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The temporomandibular joint disk in health and disease: A review

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Abstract:
Introduction: *The articular disk is the most important anatomic structure in the temporomandibular joint. It is constituted primarily by collagens and elastic fibers, peripheral nerve endings, cells and extracellular matrix. Studies describing the status of the joint disk anatomically and histologically are extremely scarce.* ***Goal:*** *Use the current literature to describe the anatomic and functional status of the joint disc in health and disease.* ***Methods:*** *The descriptors joint disk, temporomandibular joint, disk displacement, lubrication, osteoarthritis, and inflammation were entered into Google Academics database in order to search scientific papers of interest to carry out the investigation. Only papers written in the English language reviews and experimental studies were accepted, evaluated, summarized and included.* ***Outcome:*** *Sixty papers (n=60) were found but using the criteria of "insufficient information, papers about a different subject and papers written in another language", nineteen (n=19) papers were excluded. Thus, forty-one papers were used, analyzed and summarized to carry out the current investigation.* ***Conclusion:*** *The temporomandibular joint disc is made up mainly of fibrocartilage, collagen, elastic, elauinin and oxytalamic fibers with many fibroblast or chondrocyte-like cells. The periphery of the joint disc is lubricated by molecules of hyaluronic acid and lubricin. Most common cause of internal derangements of the temporomandibular joints is the presence of overloading from oral parafunctional behaviors. Disc displacement with or without reduction facilitates the development of osteoarthritic signs and symptoms.*

Keywords: *Temporomandibular joint. Joint Disk. Disk Displacement. Lubrication. Overloading. Ostoarthritis.*

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I. Introduction

The Temporomandibular joint (TMJ), is a bilateral synovial articulation of the mandibular ramus and the mandibular fossa of the temporal bone[1]. The TMJ is a hinge joint that allows for normal opening and closing of the mandible, is able to perform both lateral and protrusive movements and is essential for the masticatory system to carry out the functions of swallowing, chewing, speech and even rest of the mandible[2]. The TMJ is also considered a diarthrodial joint that allows various degrees of relative motion of the bones produced by surrounding muscle forces[3]. The TMJ is formed by the squamous portion of the temporal bone and the condyle or mandibular head of the mandible[4]. Even though TMJ is well protected from injury, lesion or degeneration by biomechanical structures (the joint disk and discal elements), by neurophysiological mechanisms (nociceptors and mechanoreceptors) and by physical means (synovial membrane and lubrication molecules), the TMJ is prone to a variety of pathologic conditions that inhibit normal jaw functions, induce protective reflexes and cause signs and symptoms, suffering and frustration.

The most important protective anatomic element within the TMJs is the joint or articulating disc. The joint disc is a tissue made up mainly of fibrocartilage , biconcave and elliptical in shape presenting the superior zone that points to the mandibular fossa and the inferior zone that points to the head of the mandible. Disc shape allows a smooth articulation between the joint condyle and the articular eminence increasing the contact area between two opposing articulating surfaces[1]. The joint disc is composed of variables amounts of cells and extracellular matrix (ECM) which in turn is made up of macromolecules and fluid[3]. The TMJ disc made up chiefly of fibrous tissue, various amounts of rounded cartilage–like cells[5], being the (ECM) the most important component that provides biomechanical characteristics associated with protection of adjacent tissues.

Main functions of the TMJ disc include the absorption of compressive loads during jaw movements[6], it provides protection to the mandibular fossa and mandibular head and allows smooth and physiologic movements between the components of the TMJ. Another function of the joint disk is to prevent wear and anatomic deformation of the hard tissues. Even though the anatomy, function, and dynamic of the TMJ disk is

relatively well known, little is known about a set of biomechanical and pathological events related to malfunctioning and pathology of such structure. Thus, this investigation was undertaken to:

1.Describe the anatomy and function of the healthy and functional TMJ disk
2.Discuss the lubrication of the joint disk in normal and pathologic conditions
3.Characterize the TMJ disk in pathological conditions including loading, lubrication and displacement.
4.Characterize the osteoarthritic disc and TMJ.

II. Methods

Research terms such as TMJs, overloading, TMJ lubrication, TMJ disc, disc displacement, anatomy and osteoarthritis were entered in the database www.google.com in order to find papers of interest to carry out the current investigation. Inclusion criteria to include papers to carry out this study papers sufficient information about the topics of interest (for instance overloading in the TMJs, or TMJ lubrication) and papers written in the English language. Literature reviews and experimental studies,were included in the current study. Papers written in other languages, insufficient information and information not published in a scientific journal were criteria used to exclude papers in this investigation. Both classic (anatomy) and recent papers about the subjects of this investigation were accepted.

III. Outcome

Using the MeSH terms mentioned previously, 60 papers were found and considered sufficient to cover all the topics of interest delineated in the current investigation. However, because of insufficient information and use of another language to write the paper, nineteen (n=19) articles were not accepted to prepare the current review. Papers included and accepted to carry out the current study were those that reviewed various topics including TMJ anatomy, joint disc and extracellular matrix, joint disc, cartilage, extracellular matrix, loading and lubrication of the TMJs.

IV. Literature review

Descriptive anatomy of the normal TMJ disk: The interarticular TMJ disc is the structure interposed between the head of the mandible and the mandibular fossa of the temporal bone. It runs anteriorly, posteriorly and laterally to protect and guide jaw movements during function. The TMJ disc is made up of fibrocartilage tissue and probably has a different ultrastructure in the anterior, intermediate and posterior bands which form the TMJ disc. Even though the joint disc is formed by a dense network of collagen fibers, such component is surrounded by delicate anatomic structures including the synovial membrane, collateral ligaments, joint capsule and its innervation and blood supply. The anterior portion of the articular disk attaches superiorly to the articular tubercle by bending with the joint capsule and inferiorly to the condyle and the upper area of the upper pterygoid muscle[1]. The joint disk is formed by the anterior band (thinner than the central and posterior band), intermediate band (the thinnest of the three bands of the joint disc) and the posterior band (the thickest band of the disc) which connects with the retrodiscal tissues. The TMJ disc is composed of variable amounts of cells and ECM constituted by macromolecules and fluid. Various types of collagen and proteoglycans are the most common molecules found in the joint disk whereas cells including fibrochondrocytyes and fibroblast-like cells can be found scattered more frequently in the periphery of the joint disc[3].

Even though collagens fibers are the main components of the joint disk, other fiber types can also be observed. Gross and colleagues[7], evaluated the localization and volume of elastic fibers in 12 articular discs removed from 12 subjects presenting with TMJ internal derangements (TMJ-IDs) and reported that elastic fibers were found predominantly in the posterior area in the transition zone from the disc to the bilaminar zone. They also reported that the anterior band is more rich in elastic fibers when compared to the intermediate and posterior zone of the disc. It is apparent that most elastic fibers in the TMJ are found in the bilaminar zone. Anteriorly, posteriorly, laterally and medially the joint disc is connected to other anatomic structures through the posterior, anterior, lateral and medial ligaments which provide stabilization and coordination to the movements of the joint disc during function. In normal conditions the movements of the condyle on the lower portion of the joint disc are smooth, coordinated and with a minimum of friction as the surfaces of the joint disc are protected with hyaluronic acid (HA) and lubricin determining a low friction coefficient.

Diskal attachments: As mentioned before, the joint disk is stabilized by six internal or collateral ligaments. The anterior portion of the articular disc attaches superiorly to the articular eminence at the same time that bends with the joint capsule. Inferiorly, the joint disc attaches to the anterior and superior portion of the joint condyle and upper part of the superior lateral pterygoid muscle. Posteriorly, the superior and posterior discal ligament attaches to the upper portion of the posterior zone of the disc, posterior fossa and squamous tympanic suture whereas the inferior and posterior ligament attaches to the posterior and inferior region of the posterior band of the disc and to the neck of the condyle[1]. Laterally and medially the joint disc attaches to both the joint capsule

and to the mandibular head[8]. The bilaminar zone is the anatomic area located in the posterior and inferior part of the disk which contains the superior and posterior discal ligament and the inferior and posterior ligament. Both ligaments are rich in collagen but superior ligament has a higher content of collagen. Loose connective tissue can be found ubiquitously in between these two lamina or ligaments[8]. Anastasi and colleagues[1] believe that in fact both lateral ligaments have their origin as projections of fibers from the joint disc which then attaches both to the joint capsule and to the mandibular head laterally and medially. They claim that such structures should be considered as true discal ligaments. Willard and colleagues[2] defend the notion that the discal attachment show many similarities with the TMJ disc itself. They both contain the same basic components, that is, collagen, GAGs, cells and elastin. Further, much of the ECM is continuous between the tissues blending seamlessly together[2]. Microscopically, collateral ligaments can be seen as forming part of the joint disc, since their fibers originate directly from the lateral and medial regions of the articular disc[1].

The ECM of the joint disk. The ECM is defined as the set of cells and the ground substance of a tissue. The three major components of the fibrocartilage matrix of the joint disc are the collagenous component, large amounts of scaterred large and small chains of proteoglycans and water or fluid[9]. At the molecular level, the TMJ is characterized by a specialized and uniform distribution of components of the ECM. Collagen fibers are not necessarily distributed in a parallel fashion with each other as such characteristic could decrease resistance to compression and deformation. Rather, fibers are arranged in both longitudinal and transverse directions in order to form a compact fibrous network and increase the capacity for absorption of water, deformation and resistance to compression.

Proteoglycans are enmeshed in the network of collagen fibers with some or none motility. Proteoglycans constitute a large family of molecules found in the ECM of the tissues. In the case of the TMJ disc, most common proteoglycans molecules of this family include biglycan, decorin, aggrecan and chondroitin dermatan sulfate[10]. The molecules in this fibrous arrangement together with the collagen, provide resistance to stretch, flexibility, capacity for deformation along the long axes of the fibers. Proteoglycans are widely distributed in both the TMJ disc and joint cartilage. They can be found in areas where resistance to tensile forces is needed. Further, proteoglycans with highly sulfated glycosaminoglycans side-chains are primarily found in areas undergoing compressive loading[11]. Proteoglycans in the TMJ disc and cartilage are responsible for a set of positive properties of the fibrocartilage including stability, deformation, tensile strength, viscosity andretention of water.

Cells in the human and normal TMJ disk. Regarding the cellular component in the TMJ disk, it seems apparent that a small amount of cells is found distributed in areas in which pressure is not applied on the joint disc. There has been a long debate about type of cells in the joint disc. There is scarcity of studies showing localization, full description and type of cells that can be found in the joint disk. In very young rats the existence of chondrocytes surrounded by type II collagen has been described previously[12]. Berkovitz and Pacey[12] demonstrated the presence of cartilage–like cells when they studied the interarticular disk in rats and marmosets. Fibrocartilage cells live somewhere in between the collagen fibers of the joint disc. Since the joint disc is neither innervated nor vascularized, fibrocartilage cells receive nourishment from the adjacent synovial membrane and fluid[3]. Cells in the articular disc are a heterogeneous combination of fibrochondrocytes and fibroblast–like cells morphologically different from chondrocytes found in hyaline cartilage[3].

In a similar study, Berkovitz and Pacey[5] evaluated the ultra structural appearance of surgically removed human intra-articular discs form three female subjects from central areas of the joint disc. They reported that "despite the large number of cells examined, only one cell showed evidence of chondrocyte-like morphology". The human TMJ inter articular disc is formed mainly by fibrous tissue, containing variable amounts of rounded cartilage-like cells[13]. The posterior, anterior, lateral and medial attachments of the joint disc are not cartilaginous as the joint disc. Thus, cells in this region are predominantly fibroblasts[14].

Synovial membrane and synovial fluid: The synovial membrane lines and protects the inner surface of the joint capsule. This TMJ component forms part of the immune system and provides nourishment for adjacent tissues including joint disk, capsule and collateral ligaments. The capsule covers all the intra-articular surfaces except those pressure–bearing areas of the cartilage[15]. It contains specialized cell types with phagocytic and immune capacity and produces the synovial fluid that provides the nutritional, metabolic and defensive requirements of the avascular tissue of the joint disc, mandibular condyle and articular eminence fibrocartilage[3]. Water and many molecules including hyaluronic acid (HA) and lubricin can be found in the synovial fluid. These molecules are pushed in the direction of the joint disc both medially and laterally, preventing direct contact between the articulating parts and thus protecting disc, mandibular head and mandibular fossa surfaces and decreasing by coefficient of friction.

The TMJ synovial membrane has some distinct histological, anatomic and pathological characteristics that differentiate it from joint membranes. For instance, it serves as a reservoir of phagocytic cells, it allows exchange of material between the TMJ and blood vessels, it nourishes the TMJ surfaces and is the source of both lubricin and HA. In pathologic conditions large amounts of some inflammatory mediators including IL-1

beta, TNF-alpha and serotonine can be found in the synovial fluid of TMJs with internal derangements (TMJs-ID)[16].

HA provides some biomechanical properties of the synovial fluid, notably the viscosity characteristic[10], thus, its presence its essential to protect the articulating surfaces of any joint. Excessive tension, compression, distension or direct trauma to the joint capsule and synovial membrane may alter the biochemical properties of the synovial fluid and thus may contribute to pain and dysfunction (capulitis and synovitis of the TMJ). Because the synovial membrane and adjacent joint capsule relates both anatomically and functionally to each other, it is very difficult if not impossible to clinically differentiate capsulitis from synovitis. Histologically, the synovial membrane consist of intimal and subintimal layers. Two types of cells can be found in the synovial membrane: fibroblasts are cells of the connective tissue that secretes collagen and proteoglycans of the synovial fluid (type B cells), whereas macrophage-like cells are specialized in phagocytosis, that is, digestion of debris, residues, cells and are referred to as (A cells)[17]. Changes in the composition of the synovial fluid are closely associated with pain, inflammation, destruction of the joint surfaces, cell death, excessive friction, degradation of cartilage and sometimes with the phenomenon of the "stuck disc". These changes increase the intra-articular friction, leading to unstable disc motion, biochemical alterations, impaired lubrication, abnormal protective mechanisms and increased coefficient of friction in the articular surfaces[4].

Normal versus pathological lubrication of the joint disk and condyle. As mentioned before, the synovial membrane is an important joint component which contributes with nourish and provides molecules that function lubricating the articulating joint surfaces including the joint disc and condyle. This is so, as those surfaces are noninnervated and non vascularized and need blood supply and nutrients from an independent source (synovial membrane). Another interesting property of the synovial fluid is the bactericidal capacity[18] probably associated with its intrinsic components and/or with the presence of phagocytic cells, for instance, macrophages and monocytes. In normal physiologic conditions, movements of the condyle and disc assembly are smooth, without joint noises, coordinated and without friction or pain. This is so, as some layers of high molecular weight molecules from the synovial fluid are interposed between the joint disc and mandibular fossa and between the joint disk and mandibular head, significantly reducing the friction coefficient of the articulating surfaces.

The synovial fluid is a viscous gel and contains mostly water. This material elaborated by some specialized cells in the synovial membrane, functions as a lubricant in the upper and lower compartment of the TMJ. Cells known as synoviocites in the synovial membrane have phagocytic and immunologic capacity and produce the synovial fluid which in normal conditions provides viscosity, nutrition, lubrication and interchange of substances between the external and internal milieu in the TMJs. In the synovial fluid of the TMJ. HA is produced by type B synovial cells being the most important molecule reducing friction in the articulating surfaces. Most researchers agree that the durability of a joint depends primarily on their ability to withstand repetitive loading and articulate with low friction, resulting in smooth and coordinate joint movements. Another essential molecule in the TMJ lubrication mechanism is a mucinous glycoprotein known as lubricin. This protein, found in the synovial fluid, provides boundary lubrication of articular surfaces under high contact pressure and quite low sliding speed[10]. Another essential molecule present in the synovial fluid is the surface active phospholipid or (SAPL) which is responsible for the boundary lubrication of the articular cartilage surface by reducing the kinetic friction[10]. Local inflammation and osteoarthritis in TMJ-ID patients, impair the capacity of the synovial membrane to produce synovial fluid. Inflammation of the synovial membrane is associated with the presence of IL-1beta, tumor necrosis factor alpha, serotonine, prostaglandin and probably histamine and bradykinin. Chronic inflammatory conditions in the TMJs cause changes in the composition of the synovial fluid which in turn increase the intra-articular friction among all main components of the TMJs, leading to unstable disc position and movements during function. These changes dramatically affect the joint lubrication mechanisms and nutritional requirements of the articular surfaces[4].

Intense parafunctional behaviors may alter the molecular composition and biomechanical properties of the synovial fluid[4] and thus, the capacity of the joint disk and cartilage to receive proper blood supply, proteins and nourishment. Destructive oral jaw habits may, indirectly contribute to increase the superficial roughness of the mandibular head, joint disc and mandibular fossa. Roughness, a higher friction coefficient and abnormal viscoelastic properties of the synovial fluid may directly contribute to the degradation and destruction of articular surfaces setting the stage for the development of OA. The development of OA signs, symptoms and degenerative characteristics occur when the capacity of repair, remodeling and neoformation is surpassed. Deterioration is most likely to occur in cases of physical injuries, a failed/traumatic surgery and in the presence of frequent, constant and intense parafunctional behaviors. Prolonged and intense loading may overcome the biomechanical limits of the joint tissues, leading to inflammation, deterioration, perforation, roughness, increased friction and deformation.

Overloading impairs the lubrication capacity of the TMJ. When the loading pressure exceeds the capillary perfusion pressure, there is a decrease in blood flow causing hypoxia in the TMJ tissues. Poor oxygen concentration stimulates the production of reactive oxygen species (ROS) which in turn inhibits the biosynthesis of HA and degrades, thus, directly impairing the capability of the joint to resist friction during jaw movements.[10] Another essential mechanism deteriorating joint surfaces is compression from overloading which increases the metabolic activity of chrondrocytes and irreversibly degrades joint cartilage[19]. A major effect of ROS in synovial joints is inhibition of the biosynthesis and degradation of HA, causing a marked reduction in the viscosity of the synovial fluid [20].

Properties of the normal joint disk: As mentioned before the joint disks is a complex structure made up mainly of fibrous cartilage, collagen, elastic, elauinin and oxytalan fibers with the predominance of a number of molecules of the ECM. The complexity of the collagen tissue and other fibers together with some molecules of the ECM plus the synovial fluid and other anatomic components of the TMJs endow the TMJ disk with a number of properties including:

1.Protective function: The joint disk is protected in both inter articular spaces by the synovial fluid and the molecules of HA and lubricin which reduce the coefficient of friction. The combination of a tridimensional arrangement of the collagen network plus HA and lubricin, allows the mandibular head to slide freely and smoothly on the lower side of the joint disc, preventing direct contact and friction in the articulating surfaces. Thus, it is the synovial fluid that confers the disc its protective functions on the articulating surfaces.

2.Deformation: The dense network of collagens and elastic fibers in the joint disc maintains the shape of the disk whereas the molecule elastin is associated with restoration of shape during unloading[10], for instance, at the end of a mandibular movement during chewing.

3.Viscosity: Molecules more frequently found in histological studies of the joint disc includes proteglycan, biglycans, decorine, dermatan sulfate, keratan and aggrecan. However, of all these proteins, aggrecan molecules posses high viscosity and large molecular size that reduce the capacity to diffuse through the collagen network resulting in the retention of large amounts of water[21]. Thus, aggrecan contributes to the viscosity and retention of water in the extracellular matrix.

4.Compressive resistance: The fact that in histological investigations many of the cells of the TMJ disk have an oval rather that a flattened outline combined with the presence of ECM molecules, indicates that such anatomical configuration is typical of cells associated with tissues subjected to compression.

5.Stress distribution: Three-dimensional finite element analysis of the human TMJ disk demonstrate that stress and strain distribution, two properties of the joint disk, were located primarily in the intermediate zone of the articular disc with the highest values in the lateral part[22].

6.Stability: The TMJ is maintained in place by six internal ligaments (collateral ligaments) located anterioly, posterior, laterally and medially. These delicate and strategically located ligaments are a source of proprioception and provides finesse and stabilization to the movements of the joint disk during function.

7.The TMJ has a natural ability to adapt to biomechanical stress[23] which is a function of the biomechanical properties of the disc. However, this capacity is to a certain extent very limited as overloading from parafunctional behaviors may lead to disc displacement, pathological alterations in the concentration of synovial fluid, increased friction, pain and inflammation. In many cases, some TMJ tissues may succumb and deteriorate under the negative influence of parafunctional behaviors.

8. The disc is subjected to a great number of different loading conditions during mandibular movements and three types of loading can be observed: compression, tension and shear. In normal physiologic conditions, the TMJ is able to resist and adapt to these three types of loading[10]. This is so as the joint disc has enormous capacity for deformation in the anterior, posterior and lateral directions. Thus, the joint disc can resist compressive, tensional, stretching and shear loading[10] .

9.Other physical and biomechanical properties of the joint disk which depend on a non homogeneous distribution of macromolecules and fluid, include viscoelastic properties, stiffness, strength and permeability[4].

10. In one investigation[22] using biomechanical tests to assess the dynamic properties of the human TMJ disc, researchers reported that the disc showed strong viscoelastic properties. Further, the resistance against deformation and the shock absorbing capabilities were larger in the intermediate zone than in regions located more anteriorly and posteriorly. Their findings provide support for point of view that the intermediate zone is the predominantly loaded region of the joint disc.

Pathological loading in the joint disk. Most researchers have accepted the point of view that the TMJs are load bearing. However, the capacity to withstand loading is limited or is not infinite as Millam put it. It is for this reasons that parafunctional behaviors in patients with TMDs are also accepted as the most common etiological factor in craniomandibular disorders (CMDs). Parafunctional behaviors vary in frequency, duration, intensity and time (nighttime, daytime) of the day in which they occur. Further, probably more than 20

parafunctional behaviors have been reported by TMDs patients and a combination of them, for instance daytime and nighttime bruxing behavior, jutting the jaw forward, and nail, cheek and tongue biting, seems to be extremely deleterious to the components of the masticatory system.

It has been reported[24] that the severity of chronic pain reported by CMDs patients is proportional to the amount of loading and damage to discs, capsule and ligament of a joint. For instance, the magnitude of capsular stretch and also of the collateral ligaments that control disc coordination and stability, are closely related to the onset, intensity and frequency of chronic pain. Because clinical cases, diagnosis and etiological factors vary from patient to patient, in many clinical cases, the frequency and magnitude of loading is such that the most frequent patients´ response is chronic pain. The disc is subjected to a multitude of different loading regions during mandibular movements including compression, tension and shear or a combination of these 3 types of loading[10]. During compressive, tensional or shear loading, the disc undergoes a deformation while internal forces are produced within the tissue. The amount of deformation is proportional to the magnitude of strain on the joint disc[10]. Shear loading is the most important loading type from a biomechanical point of view as shear loading may lead to fatigue, damage, and irreversible deformation of the joint surfaces including the disc and may result in significant collapse in the joint lubrication system[25].

Chronic overloading of the TMJs may directly affect the disc, joint capsule, synovial membrane, ligaments and cartilage of the mandibular fossa and mandibular head. In this regard, one study[26] reviewing the current literature asserts that the major direct cause of mandibular condylar cartilage breakdown and the development of osteoarthritic (OA) changes is chronic overloading. Current models of severe disk displacement and degenerative joint disease accept the importance of mechanical loading and subsequent development of severe immune and inflammatory changes which ultimately result in inflammation, pain and TMJ-OA[27].
The application of intense loading to the joint may increase the percentage of cells synthesizing DNA, alter the types of proteins being synthesized, modify the rate of bone or cartilage formation and resorption, alter calcification and inhibit osteoblastic formation[9]. It has been demonstrated[24] that degenerative pathologies in the TMJs are initiated by a complex combination of applied mechanical loading and biological cascades within the joint tissues, including the activation of mediators of inflammation, the destruction of joint disc and cartilage and the participation of elements of the immune system, for instance, IL-Ib, Il-2, and TNF. Compressive mechanical stress affects glycosamine synthesis in the rat articular disk. Further, the application of intense loading results in a significant decrease in the concentration of HA which no longer protects and lubricates the joint and disc. Alterations in glycosaminoglycans (GAGS) can also be observed[28]. When mechanical tension of any type overcomes the adaptive capacity of the joint disc and cartilage, changes in the viscoelastic properties of the disc, significant alterations in the content of the synovial fluid, deformation of the joint disc, higher coefficient of friction and roughness in the TMJs can be observed[4].

Pathological lubrication of the joint disk: As mentioned before, the joint surfaces are lubricated by HA and lubricin from the synovial fluid. During jaw movements both proteins are pushed to the upper and lower surfaces of the joint disc and other joint structures so as to produce a "cushiom effect" protecting the articular surfaces from direct contact, one with the other and thus, decreasing significantly the friction effect. In pathological conditions, for instance, when the joint disc is displaced and deformed and/or when the consistency of the synovial fluid is significantly altered, the lubricating effect of these molecules disappear. Thus, with time the joint surfaces become rough and irregular, the joint disc is no longer protective, and initial changes indicating OA may be observed using proper imaging methods.

Chronic overloading and inflammation induce significant changes in the composition of the synovial fluid. This alteration may lead to increased intra-articular friction and abnormal movements of the joint disc. Further, joint lubrication and nutritional requirements of both the disc and adjacent cartilage are significantly impaired[4] Only high viscosity synovial fluid is able to form a thick film of the order of 1 um which is able to separate the articular surface of the cartilage from the inferior or superior part of the joint disc. If this film is lost by any reason, surface roughness increases, influencing the degree of friction and the coefficient of wear and favoring the development of osteoarthritic alterations in the TMJs. It has been reported that in two articulating surfaces, the coefficient of friction is higher in joints with a high coefficient of roughness[10].

In healthy conditions SAPLs coupled with HA, synergistically enhance the lubricating property of the TMJ articulating surfaces. However, the normal friction or lubricating coefficient in the TMJ cartilage may increase due to deterioration of the lubricating mechanism which primarily depends of the production and secretion of the synovial fluid[29]. Excessive loading from parafunctional behaviors may significantly alter the concentration and or quality of both HA and SAPLs, thus impairing the lubricating mechanism in the TMJs. Upon excessive loading, the composition of the lubricating medium may change because it mixes with water from the cartilaginous tissue when cartilage is excessively compressed during prolonged parafunctional behaviors. A combination of altered composition or absence of synovial fluid, combined with a displaced disc

with abnormal shape and function may extremely impairs and alters the surface of contact between synovial fluid, cartilage and joint disc, thus contributing to the development of TMJ-OA.

When the viscoelastic properties of the normal synovial fluids are lost, the disc has changed in shape and/or there is a lower concentration of synovial fluid, the characteristics of low friction and roughness coefficient, are lost, thus, the joint disc and the synovial fluid have no longer shock absorbing capabilities, paving the way for the development of OA signs and symptoms. It is very likely that a pathologic process in the synovial membrane itself and not necessarily in the joint disc be sufficient to impair the mechanism of a normal lubrication process in the TMJs. For instance, one study[30] reported some changes in the synovial membrane including proliferation of synovial villa and subsynovial tissue, inflammation and synovitis. These changes likely cause some pathological modifications that alter the production and or quality of synovial fluid. Further, mechanical trauma to the TMJ may cause free radicals production and some of these molecules may lead to damage of important molecules in the synovial membrane of the TMJ. For instance, the superoxide anion may degrade HA [31], thus impairing the concentration of this product in the synovial fluid increasing the coefficient of friction between the joint disk and mandibular head.

Definition of TMJ-Internal derangements: The terms internal derangements of the TMJs (TMJs-ID) were coined in the 1990´s, when clinical researchers perceived that TMJs signs and symptoms were relatively well defined, that they were related to disc pathology and that many signs and symptoms were characteristics of a specific joint disorder and thus could be diagnosed and treated. In those years, researchers observed that signs and symptoms of retrodiskal, disk-attachment pain, clinical characteristics of disk displacement without reduction and so on, could be diagnosed. In the next stage and with the development of MRI, the area of TMJs-IDs became a field of prolific both clinical and theoretical research.

TMJs-IDs are usually defined as abnormalities in the normal anatomic relationship between the joint disc, the mandibular fossa and the mandibular head, usually associated with inflammation, disc deformation and displacement and abnormal function of the mandible and associated adjacent musculature. The term "derangement" refers to an alteration in the normal pathways of motion of the TMJs that largely involves abnormal function of the articular disc[4]. Worthy of mentioning is the fact that there are some disorders including capsulitis/synovitis and retrodiscal pain that in many cases do not involve significant alterations in the position and form of the joint disk.

TMJ Internal derangements etiology: The etiology of TMJs-ID is usually multifactorial. Micro, macrotrauma and chronic and severe loading from parafunctional behaviors play a major role in the etiology of pathological events which ultimately produce signs and symptoms. Any event compressing, stretching and loading the anatomic components of the TMJs most frequently the disc, can result in pain, inflammation and disc displacement. Oral jaw habits occupy a special place in the development of signs and symptoms as they progressively overload the joint tissues whose capacity for adaptation and repair is very limited. Traumatic events cause stretching, tearing, displacement, or rupture of the joint disc, lateral ligaments or even capsule[4]. Destructive alterations initially in the soft tissues progress to a point that they pathologically alter the hard tissues. Changes in the composition and viscosity of the synovial fluid cause an abnormal relationship between disc and synovial fluid paving the way for the development of the "stuck disc" phenomenon.

TMJ Internal derangement characteristics: TMJs-ID are characterized by the presence of inflammation, disk displacement and deformation, biochemical alterations of the synovial fluid and abnormal jaw functions, for instance, impaired jaw movements. TMJs-ID are usually classified according to the degree of progression and clinical characteristics. In some clinical cases one disorder progresses to the next stage but this does not occur in all patients. The best classification system is the one that classifies TMJs-ID according to the clinical characteristics and degree of severity. Further, TMJs-ID precedes the development of OA changes[10].

Interesting to note is that every stage or clinical disorder, for instance stage one I or even disk-attachment pain (stage II) has at least one sign or symptom which is characteristic of that disorder, for instance, pain described as sharp or intermittent is a characteristic of disk-attachment pain, pain described as burning is a unique characteristic of TMJ-arthralgia, pain on manipulating the mandible in the centric relation position describes retrodiskal pain and so on. Acute or chronic inflammation is a major characteristic of TMJs-ID. There are probably more than 10 diagnostic categories of TMJs-ID. However, disk displacement with and without reduction have been studied extensively in the dental and medical literature. Anterior disc displacement is the most common disorder in patients with TMJs-ID[32].

Disk displacement with reduction: With the introduction of magnetic resonance imaging (MRI) as an important tool in the diagnosis and treatment of TMDs, it became clear that disc derangements are frequently associated with pain, inflammation and functional disorders of the TMJs. Additionally, it became clear that some degree of inflammation and even subtle disc displacement precede the development of more complicated TMJs-ID and adjacent musculature. Disk displacement with reduction is defined as disk displacement during the closing stage and normal disk position during opening. This stage may or may not be accompanied by single or reciprocal clicking. Depending of the severity of disk displacement, position and

shape of the joint disc, the type of joint noise, may vary significantly. When the articular disc becomes displaced anteriorly, there is excessive stretching of the retrodiscal tissue, which then bears repeated loading from the mandibular condyle which usually displaces posteriorly. Various types of adaptive and non adaptive reactions can be observed in this area including increased number of blood vessels, hypertrophy and fibrosis of the joint disc and formation of a false disc. In more severe cases, the formation of inflammatory fluid usually associated with chronic pain has been observed frequently.

Bedran and associates[32] evaluated the MRI scans of 1038 patients presenting with TMJs-ID. They found that 14,2% of the joints demonstrated disk displacement with reduction and a frequency of 20.6% disc displacement without reduction was found. Because the frequency of reciprocal clicking is a characteristic of subjects with disk attachment pain, the frequency of disk displacement with reduction is very high in this category of TMJ-IDs. One investigation[30] evaluated the histological characteristics of 17 surgically removed disks from subjects with TMJs-ID compared to 10 symptom free persons. Even though the specific diagnosis of TMJs-ID was not mentioned in the study, researchers found that the surgically removed disks were deformed and thicker than the normal disks. The surgical specimens also showed higher density of fibroblasts and vessels and several other histological characteristics that were not observed in the normal discs of subjects with no TMDs. Mills and colleagues[33] used a method for producing disc displacement in rabbits so as to simulate changes usually observed in subjects with TMJ-IDs. They found that disc displacement was associated with gross thickening of the posterior band, shortening of the disk in the anterior and posterior direction, flexure of the intermediate zone and loss of biconcave shape. Thus, there are reasons to believe that disc displacement is strongly correlation with disc deformation.

Disk displacement without reduction (DDW_oR): DDW_oR is the condition in which the joint disk has been displaced anteriorly or anteriorly and medially and does not return to its normal position during jaw closing. There are two types of DDW_oR, DDW_{oR} that occurs following severe trauma to the TMJ, for instance, during orthognatic surgery, prolonged and difficult extraction of a third molar, motor vehicle accident and or a blow to the face and DDW_oR associated with progressive disk displacement, pain and joint inflammation, accumulation of inflammatory products in the joint spaces and sudden difficulty to open the mouth. In both categories, the disc remains anteriorly, is completely deformed and nonfunctional, severe pain is usually present, and the joint disc does not return to its normal position even when pain is eliminated completely. We shall mention in this section, that not all cases presenting disc-attachment pain and retrodiscitis progress to DDW_oR. Reducing disc displacement can also remain constant for many years, indicating that the clicking joint does not necessarily progresses to a derangement characterized by transient or permanent jaw locking[4]

In DDW_oR in which the joint disc remains anterior and inferiorly positioned in relation to the joint condyle, the bulk of the disc functions as biomechanical obstacle that prevents full mouth opening[4] and such position is associated with local inflammation and severe pain. In this condition, the mandibular head is not able to overcome the anteriorly positioned disc, thus the degree of jaw opening varies from 25 to 35mm. The anteriorly positioned disc favors the perpetuation of pain and inflammation and facilitates the development of anatomic and biochemical changes that paves the way for the development of OA signs and symptoms. This does not occur in all cases since signs and symptoms may improve with or without treatment and thus, no OA signs and symptoms develop. In chronic DDW_oR when recovery of function is observed, such change can be attributed to stretching of the retrodiscal tissues[23] in which, the posterior zone of the disc and part of the bilaminar zone hypertrophy and transforms into a false disc or ligament. This may lasts months and thus, the normal function can be restored with time. However, because pain and inflammation is severe and joint function is severely impaired, patients opt for treatment of the painful condition.

Bedran and colleagues[32] used clinical examination and MRI to evaluate 1038 patients with TMDs referred consecutively to an orofacial pain unit. Researchers reported that DDW_oR was present in 20.6% of the total sample. Researchers also reported that such disc disorder was significantly associated with disc deformity, degeneration of the condyle and mandibular fossa, and presence of inflammatory fluid.

Disk displacement and OA: OA, a form of arthritis, is characterized by chronic degeneration of the various hard and soft tissues around the TMJ[34]. OA of the TMJs is both a degenerative and inflammatory disease that probably begins in the fourth decade of life but its clinical manifestations usually are blatantly observed around the fifth and sixth decades of life. It is more frequently observed in females TMDs patients and its diagnosis may be established using clinical and radiographic methods. OA-TMJs is not a common disease in patients with TMDs and its prevalence is around 10% if patients are evaluated clinically and radiographically. Unilateral OA occurs more frequently as compared with bilateral OA and its prevalence increases dramatically after the six decade of life.

DDW_oR significantly alters the biologic and biomechanical milieu among the components of the TMJ including the disk, cartilage and synovial fluid. Supporting this point of view one investigation [32] asserts that OA of the TMJ more frequently occurs after the articular disc is displaced and bone contact is established between the

cartilage of the mandibular fossa and that of the mandibular head. This type of bone to bone relationship without the protection of the joint disk and/or synovial fluid increases the coefficient of friction and paves the way for the development of degenerative alterations

Mechanical loading that is not resisted by the articular surfaces constitutes a major etiologic factor for OA. However, a combination of factors including loss of posterior support, a flat occlusion, arafunctional behaviors, cross bite, age and probably presence of systemic musculoskeletal disorders, increases the likelihood of a certain individual to develop OA signs and symptoms. Kalladka and colleagues[34] believe that OA-TMJ has a complex and multifactorial etiology including, age, genetics, previous trauma to the face, repetitive adverse loading (parafunctional behaviors), torsional loading, and instances of prolonged micro trauma. Experimental application of mechanical loading is associated with an increase in the percentage of cells synthesizing DNA, alteration of the the types of protein being synthesized , modification of the rate of bone formation and inhibition of osteoblastic differentiation[9]. Both disk displacement with and without reduction may be associated with bony changes in the TMJs indicating OA. However, because DDW_oR involves the presence of a permanently displaced and deformed disc, such disc disorder is more frequently associated with bone changes in both the articular eminence and the mandibular head[32].

Because DDW_oR involves the presence of a deformed disc which does not return to normal position, such relationship increases the coefficient of friction in the articulating surfaces and the probability that TMJ-OA signs and symptoms develop. Increased friction coefficient is directly related to deterioration, erosion and roughness. Kuroda and associates[26], believe that TMJs-ID and associated abnormal disk position precede the development of deterioration, abrasion, thickening destruction and abnormal remodeling of joint surfaces, resulting in pain and impaired TMJs function. Sperry and associates[24] believe that degenerative joint pathology, for instance TMJ-OA, is initiated by a complex combination of applied mechanical loads and biological cascades within the joint tissues. Both trauma and age contribute to joint degeneration as joints in general are susceptible to degeneration under the influence of pathological mechanical loading, injury and aging. OA associated inflammation in the TMJ is characterized by the presence of pain, loss of cartilage, formation of osteophytes, subchondral bone changes and other degenerative and inflammatory alterations[24]. Pain in TMJ-OA is usually described as dull or aching.

V. Discussion

Anatomy and function of the healthy and functional TMJ disk

Given the biomechanical properties of the joint disc, most researchers believe that such structure is the most important anatomic component of the TMJs. The articular disc is a fibrous and cartilaginous, firm, oval structure positioned between the mandibular head and the mandibular fossa of the temporal bone. The TMJ articular disc is a biconcave structure made up of fibrocartilage tissue[1]. The TMJ disc is formed by the anterior, posterior and intermediate zone. Major constituents of the joint disc include collagen and elastic fibers, water, cells and ECM in which proteyglicans, biglycans, decorin, dermatan sulfate, chondroitin sulfate and other molecules predominate. The elastin molecule is associated with resistance and elasticity and is responsible for maintaining the shape of the disc following deformation. Elastic fibers can be found in the superior and inferior layers of the disc. Further, the amount of elastin increases from the center of the joint disc to the periphery or margins[1]. Fibrocytes, fibroblast-like cells an/or fibro-chondrocytes predominate in the articular disc of the TMJs[6] but fibroblasts can also be found the TMJ disc[5,12].

Various types of collagen molecules stratified and oriented sagitally, transversely and obliquely can be observed with the microscope[35]. Types I, II, III, VI and XII collagen forming a dense network can be found in the TMJ disc[1]. In the normal disc without TMJs-ID, elastic fibers can be found abundantly in the bilaminar zone. Elastic, elauinin and oxytalan fibers can also be observed in severely damaged discs and appear to ensure biomechanical functional properties reinforcing regions of the TMJ devoid of collagen bundles and thus, have a protective function[36]. In normal conditions, collagen fibers have the capacity to maintain the shape of the joint disc while elastin is associated with restoration of shape during unloading[10]. For instance, at the end of jaw opening, elastic fibers located in the bilaminar zone pull the disc back to the center of the glenoid fossa during closing, thus recovering position and shape of the joint disc during the jaw closing phase.

Most important functions of the TMJ disc includes its capacity to absorb compressive loading and the protection of the temporal bone and head of the mandible[6]. The fact that on the microscope many cells have and oval rather than a flattened outline indicates that this cells shape is typical of tissues subjected to compression[5,12]. The TMJ disc is maintained in place and stabilized by a set of six internal ligaments located posteriorly, anteriorly, laterally and medially[17]. Insertions of the joint disc to different anatomic components of the TMJ including the joint capsule, condyle and posterior zone of the mandibular fossa, allow the disc to adapt to rotational changes of the mandibular head by gliding across the different parts of the temporal bone[37]. To conclude, major functions of the articular disc include its capacity to resist compression, tension and shear

stress, its ability to protect the articular fossa and the mandibular head and to guide functional jaw movements during function.

2. Lubrication of the joint disk in normal and pathologic conditions

In normal physiologic conditions the synovial membrane that lines the inner surface of the joint capsule contains specialized cell types with both phagocytic and immunologic properties and produces the synovial fluid that provides the nutritional and metabolic requirements of the avascular and non innervated disc structures and fibrocartilage. As mentioned before, most common molecules found in the synovial fluid are HA and lubricin. HA is released by type B synovial cells and is pushed to the joint spaces during jaw movements[10]. The role of HA is to reduce friction and thus contributes to decrease friction and wear by producing elaborated and smooth jaw movements. Both lubricin and HA contribute to the formation of a high viscosity protective layer that is interposed between disc and fibrocartilage of the mandibular fossa and disc and fibrocartilage of the mandibular head. Thus, in normal conditions, a "pure and direct" contact between the joint surfaces, does not exist and is biomechanically undesirable. To conclude, the normal position, shape and displacement of the joint disc combined with the constant production, secretion and formation of the lubricating film guarantee protection, smooth movements of the condyle and disc assembly and a minimum of friction, roughness wear and abrasion of the functional surfaces. Severe loading, disc displacement and inflammation in some components of the TMJs, for instance, in the bilaminar zone, around the disc and in the capsule-synovial membrane complex are probably major dysfunctional events that lead to more advanced disorders and pathologic lubrication of the articular surfaces.

Because the most important functional structures of the TMJs deteriorate with aging and are subjected to dysfunctional loading from parafunctional behaviors, leading to disk displacement, the articular surfaces become roughened and eroded, the synovial fluid may negatively change its natural viscosity and its production may decrease, thus paving the way for the development of pathologic lubrication and signs and symptoms of OA alterations. Degenerative joint changes such as deterioration, abrasion, roughness and increased friction of the articulating and disc surfaces, thickening and remodeling of the underlying bone may lead to a further reduction in boundary lubrication between the articulating surfaces, thus, increasing the friction coefficient[10] in the joint and facilitating the development of osteoarthritic and perhaps osteoarthrotic changes in the TMJs.

Because the frequency of capsulitis/synovitis, retrodiscitis and disk-attachment pain are very high as compared to other TMJs-IDs, it is very likely that the process of abnormal lubrication in the TMJs initiates with molecular changes in the synovial fluid and inflammation in the synovial membranes and then progresses to a deficient or poor lubrication, increasing the coefficient of friction and facilitating the development of OA signs and symptoms. These considerations are echoed by one investigation[38] asserting that synovial inflammation or synovitis frequently accompanies intracapsular pathologic conditions of the TMJs such as TMJs-ID (disk displacement) and OA. Synovial inflammation is more likely to be associated with severer inflammatory changes in the TMJs[38]. In joints in which signs and signs and symptoms of disk displacement and OA developed, the synovial fluid has reduced viscosity due to an increase in both concentrations and molecular weight of HA[39]. This point of view is in accordance with one investigation [4] asserting that changes in the composition of the synovial fluid may affect the joint lubrication capacity of the synovial membrane leading to increased joint friction and abnormal disc movements. Furthermore, abnormal lubrication of the joint may also alter the nutritional requirements of the cells in both the disc and the joint cartilage[4].

It has been reported[9] that direct mechanical trauma (motor vehicle accidents) or indirect trauma from parafunctional behaviors may increase the production of free radicals in the hard and soft tissues of the TMJs. Free radicals are thought to induce a series of chain reactions in these molecules. Local chain reactions ultimately result in damage on important molecules in the synovial fluid. For instance, HA may be degraded, thus eliminating one important component of the lubricating mechanisms in the joints and facilitating the development of a higher friction coefficient in the joint surfaces. Further, both in fibrous and hyaline cartilage, HA, keratan sulfate and chondroitin are the principal GAGS[26]. HA present in the synovial fluid provides a high viscoelastic coefficient to the joint surfaces and its major function is to lubricate the articular surfaces, providing protection to the articular surfaces as viscoelasticity decreases the coefficient of friction. Consequently if HA is destroyed and/or biochemically altered, a number of properties in the lubricating mechanisms of the TMJs, is lost or pathologically altered.

The TMJ disk in pathological conditions including loading, lubrication, displacement.

In slightly damaged TMJ discs, which had not been subjected to serious functional pathologies, the superficial layer of the joint disc is able to maintain structural normality when compared to the already disorganized deep fibrous tissue[6]. In experimental studies from discs obtained from humans with TMDs, it was found that dysfunctional discs demonstrated a remarkable degree of modification in the macroscopic shape

with irregular and undulated surfaces. Further, collagen bundles of collagen fibers were irregularly and loosely arranged and were frequently interwoven in the superficial regions as in the deeper parts of the disc[6]. Because, researchers also observed an increase in the number of cells with abundant cytoplasm they concluded that such histological changes could be related to an attempt to reconstruct damaged tissue[6]. Deterioration of the articular surfaces not necessarily indicate a pathologic process as the may be related to an aging process. In an older patient, a degenerative process seem to be more visible in the TMJs[17].

Disk displacement and TMJs-OA

As mentioned before, a major biomechanical factor leading to pathological changes in the TMJs structures is disc displacement. This disorder has been classified by the degree of severity in disc displacement with and without reduction. The term reduction is used by researchers and clinicians to denote the capacity of the joint disk to return (reduction) or not return (no reduction) to its normal position in the mandibular fossa. Consequently and for many reasons, for instance degree of disc displacement and severity of pain, DDW_oR is considered a severer TMJ-ID as compared to disk displacement with reduction. Disc derangements and prolonged overloading associated with a poor adaptive capacity of the joint disc for repair and for maintaining the stability of the tissues, may significantly alter the repairing and functional capacities of the TMJs. When the functions of the joint disc is seriously compromised, structural changes in many components of the TMJs including OA changes may be observed.[4] Further, the presence of catching sensation, disc displacement with reduction and reciprocal clicking is a sign indicating a lower friction coefficient which paves the way for the development of OA signs and symptoms. The notion that "disk displacement with or without reduction progresses to an osteoarthritic condition in the TMJs", has been defended since the development of the first theoretical concepts about TMDs-ID. In regard to this, in patients seeking treatment for pain and dysfunction of the TMJs, the prevalence of disc displacement with and without reduction is very high. However, it has been very difficult to prove that any disc derangement with or without reduction progresses to OA changes in the TMJs.

DDW_oR and OA

DDW_{oR} is one type of TMJs-ID in which a macro or chronic micro traumatic event results in the displacement of the joint disc to an anterior and inferior position in the proximities of the articular tubercle and in which, the joint disc does not return (reduce) to its original position. This abnormality is related to severe pain and disc deformation, very limited jaw opening, no joint noise and difficulties to eat and to displace the mandible to the opposite non affected side. In chronic DDW_oR, the patient can usually recount a history consistent with acute closed-lock that resolved over time[23], thus suggesting that closed-lock may precede the development of OA signs and symptoms. The mechanisms of OA signs and symptoms in this case are deformation of the joint disc, lower coefficient of friction, roughness and poor lubrication by the synovial fluid.

Bedran and associates[32] evaluated a sample of MRI scans of 2076 TMJs of 1038 patients. They reported a prevalence of 20.6% DDW_oR, 25% of condylar degeneration indicating OA and disc deformity in 9.4% of the TMJs evalauted. In the same study, researchers reported that DDW_oR was significantly associated with disc deformity and degeneration of the condyle indicating the presence of OA signs. OA-TMJs is considered both an inflammatory and degenerative conditions affecting the TMJs and other body joints. Even though OA of the TMJs is more frequently observed in older persons, this disorder is usually the result of a long-standing inflammatory and degenerative process with a subtle onset around the fourth decade of life in which signs and symptoms are more evident both clinically and radiographically in the beginning of the six decade of life. If osteoarthritic changes do appear even in young individuals, a long-standing internal disk derangement without reduction should be ruled out[40]. OA of the TMJs is a disease that more frequently occurs after the articular disc has been displaced and bone contact has been established between the condyle and the glenoid fossa. Thus, it seems that there is an strong association between DDW_oR and degenerative TMJ bone changes[32]. TMJs-OA is a degenerative and inflammatory condition characterized by progressive cartilage degradation, subchondral bone remodeling, synovitis and chronic pain with a multifactorial etiology[41]. TMJ-OA is classified as a "low grade inflammatory arthritis" if compared with rheumatoid arthritis.

Osteoarthitic changes are the result of a long-standing and progressive process of both inflammation and degeneration of the articular surfaces and thus, is considered an advanced stage of TMJ-IDs. The role of imaging (both computerized tomography and magnetic resonance imaging) in the detection of TMJs osteoarthritic changes and diagnosis has been overemphasized in the current literature. This point of view is strongly reinforced by the observation that patient´s age, a complaint of pain, a report of a long-standing process characterized by pain and inflammation and e presence of self-reported crepitus which is also diagnosed clinically by palpation, may be sufficient clinical evidence for the diagnosis of OA of the TMJs. In this regard, it is clear that many researchers prefer to describe the radiographic changes as "diagnostic" of TMJs-OA rather than the "clinical" observations.

VI. Conclusion

Based on the literature review of current papers selected to carry out this study, the following conclusions can be drawn:

1.The joint disc is the most important structure within the TMJs. It is made up mainly of fibrocartilage and is constituted by collagen, elastic, elauinin and oxytalan fibers with many fibroblasts, fibroblast-like cells and chondrocyte-like cells scattered in different portions of the joint disc. The disc is stabilized by six collateral or internal ligaments and is nourished from the synovial fluid secreted by synoviocites and other cells found in the synovial membrane.

2.The periphery of the joint disc in the upper and lower surfaces is lubricated by molecules from the synovial fluid including HA and lubricin. The synovial fluid protects and lubricates the articulating surfaces, lowers the coefficient of friction and roughness and provides nourishment for the joint disc, cells and cartilage.

3.Internal derangements of the TMJs are usually caused by chronic overloading from parafunctional habits. Such behaviors overcome the capacity of the joint tissues to withstand compressive, tensional and shear loading. Severe loading may lead to inflammatory conditions associated with disk displacement. The severity of TMJ pain is proportional to the amount of loading and damage to disc, capsule, synovial membrane, cartilage and cellular component.

4.Disk displacement with or without reduction facilitates the development of OA signs and symptoms as such disorders are usually associated with inflammation, overloading and pain. Additional studies should be carried out to evaluate the molecular mechanisms associated with oxidative stress, cell damage and inflammation in the TMJs.

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