ARTICULAR CARTILAGE LOSS IN LONG-STANDING IMMobilisation of INTERPHALANGEAL JOINTS

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ARTICULAR cartilage undergoes changes in joints which are immobilised. These cartilage changes are seen in human hands where joint immobility has been restricted by some disease outside the joint, such as ligamentous contraction, burns scarring, tendon shortening or Dupuytren's contracture. The end-result of these changes was observed clinically by Hueston (1963) in cases of long-standing interphalangeal flexion in Dupuytren's contracture, where eventual loss of cartilage from the unused parts of the joint surface was an important factor limiting return of joint function, after fasciectomy. The experimental observation by Hall (1964) that the unused articular cartilage in the immobilised knee joints of rats gradually loses its structure confirms this clinical finding. It is very likely that similar degenerative changes occur in the hyaline cartilage whenever a joint has limited or abnormal range of movement.

Observations.—An opportunity to study this phenomenon arose when two patients had joints excised whole in the operative correction of hand deformities. One man with Dupuytren's disease had nine months' severe flexion contracture of the proximal interphalangeal joint (Fig. 1) requiring amputation of the little finger.

A mentally retarded patient had disuse contractures of all her proximal interphalangeal joints for 15 years (Fig. 2), and these joints were excised for arthrodesis. In each case the cartilage had undergone changes in those areas where, because of the extra-articular restriction of joint movement, there was no contact with the cartilage of the opposing phalanx. However the cartilage remained normal in those parts of each joint where there was contact between opposing cartilage surfaces.

The changes observed macroscopically in the unused areas were a loss of sheen, and irregularity of the cartilage surface which became pitted and eroded towards its margin (Fig. 3). In the longer standing case these changes were extensive, cartilage was lost completely from the unused area on the dorsum of the proximal phalanx, and the overlying extensor tendon was adherent to the bone (Fig. 4). The tendon could be stripped back to expose the pitted bone surface devoid of cartilage, and this area corresponded to the bone loss demonstrated radiologically (Fig. 5).

Microscopically the changes included surface irregularity of the disused cartilage, fissuring of the superficial zone and an altered staining of the chondrocytes there. The cartilage was thin, there was an overgrowth of small vessels across the surface (Figs. 6 and 7), and vascular connective tissue extended into surface defects in the cartilage matrix (Fig. 8). At the margin of the zone of altered cartilage there was increased cellularity of the periosteum and a vascular proliferation appeared to invade beneath the overhanging edge of cartilage, and to erode the subchondral bone and extend between its trabeculae (Fig. 9). In the long-standing case the tendon lay over the eroded cartilage and bone was adherent to it by an abnormal granulation tissue.

The protuberance of cartilage-capped stalks beside the eroded area, appearing superficially to resemble osteophytes, was found in fact to represent the only remaining sites of the original uneroded articular surface.
Fig. 1.—Moderate flexion deformity of little finger in Dupuytren's contracture of less than two years' duration—articular cartilage changes were found in the proximal interphalangeal joint.

Fig. 2.—Severe flexion contractures of all proximal interphalangeal joints in psychotic patient, present for 15 years—extensive articular cartilage loss was found in the proximal interphalangeal joints.

Fig. 3
Articular surface of base of middle phalanx shows (A) normal cartilage, (B) irregular surface on the unused cartilage. ×4.

Fig. 4
Head of proximal phalanx has normal volar cartilage (A) but cartilage and bone loss are seen where the extensor tendon and dorsal capsule (B) have been stripped back off the disused dorsal half of phalangeal head.
FIG. 5
Radiography of proximal phalangeal heads shows loss of bony contour dorsally.

FIG. 6
A layer of capillaries extends across the unused part of the joint surface and is covered by a strand of altered ground substance. H. & E. ×40.
FIG. 7
Capillaries in a collagen layer lie on the surface of the non-articulating cartilage matrix. ×160.

FIG. 8
Chondrocytes near the surface are oedematous, and the irregular defects in the unused cartilage are filled with connective tissue and vessels. ×120.
These changes in the articular cartilage resemble those seen by Hall (1964) in animal experiments in which he immobilised the knee joints of adult rats to study the response of cartilage to continuous compression. In these immobilised joints he also observed changes in the areas of cartilage not in contact with opposing cartilage.

After only 23 days of immobilisation changes appeared and progressed over the following months in a pattern which included the various features observed here in human cartilage.

The mechanical limitation of joint movement may bring about these changes by interfering with the cartilage nutrition. Ekholm (1955) demonstrated that hyaline articular cartilage gains its nutrition by diffusion both from the synovial fluid and from vessels of the underlying bone, and the diffusion of the nutrients through the mucopolysaccharide matrix is assisted by intermittent compression of the cartilage as the joint moves. Here the restriction of movement may impair the nutrition of the non-compressed cartilage which then undergoes atrophy. There may then be a metaplasia of hyaline cartilage in response to its altered function, no longer in gliding contact with a compressing cartilage surface, but lying beneath a synovial membrane or stiff joint capsule.

The clinical significance of these observations lies in their application to other joint situations. Prolonged immobilisation of a joint will allow this type of change to occur in the idle cartilage, and produce intra-articular problems during remobilisation. Operative procedures such as arthroplasty and osteotomy may bring back into use areas of cartilage which have undergone change. Further pain and disability can then result from attempts to use areas of articular surface repaired by fibrous tissue, fibro-cartilage or showing osteophyte formation. If cartilage loss has occurred and adhesions must be broken to mobilise the joint, hæmorrhrosis can be expected.
It would be interesting to know how reversible the changes are, once started, and to what extent hyaline cartilage can be restored by exercise. Certainly Saaf (1950) showed that exercise at first increases the number of cells present in the transitional zone of cartilage, and that the effect is less with prolonged exercise. But the slow regeneration to cope with normal wear and tear seems to be the limit of repair by actual growth of hyaline cartilage.

**SUMMARY**

Articular cartilage changes are observed in stiff joints where parts of a joint surface are not in contact with opposing cartilage. The unused cartilage surface becomes irregular, fissured and covered by a capillary network. Later defects in the matrix become filled by connective tissue and a vascular proliferation invades the cartilage margin and erodes subchondral bone, to which the overlying joint capsule becomes adherent. The changes resemble those in experimental animals, and their pathology and clinical significance are discussed.

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**REFERENCES**


