

CHAPTER 2

LITERATURE REVIEW

2.1 SYNOVIAL JOINTS

A diarthrodial joint is an encapsulated system which encloses its articulating surfaces and lubricant. The end of each bone is covered with a protective layer of articular cartilage, which serves to reduce contact stresses in the joint, protect bone surfaces from impact stresses, and minimize friction and wear in the joint [6]. The natural lubricant, called synovial fluid, is a clear, viscous fluid which serves three purposes: it lubricates the articulating surfaces, carries nutrients to the cartilage cells, or chondrocytes, and transports waste products away from the cartilage. The synovial membrane, which surrounds the joint, serves several purposes: it regulates the amount and content of the synovial fluid, it removes waste materials from the synovial fluid and allows nutrients to enter the synovial capsule, and it secretes synovial fluid and other macromolecules for lubrication of the joint [7]. A diagram of a simplified synovial joint is shown in Figure 2.1.

Cartilage is a complex material consisting of both solid and fluid components. The solid portion is composed primarily of a network of collagen fibers and brush-like proteoglycan molecules. This network traps water in the material and stores it as a gel; this gel becomes pressurized upon application of a load to the joint, and enables the cartilage to support relatively high loads. [8, 9] In addition to providing the framework for the material, the collagen network provides an ideal surface for sliding. Cartilage is more flexible than the subchondral bone that supports it, and is therefore well suited for padding the bone surfaces to reduce contact and impact stresses.

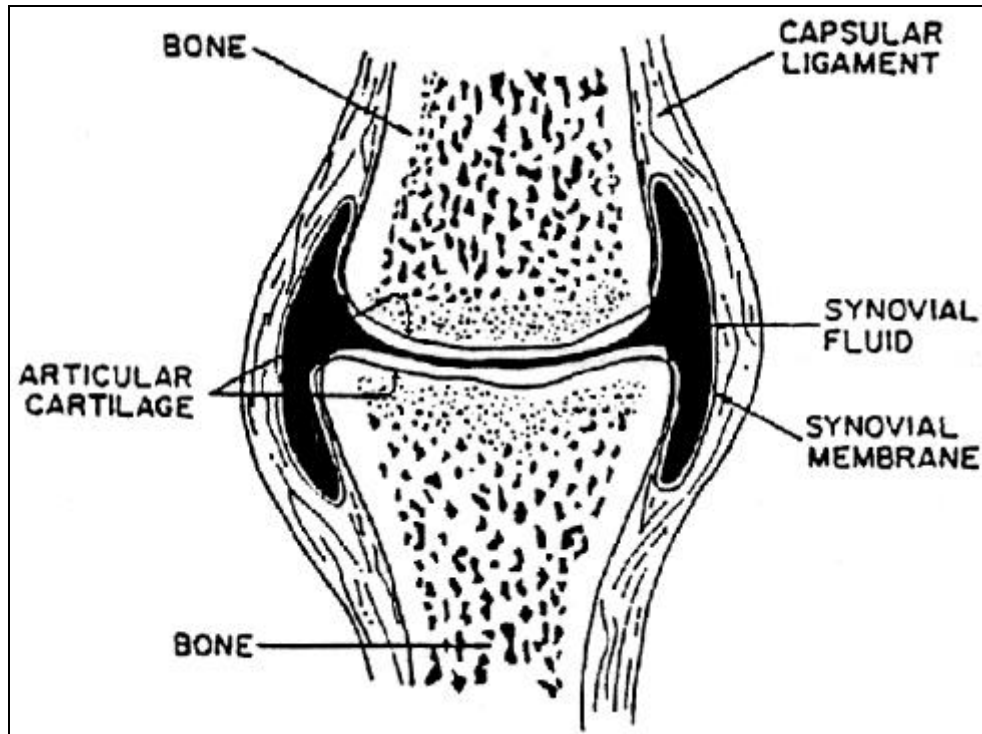


Figure 2.1: A Synovial Joint

Cartilage is unique among body tissues for several reasons. Cartilage cells, or chondrocytes, are farther from their supporting blood supply than any other cells in the body. Although cell nutrition mechanisms vary from children to adults, adult chondrocytes rely entirely on the circulation of synovial fluid to provide nutrients necessary for survival. According to Marcinko, “The [solid portion of] articular cartilage of pedal synovial joints is predominantly type II collagen and this collagen tends to be aneural, alymphatic, and avascular, receiving its nourishment from surrounding synovial fluids.” [8] The compression and decompression cycles involved in walking and other normal activity causes circulation of fluid-borne nutrients to the interior of the cartilage tissue. As a result, loss of mobility due to osteoarthritis can have serious effects on the health of chondrocytes. Another unusual characteristic of this tissue is that roughly 90% of the dry weight of adult articular cartilage is Type II collagen; this type of collagen exists in only minute proportions in other body tissues [10].

Cartilage is divided into four zones, based on the orientation of fibers and location and dispersion of chondrocytes within the material. Figure 2.2 illustrates the organization of these layers. At the surface, collagen fibers run parallel to the articulating surface of the joint, and chondrocytes are closely spaced and aligned with the cartilage surface. This region is designated Zone I, or the superficial tangential zone. In this zone, the collagen fibers serve mainly to support the tensile stresses generated when compressive loads are applied to the cartilage. In Zone II, the transitional intermediate zone, collagen fibers are randomly oriented and chondrocytes are randomly dispersed. In Zone III, the deep radial zone, the collagen fibers project radially from the bone; the chondrocytes exist as rows of

cells parallel to the collagen fibers. The calcified zone, Zone IV, is the region that connects the cartilage to the subchondral bone. Fibers nearer the bone are progressively more mineralized, and the cartilage and bone are interlaced in an interlocking mesh [8].

Research by Stachowiak [11] supports the concept of a low-friction, wear-resistant surface layer. This region, in which the collagen fibers run parallel to the cartilage surface, has been shown to possess a high resistance to wear. This superficial zone also provides a smooth articulating surface, thereby allowing movement with as little friction as possible.

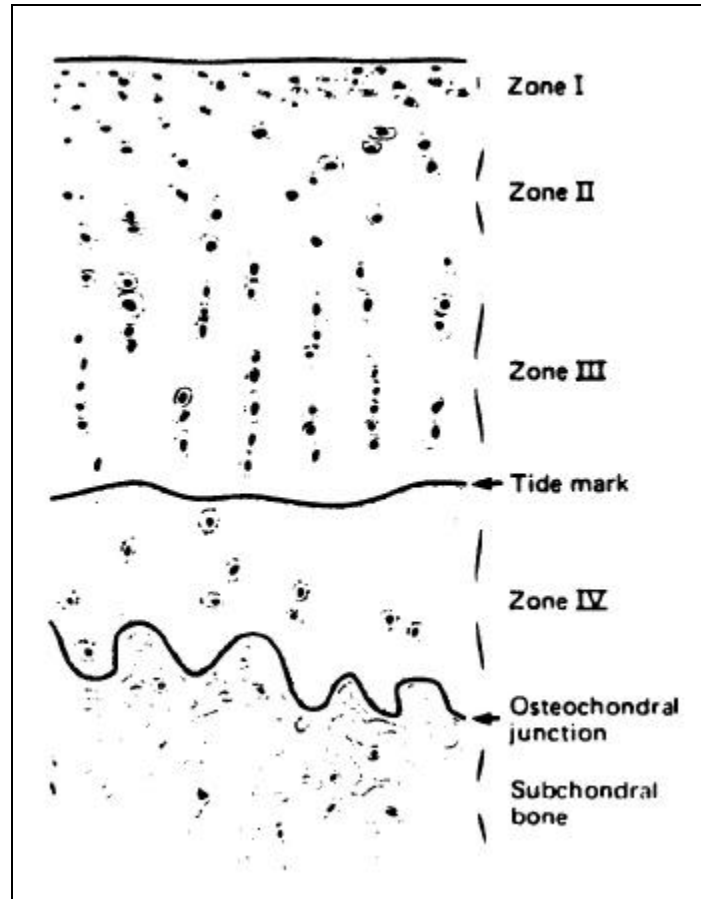


Figure 2.2: The Four Zones of Cartilage [8]

Cartilage has been shown to exhibit both viscoelastic and poroelastic mechanical properties. The viscoelastic properties of the tissue determine the rate of elastic deformation that occurs upon the application of any load. When the material is loaded, migration of water through the solid matrix causes the cartilage to deform slowly over time [12]. This property, known as poroelasticity, can cause the secretion of water that, according to the theory of weeping lubrication, pressurizes and supports the applied load.

Synovial fluid is a dialysate of blood plasma. The primary thickening agent of this lubricant is the polysaccharide chain molecule called hyaluronic acid (HA), or hyaluronan [13]. HA also acts as a partial filter, “excluding bacteria and invading inflammatory cells from the synovial space but allowing small nutrient molecules to move freely through the synovial fluid to cartilage from the capillary bed of the synovium.” [14] Hyaluronic acid treatments, in which HA was added to diseased or temporary immobilized joints in experiments by Keller [15], has been shown to reduce surface damage to some degree. Hyaluronic acid, therefore, may play an important role in both reducing damage to cartilage and aiding the nutrition of the chondrocytes.

2.2 JOINT LUBRICATION

Lubrication is defined as the process of adding a substance (solid, liquid, or gas) to reduce friction and/or wear at the interface between two surfaces in relative motion [16]. Synovial joints are lubricated by the viscous, non-Newtonian synovial fluid which is enclosed within the synovial capsule. At high rates of relative motion, the cartilage surfaces are separated by a fluid film in the hydrodynamic lubrication regime. In this regime, the viscosity of the lubricant tends to be the most important factor in determining friction and wear. At lower sliding speeds, however, the surfaces will come into contact in what is known as the boundary lubrication regime. Since the fluid film no longer supports the load and shear stresses at this lower sliding speed, wear and friction are of more concern in this regime; the biochemical properties of the lubricant are more likely to play a more important role in determining the friction and wear in the joint.

In the history of joint lubrication studies, researchers have proposed dozens of theories on joint lubrication, most based on friction measurements rather than wear. Many of the earlier theories were variations of hydrodynamic lubrication theories, which apply primarily in the case of high rates of relative motion. Hydrodynamic cartilage lubrication theories, including research by Yao [17], provide models for sliding conditions in which the surfaces come into minimal contact. McCutchen [12] proposed a theory in which pockets of fluid in the recesses of the cartilage surface pressurize and support the load, and are replenished by fluid from within the cartilage itself. This concept, called “weeping lubrication,” became one of the leading joint lubrication theories after its proposal in 1959. Another theory, known as “boosted lubrication,” introduced the idea that highly viscous accumulations of synovial fluid could accumulate in the surface pores of the cartilage, and separate the surfaces. Other major theories, such as Hlavacek’s squeeze film lubrication theory [18] and combinations of elastohydrodynamic and boundary lubrication, contributed to the many possible theories of joint lubrication. Mow et al. [19] developed a theory that considered several complex factors, including the dynamics of synovial fluid flow and its interaction with the cartilage surface. The history of several of these proposed theories is discussed by both Furey [16] and Unsworth [20].

2.3 JOINT WEAR AND DAMAGE

Cartilage wear has often been neglected in studies of both joint lubrication and the causes of osteoarthritis. In biological systems especially, however, friction and wear are not simply related phenomena [1,2]; low friction systems do not necessarily result in low levels of wear. To properly understand the behavior of a natural joint, one must consider both friction and wear.

Bergman et al. [21] and Lipshitz, et al. [22] utilized a method for measuring cartilage wear through biochemical analysis. Hydroxyproline is an amino acid that is produced in the hydrolysis of collagen. In the Lipshitz study, it was shown that the amount of hydroxyproline present in a wear sample could reliably indicate the mass of collagen in the sample. Since the proportion of hydroxyproline in hydrated cartilage remains constant at about 2%, the mass of the worn cartilage can be determined from the measured mass of hydroxyproline in each sample. This wear measurement technique has been used successfully by Furey and Schroeder [1,2,23], as well as in the present study, to measure cartilage wear from *in vitro* cartilage-on-cartilage and cartilage-on-stainless steel experiments.

The present study is undertaken to continue the ongoing research by Furey, which seeks to explore the “possible connections between tribology and the mechanisms of synovial joint lubrication as well as degeneration (e.g., osteoarthritis).” [2]. Furey’s work at the Children’s Hospital Medical Center at the Harvard Medical School in Boston investigated the connection between biochemistry and cartilage wear. In these experiments, which used a system of cartilage rubbing against stainless steel, the effects of various lubricants on cartilage wear were determined using biochemical wear analysis. The lubricants included a buffered saline reference fluid, synovial fluid, saline reference with hyaluronic acid added, reference fluid with Swann’s Lubricating Glycoprotein (LGP-I), and reference fluid with an isolated protein complex from synovial fluid. The wear results from this study are shown in Figure 2.3. It was found that synovial fluid reduced wear considerably over the reference fluid, while the hyaluronic acid produced less wear than the reference alone, but more than the bovine synovial fluid. Swann’s LGP-I, which has been shown to reduce friction in cartilage tests, produced somewhat more wear than the reference; the synovial fluid protein complex reduced wear slightly more than the synovial fluid.

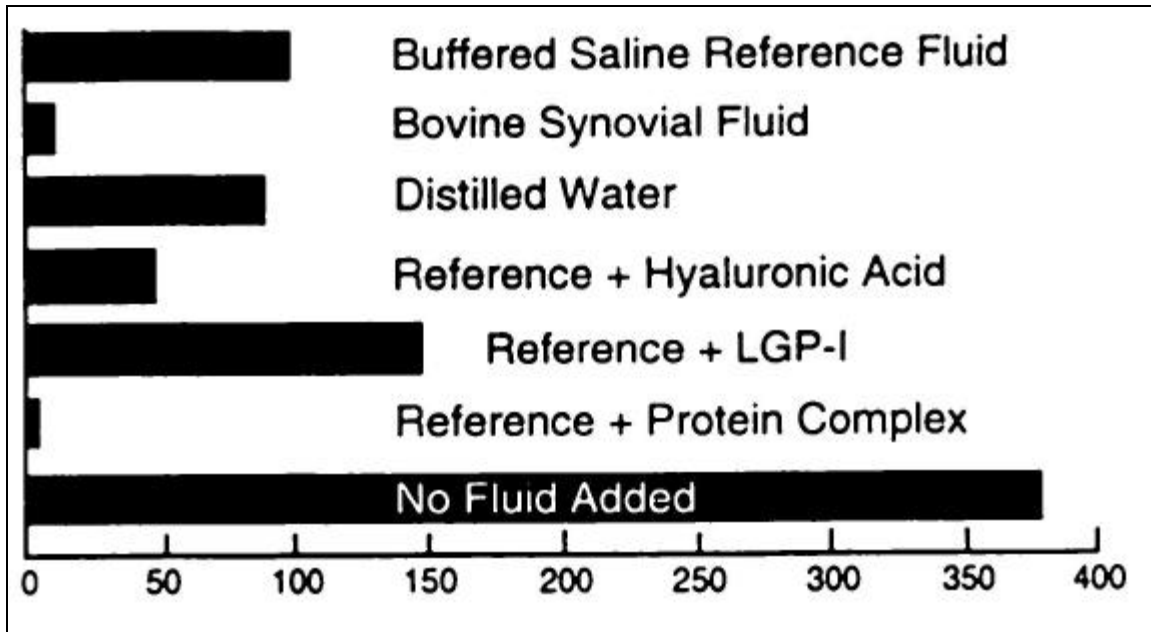


Figure 2.3: Relative Wear Results In Furey's Study [1]

In addition to these results, it was also determined that cartilage wear was linearly dependent on the number of sliding cycles in the test. Stachowiak's proposed protective surface layer was not evident in the wear data from these tests [11]; the superficial lubricating layer may have been worn away very rapidly during the beginning of the 6-hour test, or it might have remained entirely intact for the duration of the test.

One of the most significant findings of this research was that there is a relationship between the biochemical properties of the lubricants involved, and the cartilage wear and damage resulting from the tests.

Scanning electron microscopy (SEM) has been used by Furey [5], Schroeder [23], and others to observe and classify cartilage wear and damage. Clarke [24] concluded from high-magnification SEM photographs that the dead cartilage surface shows no visible difference from the living cartilage tissue. Clark [25] used this tool to demonstrate the differences in the surfaces and bulk material due to the location of the cartilage within the joint. In other studies, Chai and Pellegrini [26, 27] classified various stages of diseased cartilage degradation based on SEM photographs. SEM can be used to provide information about the specific characteristics of cartilage surface damage from sliding contact with stainless steel.

2.4 DEGENERATIVE JOINT DISEASE

The two most common forms of joint disease are rheumatoid arthritis and osteoarthritis. In joints afflicted by rheumatoid arthritis, the cartilage initially is damaged by enzymes released within the synovial capsule. This condition is chronic, and can result in capsular inflammation, pain, and eventually loss of mobility. Osteoarthritis (OA), which can be brought on by age or joint trauma, has traditionally been described as the “wear and tear” form of arthritis; the degeneration of the cartilage progresses slowly to rapidly throughout the joint, and can cause large quantities of cartilage to be worn away at an unusually high rate. OA can result in severe cartilage lesions, joint immobility, and bony growths in the joint. Although the specific causes of osteoarthritis are still unknown, several researchers have put forth theories regarding the causes and mechanisms of this type of joint degeneration.

Cartilage, like some other tissues in the body, is capable of repairing itself only in an extremely limited fashion. Damaged cartilage may be slowly repaired, but the resulting material is typically inferior in load-bearing capability to the original cartilage. “Cartilage response to injury is severely limited, in contrast to tendons and muscles, which usually heal with scar tissue.” [28]

Although osteoarthritis tends to be much more common among the elderly, joint trauma or various other factors can cause an early onset of degenerative joint disease. An instability in the joint – which could be caused by injury to the ligaments, meniscus, or cartilage, or by a structural deformity in the bone or joint – can result in “biochemical, metabolic, and mechanical property changes” that can begin the destructive cycle of osteoarthritis [6]. Early signs of osteoarthritis include cartilage fibrillation and increased hydration, thickening of the walls of the synovial capsule, and biochemical changes in the proteoglycans produced in the joint [6].

Sokoloff suggests that osteoarthritis may be an exaggerated state of the natural remodeling process within the joint [29]. Applied stresses to the cartilage result in changes in the geometry of the surface; repetitive loading may result in removal of material in some areas and growth of new material in others by natural remodeling processes. In many instances, total cartilage mass and proteoglycan production have been shown to increase during the early stages of osteoarthritis [30].

Osteoarthritic degeneration progresses through four distinct phases as the cartilage damage becomes more severe; these grades of degradation are explained by Marcinko [8]. In Grade I, surface and subsurface damage are minor, and limited to small fissures and pits. The damage can be observed only at points of highest stress, and the rest of the joint functions normally. In Grade II, more severe cartilage damage can be seen, though the damage is still confined to the areas of greatest loading. Some cartilage loss can occur in this stage. Grade III of the degradation marks the complete loss of cartilage in heavily loaded areas, and possibly the formation of bony growths. Pain in the joint would typically begin during this stage. Grade IV is the most severe level of degradation; in this

stage, large areas of bone may be completely exposed. The surfaces of the bone can become misshapen, and the articular surfaces become irregular [8].

Few treatments exist for osteoarthritis patients. Part of the difficulty in treating OA result from the difficulty in diagnosing the disease early; the pain and stiffness associated with later stages (Grades III and IV) of the disease are not generally evident until much cartilage damage has occurred. In less severe cases, a doctor may choose to remove a portion of the affected cartilage to prevent a removed cartilage flap from being released into the joint. In more serious cases of osteoarthritis, partial or total joint replacement may be required. Joint replacement is a treatment typically reserved for patients over 55 years of age, and is undertaken when no other measures can be used to save or prolong the life of the joint.

In a partial joint replacement, one of the articulating surfaces of the joint is surgically removed and replaced with an approximate artificial replica. In total joint replacements, both articulating surfaces are removed. The prosthesis design is usually chosen from a library of geometries, then modified to fit the specific patient. Several three-dimensional computer modeling tools are available to assist in the design and automated fabrication of joint replacement prostheses.

When all or a portion of a joint is replaced, a portion of the patient's bone must be cut away; the prosthesis is then mounted or cemented in place on the truncated end of the bone. In ball-socket joints, the ball end is usually replaced with a metallic replacement, such as a titanium alloy, a Co-Cr-Mo alloy, stainless steel, or a ceramic (e.g., alumina). The socket is typically replaced with ultra-high molecular weight polyethylene (UHMWPE) [32].

In an attempt to find a substitute for the extreme measure of joint replacement, several researchers have experimented with replacing patches of damaged cartilage with new cartilage grown from the patient's own cells in the laboratory. This technique has been used with some success on patients with minor cartilage damage, but the procedure is not a viable solution for most cases of degenerative joint disease [33].

Although new research continues to provide more information about the causes and mechanisms of osteoarthritis, the roles of tribology and biochemistry in the process of joint degradation must be more clearly understood before the disease can be adequately prevented or cured. Ultimately, preventative measures could become available that may force joint replacements and other such radical procedures into obsolescence.