JAOA SPECIAL COMMUNICATION

Rethinking the Model of Osteoarthritis: A Clinical Viewpoint

Greg J. Wade, MSc, DO (New Zealand)

The prevailing model of joint degeneration based on age-related, genetic, and familial factors implies inevitable progression and limited palliation from manual therapy. This model is presented to primary care physicians and the public on Web sites and in resource texts and is implicit in many published research articles. The author presents a synthesized model of the progression of osteoarthritis, combining radiographic, histologic, and clinical evidence. The revised model suggests that the progression of primary osteoarthritis is divided into an initial reversible arthrosis phase and a later arthritis phase, with both phases linked to accepted histologic and radiographic observations. The revised model also suggests a number of novel concepts, including the influence of dominance bias and laterality. The author concludes that a small change in understanding could translate into important changes in the therapeutic management of osteoarthritis, with implications for government public health policy.

J Am Osteopath Assoc. 2011;111(11):631-637

Osteoarthritis is usually thought of as an inherent and inevitable process of aging in human joint cartilage, a problem that affects most of the elderly population and is found in ancient skeletal remains from all eras and populations.[1] Current modeling of the progression of osteoarthritis is presented in standard texts such as *Rubin's Pathology*.[2] The authors of this resource describe a clear sequence of histologic changes. First in this sequence are loss of proteoglycans on the cartilage surface and death of chondrocytes in deeper layers. Next, viable chondrocytes enlarge, aggregate, and become surrounded by territorial matrix. At some later stage, surface cracks form parallel to the articular face.[2]

Financial Disclosures: None reported.

Address correspondence to Greg J. Wade, MSc, DO, Waimana Clinic, 2 Waimana Ave, Northcote Point, Auckland 0627, New Zealand.
E-mail: gregwade@actrix.co.nz

Submitted April 24, 2011; revision received June 30, 2011; accepted August 4, 2011.

These fibrillations may persist for many years before progressing further. Synovial fluid flows into defects, penetrating deeper over time. Fragments of cartilage sequester and induce inflammatory and immune reactions. The synovium becomes hyperemic and hypertrophied. Degradation of fibrillation areas eventually exposes subchondral bone, producing surface eburnation and sublayer thickening. If synovial fluid penetrates the bone marrow through defects, cysts may form. Osteophyte development occurs at joint margins.[2]

The authors of *Rubin's Pathology*[2] and other texts describe variable clinical features of osteoarthritis. Common presentations include enlarged joints that are tender and boggy, as well as postactivity deep ache that is relieved by rest. There may also be joint crepitus and periods of stiffness—especially first thing in the morning and after any activity.

Texts note that although the clinical hallmark of postactivity pain signals joint destruction, the pain arises from periarticular structures consequent to the lack of neurologic supply to the joint cartilage.[2,3] The texts also characterize restriction of movement as indicating severe disease, but a wide range of sources for the restriction is suggested, including joint or muscle contractures, intraarticular loose bodies, large osteophytes, and loss of congruity of joint surfaces. Authors are unequivocal that osteoarthritis cannot be prevented or arrested.[3] Therefore, therapeutic efforts, including exercise and weight loss programs, are to be directed supportively at specific orthopedic copresentations.

The Arthritis Foundation[3] lists the following risk factors for osteoarthritis: age, obesity, injury or overuse, genetics or heredity, muscle weakness, and other diseases and types of arthritis. The information presented to medical professionals, researchers, and the public by *Rubin's Pathology*,[2,3] the Arthritis Foundation,[4] and many other sources reinforces the prevailing view that age-related changes lead inevitably to joint degeneration, and that an individual's genetic traits play a dominant role in osteoarthritis.

Problems With the Prevailing View

This model of osteoarthritis appears to be well established in the scientific community and is accepted by most physiotherapists and by many osteopaths and osteopathic physicians. However, dissenting opinions continue among clinicians.[5-8] Why do some elderly individuals experience no joint pain at all and display no other signs of deterioration apart from some joint space narrowing? Why, if osteoarthritis follows a

purely age-related model, is there an accelerated deterioration process after trauma—even in young people, especially if the trauma affects intraarticular structures? Considering that particular occupations and sports activities produce focal and predictable osteoarthritic problems, indicating a strong "wear-and-tear" mechanism, how important is that mechanism in general presentations? With good evidence that usage and trauma play dominant roles in premature osteoarthritis, are the study participants in any cohort merely age candidates, or have they actually accumulated specific degradations?

The hereditary or genetic links for osteoarthritis are also problematic. The familial mechanism for this condition was first identified more than 60 years ago in studies of Heberden's nodes.[9] Generations of women in New Zealand have presented with Heberden's and Bouchard's nodes on their second and third digits from darning, sewing, embroidery, and knitting, though personal observation suggests that these nodes are becoming less common as fewer women engage in these activities. Thus, the transfer of habit and skills from mother to daughter produced a predictable familial association, but this association was occupational rather than genetic in nature. Other familial patterns, such as structural femoral and carrying angles in mothers and daughters, may also increase the likelihood of osteoarthritis. Earlier this year, a review[10] concluded that chromosomal associations in patients with osteoarthritis are numerous but unspecific, and despite much effort, researchers have been unable "to reach a genuine therapeutic target or even a prognostic marker of [osteoarthritis]."

Key Issues

Nutrition and tribologic issues are key in development of osteoarthritis. Being mostly avascular, articular cartilage is largely supported by diffusion. Articular margins receive nutrients from the adjacent vascular synovium and periosteum.[11] Deeper layers of articular cartilage may be fed by the blood supply to the subchondral bone, but this mechanism is less clear. The deep cartilage layers are not subject to the shear forces sustained by the superficial layer, and the deepest layer is reported to be largely calcified.[11]

The contacting surfaces in joints are pivotal in the deterioration process. Although the diffusion that the tangential zone experiences from below is contested, synovial fluid intake from the joint cavity is well established as a predominant mechanism.[12] Absorption from the surface appears to be largely osmotic, possibly assisted by a mechanical pump phenomenon, though such a process is now disputed.[12] Surface cartilage is normally more hydrated than interior layers because the presence of proteoglycans on the surface allows binding of water molecules, which are exuded under compression (ie, "weeping lubrication").[13]

Since the work of McCutchen[14] in the 1960s, the understanding of lubrication in human joints has been considerably refined. However, there are still a number of proposed mechanisms of joint lubrication under investigation,[15] and the complexities of transient, cyclic, and prolonged loadings are becoming increasingly apparent.[12,16-18]

Furey[18] has stressed that friction, lubrication, and wear are each separate issues in osteoarthritis, and abrasive forces are accompanied by delaminatory stresses and other subsurface stresses. Whatever tribologic model is followed, the pressure of the surfaces is a key factor. Increased surface pressure not only increases abrasive and shear forces and decreases lubrication, but it also adversely affects transport of nutrients and waste products if sustained. Although immobilized joints undergo atrophy of articular cartilage, presumably because they lack boosted or pumping lubrication, joints exposed to prolonged compression can also undergo atrophic changes.[11] Therefore, the effectiveness of the mechanical integrity of surface cartilage in resisting tribologic stress may be as important as nutrition and lubrication in joint deterioration.[19]

In the tangential zone, collagen is aligned parallel to the surface, providing improved shear resistance compared with vertical or oblique arrangements. Collagen fibers run obliquely to each other in tight bundles, forming a strong matting that has multidirectional resistance to shear. External to the joint, ligamentous competency is important for mechanical integrity, providing constraints to undue displacement (especially in close-packed situations) so that joint congruency is preserved.

Less well understood is how the "choreography" of muscles around a healthy joint controls the ratio between rolling and gliding motions. Rolling action involves less shear stress on the contact surfaces, and joint lubrication mechanisms are enhanced by rolling. Research published on the kinematic characteristics of the knee show the importance of muscular control,[20] but the classic example of muscular choreography involves the glenohumeral joint, in which stability is only partly established by the labrum.[21] As the arm makes complex arcs of movement, any derangement of muscular coordination leads to painful impingement syndromes (eg, frozen shoulder, painful arc syndrome, supraspinatus bursitis). Even in saddle joints and so-called gliding joints, articulation may be more rolling than gliding if the muscular and ligamentous systems are functioning optimally.

Rethinking the Process

Most studies considering laterality associations in osteoarthritis have found incidence bias. In the Johnston County Osteoarthritis Project,[22] which examined multijoint presentations, a strong association was found between contralateral knee and hip pairs, and a modest association was found with the other joint site (ie, knees vs hips). Although these findings appear to support the age-oriented model of osteoarthritis, it is more likely that compensatory gaits and the use of gait aids, such as canes and walkers, greatly increase stresses on other joints if there is restriction in a joint system. This process starts well before radiographic signs of joint degradation are visible.

Newton and Seagroatt[23] suggested that differences they observed in osteoarthritis rates between the right and left hips

(average rates: 60% right, 40% left) in 4 surveys of osteoarthritis and arthroplasty (2 from England and 2 from other countries) were caused by higher impulse loading in right-footed patients. In a survey of data in Italy, Stea et al[24] observed an overall bias toward right hip arthroplasty (rate, 58%), but a higher prevalence of right hip osteoarthritis in left-footed patients. Right-footed individuals were unbiased for monolateral arthroplasty (average rates: 50.7% right, 49.3% left), but left-footed individuals exhibited a strong differential toward arthroplasty on the right side (average rates: 76.8% right, 23.2% left). Stea et al[24] concluded that left-footed patients subjected their right sides to greater stress than right-footed patients.

Stea et al[24] supported their conclusions with a general population survey that found that left hips had slightly more loss of initial joint space in right-footed people than in left-footed people. The authors explained this survey finding as being the result of either intrinsic differences between right and left hips or of the left hip being subjected to greater mechanical stresses in right-footed people. They also postulated that early cartilage loss may target the left hip, but after a threshold is reached, joint deterioration might progress more symmetrically.

Without elaborating on the complex body patterning associated with left and right dominance, it is important to note that the conclusions of the Johnston County Osteoarthritis Project,[22] Newton and Seagroatt,[23] and Stea et al[24] do not necessarily contradict each other. Each study supports dominance usage as a factor in osteoarthritis development.

In a prospective multicenter cohort study in the United Kingdom, Birrell et al[25] considered laterality in primary care assessments of new hip pain in 195 patients who were assessed and then tracked until referral for total hip arthroplasty. Importantly, less compensatory stresses on other joints were present during the initial assessments. This cohort displayed initial bias toward right hip pain (rates: 53% right, 45% left), with only 5 of the 195 patients exhibiting bilateral pain initially.[25]

The ratios in all these studies correlate with laterality ratios for various joint replacement procedures recorded in the New Zealand National Joint Register (S. Shaw, PhD, written communication, December 2010). These data, shown in the *Table*, also indicate a greater spread in laterality ratios for the upper extremities, where usage bias is greater.

Such laterality bias in osteoarthritis serves as a strong argument for a usage-based model of the condition, especially when added to occupational, ergonomic, and sports evidence. In the age-only model of osteoarthritis, by contrast, an explanation has yet to be established for laterality bias of the preliminary arthrosis stage.

More fundamentally, the study by Birrell et al[25] shows that patients with pain scores between 3 and 7 (median, 5) on the visual analog scale, which ranges from 0 (no pain) to 10 (agonizing pain), already have their hip flexion reduced to a range of 84° to 110°, with a mean of 98°. This dramatic reduction in hip flexion means that in most sitting situations (eg, on soft furniture; in cars, planes, or the cinema), there is maximum capsular torsion. Even in sitting positions that approximate 90°, such as during dining and elimination, the joint cartilage will be in prolonged compression. With increasingly sedentary lifestyles, this is an important etiologic consideration.

Table.
Numbers and Laterality Ratios of Joint Replacement Procedures as Recorded by New Zealand National Joint Register, 1999-2009

Procedure	No.	Right:Left Ratio, %
Ankle	603	54.7:45.3
Elbow	301	57.1:42.9
Hip	63,682	53.7:46.3
Knee	46,095	52.9:47.1
Shoulder	3013	54.5:45.5

Modifying the Model

An alternative, clinically based model of osteoarthritis should incorporate accepted histologic and radiologic data. This model amends the chronologic description of the disease process and more accurately matches signs and symptoms. It also acknowledges the importance of nutrition, lubrication, tribologic forces, and kinematic factors in aging joints. I propose the following 11 points, based on clinical experience, for this revised model of osteoarthritis:

1. Aging brings natural changes in joints, including hydration loss in the tangential layer of cartilage and hydration increase in deeper layers. There are complex protein-related alterations in chemical and histologic factors, including possible chondrocyte senescence.[5] While radiographic imaging may show mild joint space narrowing, there may be neither symptoms nor loss in range of motion. However, the aging process subtly alters the biomechanical gradient between the tangential layer and sublayers, increasing the likelihood that tribologic stress will produce laminar failure.
2. Repetitive short-arc movements of a joint (eg, many individuals rarely operate the hip past the sitting position) produce limitation of capsular cartilage and pericapsular tissue within that narrowed range. Articular surfaces then show both increased compression and shear as joint play is lost, and close-packing forces increase *internal compression of the joint*. Certain activities with specific patterns of repetitive use or abuse produce distorted periarticular muscular strengths,[26] which cause torsion of the capsule and ligaments. This alteration of choreography produces *external torsional compression*, adversely altering the kinematics of the joint surfaces by changing the slip ratio in favor of gliding rather than rolling.
3. Any history of direct mechanical insult to the articular cartilage—whether by impact, trauma, or surgery—will impinge

on capsular freedom and initiate histologic changes[27] that effectively soften the joint cartilage.[28] These changes produce accelerated degradation that is irrespective of age: *post-trauma degradation*. The first clinical sign of this problem will be loss of range of motion that persists after the acute stage. Stiffness will be an early symptom.

4. Increased body weight increases loading on joint surfaces, especially in the knees. The increased contact pressure occurs without capsular torsion. Acquired obesity is widespread enough in the United States for the knee replacement rate to be 23 per 10,000, compared with a rate of 10.3 per 10,000 for total hip replacement.[29] These statistics raise interesting questions regarding ethnic-based "obesity" and acquired obesity.

Statistics from the New Zealand National Joint Register show that knee replacements in that country occur at a rate of 72% of hip replacements (*Table*), whereas in the United States the equivalent rate is 223%.[29] New Zealand is generally considered to be a society of high body weight, with Polynesians making up approximately 21% of the population.[30] However, Polynesians have a higher ratio of lean mass to fat mass compared to people of European descent.[31] Some researchers have calculated that if 25.0 is used as a benchmark body mass index (BMI) for Europeans, then 26.3 is the appropriate BMI for Americans of African descent, and 29.5 is the appropriate BMI for Polynesians.[32] Tongans, who are also well represented in New Zealand, have even higher BMI ratings, at 32 to 34.[33] Thus, it is possible that the acquiring of weight is far more of a challenge to weight-bearing joints than the carrying of a higher weight throughout adolescent development into adulthood.

5. Any infection, autoimmune reaction, chemical insult (eg, gout), or other illness may compromise the histologic integrity of cartilage, leading to immediate deterioration—*chemical degradation*—that is independent of age or biomechanical processes.[34]

6. Because mechanically compromised individuals will experience restriction, stiffness, and pain, especially after inactivity, rising after sleep and moving after maintaining a particular posture will feel restricted and painful. Pain loci are connected with periarticular trigger points rather than the joint line, and clinical testing reveals some preservation of pain-free range of movement. The disease status at this point could be termed *prearthrosis*, with few radiographic signs and probably no internal deterioration.

7. With increased abrasion, decreased tangential lubrication, and increased shear forces in the hydrated deeper layers of cartilage,[18] there is inevitable degradation. Compression also affects nutrition, both at the surface, where film effectiveness is reduced, and in deeper layers, where osmotic transport is reduced.[19] This could be termed the *arthrosis phase*. Imaging may reveal some loss of joint space but usually no other signs. During evaluation, the motion end range now exhibits a resistance incline that is above norms. Crepitus can be a feature even in early presentations, arising from a combination of reduced filming, increased surface stiffness, and tribologic stress.[35] Crepitus often reduces or disappears with later joint effusion.

During the arthrosis phase, manual treatment aimed at increasing the range of motion is effective in resolving most symptoms, including cases involving posttrauma joints and postsurgical rehabilitation. Self-management with stretching is effective in indefinitely prolonging treatment efficacy.

8. In hypermobile (ie, ligamentously lax) joints there is less intraarticular pressure than in normal joints, but lack of congruency of movement of the joint axis produces substantial surface abrasion and capsular distortion.[36] Therefore, synovial inflammation and trigger point activity in the periarticular region occur early and acutely. Osteophyte development may also be precocious in hypermobile presentations. Age-related sarcopenia can be contributory to hypermobile joint degeneration. In addition, as weight-loading of hypermobile joints simultaneously produces anomalous close-packing and abrasion, leading to painful "nips" in movement arcs, exercise requires careful guidance.

9. True osteoarthritis commences with the breakdown of the tangential layer. With reduced regenerative activity and lubrication, surface stresses will eventually cause fissures. The arrangement of fibers at the surface may be resistant, and the first ruptures may be plastic failure of sublayers under high lateral shear forces (ie, fibrillation), but microtears will eventually appear at the surface. After the tangential zone is ruptured and allows more movement, vertical fissuring accelerates. Fragments can sequester and produce inflammatory and immunologic responses, causing the onset of the *arthritis phase*.

In this phase, there is onset of active muscle guarding around the joint, with pain focused on the joint itself and evident throughout the range of motion. The joint space may become effused, producing a boggy end-feel to motion in passive testing. Manual therapy after this stage produces only temporary improvement in range and less reduction in joint locus pain.

10. Surface fissuring allows more movement within deeper layers of the transitional and radial zones. Cartilage degradation continues until sites of subchondral bone are exposed and characteristic end-stage radiologic changes manifest. The patient has now entered the *presurgical phase*.

11. In the presurgical phase, patient pain ratings increase substantially, especially in weight-bearing joints. Within the hip joint, the head may remodel or collapse.[37] Walking becomes difficult, with restricted motion and a flexed stance, and hip or knee arthroplasty is probably inevitable. Neighboring joints are inevitably affected at this stage, as shown in the Johnston County study.[22]

Garbuz et al[38] found that waiting time for surgery was strongly associated with reduced outcome scores after total

hip arthroplasty, with an 8% average reduction in outcome score with every additional month of waiting. The worst outcomes occurred in patients who had more pain, lower function, and more hip stiffness preoperatively. Other researchers have found strong associations between postoperative outcome and preoperative walking time and hip flexion arc. Patients with worse preoperative scores in these categories had greater likelihood of postoperative pain and lower functional outcomes.[39,40] These results support the hypothesis that joint degradation accelerates dramatically after the arthritis phase is reached.

Comment

There are substantial clinical implications in this proposed alteration of the osteoarthritis model (*Figure*). In this revised model, osteoarthritis is not an inevitable disease of aging but the result of a definable history in the individual. Age-related changes do not lead to osteoarthritis by themselves. Rather, there must be some additional stress or insult to cause the condition. The greatest risk factor for osteoarthritis is not age but duration and intensity of stressing. Obviously, family members will have similar pain-related biochemical characteristics, similar susceptibilities to mechanical and histologic breakdown, and similar inflammatory and immunologic responses. Nevertheless, osteoarthritis—first and foremost—depends on the individual's own history.

Another implication is that joint restriction is causal rather than symptomatic, with restriction around the joint associated with early changes in the joint rather than late-stage degeneration.

Furthermore, the altered model implies a reversible arthrosis phase before the irreversible arthritis phase. The traditional model maintains that osteoarthritis cannot be prevented or arrested, let alone reversed. Yet recent in vivo research using hydropolymers as scaffolding for chondrocyte seeding and cartilage regeneration shows that, given some relief from compression and shear, chondrocyte activity may be recoverable.[41] This finding bodes well for joint rehabilitation in the absence of subchondral exposure, as well as for manual therapy aimed at improving capsular freedom.

Treatment intervention points are earlier. Preserving joint mobility offers the possibility of arresting further degradation, especially in earlier phases of osteoarthritis. Therefore, intervention points and goals of physical therapy are altered dramatically. As soon as maintenance pain relief is required in joint-related presentations, physical therapy should be introduced, combined with a program of effective stretching. Posttrauma and postsurgery rehabilitation needs to be immediate, comprehensive, and aimed at restoring articular freedom as well as limb function.

Finally, some therapeutic interventions used for patients with osteoarthritis need to be reconsidered. Many resistance and weight-loaded exercises (including outdoor walking, treadmill sessions, and exercycles) involve short-arc repetitive motion, which tends to decrease joint mobility for the same reasons involved in repetitive occupational[42] and sporting activities.[43] Physical therapy has traditionally concentrated on countering muscle strength imbalance in the agonist muscles around an affected joint system[44] by strengthening weak muscles and lengthening strong muscles. However, there is evidence that such muscular imbalance can originate from disturbance of joint proprioception,[45] and it is likely that short-arc exercise programs cause shortening of muscle action and place further stress on the joint capsule and joint surfaces.

Although exercise of this type may improve congruency, such exercise is contraindicated in many cases, especially those cases involving joints that do not bear weight, such as the shoulder joint. Sustained static stretching of strong muscles, which also improves congruency, together with range-of-motion exercises are the preferred remedial choices for patients

- Osteoarthritis is the result of a definable history in the individual, not an inevitable disease of aging.
- The greatest risk factor for osteoarthritis is duration and intensity of joint stressing, not age.
- Joint restriction is usually causal rather than symptomatic.
- Osteoarthritis has a reversible arthrosis phase before the irreversible arthritis phase. Primary osteoarthritis might be preventable with improved body maintenance.
- Treatment interventions need to be initiated earlier in the disease progression.
- In the arthrosis phase of osteoarthritis, manual therapy should target joint range, congruency, and periarticular muscle coordination. After the arthritis phase begins, manual therapy becomes palliative at best.
- Resistance and weight-loaded exercises (eg, outdoor walking, treadmill sessions, exercycles) tend to decrease joint mobility because of repetitive short-arc action. Physical therapy concentrating on increasing competency of agonist muscles around an affected joint system may shorten muscle action and place further stress on the joint capsule and joint surfaces.
- Sustained static stretching improves congruency and is the best remedial choice for patients with osteoarthrosis. Low-resistance loaded exercise is the treatment of choice in early-stage hypermobile situations. For weight-bearing joints, low-resistance exercises (eg, aquaerobics, aquajogging, swimming) have a supportive role for patients who are obese, who have comorbid conditions, or who are elderly with sarcopenia.
- Costs of surgical interventions could be reduced by government information programs highlighting stretching and mobility as important aspects of health. Such programs could be enhanced by conducting simple screening tests (eg, supine hip flexion) and manual interventions (eg, facilitated positional release, muscle energy, strain-counterstrain).

Figure. *Clinical implications of altering the model of osteoarthritis as proposed by the author.*

in these situations. Low-resistance loaded exercise would be the treatment of choice in early-stage hypermobile situations. For weight-bearing joints, low-resistance exercises such as aquaerobics, aquajogging, and swimming have a supportive role for patients who are obese, who have comorbid conditions, or who are elderly with sarcopenia.[46]

Although there has been recent criticism of primary care physicians for neglecting weight-reduction and exercise programs[47] in managing osteoarthritis, most current exercise regimens tend to produce varied results for the previously mentioned reasons. In my experience, managing osteoarthritis in patients with comorbid obesity is a major clinical challenge that requires a carefully planned and multifaceted approach, even in early stages. While aerobic exercise is vital, the factors discussed in the present article need to be taken into account, often making tailoring of specific exercises necessary. The case for preventive management is therefore strong.

Strapping may palliate pain simply by restricting movement, but it does not alleviate contact pressure, and it introduces novel congruency patterns.[8] Lack of movement also promotes cartilage atrophy.[11] Strapping may retain a niche treatment role in hypermobile presentations, but its drawbacks limit its usefulness. In hypermobile presentations of osteoarthritis, as indicated by the Johnston County Osteoarthritis Project[22] and the Italian study by Stea et al,[24] contralateral transfer of stress must be considered, and bilateral evaluation should be routine. Restriction in 1 joint system, such as the hip, transfers stress to other joint systems that are functionally linked and more mobile, such as the knee, as noted in the Johnston County study.[22] The shoulder is similarly linked to grip, wrist, and forearm function. In addition, walking aids transfer stresses to other joint systems as the user assumes a flexed stance, producing additional problems in long-term use.

In the arthrosis phase of osteoarthritis, manual therapy should target joint range, congruency, and periarticular muscle coordination to reduce contact (ie, capsular) pressure and surface friction. After the arthritis phase has been reached, manual therapy becomes palliative at best. Although it is difficult to determine the cut-off point for effectiveness of conservative manual therapy in remedying the problem, a useful clinical measure is that improvements in range of motion do not last between treatment sessions, and overall improvement reaches a plateau. From that point on, delaying surgery produces higher ancillary costs and worse outcomes.[38-40]

Conclusion

This revised model of osteoarthritis allows for the possibility that primary osteoarthritis might be preventable with improved body maintenance. Some of the costs of surgical interventions could be usefully transferred to government information programs highlighting stretching and mobility as important aspects of public health. The effectiveness of such information programs could be enhanced by conducting simple screenings (eg, supine hip flexion) and manual interventions (eg, facilitated positional release, muscle energy, strain-counterstrain) based on an improved understanding of the processes involved in joint degradation.

As physicians and other healthcare providers manage populations that are not only growing older and increasing in BMI, but also becoming more sedentary, policymakers face the "perfect storm." On the basis of arthroplasty projections for the United States,[48] primary hip replacements are expected to increase by 174% and knee replacements by 673% by 2030. The prevailing model of osteoarthritis does not correlate well with clinical experience, and it is hindering progress in improving these arthroplasty statistics. A new osteoarthritis model is overdue.

Acknowledgments

I wish to acknowledge the cooperation of the New Zealand National Joint Registry in releasing base data for 1999 to 2009, as well as the assistance of Susan Shaw, PhD, associate dean of health at Auckland University of Technology, in communicating with the New Zealand National Joint Registry.

References

1. Dequeker J, Luyten FP. The history of osteoarthritis-osteoarthrosis. *Ann Rheum Dis*. 2008;67(1):5-10.

2. Hoch BL, Klein MJ, Schiller AL. Bones and joints. In: *Rubin's Pathology: Clinicopathologic Foundations of Medicine*. 5th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2008:1134.

3. Hoch BL, Klein MJ, Schiller AL. Bones and joints. In: *Rubin's Pathology: Clinicopathologic Foundations of Medicine*. 5th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2008:1136.

4. Who gets osteoarthritis—and why? Arthritis Foundation Web site. http://www.arthritis.org/who-gets-osteoarthritis.php. Accessed October 5, 2011.

5. Anderson AS, Loeser RF. Why is osteoarthritis an age-related disease? *Best Pract Res Clin Rheumatol.* 2010;24(1):15-26.

6. Felson DT. Developments in the clinical understanding of osteoarthritis. *Arthritis Res Ther*. 2009;11(1):203-214.

7. Sims K. The development of hip osteoarthritis: implications for conservative management. *Man Ther*. 1999;4(3):127-135.

8. Block JA, Shakoor N. The biomechanics of osteoarthritis: implications for therapy [review]. *Curr Rheumatol Rep*. 2009;11(1):15-22.

9. Cassel CK, Leipzig R, Cohen HJ, Larson EB, Meier DE, eds. *Geriatric Medicine: An Evidence-Based Approach.* 4th ed. New York, NY: Springer; 2003:581.

10. Akune T, Kawaguchi H. Human genetic studies on osteoarthritis from clinician's viewpoints [published online ahead of print December 22, 2010]. *Osteoarthritis Cartilage*. 2011;19(3):251-253.

11. Hertling D, Kessler RM. *Management of Common Musculoskeletal Disorders: Physical Therapy Principles and Methods*. 4th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2006:39-40.

12. Krishnan R, Mariner EN, Arteshian GA. Effect of dynamic loading on the frictional response of bovine articular cartilage. *J Biomech*. 2005;38(8):1665-1673.

13. Lewis PR, McCutchen CW. Experimental evidence for weeping lubrication in mammalian joints. *Nature*. 1959;24(184):1285.

14. McCutchen CW. Why did nature make synovial fluid slimy? *Clin Orthops Rel Res*. 1969;64:18-20.

15. Jin Z. Lubrication of synovial joints. In: Luo J, Meng Y, Shao T, Zhao Q, eds. *Advanced Tribology: Proceedings of CIST2008 & ITS-IFToMM2008*. Beijing, China: Tsinghua University Press/Springer; 2010:871-872.

16. Ateshian GA. A theoretical formulation for boundary friction in articular cartilage. *J Biomech Eng*. 1997;119(1):81-87.

17. Solz MA, Ateshian GA. Interstitial fluid pressurization during confined compression cyclical loading of articular cartilage. *Ann Biomed Eng*. 2000;28(2):150-159.

18. Furey MJ. Joint lubrication. In: Bronzino JD, ed. *The Biomedical Engineering Handbook*. 2nd ed. Boca Raton, FL: CRC Press/Springer; 2000;21:1-21.

19. Caligaris M, Ateshian GA. Effects of sustained interstitial fluid pressurization under migrating contact area, and boundary lubrication by synovial fluid, on cartilage friction [published online ahead of print April 18, 2008]. *Osteoarthritis Cartilage*. 2008;16(10):1220-1227.

20. Hollman JH, Deusinger RH, Van Dillen LR, Matava MJ. Gender differences in surface rolling and gliding kinematics of the knee. *Clin Orthop Relat Res*. 2003;413:208-221.

21. Karduna AR, Williams GR, Williams JL, Iannotti JP. Kinematics of the glenohumeral joint: influences of muscle forces, ligamentous constraints, and articular geometry. *J Orthop Res*. 1996;14(6):986-993.

22. Sayre EC, Jordan JM, Cibere J, et al. Quantifying the association of radiographic osteoarthritis in knee or hip joints with other knees or hips: The Johnston County Osteoarthritis Project. *J Rheumatol*. 2010;37(6):1260-1265.

23. Newton J, Seagroatt V. Why is osteoarthritis of the hip more common on the right? *Lancet*. 1993;341(8838):179.

24. Stea S, Bordini B, Viceconti M, Traina F, Cervini A, Toni A. Is laterality associated with a higher rate of hip arthroplasty on the dominant side? *Artif Organs*. 2008;32(1):73-77.

25. Birrell F, Afzal C, Nahit E, et al. Predictors of hip joint replacement in new attenders in primary care with hip pain. *Br J Gen Pract*. 2003;53(486):26-30.

26. Wang H-K, Cochrane T. Mobility impairment, muscle imbalance, muscle weakness, scapular asymmetry and shoulder injury in elite volleyball athletes. *J Sports Med Phys Fitness*. 2001;41(3):403-410.

27. Nelson F, Billinghurst RC, Pidoux I, et al. Early post-traumatic osteoarthritis-like changes in human articular cartilage following rupture of the anterior cruciate ligament [published online ahead of print October 20, 2005]. *Osteoarthritis Cartilage*. 2006;14(2):114-119.

28. Ewers BJ, Newberry WN, Haut RC. Chronic softening of cartilage without thickening of underlying bone in a joint trauma model. *J Biomech*. 2000;33(12):1689-1694.

29. Table 103. US Department of Health and Human Services, National Center for Health Statistics. *Health, United States, 2009: With Special Feature on Medical Technology*. Hyattsville, MD: National Center for Health Statistics; 2010:368-371. http://www.cdc.gov/nchs/data/hus/hus09.pdf. Accessed October 5, 2011.

30. Ministry of Health. *The Health of New Zealand: Total Population*. Wellington, New Zealand: Ministry of Health; June 2004:2,7,8.

31. Swinburn BA, Ley SJ, Carmichael HE, Plank LD. Body size and composition in Polynesians. *Int J Obes Relat Metab Disord*. 1999;23(11):1178-1183.

32. Deurenberg P, Yap M, van Staveren WA. Body mass index and percent body fat: a meta analysis among different ethnic groups. *Int J Obes Relat Metab Disord*. 1998;22(12):1164-1171.

33. Craig P, Samaras K, Freund J, Culton N, Halavatau V, Campbell L. BMI inaccurately reflects total body and abdominal fat in Tongans. *Acta Diabetol*. 2003;40(suppl 1):S282-S285.

34. Pritzker KP. Crystal deposition in joints: prevalence and relevance in arthritis. *J Rheumatol*. 2008;35(6):958-959.

35. Robertson CJ. Joint crepitus—are we failing our patients [published online ahead of print October 28, 2010]? *Physiother Res Int*. 2010;15(4):185-188. doi:10.1002/pri.492.

36. Grahame R. How often, when and how does joint hypermobility lead to osteoarthritis? *Br J Rheumatol*. 1989;28(4):320.

37. Resnick D. Patterns of migration of the femoral head in osteoarthritis of the hip: roentgenographic-pathologic correlation and comparison with rheumatoid arthritis. *Am J Roentgenol Radium Ther Nucl Med*. 1975;124(1):62-74.

38. Garbuz DS, Xu M, Duncan CP, Masri BA, Soboley B. Delays worsen quality of life outcomes of primary total hip arthroplasty. *Clin Orthop Relat Res*. 2006;447:79-84.

39. Röder C, Staub LP, Eggli S, Dietrich D, Busato A, Müller U. Influence of preoperative functional status on outcome after total hip arthroplasty. *J Bone Joint Surg Am*. 2007;89(1):11-17.

40. Johansson HR, Bergschmidt P, Skripitz R, Finze S, Bader R, Mittelmeier W. Impact of preoperative function on early postoperative outcome after total hip arthroplasty. *J Orthop Surg (Hong Kong)*. 2010;18(1):6-10.

41. Rampichová M, Filová E, Varga F, et al. Fibrin/hyaluronic acid composite hydrogels as appropriate scaffolds for in vivo artificial cartilage implantation. *ASAIO J*. 2010;56(6):563-568.

42. Rossignol M, Leclerc A, Allaert FA, et al. Primary osteoarthritis of hip, knee, and hand in relation to occupational exposure. *Occup Environ Med*. 2005;62(11):772-777.

43. Lequesne MG, Dang N, Lane NE. Sport practice and osteoarthritis of the limbs. *Osteoarthritis Cartilage*. 1997;5(2):75-86.

44. Arokoski MH, Arokoski JP, Haara M, et al. Hip muscle strength and muscle cross sectional area in men with and without hip osteoarthritis. *J Rheumatol*. 2002;29(10):2185-2195.

45. Hurley MV, Newham DJ. The influence of arthrogenous muscle inhibition on quadriceps rehabilitation of patients with early, unilateral osteoarthritic knees. *Br J Rheumatol*. 1993;32(2):127-131.

46. Rahmann AE. Exercise for people with hip or knee osteoarthritis: a comparison of land-based and aquatic interventions. *Open Access J Sports Med*. 2010;1:123-135. doi: http://dx.doi.org/10.2147/OAJSM.S6941.

47. Hunter DJ, Neogi T, Hochberg MC. Quality of osteoarthritis management and the need for reform in the US [published online ahead of print June 25, 2010]. *Arthritis Care Res (Hoboken)*. 2011;63(1):31-38. doi:10.1002/acr.20278.

48. Kurtz S, Ong K, Lau E, Mowat F, Halpern M. Projections of primary and revision hip and knee arthroplasty in the United States from 2005 to 2030. *J Bone Joint Surg Am*. 2007;89(4):780-785.