness and the precise state of its overlying cartilage. It is interesting to note that there is a correlation between the gradients of bone stiffness in the horizontal plane and the location and progression of damage in articular cartilage. The results suggest that the cartilage degeneration on both medial and lateral facets is interrelated. Apart from the peripheral cartilage changes, the first observable cartilage degeneration occurs on the central medial facet area; this area of fibrillation is limited and only rarely progresses to bare bone.

There is a relative bony incongruity of the patellofemoral joint. In order to achieve the substantial contact area known to occur with flexion of the knee, the articular cartilage of the patella is subjected to considerable deformation. Because osteopenic bone is less stiff than normal bone it will press back against the cartilage with less than normal force. Thus when osteopenic bone is adjacent to normal bone, or when normal bone is adjacent to stiff bone, the difference in resistance to deformation will give rise to local shear strains within the cartilage (Fig. 10). These distortions are a likely basis for cartilage damage, and the observed stiffness gradients in the underlying bone are a potential basis for progressive destruction of cartilage. With time, the location and magnitude of these stiffness gradients may change. Observation of relative osteopenia under the medial facet of the patella is consistent with the observations of Stougård (1975). The rate of progress of the cartilage changes on this facet may also be limited by the underlying relative osteopenia.

If our hypothesis on the role of stiffness gradients in cartilage damage is correct, then one would expect the
first cartilage damage to occur where the stiffness gradient is greatest. Extrapolating from Table I, the predicted patterns of cartilage damage are consistent with our necropsy observations (Fig. 4).

A precipitous decrease in stiffness would cause a corresponding increase in the strain on the bone, assuming the loads on the joint remain the same. Resulting overstrain of trabecular bone may be one cause of retropatellar pain (Outerbridge 1961; Townsend et al. 1977).

It is universally agreed that the majority of young patients with pain in the patellofemoral joint have symptoms of a transient nature which respond to conservative measures or which settle spontaneously. Two of us (P. J. Abernethy and E. L. Radin) examined the knees of first-year medical students under the age of twenty-five years. We found asymptomatic patellofemoral crepitus present in 62 per cent of 123 students. Only 29 per cent of the total admitted to previous transient discomfort, and 3 per cent had chronic patellofemoral pain.

The remarkably high necropsy incidence (85 per cent) of cartilage fibrillation of the central medial facet of the patella and the low incidence of chronically symptomatic patellofemoral joints in the general population suggest that the surgical finding of softening of the articular cartilage of the medial facet should be considered an incidental finding. We believe that persistent pain and tenderness in the patellofemoral joint would be better regarded not as “chondromalacia” but as some other disorder such as an odd facet lesion, an osteochondral injury resulting from trauma, a patella malalignment syndrome, synovial impingement or an arthritic disorder. We anticipate that the confusion presently surrounding the clinical entity of chondromalacia of the patella could be reduced by considering the medial facet changes as an asymptomatic, age-related physiological change which does not require surgical intervention. In our opinion, patients with persistently painful, tender patellofemoral joints should be specifically investigated for alternative causes of pain, and these causes should be specifically treated.

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REFERENCES


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