

## Preface



David J. Hunter, MBBS, MSc, PhD  
*Guest Editor*

The evolution of our society has been accompanied by increasing obesity and aging, and, with this, increasing prevalence of osteoarthritis (OA). With these societal trends, new insights are developing into the pervasive disease we know as OA. Critically, the disease is no longer viewed as a passive, degenerative disorder, but rather an active disease process driven primarily by mechanical factors. Many define OA as a condition that primarily affects hyaline articular cartilage, including William Hunter, who in 1743 stated soberly “From Hippocrates to the present age it is universally allowed that ulcerated cartilage is a troublesome thing and that once destroyed, is not repaired.”<sup>1</sup> We now conceptualize OA as a disease of the whole joint organ.

Drs. Brandt, Dieppe, and Radin have coauthored a provocative review critiquing the current definition and understanding of OA and providing important insights into the etiopathogenesis of the pathologic changes associated with disease, the tissue changes important for symptoms, and how this should inform disease management. Hopefully the diminishing remnants of our scientific community who hold so firmly to the centrality of cartilage and terms such as “degenerative and passive” will be better informed after reading this sagely work by founding members of our field.

With the centrality of cartilage now cast aside, and a more thoughtful focus on the whole joint organ, two tissues that are worthy of more extensive investigation include the bone and meniscus. Dr Goldring provides a stimulating review of the specific skeletal features of OA and the putative mechanisms involved in their pathogenesis. In addition, the relationship of these boney changes to the alterations in other tissues comprising the diarthrodial joint are appraised. Dr Englund has provided a thorough review of the critical role of meniscus in joint function, the role they play in the incipient development of OA, and the overwhelming need to preserve their integrity rather than the penchant to attribute knee symptoms to them and remove them.

OA has a strong hereditary component that is likely polygenic in nature. In recent years several linkage analysis and candidate gene studies have been carried out and unveiled some of the specific genes involved in disease risk. Much of this work

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A version of this article originally appeared in the 34:3 issue of the *Rheumatic Disease Clinics of North America*.

Med Clin N Am 93 (2009) xv–xviii

doi:10.1016/j.mcna.2008.09.010

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has been led by Drs. Valdes and Spector, and in their considered review they appraise what we know and what impact future genome wide scans will have on providing further insights.

Mechanics plays a critical role in the initiation, progression, and successful treatment of OA. Dr. Wilson, a true pioneer in this field, and colleagues summarize the methods for assessing joint mechanics, describes the current evidence for the role of mechanics in OA initiation and progression, and further describes some current treatment approaches that focus on modifying joint mechanics.

OA causes substantial physical and psychosocial disability. I had the privilege of coauthoring a review with Drs. McDougall and Keefe that delineates the characteristic symptoms and signs associated with OA and how they can be used to make the clinical diagnosis. We also describe what we know causes pain in OA and what contributes to its severity.

Conventional radiography has played an important role in confirming the diagnosis of OA demonstrating late bony changes and joint space narrowing and has been applied as an endpoint for disease progression in clinical trials. However, OA is a disease of the whole joint, including cartilage, bone, and intra- and peri-articular soft tissues. Thus, the importance to image and assess all joint structures has been recognized in recent years largely using magnetic resonance imaging. Leaders in this field, Drs. Guermazi, Burstein, Conaghan, Eckstein, Hellio Le Graverand-Gastineau, Keen, and Roemer review radiography and MRI in OA and also give insight into other modalities (such as ultrasound, scintigraphy and computed tomography (CT), and CT-arthrography), discussing their role in the diagnosis, follow-up, and research in OA.

Sir William Osler, considered to be the “Father of Modern Medicine,” once said “osteoarthritis is an easy disease to take care of—when the patient walks in the front door, I walk out the back door.”<sup>2</sup> No one denies that the management of OA is a challenge, however modern clinicians are armed with a plethora of effective treatment options. We are also charged with discerning what agents are less effective yet still receive generous publicity rigorously eulogizing their benefits. The review of the management of OA provides an overview of what is available to the clinician managing OA. It is not comprehensive relying on subsequent articles (on imaging, weight management, exercise, braces and orthotics, pharmacologic intervention, and surgery) to provide more detail, but rather provides the clinician an opportunity to put the multitude of therapeutic options in perspective. I and Dr. Lo encourage active clinician involvement, to instill much of what are self-management strategies in patients, to further promote more effective long-term treatment of this pervasive disease.

For practicing clinicians, arming themselves with evidence for disease management is critical, and the ensuing articles—particularly those on obesity, muscle, and device use—are critical, as these are far too frequently overlooked in clinical practice. It is important that symptomatic improvement serve the purpose of increasing tolerance for functional activity. Ultimately, an efficacious treatment for any progressive disorder should also control the factors and forces that drive disease progression. These sections highlight this need.

The impact of and mechanisms by which obesity affects osteoarthritis are of great concern. Dr. Messier, a master in this field, reviews the physiologic and mechanical consequences of obesity on older adults with knee OA; the effects of long-term exercise and weight-loss interventions; the most effective nonpharmacological treatments for obesity; and the utility and feasibility of translating these results to clinical practice.

Drs. Bennell, Hunt, Wrigley, Lim, and Hinman provide a thorough review of the influence of muscle activity on knee joint loading, describe the deficits in muscle

function observed in people with knee OA, and summarize available evidence pertaining to the role of muscle in the development and progression of knee OA. They focus on whether muscle deficits can be modified in knee OA and whether improvements in muscle function lead to improved symptoms and joint structure, then conclude with a discussion of exercise prescription for muscle rehabilitation in knee OA.

The goal of many noninvasive devices for knee OA is to alter joint biomechanics in such a way as to limit regional exposure to potentially damaging and provocative mechanical stresses. Because of their targeted intention, optimal prescription of most noninvasive devices requires that we first specify which mechanical stresses we wish to reduce, and in which knee region. Drs. Gross and Hillstrom lend their expertise and review several of the most important devices currently used in the treatment of knee OA.

Ultimately, many patients seek assistance for pain relief in the form of pharmacologic intervention. Dr. Harvey and I review the current trends and controversies related to pharmacologic management, including the use of oral, topical, and injectable agents.

Failing prior interventions for knee OA, surgery may become necessary. While the indications for arthroscopy have narrowed, joint replacement continues to play a pivotal role in disease management. Dr. Richmond reviews the plethora of surgical options and the evidence to support their efficacy. Orthopedic surgeons continue to explore options less invasive than total knee replacement for isolated unicompartmental arthritis of the knee joint.

The Holy Grail for many in this field is to modify the underlying structural changes. Dr. Hellio Le Graverand-Gastineau and I review the evidence to suggest we can modify the disease, and if the current tissue we are predominantly focused upon, namely cartilage, is an appropriate target. We will also consider the methodologic approaches and other obstacles to demonstrating efficacy of these agents in clinical trials.

Looking forward, we are reminded by the late Sir Henry Tizard that “the secret of science is to ask the right question, and it is the choice of problem more than anything else that marks the man of genius in the scientific world.” We have been afforded an opportunity to study a much maligned disease that is rapidly evolving. Let’s learn from the insights our research is providing to focus even more on important modifiable risk factors, such as mechanics and obesity, as we develop the therapeutic armamentarium of the 21st century. Assuming we maintain a meaningful motivation with the patient at the forefront of our mind, we have an opportunity to make a difference in millions of people’s lives. I look forward to the evolution ahead.

I would sincerely like to thank my friends and colleagues for their valuable contributions to this issue of *Medical Clinics of North America*. They were a pleasure to work with, and I am sure you will see that the contents here reflect wonderful insight and appraisal of a complex and developing field.

David J. Hunter, MBBS, MSc, PhD  
Division of Research  
New England Baptist Hospital  
125 Parker Hill Avenue  
Boston, MA 02120, USA

E-mail address:  
[djhunter@caregroup.harvard.edu](mailto:djhunter@caregroup.harvard.edu) (D.J. Hunter)

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