INTRODUCTION

Articular cartilage, the resilient load-bearing tissue that forms the articulating surfaces of diarthodial joints, provides these surfaces with the low friction, lubrication, and wear characteristics required for repetitive gliding motion.

It also absorbs mechanical shock and spreads the applied load onto subchondral bone. In most synovial joints, articular cartilage provides this essential biomechanical function for eight decades or more. No synthetic material performs this well as a joint surface [1].

Articular cartilage is a highly specialized tissue that allows for unique functions within synovial joints. It has been known for many years that the healing potential of articular cartilage after injury is poor if the structural integrity is disrupted. Disruption of the cartilage framework results in alteration of its biomechanical properties. These changes, in turn, can lead to the patient's perception of pain, loss of motion, strength, or instability. Although recently a great deal of research has been performed in this area, our understanding of this tissue is far from complete, and treatment of articular cartilage injuries remains challenging and controversial. [2].

In many instances, the degeneration of articular cartilage and alteration in other joint tissues that result from the loss of structure and function of articular cartilage cause pain and loss of motion. This occurs most frequently in the clinical syndrome of idiopathic or primary osteoarthrosis, but it may also result from joint injury or from developmental, metabolic, and inflammatory disorders that destroy the articular surface, causing secondary osteoarthrosis[1].

The treatment of focal full thickness articular defect in the knee has continued to present a challenge, with no traditional treatment method providing consistent acceptable long term clinical results, patient with significant chondral defects frequently have persistent joint pain, swelling and catching in the knee [3].

Analgesics and anti-inflammatory medications, chondro-protective agents, activity modification, and physical therapy may provide partial symptomatic relief, but they don't restore damaged articular cartilage to its normal state; thus, they rarely allow patients to return to full function for prolonged periods of time.

It has been shown that fibrous tissue and fibro-cartilage (formed by introduction of new cartilage-forming cells by perforation of subchondral bone) lack the durability and mechanical properties necessary for weight bearing surfaces.

Experimentally, cartilage repair has been stimulated successfully with allografts of periosteum, witch serve as sources of cells with chondrogenic potential; introduction of cells grown in culture(stem cells or chondrocytes); stimulation by fibrin clot formation; artificial collagen matrices combined with cell transplants; and chondrogenic growth factors[4].

The goal of developing methods to restore damaged articular surface has generated a tremendous amount of interest among scientists, clinicians, and patients. Recently a number of new articular cartilage repair techniques have emerged, some of which appear to be very promising. Although the final products are still in their infancy, the systemic sequential progression of developing these methods will yield improved treatment options [5].

Osteochondral autograft transfer system (*OATS)* offers a comprehensive surgical treatment for most full thickness femoral condylar defects in the knee. The OATS technique utilize a series of thin walled cutting tubes to harvest autogenously plugs of bone with healthy hyaline cartilage witch will be transferred to the damaged area [6].

AIM OF THE WORK

The aim of this work is to evaluate Osteochondral Autogenous Transfer grafts for the treatment of full thickness articular surface defect of the knee