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Not the Last Word

Not the Last Word: Arthrostasis

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Whenever I encounter yet another article chiding surgeons for performing too many spinal fusions for back pain—the "bane of health wonks everywhere" [9]—I try to think of orthopaedic operations that really work. I think about hip replacement, the "operation of the century" [8], and the patients who would be stuck on the couch without it. I think about surgical drainage of joint infections, and its powers to preserve articular cartilage. I think about the nailing of femur fractures—an advance over casting or traction that is almost immeasurable.

These three operations share a special feature: Clinical effectiveness. Yet upon closer examination, hip replacement does not belong with the other two. Hip replacement does not restore the joint to health; rather, the surgeon has to destroy the joint in order to save it. (As Wroblewski [10] noted, "It is essential to understand and accept the very simple fact: The arthroplasty is a foreign body bursa housing a neuropathic spacer"). By contrast, fracture fixation and drainage procedures are operations that seek to restore homeostasis.

A fractured femur fixed with a nail invokes the steps of endochondral ossification and can (with at least some patterns of injury) heal the break with scant evidence of the original damage. An infected joint that is drained expeditiously can clear the bacteria, dilute the inflammatory enzymes, and let a normal joint surface endure. Next to these marvels, hip replacement—cutting out the proximal femur, excavating the acetabulum, and pounding in pieces of metal and plastic—is biologically barbaric.

Shouldn't we care more about restoring joint homeostasis? Why wouldn't we?

I submit that the best treatment of osteoarthritis will be one that enhances joint homeostasis, restoring normal anatomy and biology—a process I will label "arthrostasis." At the same time, I will fully concede that conceiving, implementing, and validating arthrostatic treatments will be inordinately difficult.

The first road block is human physiology. Osteoarthritis is a complex disease of synovium, bone, and cartilage. Among these, the problem of healing cartilage alone looks insurmountable. For reasons we can only guess at, evolution has decided to take us this far without a means for regenerating articular cartilage. As such, if we aim to develop an arthrostatic treatment for osteoarthritis, scientists will first have to devise biological processes that do not naturally occur.

And how are we going to convince patients to accept such a treatment? At present, we can offer an operation for hip arthritis that reliably relieves pain and restores function. Even with the risks of postsurgical complications and implant failure over time, hip replacement is still an attractive bargain for patients who will be otherwise

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sentenced to inactivity. Arthrostasis, on the other hand, will be a much harder sell: The benefits of restoring normal anatomy and biology might include long-term prevention but perhaps less immediate relief. (The discounted value ascribed to late benefits is critical, as most patients have not mastered deferred gratification. More than one patient in my own practice, for example, requested partial meniscectomy over meniscal repair: They were willing to forgo a repair that sought to restore homeostasis just to get back on the court a few months sooner).

We also have the problem of validation. The major benefit of procedures that enhance arthrostasis is the decreased incidence of end-stage arthritis, yet even without an intervention, arthritis will not arise in all patients at risk. Accordingly, studies that can validate that these treatments will be, by their very nature, large-scale and long-term, with plenty of noise. And until those hard-to-obtain data are in fact, obtained, nudniks like me [1] might question whether the intervention is really more myth than fact.

Despite all of this, I am optimistic. The dentists have shown us that therapeutic paradigms can shift. Fifty years ago, the problem of dental cavities—rotting teeth—was addressed by filling small defects and resecting and replacing larger ones. Yet at the same time, an assault on a second front, prevention, was carried out with water fluoridation and hygiene programs. These efforts have succeeded admirably.

We can probably rest easy when health wonks pick on surgery for back pain. Spinal fusion is not only the opposite of homeostatic, it is also terribly expensive, inconsistently applied, and variably effective. (Extracting rotten teeth was equally lacking in homeostasis, but at least it was cheap and effective). Surgical treatments for back pain will likely fade on their own accord. When that happens, a more arthrostatic approach can thrive in its place. Hip replacement is a much harder problem—precisely because it is so good. For some conditions like hip arthritis, we are prisoners of our own success.

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I am pleased that Dr. Bernstein has taken the bold step of recognizing and recommending the concept of joint homeostasis (or "arthrostasis") as a viable and desirable therapeutic goal for orthopaedic surgeons. Most orthopaedists are unfamiliar with the concept of tissue/joint homeostasis, as they have been exposed almost exclusively to imaging modalities that provide only structural and anatomic data (like radiographs, CT, and MRI) resulting in a limited and constrained view of musculoskeletal pathology. However, tissues are more than just structural components of joints: They represent volumes of living cells that are metabolically active in order to maintain a specific range of physiological characteristics under normal circumstances (homeostasis), and to repair themselves if damaged (loss of homeostasis). A joint in homeostasis is, by definition, pain-free.

We currently require a metabolically oriented imaging modality, such as a Technetium bone scan (my favorite because it is widely available, and has relatively low radiation exposure), SPECT-CT, or PET-CT, to assess the quality of homeostasis in living joints. Yet we do not need such sophisticated imaging to effectively manage many clinical conditions from a joint homeostasis perspective, such as symptomatic osteoarthritis of the knee. If a knee is hot and swollen, imaging to determine that the joint is not in homeostasis is unnecessary. Often the simple, logical (but clinically powerful) approach of decreasing the load across the knee to within that joint's "envelope of function or envelope of load acceptance" [3], along with cooling and some form of

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antiinflammatory medication, can be surprisingly effective.

We have even shown that with the religious use of a simple lateral sole wedge, not only was the considerable pain of advanced osteoarthritis of the medial compartment of the knee nearly completely resolved, but that parts of the "hot" pretreatment bone scan activity diminished substantially as well, demonstrating "disease-modifying" properties of the gentle, nonoperative, joint homeostasis approach as espoused by Dr. Bernstein.

The value of a joint homeostasis perspective extends well-beyond the treatment of established osteoarthritis, however. This new, emerging musculoskeletal paradigm can provide clarity of etiology as well as safe therapeutic recommendations for a variety of conditions of great interest to orthopaedic surgeons. Some common examples include patients with patellofemoral pain [4]; when to safely "return to sport" following injury and/or surgery [7]; and the early detection and prevention [5] of posttraumatic osteoarthritis at a "prearthritis" stage [2].

Finally, this new paradigm has even proven its worth in a difficult spinal condition. I know a middle-aged physician who was encouraged by three different spine surgeons to have a 3-level cervical fusion with multiple foraminotomies for intermittent neck pain. A bone scan revealed, however, that only one facet pair demonstrated increased radiotracer uptake, which matched the site of his symptoms. He decided to "get into his symptomatic joint's envelope" by persistently avoiding certain neck positions. Eighteen months later, his symptoms had fully resolved along with his abnormal bone scan without having to resort to inherently dangerous surgery [6].

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A few weeks ago, I was telling my sports medicine friend about the newfound knee pain I felt while running. I carefully described the location (or its vagueness), quality, intensity of the discomfort, and embellished it with mitigating and exacerbating factors. I was so sure that the sharp intellect of my friend-healer would provide an anatomic diagnosis and treatment.

"Welcome to your 40s," he said. "Alter your foot-strike pattern and take some NSAIDs." Lo and behold, it worked! But I am certain that my knee did not return to an arthrostatic state. I know this because my joint, ever since it was shocked into disequilibrium by an ACL disruption more than two decades earlier, has been slowly working itself away from arthrostasis. My joint has been undergoing perhaps an accelerated form of aging—an inexorable end for all tissues and especially cartilage. That is where a real challenge lies.

Getting a femur fracture to heal is easy (I am a trauma surgeon) because biology is on our side. Despite the fact that fractures do not heal as well with age, the cellular and molecular pathways that allow for homeostasis are mostly preserved from embryogenesis; Hedgehog, Notch, BMP, and Wnt pathways all fire up their engines—although some undergo age-dependent decline—with injury. Regenerative mesenchymal stem cells become active, replicate, migrate, and do what they need for bone. With cartilage, not so much. To me, that means "prophylactic" therapy must be initiated in childhood before the cells forget their days of replenishment and rejuvenation. Otherwise, we are, as Dr. Bernstein alludes, trying to fool the body into doing something it naturally cannot. The halting and reversal of joint degeneration poses perhaps even more formidable obstacles—to stop and reverse the seemingly inexorable march of aging or a chronic time-dependent decline of tissue properties and function. When you consider this, our barbaric resection of degenerative tissues (like a tumor) seems pretty liberating. Is the best we can hope

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(strive) for now something like a statin drug for joints? Can we achieve this systematically and methodically? Perhaps, but it would certainly help to have a few strokes of Nobel-worthy genius a la Brown and Goldstein (who were recognized for their work on cholesterol metabolism with the 1985 Nobel prize). Any takers out there?

When I teach medical students, we talk about joints as a machine that allow for controlled rotational motion. We could theorize that if the mechanical integrity is there (avascular necrosis with collapse would be a case of loss of this integrity), the joint could still function, albeit not as efficiently as if the cartilage were there, if we could alleviate the associated pain. In this sense, the achievement of a painless state could be a goal for a new functional arthrostasis. Although certainly not the Holy Grail, this could still be an alternative and more realistic next step. In the future, could a combination of engineering and biological feats allow for a living (and cartilage surfaced) replacement with a 50-year survivorship? This would not obviate the need to cut, dissect, and resect, but it would be yet closer to true arthrostasis.

Finally, I do hope that someday we can liberate the power contained within the cells that produces and maintains cartilage as every nucleus has all the coding information it needs to recapitulate embryology. After all, its mineralized mesenchymal sibling has known all along how to strive for osteostasis.

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Dr. Bernstein's column describes the potential advantages and possible difficulties with interventions that seek "arthrostasis." After reading his column, I came to the conclusion that treatment and prevention are often confused, and that we need to invest more in prevention. This is particularly true where tissues that cannot regenerate are concerned—pancreatic cells, neurons in the brain and spine, and the myocardium all come to mind. For now, and probably for the forseeable future, cartilage is on this list. When those cells are damaged, we have no treatment that can heal them. For those tissues, prevention is much more important than treatment, because treatment can do little more than provide a fair (or poor) substitution for those organs' original function, or even worse, merely relieve (or palliate) symptoms.

We should draw a clear distinction between treatment and prevention. The goal of prevention is to avoid the future development of the pathology, whereas treatment heals or mitigates the suffering and consequences of the established disease.

To analyze therapies for an established disease, we must first: (1) Understand the degree, extension, and pathology of the disease, and (2) Come to know the results, benefits, risks, advantages and disadvantages of the treatment we propose. In practice, doing this well is very, very difficult, and doing it incompletely renders our patients vulnerable to the many unintended consequences of treatment we observe in daily practice. For this reason, it is important that we focus more energy on preventing these problems from arising in the first place.

To prevent musculoskeletal disease, we must promote musculoskeletal health with the same vigor as physicians who promote cardiovascular health. Our patients must understand that a sedentary lifestyle will likely result in chronic pain and functional disability as they get older.

Until we truly have treatments that deliver on the promise of arthrostasis, the best way to prevent osteoarthritis is to promote musculoskeletal health.

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