CHAPTER

5

Biomechanics of Cartilage

JOSEPH M. MANSOUR, PH.D.

The materials classed as cartilage exist in various forms and perform a range of functions in the body. Depending on its composition, cartilage is classified as articular cartilage (also known as hyaline), fibrocartilage, or elastic cartilage. Elastic cartilage helps to maintain the shape of structures such as the ear and the trachea. In joints, cartilage functions as either a binder or a bearing surface between bones. The annulus fibrosus of the intervertebral disc is an example of a fibrocartilaginous joint with limited movement (an amphiarthrosis). In the freely moveable synovial joints (diarthroses) articular cartilage is the bearing surface that permits smooth motion between adjoining bony segments. Hip, knee, and elbow are examples of synovial joints. This chapter is concerned with the mechanical behavior and function of the articular cartilage found in freely movable synovial (diarthroidal) joints.

In a typical synovial joint, the ends of opposing bones are covered with a thin layer of articular cartilage (Fig. 5.1). On the medial femoral condyle of the knee, for example, the cartilage averages 0.41 mm in rabbit and 2.21 mm in humans [2]. Normal articular cartilage is white, and its surface is smooth and glistening. Cartilage is aneural, and in normal mature animals, it does not have a blood supply. The entire joint is enclosed in a fibrous tissue capsule, the inner surface of which is lined with the synovial membrane that secretes a fluid known as *synovial fluid*. A relatively small amount of fluid is present in a normal joint: less than 1 mL, which is less than one fifth of a teaspoon. Synovial fluid is clear to yellowish and is stringy. Overall, synovial fluid resembles egg white, and it is this resemblance that gives these joints their name, *synovia,* meaning "with egg."

Cartilage clearly performs a mechanical function. It provides a bearing surface with low friction and wear, and because of its compliance, it helps to distribute the loads between opposing bones in a synovial joint. If cartilage were a stiff material like bone, the contact stresses at a joint would be much higher, since the area of contact would be much smaller. These mechanical functions alone would probably not be sufficient to justify an in-depth study of cartilage biomechanics. However, the apparent link between osteoarthrosis and

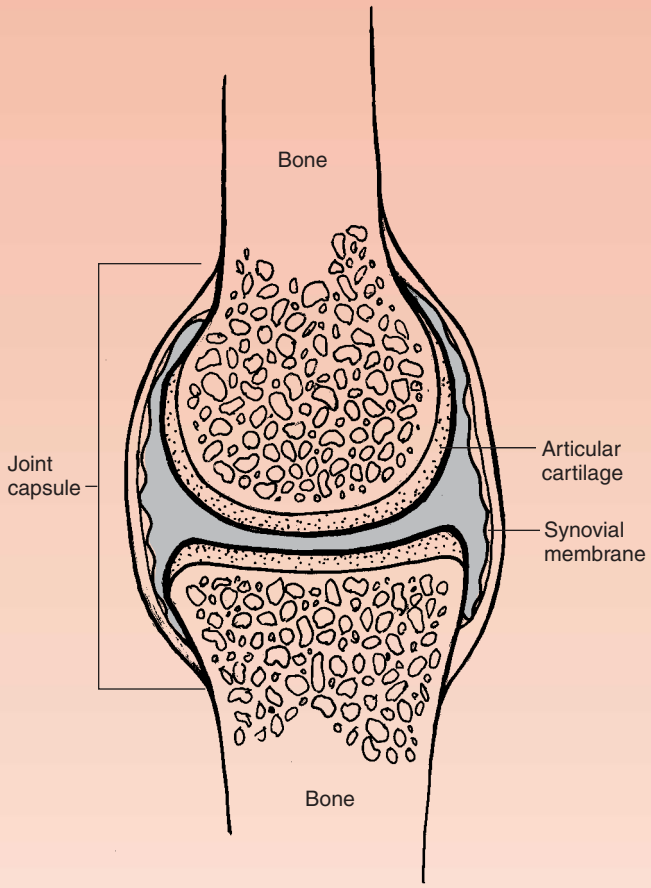


Figure 5.1: Schematic representation of a synovial joint. Articular cartilage forms the bearing surface on the ends of opposing bones. The space between the capsule and bones is exaggerated in the figure for clarity.

mechanical factors in a joint adds a strong impetus for studying the mechanical behavior of articular cartilage.

The specific goals of this chapter are to

- **Describe the structure and composition of cartilage in relation to its mechanical behavior**
- **Examine the material properties of cartilage, what they mean physically, and how they can be determined**
- **Describe modes of mechanical failure of cartilage**
- **Describe the current state of understanding of joint lubrication**
- **Describe the etiology of osteoarthrosis in terms of mechanical factors**

A comment on terminology seems appropriate. *Osteoarthritis* is the term commonly used to describe the apparent degeneration of articular cartilage. Radin has argued that this is a misnomer since osteoarthritis does not directly involve inflammation. He suggests the term *osteoarthrosis,* which is defined as "loss of articular cartilage with eburnation of the underlying bone associated with a proliferative response [68,69]." In this chapter, the term *osteoarthrosis* is used in place of *osteoarthritis*. Before proceeding through this chapter, the reader should be familiar with the basic concepts and terminology introduced in Chapters 1 and 2.

COMPOSITION AND STRUCTURE OF ARTICULAR CARTILAGE

Articular cartilage is a living material composed of a relatively small number of cells known as *chondrocytes* surrounded by a multicomponent matrix. Mechanically, articular cartilage is a composite of materials with widely differing properties. Approximately 70 to 85% of the weight of the whole tissue is water. The remainder of the tissue is composed primarily of proteoglycans and collagen. Proteoglycans consist of a protein core to which glycosaminoglycans (chondroitin sulfate and keratan sulfate) are attached to form a bottlebrush-like structure. These proteoglycans can bind or aggregate to a backbone of hyaluronic acid to form a macromolecule with a weight up to 200 million [61] (Fig. 5.2). Approximately 30% of the dry weight of articular cartilage is composed of proteoglycans. Proteoglycan concentration and water content vary through the depth of the tissue. Near the articular surface, proteoglycan concentration is relatively low, and the water content is the highest in the tissue. In the deeper regions of the cartilage, near subchondral bone, the proteoglycan concentration is greatest, and the water content is the lowest [43,51,59]. Collagen is a fibrous protein that makes up 60 to 70% of the dry weight of the tissue. Type II is the predominant collagen in articular cartilage, although other types are present in smaller amounts [16]. Collagen architecture varies through the depth of the tissue.

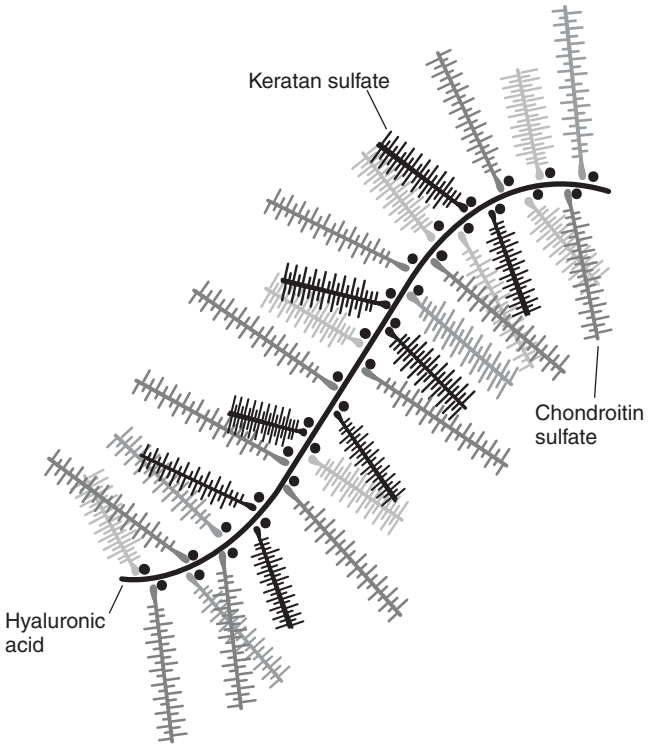


Figure 5.2: A proteoglycan aggregate showing a collection of proteoglycans bound to a hyaluronic backbone. Proteoglycans are the bottlebrush-like structures consisting of a protein core with side chains of chondroitin sulfate and keratan sulfate. Negatively charged sites on the chondroitin and keratan sulfate chains cause this aggregate to spread out and occupy a large domain when placed in an aqueous solution.

The structure of articular cartilage is often described in terms of four zones between the articular surface and the subchondral bone: the surface or superficial tangential zone, the intermediate or middle zone, the deep or radiate zone, and the calcified zone (Fig. 5.3). The calcified cartilage is the boundary between the cartilage and the underlying subchondral bone. The interface between the deep zone and calcified cartilage is known as the *tidemark*. Optical microscopy (e.g., polarized light), scanning electron microscopy, and transmission electron microscopy have been used to reveal the structure of articular cartilage [6,7,26,27,61,85]. While each of these methods suggests somewhat similar collagen orientation for the superficial and deep zones, the orientation of fibers in the middle zone remains controversial.

Using scanning electron microscopy to investigate the structure of cartilage in planes parallel and perpendicular to split lines, Jeffery and coworkers [27] have given some new insights into the collagen structure (Fig. 5.3). **Split lines** are formed by puncturing the cartilage surface at multiple sites with a circular awl. The resulting holes are elliptical, not circular, and the long axes of the ellipses are aligned in what is called the *split line direction*. In the plane parallel to a split line, the collagen is organized in broad layers or leaves, while in the plane orthogonal to the split lines the structure has a ridged pattern that is interpreted as the edges of the leaves (Fig. 5.3). In the calcified and deep zones, collagen fibers are oriented radially and are arranged in tightly packed bundles. The bundles are linked by numerous fibrils. From the upper deep zone into the middle zone, the radial orientation becomes less distinct, and collagen fibrils form a network that surrounds the chondrocytes. In the superficial zone, the fibers are finer than in the deeper zones, and the collagen structure is organized into several layers. An amorphous layer that does not appear to contain any fibers is found on the articular surface. The mechanical behavior of articular cartilage is determined by the interaction of its predominant components: collagen, proteoglycans, and interstitial fluid.

MECHANICAL BEHAVIOR AND MODELING

In an aqueous environment, proteoglycans are polyanionic; that is, the molecule has negatively charged sites that arise from its sulfate and carboxyl groups. In solution, the mutual repulsion of these negative charges causes an aggregated proteoglycan molecule to spread out and occupy a large volume. In the cartilage matrix, the volume occupied by proteoglycan aggregates is limited by the entangling collagen framework. The swelling of the aggregated molecule against the collagen

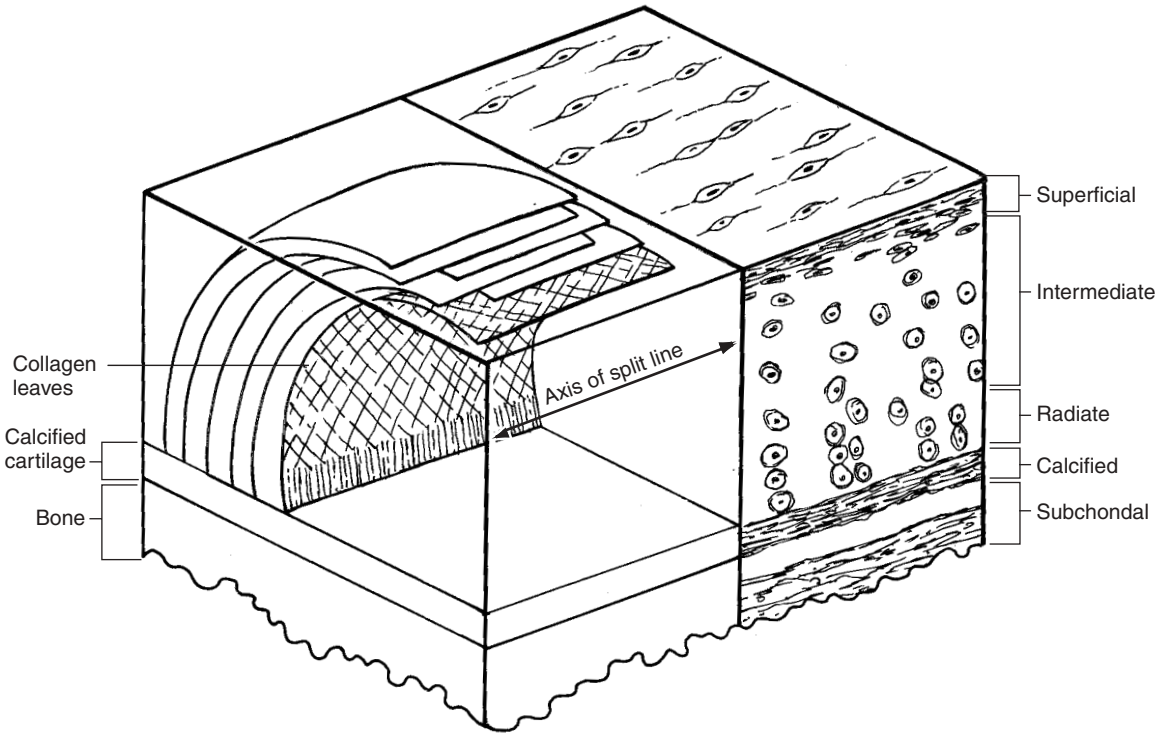


Figure 5.3: Cross sections cut through the thickness of articular cartilage on two mutually orthogonal planes. These planes are oriented parallel and perpendicular to split lines on the cartilage surface. The background shows the four zones of the cartilage: superficial, intermediate, radiate, and calcified. The foreground shows the organization of collagen fibers into "leaves" with varying structure and organization through the thickness of the cartilage. The leaves of collagen are connected by small fibers not shown in the figure.

framework is an essential element in the mechanical response of cartilage. When cartilage is compressed, the negatively charged sites on aggrecan are pushed closer together, which increases their mutual repulsive force and adds to the compressive stiffness of the cartilage. Nonaggregated proteoglycans would not be as effective in resisting compressive loads, since they are not as easily trapped in the collagen matrix. Damage to the collagen framework also reduces the compressive stiffness of the tissue, since the aggregated proteoglycans are contained less efficiently.

The mechanical response of cartilage is also strongly tied to the flow of fluid through the tissue. When deformed, fluid flows through the cartilage and across the articular surface [42]. If a pressure difference is applied across a section of cartilage, fluid also flows through the tissue [51]. These observations suggest that cartilage behaves like a sponge, albeit one that does not allow fluid to flow through it easily.

Recognizing that fluid flow and deformation are interdependent has led to the modeling of cartilage as a mixture of fluid and solid components [59–61]. This is referred to as the *biphasic model of cartilage*. In this modeling, all of the solid-like components of the cartilage, proteoglycans, collagen, cells, and lipids are lumped together to constitute the solid phase of the mixture. The interstitial fluid that is free to move through the matrix constitutes the fluid phase. Typically, the solid phase is modeled as an incompressible elastic material, and the fluid phase is modeled as incompressible and inviscid, that is, it has no viscosity [60]. Under impact loads, cartilage behaves as a single-phase, incompressible, elastic solid; there simply isn't time for the fluid to flow relative to the solid matrix under rapidly applied loads. For some applications, a viscoelastic model is used to describe the behavior of cartilage in creep, stress relaxation, or oscillating shear. Although the mathematics of modeling cartilage is outside the scope of this chapter, some examples illustrate the fundamental fluid–solid interaction in cartilage.

MATERIAL PROPERTIES

A confined compression test is one of the commonly used methods for determining material properties of cartilage (Fig. 5.4). A disc of tissue is cut from the joint and placed in an impervious well. Confined compression is used in either a "creep" mode or a "relaxation" mode. In the creep mode, a constant load is applied to the cartilage through a porous plate, and the displacement of the tissue is measured as a function of time. In relaxation mode, a constant displacement is applied to the tissue, and the force needed to maintain the displacement is measured.

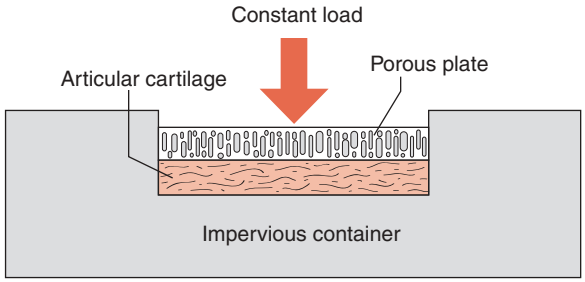


Figure 5.4: Schematic drawing of an apparatus used to perform a confined compression test of cartilage. A slice of cartilage is placed in an impervious, fluid-filled well. The tissue is loaded through a porous plate. In the configuration shown, the load is constant throughout the test, which can last for several thousand seconds. Since the well is impervious, flow through the cartilage will only be in the vertical direction and out of the cartilage.

In creep mode, the cartilage deforms under a constant load, but the deformation is not instantaneous, as it would be in a single-phase elastic material such as a spring. The displacement of the cartilage is a function of time, since the fluid cannot escape from the matrix instantaneously (Fig. 5.5). Initially, the displacement is rapid. This corresponds to a relatively large flow of fluid out of the cartilage. As the rate of displacement slows and the displacement approaches a constant value, the flow of fluid likewise slows. At equilibrium, the displacement is constant and fluid flow has stopped. In general, it takes several thousand seconds to reach the equilibrium displacement.

By fitting the mathematical biphasic model to the measured displacement, two material properties of the cartilage are determined: the aggregate modulus and permeability. The aggregate modulus is a measure of the stiffness of the tissue at equilibrium when all fluid flow has ceased. The higher the aggregate modulus, the less the tissue deforms under a given load. The aggregate modulus of cartilage is typically in the range of 0.5 to 0.9 MPa [2]. There is no analogous material constant for solid materials, but using the aggregate modulus and representative values of Poisson's ratio (described below), the Young's modulus of cartilage is in the range of 0.45 to 0.80 MPa. For comparison, the Young's modulus of steel is 200 GPa and for many woods is about 10 GPa parallel to the grain. These numbers show that cartilage has a much lower stiffness (modulus) than most engineering materials.

In addition to the aggregate modulus, the permeability of the cartilage is also determined from a confined compression test. The permeability indicates the resistance to fluid flow through the cartilage matrix. Permeability was first introduced in the study of flow through soils. The average fluid velocity through a soil sample (v_{ave}) is proportional to the pressure gradient (∇p) (Fig. 5.6). The constant of proportionality (k) is called the *permeability*. This relationship is expressed by Darcy's law,

$$v_{ave} = k\nabla p \qquad \text{(Equation 5.1)}$$

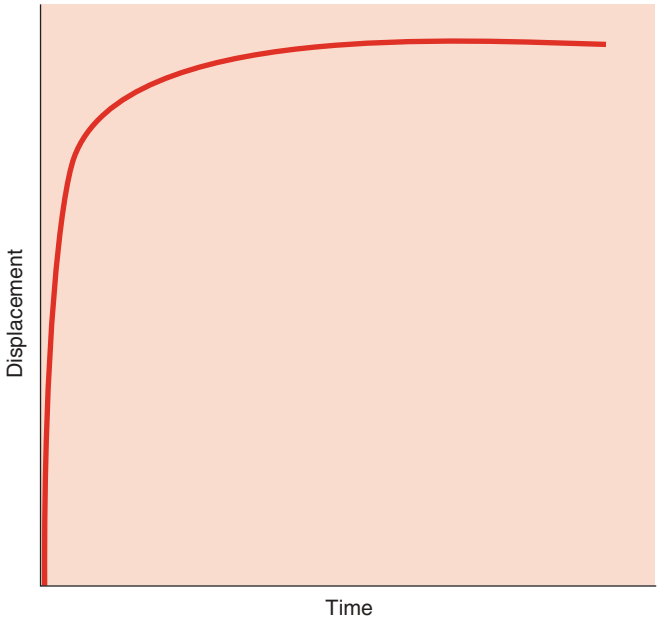


Figure 5.5: Typical displacement of cartilage tested in a confined compression test. A constant load is applied to the cartilage, and the displacement is measured over time. Initially, the deformation is rapid, as relatively large amounts of fluid are exuded from the cartilage. As the displacement reaches a constant value, the flow slows to zero. Two material properties are determined from this test.

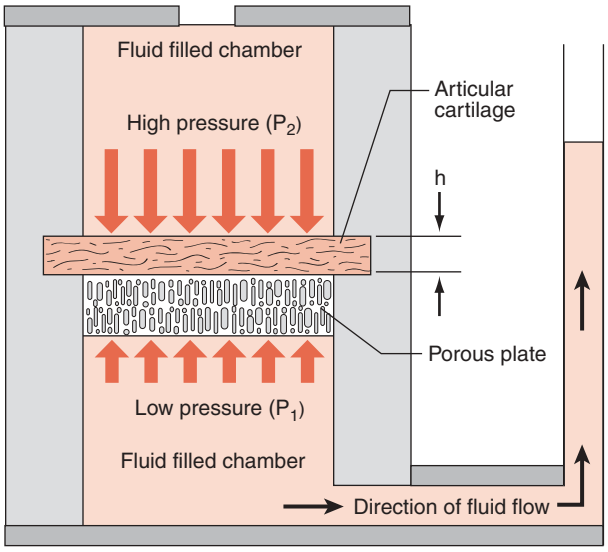


Figure 5.6: Schematic representation of a device used to measure the permeability of cartilage. A slice of cartilage is supported on a porous plate in a fluid-filled chamber. High pressure applied to one side of the cartilage drives fluid flow. The average fluid velocity through the cartilage is proportional to the pressure gradient, and the constant of proportionality is called the *permeability*.

where the pressure gradient is approximated by

$$\nabla p \approx \frac{P_2 - P_1}{h} \qquad \text{(Equation 5.2)}$$

In SI units, the permeability of cartilage is typically in the range of 10^{-15} to 10^{-16} m^4/Ns. If a pressure difference of 210,000 Pa (about the same pressure as in an automobile tire) is applied across a slice of cartilage 1 mm thick, the average fluid velocity will be only $1 \cdot 10^{-8}$ m/s, which is about 100 million times slower than normal walking speed.

Permeability is not constant through the tissue. The permeability of articular cartilage is highest near the joint surface (making fluid flow relatively easy) and lowest in the deep zone (making fluid flow relatively difficult) [50–52]. Permeability also varies with deformation of the tissue. As cartilage is compressed, its permeability decreases [37, 47]. Therefore, as a joint is loaded, most of the fluid that crosses the articular surface comes from the cartilage closest to the joint surface. Under increasing load, fluid flow will decrease because of the decrease in permeability that accompanies compression.

CLINICAL RELEVANCE: VARIABLE PERMEABILITY

Deformation-dependent permeability may be a valuable mechanism for maintaining load sharing between the solid and fluid phases of cartilage. If the fluid flowed easily out of the tissue, then the solid matrix would bear the full contact stress, and under this increased stress, it might be more prone to failure.

An indentation test provides an attractive alternative to confined compression [20, 21,33,45,58,82] (Fig. 5.7). Using an indentation test, cartilage is tested in situ. Since discs of cartilage are not removed from underlying bone, as must be done when using confined compression, indentation may be used to test cartilage from small joints. In addition, three independent material properties are obtained from one indentation test, but only two are obtained from confined compression. Typically, an indentation test is performed under a constant load. The diameter of the indenter varies depending on the curvature of the joint surface, but generally is no smaller than 0.8 mm. Under a constant load, the displacement of the indenter resembles that for confined compression and requires several thousand seconds to reach equilibrium. By fitting the biphasic model of the test to the measured indentation, the aggregate modulus, Poisson's ratio, and permeability are determined. Poisson's ratio is typically less than 0.4 and often approaches zero. This finding is a significant departure from earlier studies, which assumed that cartilage was incompressible and, therefore, had a Poisson's ratio of 0.5. This assumption was based on cartilage being mostly water, and water may often be modeled as an incompressible material. However, when cartilage is loaded, fluid flows out of the solid matrix, which reduces the volume of the whole cartilage. Recognizing that cartilage is a mixture of a solid and fluid leads to the whole tissue behaving as a compressible material, although its components are incompressible.

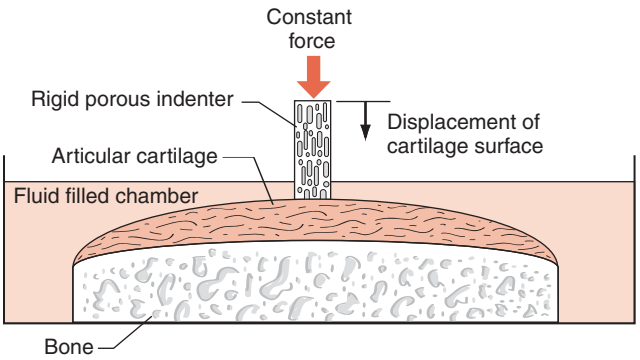


Figure 5.7: Schematic representation of an apparatus used to perform an indentation test on articular cartilage. Unlike the confined compression and most permeability tests, the cartilage remains attached to its underlying bone, which provides a more natural environment for testing. A constant load is applied to a small area of the cartilage through a porous indenter. The displacement of the cartilage is similar to that shown in Figure 5.6. Three material properties are determined from this test.

The equilibrium displacement is determined by the aggregate modulus and Poisson's ratio. The permeability influences the rate of deformation. If the permeability is high, fluid can flow out of the matrix easily, and the equilibrium is reached quickly. A lower permeability causes a more gradual transition from the rapid early displacement to the equilibrium. These qualitative results are helpful for interpreting data from tests of normal and osteoarthrotic cartilage.

CLINICAL RELEVANCE: PERMEABILITY OF OSTEOARTHROTIC CARTILAGE

The lower modulus and increased permeability of osteoarthrotic cartilage result in greater and more-rapid deformation of the tissue than normal. These changes may influence the synthetic activity of the chondrocytes, which are known to respond to their mechanical environment. [8,87,96]

Pure shear provides a means for evaluating the intrinsic properties of the solid matrix. Small torsional displacements of cylindrical samples (which produce pure shear), result in no volume change of the cartilage to drive fluid flow. Furthermore, the interstitial fluid is water. It has low viscosity and does not make an appreciable contribution to resisting shear. Therefore, the resistance to shear is due to the solid matrix. Tests of cartilage in shear show that the matrix behaves as a viscoelastic solid [18–20,80]. Mathematical models of cartilage deformation also suggest that the matrix may behave as a viscoelastic solid [44,80,83].

Studying the tensile properties of cartilage illustrates its anisotropy, inhomogeneity, some surprising age-dependent

changes in mechanical behavior, and additional collagen–proteoglycan interaction. Tensile tests of cartilage are performed by first removing the cartilage from its underlying bone. This sheet of cartilage is sometimes cut into thin slices (200–500 μm thick) parallel to the articular surface, using a microtome. Dumbbell-shaped specimens are cut from each slice with a custom-made cookie cutter.

A particularly thorough study of the tensile properties of cartilage shows that samples oriented parallel to split lines have a higher tensile strength and stiffness than those perpendicular to the split lines. In skeletally mature animals (closed physis), tensile strength and stiffness decrease from the surface to the deep zone. In contrast, tensile strength and stiffness increase with depth from the articular surface in skeletally immature (open physis) animals [76].

The relative influence of the collagen network and proteoglycans on the tensile behavior of cartilage depends on the rate of loading [77]. When pulled at a slow rate, the collagen network alone is responsible for the tensile strength and stiffness of cartilage. At high rates of loading, interaction of the collagen and proteoglycans is responsible for the tensile behavior; proteoglycans restrain the rotation of the collagen fibers when the tissue is loaded rapidly.

RELATIONSHIP BETWEEN MECHANICAL PROPERTIES AND COMPOSITION

In addition to the qualitative descriptions given above, quantitative correlations between the mechanical properties of cartilage and glycosaminoglycan content, collagen content, and water content have been established. The compressive stiffness of cartilage increases as a function of the total glycosaminoglycan content [35] (Fig. 5.8). In contrast, there is no correlation of compressive stiffness with collagen content. In these cases, compressive stiffness is measured in creep, 2 seconds after a load is applied to the tissue. Permeability and compressive stiffness, as measured by the aggregate modulus, are both highly correlated with water content. As the water content increases, cartilage becomes less stiff and more permeable [1] (Fig. 5.9). Note that the inverse of permeability is plotted in Figure 5.9*B*. This is done for convenience,

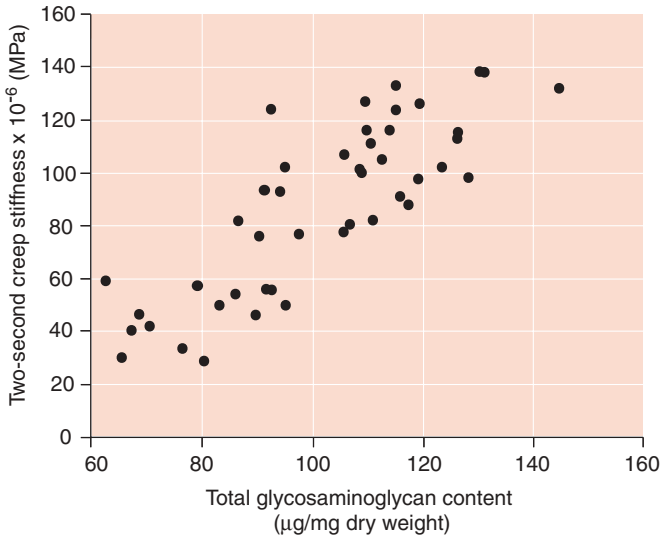
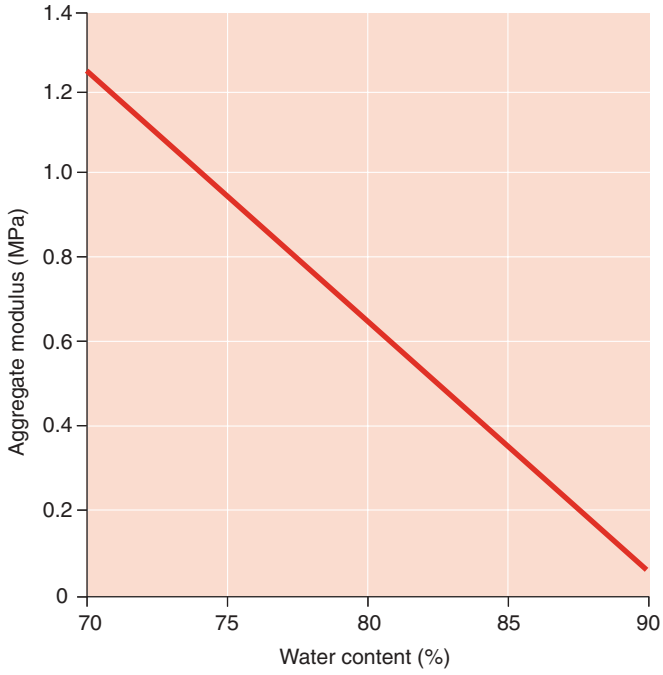


Figure 5.8: Correlation of compressive stiffness with the total glycosaminoglycan concentration. As the total glycosaminoglycan concentration decreases, the compressive stiffness also decreases.



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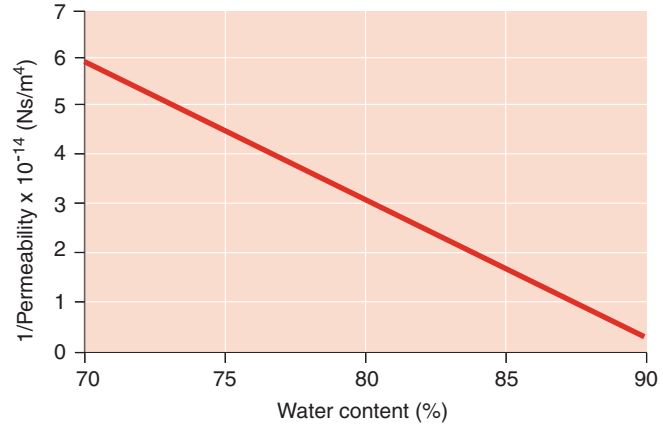

B

Figure 5.9: **A.** Correlation of the aggregate modulus with water content of articular cartilage. A regression line obtained from tests of a large number of samples is plotted. As the water content increases, the aggregate modulus decreases. **B.** Correlation of the inverse of permeability with water content. A regression line obtained from tests of a large number of samples is plotted. As the water content increases, the permeability increases.

since the permeability becomes very large as the water content increases.

CLINICAL RELEVANCE: MATERIAL PROPERTIES OF CARTILAGE

The relationships between material properties and water content help to explain early cartilage changes in animal models of osteoarthrosis. Proteoglycan content and equilibrium stiffness decrease and the rate of deformation and water content increases in these models [38,56]. Decreasing proteoglycan content allows more space in the tissue for fluid. An increase in water content correlates with an increase in permeability. Increasing permeability allows fluid to flow out of the tissue more easily, resulting in a more rapid rate of deformation.

Using confined compression, indentation, tension, and shear tests, the mechanical properties of cartilage can be determined. These properties are necessary for any analysis of stress in the tissue. However, material properties do not give any indication of the failure of cartilage. For example, simply knowing the value of aggregate modulus or Poisson's ratio is not sufficient to predict if cartilage will develop the cracks, fissures, and general wear that is characteristic of osteoarthrosis. Various loading conditions have been used to gain better insight into the failure properties of cartilage.

MECHANICAL FAILURE OF CARTILAGE

A characteristic feature of osteoarthrosis is cracking, fibrillation, and wear of cartilage. This appears to be a mechanically driven process, and it motivates numerous investigations aimed at identifying the stresses and deformations responsible for the failure of articular cartilage. Since cartilage is an anisotropic material, we expect that it has greater resistance to some components of stress than to others. For example, it could be relatively strong in tension parallel to collagen fibers, but weaker in shear along planes between leaves of collagen.

Tensile failure of cartilage has been of particular interest, since it was generally believed that vertical cracks in cartilage were initiated by relatively high tensile stresses on the articular surface. More-recent computational models of joint contact show that the tensile stress on the surface is lower than originally thought, although tensile stress still exists within the cartilage [13–15]. It now appears that failure by shear stress may dominate. Studies of the tensile failure of cartilage are primarily concerned with variations in properties among joints, the effects of repeated load, and age.

Kempson and coworkers report a decrease in failure stress with age for cartilage from hip and knee [30–32, 34]. However, they find no appreciable age-dependent decrease in tensile failure stress for cartilage from the talus (Fig. 5.10).

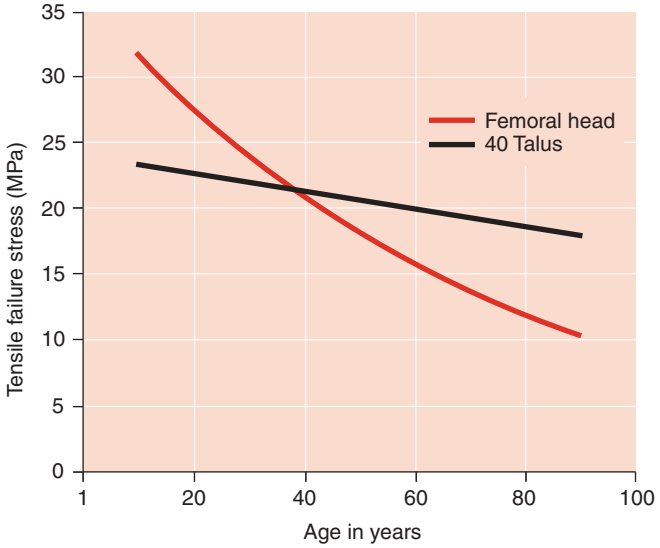

Figure 5.10: Comparison of the tensile failure stress of cartilage from the hip and talus. There is a statistically significant drop in the failure stress, as a function of age, for cartilage from the hip, but not for cartilage from the talus. Interestingly, there is a relatively high occurrence of osteoarthrosis in the hip compared with that in the ankle (talus).

CLINICAL RELEVANCE: INCIDENCE OF OSTEOARTHROSIS AT THE ANKLE

There is a low incidence of osteoarthrosis in the ankle compared with the hip or knee. The maintenance of tensile strength of cartilage from the ankle may play a role in the reduced likelihood of degeneration in this joint.

Repeated tensile loading (fatigue) lowers the tensile strength of cartilage as it does in many other materials. As the peak tensile stress increases, the number of cycles to failure decreases (Fig. 5.11) [93–95]. For any value of peak stress, the number of cycles to failure is lower for cartilage from older than younger individuals.

Repeated compressive loads applied to the cartilage surface in situ also cause a decrease in tensile strength, if a sufficient number of load cycles are applied [53]. Following 64,800 cycles of compressive loading there is no change in the tensile strength of cartilage, but after 97,200 cycles, tensile strength is reduced significantly. Surface damage is not found in any sample. This shows that damage may be induced within the tissue before any signs of surface fibrillation are apparent.

Some caution must be exercised when interpreting the results of tests in which a large strain is applied to cause failure of samples removed from the joint. The strain to failure may be greater than that experienced in vivo. In addition, the properties of most biological materials change with the applied strain; the collagen network becomes aligned with the direction of the tensile strain, and the material becomes strongly anisotropic.

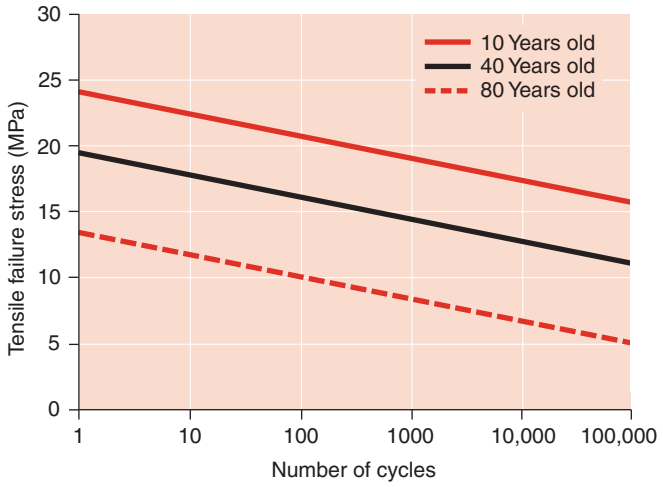

Figure 5.11: The effects of repeated tensile loading on the tensile strength of cartilage. As the tensile loading stress increases, fewer cycles of loading are needed to cause failure. Age is also an important factor. Cartilage from older individuals fails at a lower stress than that from younger people. Regression lines fit to multiple tests are plotted.

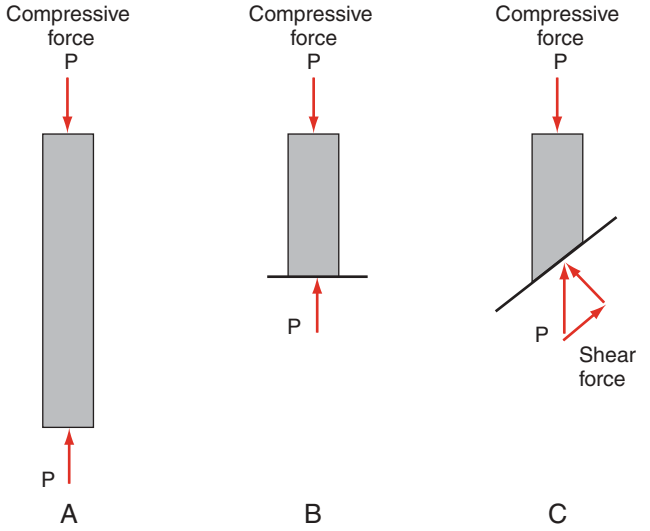

Figure 5.12: Illustration of shear stress in a simple loading condition. **A.** A free body diagram of a bar loaded in compression. **B.** A free body diagram of the same bar cut perpendicular to the load at an arbitrary location. On the cut surface, the resultant force must be *P* to maintain equilibrium. **C.** The same bar cut at an arbitrary angle. Again, to be in equilibrium the resultant force parallel to the bar must be equal to *P*. This force can always be decomposed into components parallel and perpendicular to the cut. The component parallel to the cut is a shear force that gives rise to a shear stress on the inclined surface.

Rather than assume that tensile stress is responsible for fibrillation of the articular surface, the feasibility of several criteria is considered in a combined experimental and computational approach to cartilage failure [3–5]. Dropping three different-sized spherical indenters (2, 4, and 8 mm) onto the articular surface produces three different states of stress and, in some instances, a crack through the surface. Based on the stresses in the cartilage in each test and the presence or absence of a crack, a regression is used to determine the condition that is most likely to cause a crack to develop. The maximum shear stress in the cartilage is the most likely predictor of crack formation based on the location of the crack with respect to the calculated stresses. Since cartilage is loaded in compression, the idea of failure by shear stress may seem unrealistic. Shear stresses do exist in cartilage, although the orientation of these stresses is not always obvious. To illustrate this, imagine a loading situation that is simpler than a joint, namely a straight bar loaded in compression (Fig. 5.12). If the bar is cut by a plane perpendicular to its length, then the resultant force on the cross section must also be compressive and equal to the applied force to maintain equilibrium. Now imagine the bar is cut at a 45° angle to its length (the exact angle is not important). The resultant force must still be equal to the applied force. Resolving the resultant force into components parallel and perpendicular to the cut surface gives rise to a shear force and a normal force. The shear stress (force per unit area) comes from the shear force acting over the inclined cut area of the bar. The same concept applies in any loading situation, including the cartilage in a synovial joint. However, in a synovial joint the stresses are multiaxial, not uniaxial as in the bar.

Radin and coworkers also show that cartilage failure could be induced by shear stress [69]. However, they are particularly interested in failure at the cartilage–bone interface, not the articular surface. Motivation for this investigation comes from postmortem studies that show cracks at the cartilage–bone interface and the recognition that under rapid loading, cartilage behaves as an incompressible elastic material, that is, its Poisson's ratio is 0.5. The relatively compliant, but incompressible

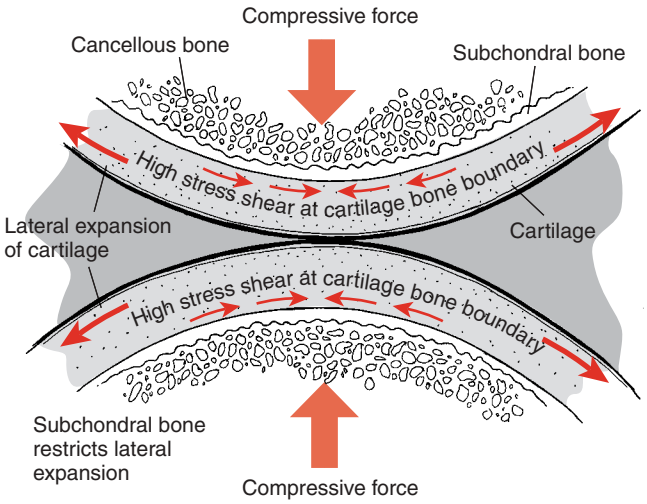

Figure 5.13: Under impulsive compressive loads, the cartilage experiences a relatively large lateral displacement due to its high Poisson's ratio. This expansion is restrained by the much stiffer subchondral bone, causing a high shear stress at the cartilage bone interface.

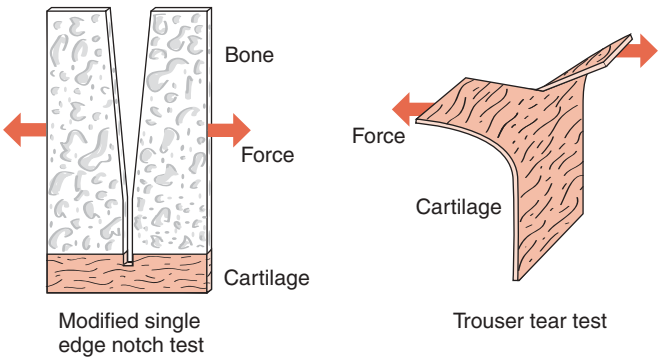

Figure 5.14: Sample shape and load application for the modified single-edge notch and trouser tear tests. Each test yields a specific measure of fracture, the energy required to propagate a crack in the material.

cartilage experiences large lateral displacement (due to its high Poisson's ratio) when loaded in compression, but this expansion is constrained by the stiff underlying bone (Fig. 5.13). Under these conditions, high shear stress develops at the cartilage–bone boundary.

Most studies of cartilage failure are based directly on the values of ultimate stress or strain. An alternative is to use parameters that more directly represent the propagation of a crack in a loaded material sample. The feasibility of using two methods to determine fracture parameters of cartilage is evaluated extensively by Chin-Purcell and Lewis (Fig. 5.14) [9]. The so-called J integral is a measure of the fracture energy dissipated per unit of crack extension. As used, the J integral also assumes that a crack propagates in the material, as opposed to deformation or flow of the material, which results in a more ductile failure. Since cracks may not propagate in soft biological materials, a tear test is also evaluated. The tear test yields a fracture parameter similar to the J integral. As with tensile-stress-based ideas of failure, it is necessary to apply large strains to cause failure of the samples: these strains may be far greater than those found in any in vivo loading conditions. To date, the application of these fracture parameters is limited to the normal canine patella.

JOINT LUBRICATION

Normal synovial joints operate with a relatively low coefficient of friction, about 0.001 [40,54,86]. For comparison, Teflon sliding on Teflon has a coefficient of friction of about 0.04, an order of magnitude higher than that for synovial joints. Identifying the mechanisms responsible for the low friction in synovial joints has been an area of ongoing research for decades. Both fluid film and boundary lubrication mechanisms have been investigated.

For a fluid film to lubricate moving surfaces effectively, it must be thicker than the roughness of the opposing surfaces. The thickness of the film depends on the viscosity of the fluid, the shape of the gap between the parts, and their relative velocity, as well as the stiffness of the surfaces. A low coefficient of friction can also be achieved without a fluid film through a mechanism known as boundary lubrication. In this case, molecules adhered to the surfaces are sheared rather than a fluid film.

It now appears that a combination of boundary lubrication (at low loads) and fluid film lubrication (at high loads) is responsible for the low friction in synovial joints [41,74,75]. This conclusion is based on several important observations. First, at low loads, synovial fluid is a better lubricant than buffer solution, but synovial fluid's lubricating ability does not depend on its viscosity. Digesting synovial fluid with hyaluronidase, which greatly reduces its viscosity, has no effect on friction. This shows that a fluid film is not the predominant lubrication mechanism, since viscosity is needed to generate a fluid film. In contrast, digesting the protein components in synovial fluid (which does not change its viscosity) causes the coefficient of friction to increase. This result suggests that boundary lubrication contributes to the overall lubrication of synovial joints. A glycoprotein that is effective as a boundary lubricant has been isolated from synovial fluid [84]. Newer evidence suggests that phospholipids may be important boundary lubricant molecules for articular cartilage [17,65,78]. At high loads, the coefficient of friction with synovial fluid increases, but there is no difference in friction between buffer and synovial fluid. This suggests that the boundary mechanism is less effective at high loads and that a fluid film is augmenting the lubrication process. Numerous mechanisms for developing this film have been postulated [12,28,48,54,89,91,92]. If cartilage is treated as a rigid material, it is not possible to generate a fluid film of sufficient thickness to separate the cartilage surface roughness. Treating the cartilage as a deformable material leads to a greater film thickness. This is known as *elastohydrodynamic lubrication:* the pressure in the fluid film causes the surfaces to deform. However, as the surfaces deform, the roughness on the surface also deforms and becomes smaller. Models, which include deformation of the cartilage and its surface roughness, have shown that a sufficiently thick film can be developed [28]. This is known as *microelastohydrodynamic lubrication*. Deformation also causes fluid flow across the cartilage surface, which modifies the film thickness, although there is some question as to the practical importance of flow across the surface [22,23,28].

MODELS OF OSTEOARTHROSIS

Animal models are used to provide a controlled environment for studying the progression of osteoarthrosis. Although osteoarthrosis may be induced by numerous means, models based on disruption of the mechanical environment of the joint, either by surgical alteration of periarticular structures or by abnormal joint load, are commonly used [24,25,57,66,72,73,81].

Surgical resection of one or combinations of the anterior cruciate ligament, the medial collateral ligament, and a partial medial meniscectomy produce osteoarthrosis of the knee. These models are thought to produce an unstable joint, but kinematic studies show varying degrees of deviation from normal joint kinematics.

Small differences in kinematics between control and operated knees (anterior cruciate ligament release and partial medial meniscectomy) are reported in rabbit [49] . At 4 weeks after surgery, there is a statistically significant change in the maximum anterior displacement of the knee, but anterior displacement is not significantly different from normal at 8 or 12 weeks after surgery. The most notable kinematic changes are in external rotation at 8 weeks and adduction at 4, 8, and 12 weeks after surgery. In dog, which has a more extended knee, greater anterior-posterior drawer is found after anterior (cranial) cruciate ligament release [36,88]. The relatively small changes in kinematics in unstable joints (particularly in rabbit) suggests that altered forces and possibly sensory input may be more important than joint displacements in the development of osteoarthrosis [29].

Repetitive impulse loading also produces osteoarthrosis in animal joints [70,72,73,81]. An advantage of this model is that it is more controlled than surgical models; the force applied to the limb is known and can be altered. This model has demonstrated the effect of loading rate on the development of osteoarthrosis. Impulsively applied loads were found to produce osteoarthrosis, while higher loads applied at a lower rate do not. The importance of impulsive loading to the development of osteoarthrosis also appears in humans; persons with knee pain, but no history to suggest its origin, load their legs more rapidly at heel strike than persons without knee pain.

Although biochemical, metabolic, and mechanical assays have been used to evaluate the properties of cartilage from animal models of osteoarthrosis, this chapter concentrates on the mechanical properties of cartilage. Following resection of the anterior cruciate ligament in dog, tensile stiffness, aggregate modulus, and shear modulus are lower than those in cartilage from unoperated control joints [79]. Permeability increases significantly 12 weeks after surgery. There is a significant increase in water content of samples from the medial tibial plateau and the lateral condyle and femoral groove.

In summary, various mechanical alterations of a joint lead to the development of osteoarthrosis. The kinematic instability induced by surgical alterations may be small, suggesting that altered forces are primarily responsible for the developing osteoarthrosis. Models based solely on abnormal joint loading support the view that alterations in force can lead to osteoarthrosis. Following resection of the anterior cruciate ligament, cartilage is less stiff in both compression and shear, and fluid flows more easily through the tissue in joints with osteoarthrosis. This implies greater displacement of osteoarthrotic cartilage than normal (decreased stiffness) and a greater rate of deformation (increased permeability).

CLINICAL RELEVANCE: OSTEOARTHROSIS

Osteoarthrosis is a leading cause of disability in developed countries [10]. In the United States, it is second to cardiovascular disease as the most common cause of disability [63]. Despite the widespread occurrence of osteoarthrosis, it is difficult to study in human populations. Early physical symptoms such as fibrillation and cracking of the articular surface cannot be detected by an individual, since cartilage is aneural. Insults to the cartilage may take years to progress to the point where symptoms are detected by the surrounding joint structures and underlying bone. Although numerous epidemiological studies of osteoarthrosis have been performed, they have been described as "disappointing," since they have not lead to an explanation of the mechanisms underlying the development of osteoarthrosis [63]. However, what seems to be clear is that the development of osteoarthrosis depends on a combination of factors including age, sex, heredity, joint mechanics, and cartilage biology and biochemistry [11,46,55].

Although it is not an inescapable consequence of aging, osteoarthrosis is more prevalent in the elderly [62,64]. In the United States, approximately 80% of people over the age of 65 and essentially everyone over the age of 80 has osteoarthrosis, although it is uncommon before the age of 40. After 55 years of age, osteoarthrosis is more common in women than in men. Typically the interphalangeal, first carpometacarpal and knees are the first joints that are affected [62]. However, specific links between aging and osteoarthrosis are not known. Excessive mechanical loading may also predispose joints to osteoarthrosis. Some studies have shown workers in physically strenuous occupations (coal miners) have a higher incidence of osteoarthrosis than those in less strenuous lines of work (office workers) [63]. Interestingly, osteoarthrosis of the shoulder and elbow have been found in relatively young individuals in ancient populations who depended on hunting [63]. However, strenuous work may not be the only risk factor for osteoarthrosis, since persons who use pneumatic drills or physical education teachers do not have an increased risk of osteoarthrosis [63].

Obesity has also been found to increase the risk of osteoarthrosis, particularly in the tibiofemoral, patellofemoral, and carpometacarpal joints [10]. Although increased weight would be expected to increase the load on joints of the lower extremity and possibly predispose an individual to osteoarthrosis, obesity would have no direct mechanical effect on the carpometacarpal joint.

Injuries to the anterior cruciate ligament, collateral ligament, or meniscus have been implicated in the development of osteoarthrosis in the knee [39]. Loss of the anterior cruciate ligament may impair sensory function and protective mechanisms at the knee. Disruption of internal joint structures may alter joint alignment and the areas of

cartilage that are loaded. If ligament damage results in a loss of joint stability, then joint loads may be increased by active muscle contraction trying to stabilize the joint. Partial or total meniscectomy can also be expected to increase the stress on the joint, since the joint force is concentrated over a smaller area [90].

While there appears to be an increased risk of osteoarthrosis in situations that entail abnormal or excessive loading this is clearly not universal. Radin has argued that it is not the magnitude of the load, but the loading rate that is the determining factor in the development of osteoarthrosis. Osteoarthrosis develops only when impulsive loads are applied; that is, the load reaches its maximum value over a relatively short time. This has been clearly demonstrated in animal models by use of externally applied loads and in sheep walking on soft and hard surfaces [67,71–73,75,81]. The role of impulsive rather than more-slowly applied loads is also supported by tests in humans. Individuals with knee pain who are diagnosed as "prearthrotic" have a higher loading rate at heel strike than normal subjects [69]. These studies suggest that particular activities alone do not necessarily predispose an individual to osteoarthrosis. Rather, the way in which the activity is performed may be the factor that determines if osteoarthrosis will develop.

SUMMARY

In summary, articular cartilage provides an efficient load-bearing surface for synovial joints that is capable of functioning for the lifetime of an individual. The mechanical behavior of this tissue depends on the interaction of its fluid and solid components. Numerous factors can impair the function of cartilage and lead to osteoarthrosis and a painful and nonfunctional joint. Mechanical factors are strongly implicated in the development of osteoarthrosis, although exact mechanisms are still not known.

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