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Chronic Repetitive Trauma: A Cause of Atypical Degenerative Joint Disease

David B. Hellmann, M.D.[1], Clyde A. Helms, M.D.[2], and Harry K. Genant, M.D.[1, 2]

[1] Department of Medicine, University of California, San Francisco, California
[2] Department of Radiology, University of California, San Francisco, California, USA

Abstract. Six cases of amateur athletes who have severe atypical degenerative joint disease (DJD) are presented; their histories suggest that chronic, repetitive trauma was causative in the development of their arthropathy. Although many examples of this process have been reported in professional athletes, it has not been reported in amateurs. As participation in athletic activities increases we can, perhaps, expect to see more of this type of DJD in the future.

Key words: Degenerative joint disease – Trauma – Sports injuries

Osteoarthritis (OA) is the most common rheumatic disease. It is characterized pathologically by focal ulceration of articular cartilage and marginal new bone formation, and clinically by joint pain and stiffness. Disagreements over the pathogenesis of OA have generated a confusing terminology and classification. In this report, osteoarthritis and degenerative joint disease (DJD) are used interchangeably. OA is classified as primary or secondary depending on whether or not a predisposing factor is absent or present, respectively.

It now appears that many different types of insults can precipitate OA [3]. Studies on professional athletes and heavy manual laborers suggest that very strenuous physical activities may lead to DJD [2, 4–7, 10]. The role that less vigorous but more commonly performed activities play in the development of OA has not been well studied. We have recently seen six patients whose OA seems to have resulted from their participation in certain hobbies and amateur sports. Increasing leisure time and interest in physical fitness ensure that the number of participants in amateur sports and physically demanding hobbies will rise. Our case reports emphasize that such common activities may predispose patients to the development of OA.

Address reprint requests to: Harry K. Genant, M.D., Department of Radiology, C-309, University of California, San Francisco, CA 94143, USA

Case Reports

Case 1

P.S. is a right-handed 38-year-old male who presented with diffuse right shoulder pain. He had been lifting weights regularly for several years when in December 1978 he gradually noted diffuse right shoulder pain that was aggravated by exercise, especially bench or military presses. He had no other symptoms and had previously been well. The shoulder was not swollen but was diffusely tender. Abduction was moderately limited by pain.

X-rays of the right shoulder showed glenohumeral joint space narrowing, a large cyst in the glenoid, and a loose body (Fig. 1A). Films of the left shoulder showed spur formation at the humeral head and subchondral sclerosis of the glenoid (Fig. 1B). A computed tomography (CT) scan of the right shoulder revealed that the cyst was subchondral (Fig. 1C) and the left shoulder CT scan demonstrated glenoid spurs and humeral head sclerosis which were not seen on the routine films (Fig. 1D).

Comment. Shoulders are rarely involved in primary OA, making it likely that weight lifting played an important role in the development of this patient's OA. The CT scans disclosed findings not present on the routine radiographs.

Case 2

R.A. is a 67-year-old male who presented because of left shoulder pain. He had been a life-long polo player. Eighteen months before entering the clinic, he had diffuse left shoulder pain that was especially severe at night. His only other symptomatic joint was the first right carpometacarpal joint which had ached intermittently for eight years. Physical examination

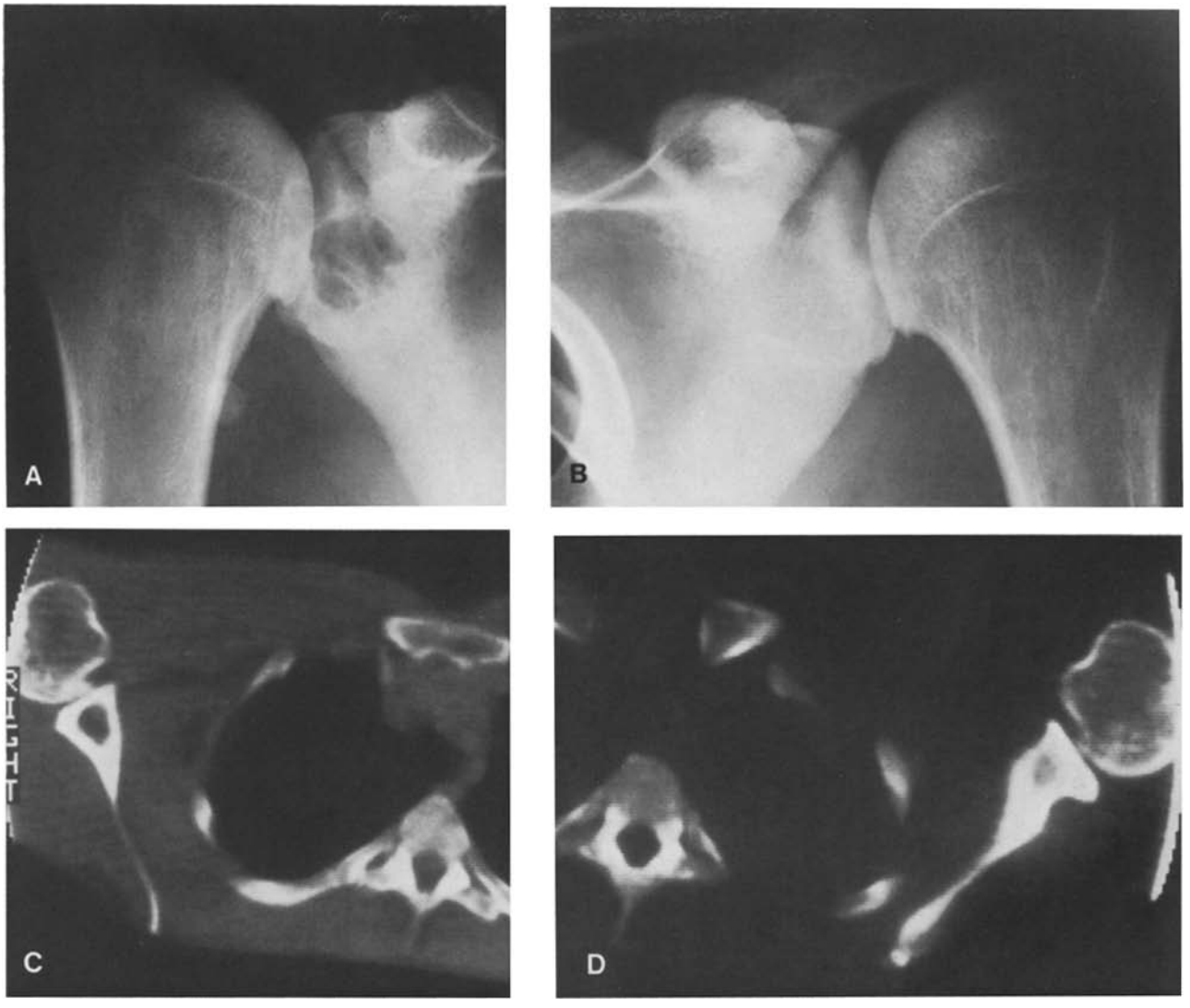

Fig. 1A–D (*Case 1*). **A** Anteroposterior view (AP) of the right shoulder; glenohumeral joint space narrowing is present with an osteophyte noted on the humerus. A large subchondral cyst is present in the glenoid. A loose body is also noted. **B** AP view of left shoulder; an osteophyte is noted on the humeral head adjacent to the inferior glenoid. Subchondral sclerosis is present throughout the glenoid. These findings are consistent with DJD. **C** CT scan through the right shoulder: a large subchondral cyst seen on the plain film in the glenoid is demonstrated. An osteophyte and subchondral sclerosis are seen in the humerus. **D** CT scan of the left shoulder: subchondral sclerosis and spurring are seen on the glenoid. Minimal subchondral sclerosis and spurring are also seen in the humeral head

showed that the right shoulder range of motion was descreased moderately in all planes. The right first carpometacarpal area was slightly tender.

The ESR, CBC, and SMA-12 were within normal limits. Radiographs showed marked degenerative changes involving the radiocarpal joints bilaterally, all the right carpal bones (Fig. 2A), and the shoulders bilaterally (Figs. 2B and 2C). Advanced degenerative changes at the first MTP of the feet with complete loss of cartilage and destruction and eburnation of subarticular bone was present (Fig. 2D).

Comment. Primary degenerative arthritis usually spares the wrists and shoulders. In polo players these joints are subject to repetitive impacts which probably accelerate the development of OA. Additionally, advanced degenerative arthrosis of the first MTP joints is uncommon in the absence of hallux valgus deformity. One can speculate that repetitive trauma and pressure from the stirrups might predispose to accelerated OA at this site.

Case 3

J.T. is a right-handed 45-year-old male who was admitted in May, 1980 for arthroplasty of the right elbow. He had been an enthusiastic handball player for 15 years. Since 1976, he had increasing pain and stiffness in the right elbow, not responsive to physical therapy or intra-articular steroids. An arthrogram in 1976 was reported to be normal. He had no other symptoms. Physical findings were limited to the right elbow. There was no tenderness or swelling. Flexion and extension were limited slightly. Pronation and supination were normal.

The CBC, ESR, SMA-12, and chest X-ray were normal.

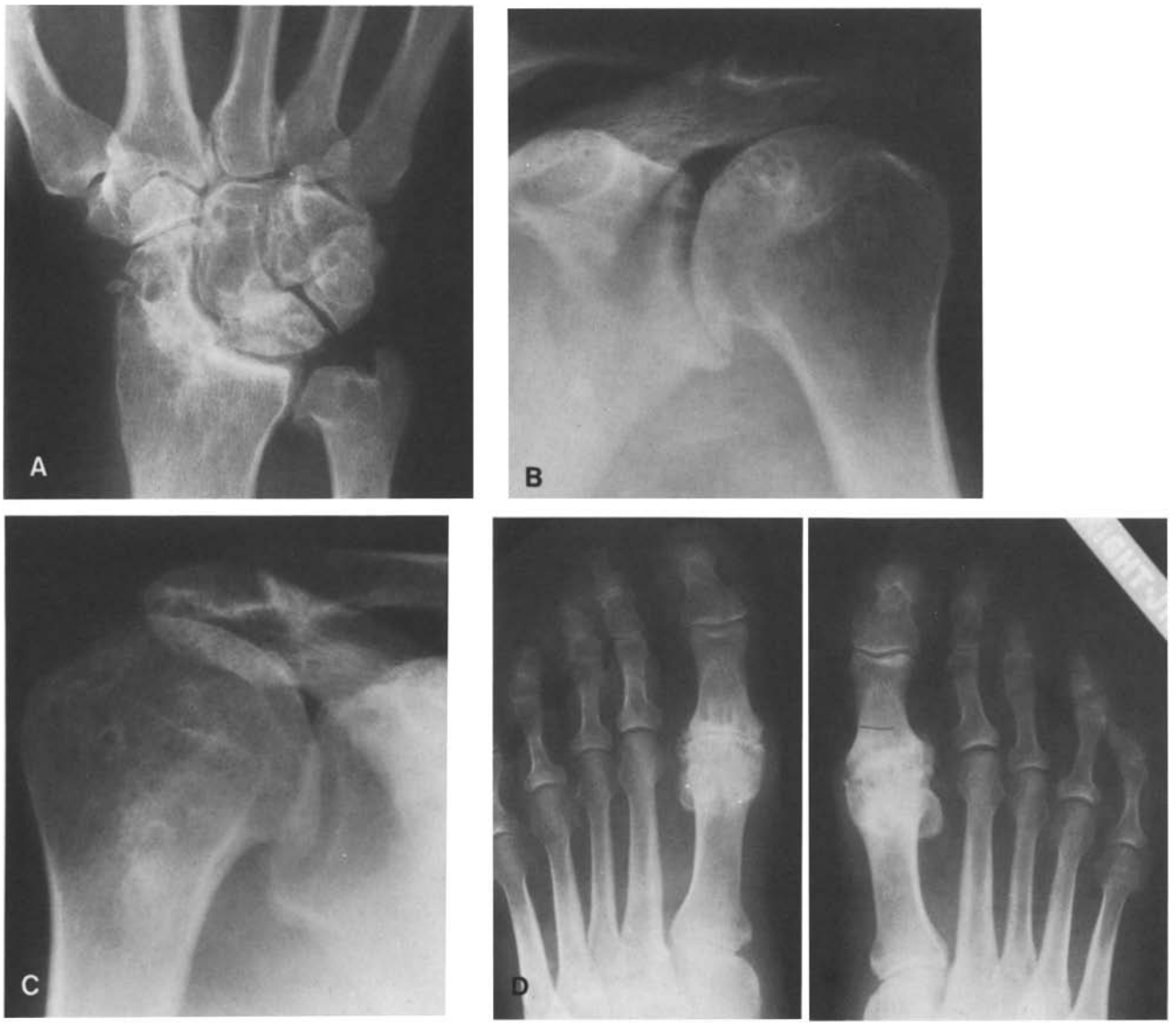

Fig. 2A–D. (*Case 2*). **A** AP view of right wrist: extreme radiocarpal joint space narrowing with similar joint space narrowing and subchondral sclerosis is seen throughout the wrist. This is typical of advanced degenerative disease. **B** AP view of left shoulder: marked glenohumeral joint space narrowing is present with subchondral sclerosis and geode formation indicating advanced degenerative disease. **C** AP view of right shoulder: osteophytes are present on the glenoid and humeral head with marked narrowing of the space between the humeral head and the acromion indicating probable atrophy of the rotator cuff. These findings are indicative of advanced degenerative disease. **D** AP view of both fett: marked joint space narrowing with subchondral sclerosis and osteophytosis is noted at both first MTP joints. No other significant abnormalities are noted in the feet. These findings are typical of advanced degenerative disease

Radiographs of the right elbow demonstrated irregularity and sclerosis of the humero-ulnar joint surface. Osteophytes and loose bodies were also evident (Figs. 3A and 3B).

Comment. This otherwise healthy man developed disabling degenerative changes in the right elbow after 15 years of playing handball. Like the shoulder and wrist, the elbow is an unusual site for primary osteoarthritis.

Case 4

J.O. is a 36-year-old male with right hip pain. He was an outdoor enthusiast who had done extensive mountain climbing, back packing, bicycling, and jogging. In 1975, he developed right hip pain. He discontinued most of this activities, but the pain gradually progressed, such that by April, 1980, he had disabling pain after walking a half mile. He had had no joint symptoms during childhood and there was no history of major hip trauma. Outside of his skeletal complaints, he was well. Examination in April, 1980 showed moderate decreased range of motion in the right hip in all planes except for extension and adduction. By January, 1981, there was a positive Trendelenburg sign of the right. The right leg was one-quarter inch shorter than the left.

Hip films from 1976 showed minimal acetubular dysplasia, eburnation, and marginal osteophytosis on the right (Fig. 4A). Repeat films in 1980 showed a large right subchondral cyst

with a thin sclerotic border (Fig. 4B). The acetabulum and femoral head joint surface were sclerotic and the femoral head was subluxed superolaterally to the acetabulum.

Comment. The hip is a common site of osteoarthritis. Alterations in joint architecture may prediscope to development of OA as they have been found in 65% of patients with hip OA [8]. Still symptomatic hip OA is unusual before age 50 years. The 1980 films show a shallow right acetabulum, suggesting this patient had a mild form of congenital hip dysplasia. The occurrence of severe OA by age 30 years and the rapid progression of destructive changes implicate chronic trauma in the pathogenesis of his hip OA.

Case 5

S.G. is a 48-year-old female who presented with a painful, swollen right knee. Since adolescence she had been an avid tennis player, playing up to six hours per day. The knee first became painful and swollen when she was 26 years old. Over the next seven years, she had intermittent pain, swelling, and redness of the right knee and the right third DIP joint, which were treated off and on with salicylates, oral and intra-articular steroids. At age 33 years, the symptoms abated and all medical therapy was stopped. She continued to play tennis and was free of symptoms until age 48 years when swelling of the right knee and the right second and third DIPs recurred. She denied having psoriasis, morning stiffness, or gastrointestinal complaints.

The general physical examination was negative except for the joints. The second and third right DIPs showed synovial thickening. The right knee had a small effusion but heat, redness, and tenderness were absent. The range of motion was normal although crepitus was present. No instability of the joint was appreciated. A firm, mobile nodule was overlying the lateral tibial plateau.

The CBC, SMA-12, and ESR were normal. The RF and ANA were negative. X-rays of the right knee showed marked lateral compartment and patellofemoral joint space narrowing with sclerosis and spur formation. Views of the right hand (dominant) showed marked peri-articular soft-tissue swelling accompanied by advanced cartilage and bone destruction at the second and third DIPs (Fig. 5C).

Comment. This avid tennis player developed degenerative changes in her right knee. She also had an undifferentiated arthritis. Probably both the tennis playing and the inflammatory arthritis influenced the development of the degenerative knee changes. As in the previous case, the degenerative changes are somewhat atypical with marked bony destruction and massive secondary osteochondromatosis.

Case 6

R.B. was 20 years old in 1974 when he presented with right ankle pain. For several years, he had participated in cross-country motorcycling during which he experienced sharp pains in his right hind foot. At that time, the physical examination showed the right ankle to be stable and nontender. There was slight fullness in the region of the anterior talofibular ligament. The range of motion was full. X-rays of the right ankle in 1974 were normal. Because of persistent symptoms, the X-rays were repeated in 1979 and were again normal (Fig. 6A). However, tomograms and CT scans of the ankles showed degenerative arthrosis of the posterior facet of the subtalar joint with subchondral sclerosis, marginal osteophytosis, and pseudocyst formation (Figs. 6B and 6C).

Comment. It is likely that the repetitive pounding absorbed by the ankles in off-road motorcycle racing resulted in an accelerated degenerative arthrosis, possibly related to repetitive impaction injuries of the subtalar joint.

Discussion

Our patients suggest that chronic, repetitive trauma from participation in hobbies and nonprofessional sports can promote the development of OA. Participation in these activities and development of OA are both common events and, therefore, chance alone could theoretically dictate their occuring together. However, several lines of evidence suggest that our patients' activities played some role in the development of their OA. First of all, it is unlikely that these patients simply had idiopathic or primary DJD. Most of the patients reported here are young (four are less than 40 years old). Though radiographic evidence of OA has been found in 10% of persons between the ages of 15–24 years, clinically severe OA such as that found in our patients rarely occurs in young patients [6]. Also primary OA usually does not involve shoulders, elbows, wrists, or ankles – the sites of degenerative changes in 66% of our patients. Indeed, the one patient over age 60 years had OA of the wrists and shoulders. Thus, the age of our patients and the distribution of joint involvement make it unlikely that they had primary OA.

Second, of the many factors which have been causally related to OA, repetitive trauma appears to be the most prominent one in our patients. In four of the patients, repetitive trauma is the only contributing factor, as other conditions associated with the development of OA (e.g., CPPD, hemophilia, hemochromatosis) were not present. In the other two patients, trauma was important but probably not the sole influence on the development of OA. The mountain climber for instance, may have had a very mild form of congenital hip dysplasia. Studies have shown that 65% of patients with hip OA have hip architectural abnormalities such as hip dysplasia or pelvic tilt, but do not usually have symptoms before age 50 years [8]. That the patient in this study had hip OA by age 30 years and that the degenerative changes progressed so rapidly, strongly suggest that trauma contributed to the osteoarthritic process. Likewise, the tennis player may have had an underlying inflammatory arthritis, but it would be unusual for inflammation alone to produce such extensive degenerative changes, indicating that both inflammation and trauma contributed to the final X-ray appearance.

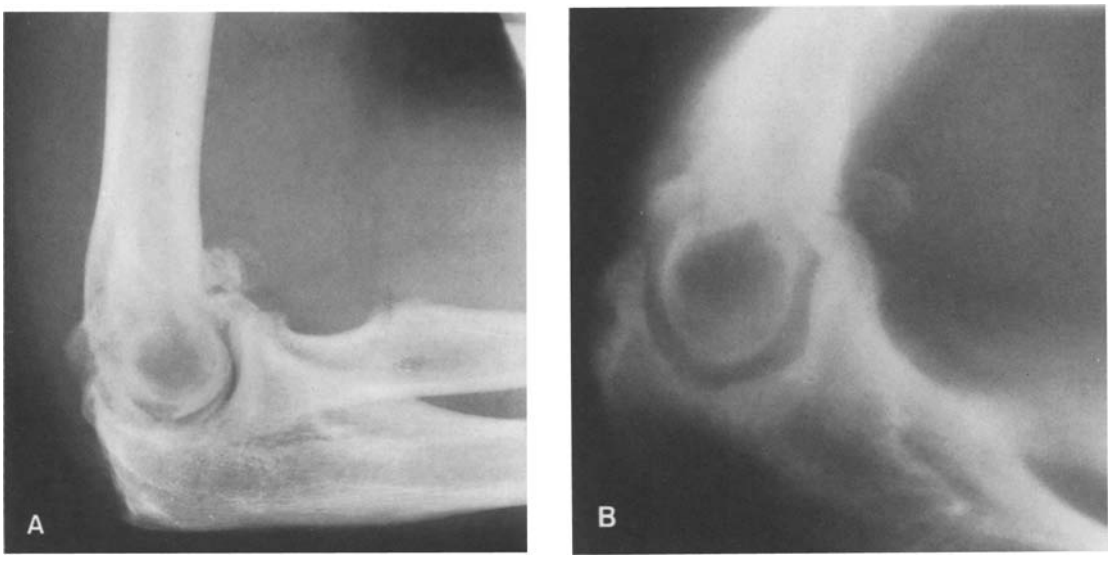

Fig. 3A, B (*Case 3*). **A** Lateral view of right elbow: joint space narrowing, subchondral sclerosis, and osteophytosis are present. Several loose bodies are also noted. These findings are typical of degenerative disease. **B** Lateral tomogram of right elbow: joint space irregularity, sclerosis, and osteophytosis are noted. Loose bodies in both the anterior and posterior portions of the joint are identified

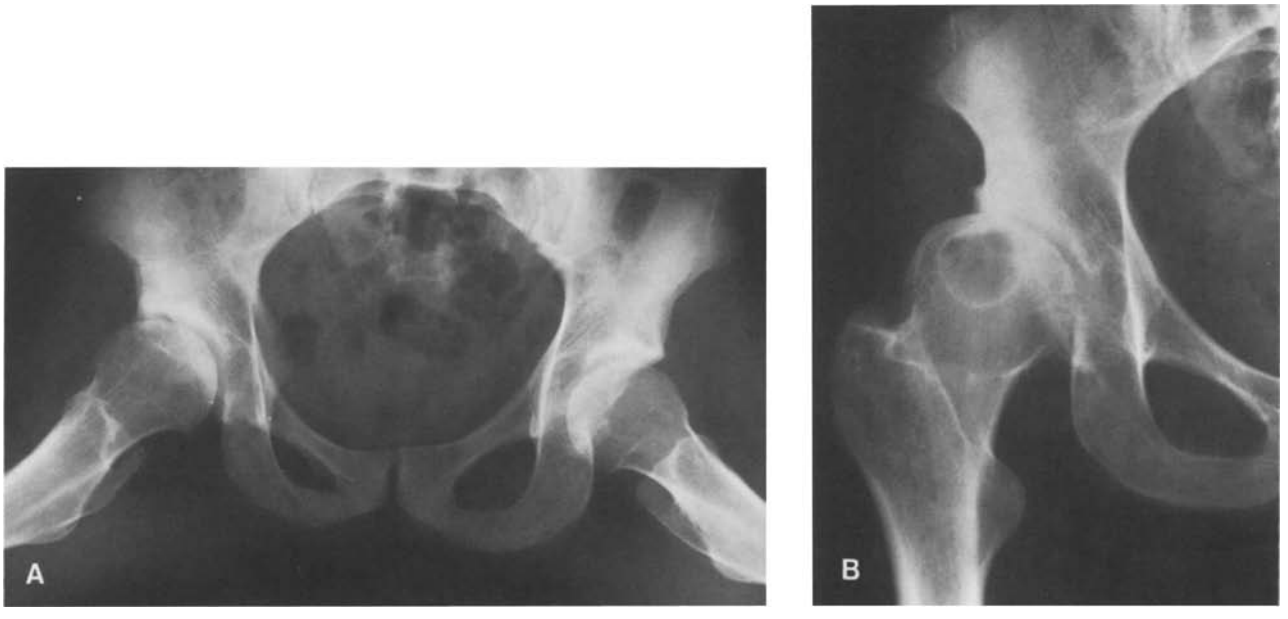

Fig. 4A, B (*Case 4*). **A** AP view of hips (1976): the left hip appears normal. Minimal acetabular dysplasia with subchondral sclerosis and femoral head osteophytes indicates minimal degenerative disease of the right hip. **B** AP view of right hip (1980): advanced superior joint space narrowing with osteophytosis and a large subchondral cyst is noted. Superolateral migration of the hip has also occurred. These changes are characteristic of degenerative disease

The hypothesis that excessive physical activity can lead to OA is a controversial one, championed by some, denounced by others. Studies on marathon runners and soccer players have not detected an increased incidence of OA [1, 11]. However, most reports on professional athletes and certain manual laborers support the hypothesis, showing that the joints involved in these very strenuous activities have an increased incidence of OA. The possibility that non-professional or less strenuous activities can predispose to OA obtains support not only from our study but also from that of Murray and Duncan [9] which showed that boys attending schools with compulsory athletics have a significant increase in tilt deformities – an abnormality implicated in the pathogenesis of OA of the hip.

Results from animal experiments have illus-

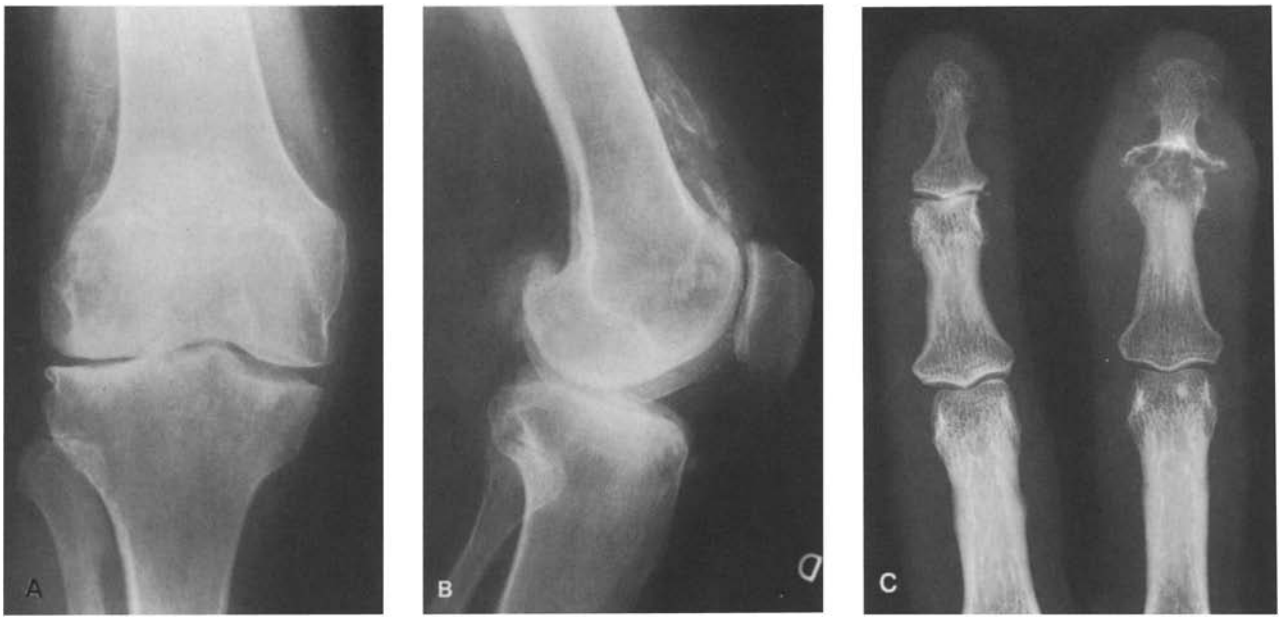

Fig. 5A–C (*Case 5*). **A** AP view of right knee: marked lateral compartment joint space narrowing with subchondral sclerosis and osteophytosis is seen. Multiple loose bodies are present in the suprapatellar joint space laterally. **B** Lateral view of right knee: the osteophytosis and large loose bodies as well as the patellofemoral joint space narrowing are seen. **C** AP view of second and third digits of right hand: marked periarticular soft tissue swelling is present at the third DIP with underlying joint destruction. A moderate joint space narrowing, sclerosis, and osteophytosis are seen at the second DIP

trated the effects activity has on articular cartilage. Not all stresses are equally injurious to cartilage. Radin and Paul have shown that joints can be oscillated or rubbed for long periods without damaging cartilage [12]. However, impact loading or pounding of joints quickly leads to cartilage wear. Activity may also be beneficial. Since cartilage is avascular, its nutrients and waste products may be transported by synovial fluid pumped in and out of cartilage by joint movement. Also regular exercise produces hypertrophy of bone and muscle – structures which absorb most of the stress across joints and thereby protect cartilage [14].

The net effect of activity on cartilage, then, may vary depending on the types of stress applied, and the presence of other mechanisms which can protect or injure cartilage. For example, the importance of the type of stress is shown in jack hammer operators who develop OA in the joints subject to impact loading – shoulder, elbows, but not in the joints which are merely oscillated – fingers [13]. A marathon runner may develop hypertrophy of muscle and bone which helps prevent cartilage damage. A mountain climber may also develop bone and muscle hypertrophy; but since he frequently repels over an uneven surface, he often experiences jarring, or impact loading, of the hip. This may occur so rapidly that protective muscle reflexes cannot be recruited quickly enough, resulting in a large force delivered to the articular cartilage. If a mountain climber also has a dysplastic or inflammed hip, then the likelihood of the activity injuring the cartilage is even greater.

Motivation may also be a factor. It is tempting to speculate that in several of our patients the tenacious pursuit of their activities, despite discomfort, contributed to the development of advanced destruction of bone and cartilage. It is not surprising then, that different studies on different sports have reached different conclusions about the influence of that activity on OA. It is probably the interplay of forces stressing and protecting cartilage which determines whether or not OA develops.

Though some activities may promote cartilage health, our study suggests that chronic, repetitive trauma from participation in certain hobbies and nonprofessional sports can predispose to the development of OA. These results are important for two reasons. First, they indicate that participation in some nonprofessional sports can lead to OA; most reports linking sports-trauma to OA have been on professional athletes. Second, our study suggests that finding DJD in a young person or in a joint usually spared by OA should prompt not only a search for CPPD, hemophilia, and hemochromatosis, but also an inquiry into the patient's previous activities. X-ray evidence of atypical, extensive degenerative changes, particularly in

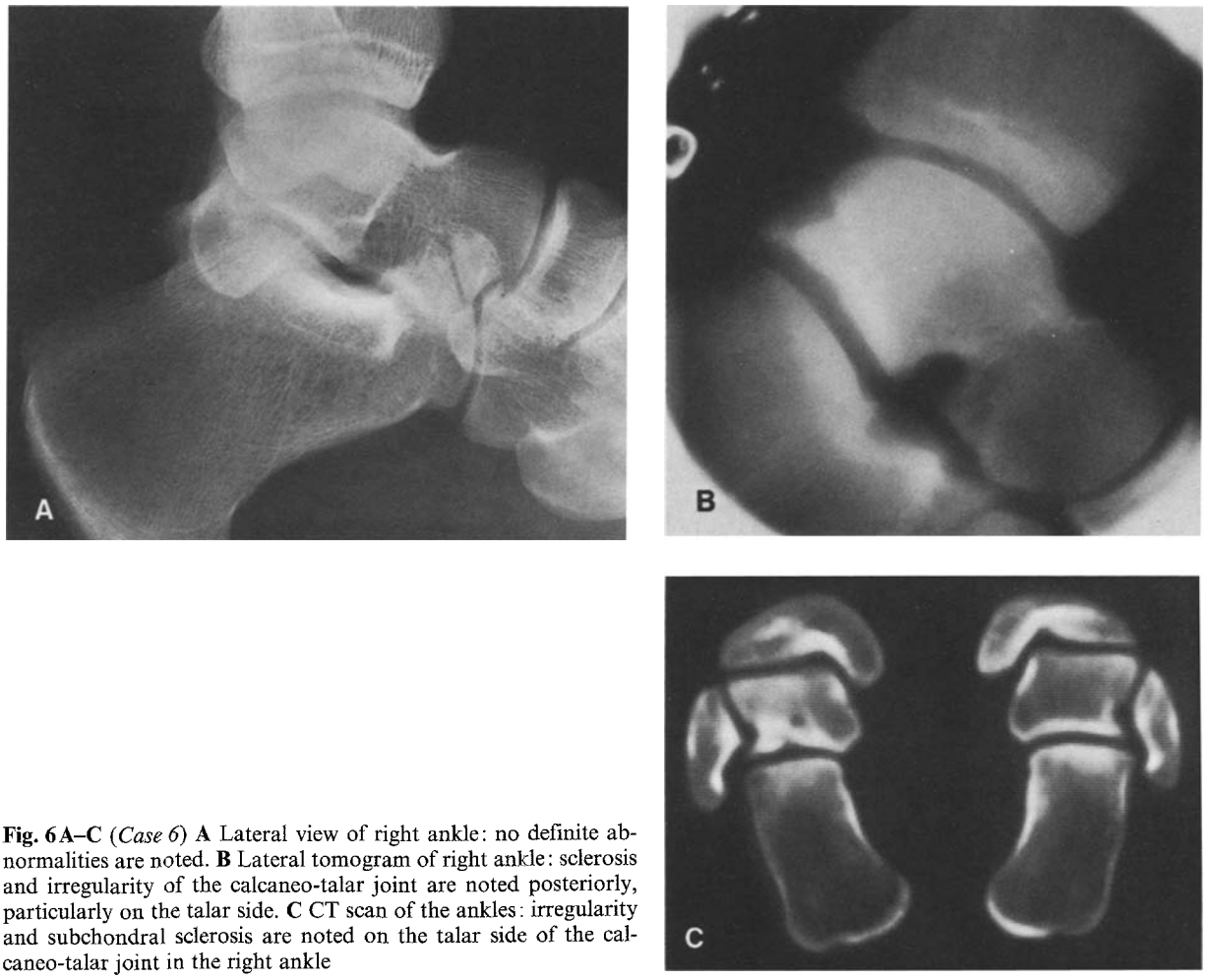

Fig. 6A–C (*Case 6*) **A** Lateral view of right ankle: no definite abnormalities are noted. **B** Lateral tomogram of right ankle: sclerosis and irregularity of the calcaneo-talar joint are noted posteriorly, particularly on the talar side. **C** CT scan of the ankles: irregularity and subchondral sclerosis are noted on the talar side of the calcaneo-talar joint in the right ankle

a young person, may be suggestive of chronic, repetitive trauma from sports.

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