Osteoarthritis today, and probably in past centuries also, is the most common type of arthritis. Since the turn of the twentieth century and with the advent of x-rays, the "osteoarthritis group of diseases" has been separated more clearly from other types. Although some accurate descriptions of osteoarthritis had been recorded previously, much overlap and confusion existed with many diseases which we now separate from each other. For instance, in 1859 Adams separated these two types of arthritis, which together were called "rheumatic gout," from true gouty arthritis by means of the uric acid test. In the early 1900s, with the aid of x-rays, it was possible to show that the bone changes of osteoarthritis were hypertrophic and contrasted sharply with those of rheumatoid arthritis, where the bone was atrophic or eroded.

The term osteoarthritis has numerous synonyms, but at present no one has a great advantage over another. These terms include degenerative joint disease, hypertrophic arthritis, traumatic arthritis, osteoarthritis deformans, gonarthrose, and even the more recent osteoarthrosis, all of which refer to similar clinical situations. Even with the refined techniques of present day x-ray and biochemical analysis, there is still debate about the best sub-classification of this group of diseases, for numerous factors play a role in the development, if not the etiology, of the disease (see Table I). When one considers the number of joints which could be involved in different types of motion and trauma and then considers the duration of this activity, the age, the sex, the genetic influence, the metabolic disturbances, etc, the complex etiology of osteoarthritis is evident. Two major clinical classifications exist: 1) primary osteoarthritis, which develops spontaneously, and 2) secondary osteoarthritis, in which trauma or some predisposing factor can be clearly identified.

<table>
<thead>
<tr>
<th>FACTORS INFLUENCING DEVELOPMENT OF OSTEARTHRITIS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Primary</strong></td>
</tr>
<tr>
<td>Age</td>
</tr>
<tr>
<td>Sex</td>
</tr>
<tr>
<td>Ethnic Group</td>
</tr>
<tr>
<td>Metabolic Disturbances</td>
</tr>
<tr>
<td>Diabetes</td>
</tr>
<tr>
<td>Obesity</td>
</tr>
<tr>
<td>Stress Trauma</td>
</tr>
</tbody>
</table>

In a field survey in Leigh, England, Kellgren, using x-ray evidence and clinical history, found that 20% of the population over 55 years of age had "osteoarthritis."

Danielsson, reviewing 3,903 radiographs taken for bowel studies, noted a 3.9% incidence of x-ray changes classified as osteoarthritis of the hip. In the white population in North America, there is a higher frequency of osteoarthritis in men when the large joints are considered. However, for small joints of the hands, particularly the distal phalangeal joints (Heberden nodes), a dominant hereditary pattern is evident which shows a female involvement of some 30%, as compared with a 3% frequency for men. In the Blackfoot Indians, it has been reported that women are more frequently afflicted with osteoarthritis than men, whereas in the Pima Indians, the reverse tendency occurs, men being more frequently affected.

**Primary Osteoarthritis**

Recent experimental studies have used a specific strain of mice which are predisposed to osteoarthritis to evaluate the influence of weight, aging, and other factors upon the development of the disease. Mechanical stress has probably been the most important single factor. Epidemiological studies confirm what would be obvious to most observers,
that osteoarthritis in the hands and wrists develops in a population whose working pattern requires repetitious, strenuous activity with strains in the forearms and fingers. Again, experimental studies using repetitious impulse loading can produce these symptoms, but exactly why these reactions occur is difficult to elucidate. If the whole process were simply a degeneration of the joint or cartilage due to use, it would be reasonable to assume that the weight-bearing joints would always undergo symmetrical changes and that the cartilage surfaces in contact with one another would be the primary sources of degeneration. However, neither of these observations is always correct.

To date, we have not identified the pathophysiology of osteoarthritis. Is it a disease of cartilage, of bone, of synovium, of all, or of none of these structures? Let us look at some of the facts which have emerged recently in numerous studies of osteoarthritis to explain some of the variations referred to previously. While osteoarthritis occurs more often in older age groups, many young persons may also be involved. Specifically, a rapid wearing of cartilage is often seen in young athletes between 10 and 30 years of age, occurring behind the patella. Recognized as chondromalacia, this condition is similar to that found in the wearing of the cartilage of typically osteoarthritic joints. In this process, there is initial discoloration, turning from white to pale yellow, after which the cartilage becomes soft and ultimately may erode and be worn down to underlying bone.

Normal articular cartilage is of varying thickness with a unique structure (Figure 1). It consists of a framework of collagen fibers anchored at one end to the bone and rising perpendicularly for some distance, then arching so that the arch becomes tangential to the cartilage surface. Between these collagen fibers is a matrix arranged around the chondrocytes which seem to nourish and control repair in the adjacent region. The matrix consists of chains of hyaluronic acid and various proteoglycans which have water molecules and other nutrients derived from the joint fluid interspersed between them. This interstitial matrix has recently been shown to be very active metabolically and to undergo constant renewal. In adverse circumstances, the biochemical replacement of these proteoglycans may become impaired, so that the cartilage becomes weaker and the repair process lags behind the damage produced by normal use of the joint. Inevitably, microscopic changes develop. Splitting and fibrillation of the cartilage occur. Enzymes, synovial fluid, cells, and antibodies intrude into the depths of the cartilage which is not normally exposed to adverse inflammatory reactions, and the chondrocytes themselves undergo changes. These circumstances are far more common in metabolic disturbances such as diabetes. In these early changes of osteoarthritis, there may be no specific x-ray evidence beyond narrowing of the joint space. However, we appreciate that in the paleopathological field no evidence for this type of reaction would remain in most specimens.

Cartilage may be involved not only on its free surface, but in many instances the bone cells at the osteochondral junction may promote calcification in the depths of the cartilage. The bone appears to advance into the cartilage, thus reducing its thickness and effectiveness as a "shock absorber." Much of the shock absorber effect in a joint is actually related to the lattice work of bony trabeculae beneath the cartilage surface and its junction with the bone. Radin, expanding on Frost's concepts of microfractures, suggests that this mechanism causes the sclerosis and hardening of bone seen around the joints in the long-standing osteoarthritic patient. Trauma, such as sudden jolts from missing a small step of several inches, and comparable repetitious experiences, may crack one or two trabeculae which at this site produces callus and accumulates more new bone. This region then becomes less flexible, more firm and solid ("sclerosed" on the x-ray), and more rapid cartilage wear results. The effect of subsequent stresses on the cartilage can be compared to the difference between bouncing on a bed mattress supported only by a bedboard and jumping on one with springs under the mattress.

In certain circumstances and some areas, bone tends to invade the cartilage from the lateral aspect and ultimately produces osteophytes, which occur as a ridge in and around ligamentous attachments at the periphery. This confirms Danielsson's observation that osteophytes may develop in
Osteoarthritis

the absence of identifiable joint space narrowing. In a study of symptomatic patients whose clinical diagnosis was “hip disease,” he found that 86 of 214 subjects had osteophytes present but no narrowing of the joint space. More interestingly, when re-evaluated ten years later, only one patient in this group of 86 had a further narrowing of the joint space. The important point in this observation was that although osteophytes were present, evidence for actual osteoarthritis of the hip joint itself was not.

In the spine, spondylosis or Forestier’s type of hyperostosis (Figure 2) is often confused with ankylosing spondylitis and also with osteoarthritis, for they all impair the motion of the spine and produce various types of osteophytes. Yet osteoarthritis and osteophytosis frequently do occur together. Forestier’s “ankylosing hyperostosis of the spine” is usually entirely asymptomatic, as are many osteophytes in other areas.

Secondary Osteoarthritis

The causes of secondary osteoarthritis are quite numerous. Any joint disease (gout, septic arthritis, rheumatoid arthritis, etc) by reason of the damage to the joint surface may be predisposed to accelerated degeneration and subsequent development of some feature of osteoarthritis. Also, any previous fracture through a joint promotes more rapid degeneration of that joint. Definition of the primary etiology, such as infection, rheumatoid arthritis, or gout, by examining an osteoarthritic joint may be quite difficult, but it may be inferred by more specific changes in other parts of the body. On many occasions, tuberculous joint disease was associated with the same affliction of the lung. Infections of joints do not have specific bony changes which can distinguish one type from another. A single joint affliction may be of varying degree, may have healed, and the degenerative processes of osteoarthritis superimposed on the already damaged joint. This damage will so increase with use and age that the original infectious etiology could easily be disguised.

Because many factors influence the development of osteoarthritis, it may be impossible when examining the damaged bone to determine which predisposing factor was primary. Although osteophyte production in many instances parallels the degree of osteoarthritis, the size, number and distribution of osteophytes should not be used as major criteria for deciding on the degree, type and magnitude of osteoarthritis.

Fig 2. Ankylosing Hyperostosis (Forestier’s Syndrome)

Note the marked bony proliferation and its asymmetrical distribution in lumbar region. Osteoarthritis is seen at the hip joints with narrowed joint space and slight osteophytosis.
References


