

Osteoarthritis and Exercise: Does Increased Activity Wear Out Joints?

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Introduction

Exercise is one of the most effective ways of improving and maintaining health. High levels of physical activity have been correlated with lower risk for cardiovascular disease, lower blood pressure, and weight reduction as well as increased sense of well-being. More and more people are becoming active in exercise programs and are trying to maintain a high level of physical activity throughout life. A common concern of athletes—and especially of aging athletes—is that increased joint stress may lead to premature “wearing out” of the joints and osteoarthritis.

Background

Osteoarthritis is defined as a noninflammatory, degenerative joint disease characterized by loss of articular cartilage and marginal hypertrophy of bone accompanied by pain and stiffness that is aggravated by prolonged activity. Some other joint problems resemble this definition but do not progressively worsen and do not represent osteoarthritis. An example is development of anterior tibiotalar osteophytes in kicking-type sports such as soccer. Formation of the osteophytes is thought to be caused by repeated traction injury to the attachment of the joint capsule. This condition can be treated and symptoms resolves by removing the osteophytes (a procedure not effective with osteoarthritis). The osteophytes may reform years later, but the ankle typically does not become diffusely arthritic.

Biomechanical Studies

The effects of various forms of stress on articular cartilage have been evaluated biomechanically. Articular cartilage is composed of several layers. The superficial layer provides a smooth, gliding surface. The deeper layers have high concentrations of hydrophilic macromolecules (glycosaminoglycans), which absorb large amounts of water. When mechanical load is applied to the cartilage, the water is squeezed out. This removal of water helps to dissipate the load and provides increased lubrication for the joint.

Cartilage can accommodate a slowly applied load better than an impact load. Impact loads above a certain threshold can acutely disrupt cartilage sur-

faces. The magnitude of load required to acutely disrupt the cartilage surface is reported to be 25 MPa (approximately 3600 psi).¹ The injury may not be initially apparent: Impact sufficient to cause death of chondrocytes and degradation of the matrix may result in changes not seen until months or even years later. Thompson et al² evaluated the effect of a transarticular impact load of 2170 N (approximately 477 lb) to the patellofemoral joint in dogs and noted initial formation of minute fractures of the subchondral bone without visible damage to the cartilage surfaces. During the next six months, changes in the patellar cartilage consistent with osteoarthritis developed. Repeated application of impact loads below the threshold also can lead to disruption of the cartilage surface. Zimmerman et al³ evaluated a cyclic load on the human patella in vitro and with a load of 1000 psi found surface abrasions after application of 250 cycles. The cartilage did not disintegrate until 8000 cycles had been applied.

Animal Studies

Other injuries to the joint also affect the capacity of the articular cartilage to withstand stress. Two important factors in this ability are the surrounding ligamentous restraints and the protective effect of the musculature. O'Connor et al⁴ compared dogs with normal knees to three other groups of dogs: 1) dogs with injury to the anterior cruciate ligament, 2) dogs in whom sensory input was removed from the knee joint, and 3) dogs with both injury to the anterior cruciate ligament and removal of sensory input from the knee joint. In the dogs with normal knees but no articular sensory input, no signs of arthritis had developed after 64 weeks. By eight weeks, dogs with sensory input and injury to the anterior cruciate ligament showed early stages of arthritis, which progressed to moderate-to-severe arthritis by 18 weeks. Dogs with neither knee sensation nor a functioning anterior cruciate ligament showed signs of arthritis in only two weeks and severe arthritis by eight weeks.

Exercise (even strenuous exercise) on normal joints does not result in a substantially increased likelihood of arthritis. In a study evaluating the knees of beagle dogs who ran as much as 40 km/day for a year, Arokoski et al⁵ identified a decrease in the concentration of glycosaminoglycans in the knee but saw no signs of degeneration of the articular cartilage. In a study of beagle dogs who ran on treadmills for as much as 15 km/day at a 15° uphill angle for 40 weeks, Kiviranta et

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ROBERT H SANDMEIER, MD is the Chief of Orthopedics for Northwest Permanente, and specializes in sports medicine. E-mail: robert.sandmeier@kp.org



al⁶ found that cartilage thickness and glycosaminoglycan concentration were both decreased compared with controls. This result contrasted with a previous study,⁷ in which the same authors found an increase in both cartilage thickness and glycosaminoglycan concentration after a more modest running program. Newton et al⁸ found no difference in cartilage thickness or mechanical properties of the cartilage at the end of a study in which 11 dogs ran on a treadmill at 3 km/hr for 75 minutes for 527 weeks (ten years) while wearing weight jackets (weighing 130% of the dog's body weight). Arthritis did not develop in any of the dogs. This research suggests a threshold after which changes are seen in the cartilage and that these changes are probably adaptive rather than pathologic. Even in these studies of long-term, very vigorous exercise, no arthritis was seen in otherwise normal joints.

Human Studies

Research has also been done in humans, but these studies are much more anecdotal in nature because the subjects cannot be controlled as the animals were. It is difficult to find a group that engages in similar activities at similar intensities. In addition, there is currently no easy way to determine who may be susceptible to osteoarthritis.

That previous injury to the joint can result in arthritis is unequivocal. Injury to the meniscus resulting in early signs of arthritis in the knee was described by Fairbank⁹ at a time when the meniscus was still believed to be a vestigial structure. Recently, Daniel et al¹⁰ documented an increased risk of arthritis after injury to the anterior cruciate ligament. In that study,¹⁰ reconstruction of the ligament actually increased the amount of arthritic change.

The risk of osteoarthritis developing in athletes without associated injury to the knee is thought to be minimal. However, this belief cannot be confirmed from the medical literature, because most studies do not separately analyze athletes who have previous knee injuries and those with uninjured knees.

Lane et al¹¹ compared 41 runners aged 50–72 years with matched controls. The comparisons were made on the basis of radiographic changes as well as by clinical symptoms of osteoarthritis. In that study,¹¹ runners had a 40% mean increase in bone density compared with nonrunners. No clinically significant difference between groups was seen in the incidence of osteoarthritis detected either clinically or radiographically. Women runners did have an increased amount of sclerosis and spur formation about the knee, but this difference was of doubtful clinical significance. A

second radiologic study comparing runners with age-matched controls¹² showed no difference in frequency or severity of radiographic changes.¹²

The same author¹³ compared 498 runners with 365 community controls. Runners had less physical disability and higher functional capacity than age-matched controls. The runners sought medical attention less frequently and developed less disability as they aged. These differences were present even after subjects with clinically significant medical problems were excluded and after adjustments were made for age, sex, and occupation.¹³ Spector et al,¹⁴ in a comparison of 81 athletes and more than 900 controls, found slightly increased signs of osteoarthritis by radiographic criteria in the athletes, but the athletes had fewer symptoms than did controls. Although these results are encouraging, it is not possible to determine whether the runners were a self-selected group who were able to continue running because they have fewer musculoskeletal problems or if runners have fewer musculoskeletal problems because they run.¹⁴

Several authors have attempted to differentiate between weightbearing and nonweightbearing activity. Sohn and Micheli¹⁵ attempted to control for the effect of weightbearing exercise by comparing 504 former college runners with 287 swimmers and found no difference in the incidence of osteoarthritis. Kujala et al¹⁶ found radiographic signs of osteoarthritis in 3% of the shooters studied, 29% of the soccer players, 31% of the weightlifters, and 14% of the runners. The authors¹⁶ felt that the majority of the differences in the incidence of osteoarthritis could be explained by the higher rate of injury in soccer players and by increased body weight in weightlifters.¹⁶ Knee injuries resulted in a fivefold increased risk of osteoarthritis.¹⁶ Kujala et al¹⁷ also reported on 2049 athletes who competed in the Olympic Games from 1920 to 1965, comparing the athletes with 1403 matched controls. In this study, the endpoint (presenting for joint replacement) takes into account symptoms as well as radiographic criteria. Endurance athletes (runners) had a relative risk of 1.73, participants in mixed-type sports (ex-soccer players) had a relative risk of 1.9, and participants in power sports (weightlifting, wrestling) had a relative risk of 2.17.¹⁷ Incidence of injury was not reported.

Conclusions

Impact loads are the most likely to result in injury to articular cartilage. Having well-developed muscles decreases the loading on the cartilage and thus has a protective effect. Animal research suggests that exer-

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cise—at least when done in the form of running—is not harmful to normal joints even under high loads and over long distances. In contrast, similar exercise of an injured joint leads to arthritic change. The literature suggests that in humans, athletic activity is associated with a slightly increased risk of osteoarthritis. Athletic individuals seem to tolerate similar radiographic levels of osteoarthritis with less disability than nonathletic individuals. Joint injury is the primary factor that increases the risk of arthritis developing in athletes.

Activities that maintain flexibility, muscle strength, and coordination protect the cartilaginous surfaces and help to maintain joint function in joints that have already been injured and in which arthritic changes have developed or are developing. The forms of exercise that meet these criteria include bicycling, weightlifting (with emphasis on closed-kinetic-chain exercises), and pool exercises.

A good program to start with is an exercise bike with the seat positioned high and with resistance set to a low level. After the patient is able to spend 20 minutes on the bike, the seat may be lowered to deepen flexion, and the level of resistance may be increased. The patient may then add leg presses using a low weight and with a high number of repetitions (start with 20 repetitions at a time). Patients may progressively add weight to the leg press until lifting to their tolerance. I tell them to avoid knee extensions despite the fact that these machines are found everywhere. Reactive forces on the patellofemoral joint exceed body weight, even when light weights are used. For patients without access to exercise equipment, straight-leg raises are a good start. Wall sits are a substitute for leg presses, although it is often difficult for patients to start out with wall sits because they cannot exercise using less than their body weight. Patients should also work on a stretching program to maintain full extension of the knee.

For patients who have suffered a significant injury to the knee but who do not have arthritis, activities that include prolonged, repetitive impact (eg, distance running) are not the best choice for maintaining fitness. Other activities that the patient enjoys and that maintain physical strength and flexibility are probably acceptable if they do not cause pain. The best choices are bicycling, swimming, and weightlifting. Runners usually find this recommendation difficult to accept; many dedicated runners do not feel that any other activity makes them feel as good as running does. Sometimes a difficult decision must be made, however, and they must recognize that they exercise for many reasons and that the possibility that arthritis may develop may be offset by the car-

diovascular benefit and the sense of well-being that they get from running. Doing any exercise—even one that is not especially recommended—is better than doing no exercise. If the choice were running or nothing, I would run. ♦

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