Osteoarthrosis—the orthopedic surgeon's perspective

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The clinically useful definition of osteoarthrosis (OA) is symptomatic loss of significant articular cartilage in a habitual load bearing area of a joint associated with subchondral sclerosis and osteophyte formation. In the large joints, significant articular cartilage loss means full thickness loss of articular cartilage in an area greater than about 1 cm^2. The presence of fibrillated articular cartilage, by itself, is not necessarily progressive. Cartilage fibrillation and softening, with significant proteoglycan loss and markedly increased water content, i.e. chondromalacia, can persist indefinitely (Byers 1974, Meachim 1963). Chondromalacia is a pathologic rather than a clinical diagnosis, since it is asymptomatic, and is not necessarily pre-arthrotic (Abernathy et al. 1978). Trueta and his colleagues describe such changes, particularly in the usually unloaded areas of joints, believing that these changes were the signs of the earliest arthrotic involvement of the joints (Trueta 1963). They are not!

Subchondral bone changes are integral to the arthrotic process. Subchondral sclerosis is the first reliable radiographic sign of arthrosis and precedes joint space narrowing (Pauwels 1976). It is useful to differentiate arthrosis from arthritis, the latter being joint failures from a primarily inflammatory process, whereas arthrosis are joint failures from primarily mechanical factors. Obviously, a progressive arthritis eventually causes mechanical aberrations in the joint, when the inflammatory process destroys enough tissue, as for example when rheumatoid pannus erodes the collateral ligaments of the MCP joints, which then sublux. From a clinical point of view, it is critically important to differentiate this secondary arthrosis, following a primary arthritis, from a primary arthrosis in which mechanical debris causes a secondary inflammation. The pathophysiology of the arthroses is markedly different from the arthritides and, with the exception of total replacement of a joint done for end-stage change, the treatments of the two classes of conditions should be different. For example, it would be a mistake to transplant an articular cartilage surface into an arthritic joint, as the underlying inflammatory process would destroy the transplant, just as it had the original articular cartilage. However, in an arthrotic joint, if there are no concentrations of stress, transplanting a new articular surface would make a great deal of sense. The same is true for pressure reducing surgery, including realignment osteotomies. They are not indicated for the treatment of arthritic joints but are sometimes useful in the treatment of arthrotic ones.

It is important, if we are to make progress in our understanding and treatment of OA, to put limits on what we diagnose as OA. The usual patients with OA are usually middle-aged and present with pain in a single joint, or sometimes in a pair of joints. If there was trauma, the arthrosis is in the damaged joint. Patients with what is usually diagnosed as OA do not initially present with widespread joint involvement or a florid inflammatory component. Such patients usually have some systemic pathology which is manifesting itself in the joints. Arthrotic should mean mechanically caused. Although it is attractive to think of osteoarthrotic joints as inherently biologically abnormal, populated by atypical matrix molecules such as aberrant type II collagen or rogue proteoglycans, waiting for the right mechanical moment to "come out of the closet" and wear away, the presentation of patients with such abnormal matrix molecules is quite different than that of the usual patient with OA (Schaffler and Radin 1991). The patients with abnormal type II collagen present with involvement of almost all their joints and tend to be fairly young. Obviously, structurally weakened joint tissues will fail even under physiologic intra-articular stress, but they are not primarily mechanical joint failures, rather metabolic ones. Such joint failures should be placed in the same category as joint failure from ochronosis, gout, and rickets. The same is true of "gener-

alized inflammatory osteoarthritis" and "disseminated idiopathic sclerosing hypertrophy" of the spine. They are not arthroses in the useful sense. The term "osteoarthritis" is confusing and is best abandoned.

OA primarily involves the hips, knees, first metatarsophalangeal joint, first metacarpophalangeal joint and, most commonly, the distal interphalangeal joints of the fingers. The epidemiology of OA has been hampered by the lack of a good marker. Distal interphalangeal joint involvement (Heberden nodes) is a weak marker, there being several other causes for them besides OA: an isolated hereditary form of Heberden nodes, compression fracture or the result of contracture of the extensor mechanism with "jamming" of the joint on repeated flexion (Smyth 1983). Osteophytes are not pathognomonic for OA (Schaffler and Radin 1991).

There is no relationship between x-ray appearance of an arthrotic joint and the patient's symptoms, since the factors which produce the discomfort will not be apparent on x-ray. These are secondary inflammation caused by breakdown of the articular tissues of the joint and increased interosseous venous pressure caused by bony remodeling (Radin et al. 1993).

The most common causes of OA are following trauma from mal-alignment or deformity, or are so-called idiopathic. This latter category need no longer be a mystery. It is due to accumulated microtrauma secondary to subclinical neuromuscular incoordination, i.e. "microklutziness" (Radin et al. 1991). Symptomatic OA does not appear to be related to occupation or to the level of an individual's activity (Sokoloff 1969).

The joint is an organ, and, as in all organs, there is a functional interdependence of the tissues of which it is composed. The health of the articular cartilage is dependent upon several tissues. The synovia lines the joint cavity, and, if it becomes inflamed enough, can actually invade the articular cartilage and destroy it. The ligaments and capsule of the joint keep it stable and limit the sheer stress on and the tensile strain in the articular cartilage. The calcified base upon which the articular cartilage is mounted is a layer of calcified cartilage supported by a bony plate, itself supported by a network of struts. Internal damage to articular cartilage results in hypertrophy and cloning of chondrocytes. The pathologic hallmarks of OA are osteophytes (caused by tension on the pleopotential cells of the synovia/capsule at its insertion to the edge of the articular surface) and tidemark advance. The tidemark is the junction between the calcified cartilage and the subchondral bony plate, and its duplication and advance toward the articular surface was described long ago (Ogston 1876, Johnson 1956). Tidemark advance is a significant phenomenon. It is the sine que non of the osteoarthrotic process. Thinned articular cartilage is subjected to significant deep tensile stresses which can lead to horizontal splits and the breaking off of chunks of this tissue (O'Connor and Johnston 1993).

The end of a bone is generally created by what embryologists call a secondary center of ossification which can be considered an oval of circumferentially growing endochondral bone. This forms the articular end of the bones, and obviously must grow in concert with longitudinal skeletal growth, otherwise the joints will not grow. At adulthood these growth processes stop. The primary center of ossification, the epiphysial plates fuse and growth cartilage disappears. But the secondary center of ossification, because it makes up the sides and top the articular surface, retains its cartilage and thus the potential for additional joint growth. In the adult skeleton, additional joint growth is not a good idea. There is no coordination, and these larger parts no longer fit. This happens, for example in acromegaly, as the result of a pituitary gland tumor which fills the adult body with adolescent or higher levels of growth hormone (Sokoloff 1969). Malfit joints can cause interarticular stress concentrations, and OA can result. OA is a hallmark of this condition and is one of the patient's major sources of symptoms. Another factor which can cause reactivation of the secondary centers of ossification, albeit locally, is cumulative microtrauma.

Articular cartilage and subchondral bone are viscoelastic materials, meaning that their fluid components, water in the case of cartilage and marrow in the case of bone, contribute to the tissues' mechanical properties. If viscoelastic materials are slowly squeezed, their fluid phase flows, which spares the strains on the nonfluid portions of the matrix. However, if one applies the loads too rapidly for these fluid phases to flow, the matrix of these tissues can be damaged. This is the basis of the deleterious effect of impulsive loading on the joints of animals, as opposed to loads applied at slower rates. Working with colleagues in the Oxford Orthopaedic Engineering Center in 1985-86, and, subsequently, in the Motion Analysis Laboratory at West Virginia University, we have been able to show that about a third of normal individuals have, probably for a variety of reasons, a minor incoordination (microklutziness), which interferes with the timely deceleration of limbs about to hit a solid surface, such as the legs in walking or the fingers in typing. These individuals repetitively, impulsively load their joints (Radin et al. 1990, Radin et al. 1991). If you want to

create OA in an animal without operating on it and without injecting harmful substances into its joints, the only way you can do it is by repetitive impulsive loading (Radin et al. 1973). You cannot do it by running the animal, and you cannot do it by making the animal walk around wearing saddlebags with heavy weights in them. And, amazingly repetitive impulsive loading creates OA very quickly with loads well within physiological limits. Years ago we showed that rabbits, subjected to repetitive impulsive load of 1.2 times the body weight at 60 times a minute (1 Htz) for forty minutes/day for six weeks, if then left for another six weeks, develop OA of their knees (Radin et al. 1973). And we have subsequently shown that individuals who are microklutzes tend to have prearthrosis (Radin et al. 1991). We have also found that people with OA, who do not limp tend to be microklutzes (Tashman unpublished). Limping is an excellent protective mechanism which, if absolved by relieving pain, may lead to a potentiated progression of the OA.

The diagnosis of OA is made on the basis of the history of pain, primarily with use of the joint, and relief of this pain with rest. Physical examination shows minimal inflammation, diminished joint range of motion, crepitus, and sometimes instability and deformity. The characteristic x-ray appearance is of subchondral sclerosis, joint space narrowing and osteophyte formation.

Nonoperative treatment works well in most patients. Life-style changes and analgesics, taken on a regular basis, can diminish the patient's symptoms. Considerable success has been achieved by shock absorption with the use of soft soled footwear (Voloshin and Wosk 1982), and the training of patients with biofeedback techniques to stop microklutzes, i.e. to stop impulsively loading their joints (Tashman unpublished).

The pathology of OA, loss of articular cartilage surrounded by its local hypertrophy, and the formation of new bone, and osteophytic hyaline cartilage, suggests that joints can heal. The pathology of OA is best thought of as an attempt at healing, which, because stress concentrations or adverse strain circumstances are still operant, is not successful. In cases where the intra-articular stress can be brought down to within physiological limits, joints, even without any articular cartilage, can clinically heal. They reestablish a fibrocartilaginous bearing surface, which, in the presence of well distributed physiologically reasonable stress, will survive (Radin and Burr 1984, Radin et al. 1990). The difficulty that many scientists in the field seem to have is a failure to understand that OA is usually caused by injury, and biochemically and pathologically is a repair mechanism, not a reconstitution of a normal joint. But, unless those studying articular healing take mechanical factors into consideration, pay attention to the interrelationship between articular cartilage and its subchondral bed, appreciate that changes in the subchondral bed affect what happens to the overlying articular cartilage, and understand that the pathology of OA is a response to injury, the field will remain confused. The orthopaedic community needs to understand how and when to perform pressure reducing operations, and to perform these operations in such a way that the interarticular pressures are reduced.

OA then is not a common final pathway, as there are a multiple number of routes to get to the result. Therefore, the chances of finding biological markers, which will be common to all, or even most early cases, cannot be great. The molecular markers of inflammation and healing are not pathognomonic in OA. The critically important studies on OA need to be based on the relationship between cellular metabolism, cell differentiation, and mechanical factors. And what we should be looking for is lasting repair and not regeneration.

The continuing confusion in OA research is based upon failures to limit the diagnostic criteria, to understand that we are dealing with a biological healing process that is driven mechanically, and the apparent failure to understand that, although chondrocytes are highly differentiated, they still have many things in common with other connective tissue cells. Based on an orthopedic surgical perspective and clinical experience, the hunt for molecular markers implies a disease, rather than the organ failure that OA is.

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