Perception is everything

Perhaps the most important change in OA over the past 60 years has been in attitudes rather than knowledge. OA has changed from being a boring, degenerative form of joint pathology, for which nothing could be done [1, 2], to an exciting clinical disorder of the joints that can be manipulated and improved [3, 4]. Unfortunately, that change has yet to be fully embraced or understood by all the health-care professionals dealing with musculoskeletal disorders, or by the vast numbers of people in the community with pain and disability associated with their OA. However, as perception is everything, one of our major current challenges is to spread the word.

OA has become exciting.

This change in attitude has resulted from the introduction of new technologies—such as MRI and biomarkers for OA—and from clinical research in patients and populations.

New technologies leading to new concepts

Until relatively recently, there were only two ways of looking at joints affected by OA: X-rays [5] or gross anatomy [1]. A striking pathological feature of joints affected by OA is focal areas of cartilage destruction. If advanced, this leads to joint space narrowing on the X-ray. Therefore, we should not be surprised that interest in OA centred on this destruction of articular cartilage, as all we had to go on was X-ray changes and gross pathology.

Arguably, however, that obsession with pathology, X-rays and cartilage damage, which was led from the UK, and that stretched from the 1950s to the beginning of this century [1, 5, 6], set the field back, perpetuating a negative view of the condition among clinicians. Newer imaging modalities, including bone scans and MRI [7], as well as biomarkers [8], have made it abundantly clear that the OA process affects all joint tissues, and, along with more sophisticated approaches to pathology, have shown us that it is an active repair process and not purely a degenerative or destructive one [9, 10, 11].

- OA is not a cartilage problem [10].
- OA is not a degenerative disorder [11].

During the years of focus on articular cartilage, fundamental research into OA was dominated by biochemists and cellular biologists who did wonders in sorting out the biology of cartilage, but largely failed to understand that OA is primarily a mechanical problem. There was a failure to appreciate the importance of the old observation that OA pathology is focal, not generalized, and that the damage only occurs in habitually loaded areas of a joint.

Joint failure and abnormal mechanics

OA is not a discrete disease entity, it is joint failure, akin to cardiac or kidney failure; and, like heart or kidney failure, it can be asymptomatic. Furthermore, just as the heart can fail as a result of primary problems in the endocardium, myocardium or epicardium, joint failure can result from problems in subchondral bone, cartilage, ligaments, periarticular muscles, nerves or synovium, and OA can originate in any of these or other tissues [11].

The joint is a mechanical structure, and the key to understanding OA is abnormal mechanical stress: joint failure is the pathophysiological response of a synovial joint to mechanical insult, and the attempt of the joint to repair the damage caused by local abnormalities in force/unit area. The abnormalities in cytokines, degradative enzymes, toxic radicals and the like, which are being studied as the cause of OA, are rather the result of this attempted repair [9, 11]. Therefore, thinking about OA is moving from biochemistry of the articular cartilage to the mechanobiology of the whole joint.

- OA is joint failure rather than a disease.
- Joint failure is driven by abnormal joint loading.

From patients to populations

Everyone knows that OA is common, that it can be asymptomatic, and that most of those who seek help are managed in primary care. What is less well known is the fact that many of those who become symptomatic never seek any medical help for their OA-related problems [12]. Therefore, if we confine our studies to patients with OA, we will get a strangely biased view of the condition. Until recently, that is exactly what was going on, but one of the big changes that occurred over the last 50 or 60 years has been the development of population-based studies of OA, from which we have gained new insights into risk factors, progression and outcomes [13].

One of the more important findings from such studies has been the fact that OA is not necessarily a progressive disorder. After a period of time during which the OA process (attempted containment of abnormal forces and
repair of damage) is active, and the anatomy of the joint changes, the condition then stabilizes in most cases, although it can reactivate years later. This is not apparent from X-rays, as they will show the same anatomical changes, whether the process is active or stable, but dynamic imaging modalities, such as bone scintigraphy, have clearly shown that the process can switch itself on and off [14].

- OA is not always a progressive disease process.

In retrospect, this is obvious. If we compare the numbers of people who have radiographic evidence of OA in the population with the numbers who have severe pain or disability, or the numbers who come to joint replacement, there is a huge mismatch [15]. Thus, most people who develop changes evidenced by X-rays must stabilize.

**Understanding pain and disability**

OA would not pose so much of a problem if it was not associated with pain and disability: sometimes it is and sometimes it is not. We have known for years that many people in the community have radiographic evidence of severe OA but have no symptoms [5, 16]. This has confused those of us wedded to the biomedical model of disease—to the belief that diseases cause tissue damage, which results in symptoms. That thinking is still apparent in the rather desperate attempts that some researchers are currently making to show that if we could only image or assess the pathology of OA better, we would find that there is a good correlation between joint changes and symptoms.

But pain and disability are more complex and more interesting than that. Studies of pain in OA are helping rheumatologists and orthopaedic surgeons understand some of the elegance and complexity of nocioception and its sensitization and control within the peripheral nervous system and CNS [17]. For example, if the OA process sets off noxious pain (which it sometimes does), this can result in pain sensitization and perpetuation of the pain even if the noxious drive (the OA process) stops [16, 17]. Similarly, we are gaining new insights into the complex determinants of disability in people with OA. It would appear that having OA alone is generally not a cause of much disability (particularly if it is not causing pain), but if painful OA occurs in combination with some other health problem, such as reduced vision, severe disability can be the outcome [18]. These insights will open up new approaches to the management of OA.

- OA alone does not cause pain or disability.

**Future management**

What of the future? It seems to me that after years in the doldrums, we are now in a position to move forward in OA, and find new approaches to the control of the disease process and of pain and disability. But these advances will not come if we only use the biological approaches that have so successfully led to the control of inflammatory arthritis. The OA process is driven by abnormal mechanical forces on a joint, so the answer must come from biomechanics as much as biology. Proof-of concept studies are already available, through, for example, the improvements that can occur in both symptoms and joint pathology following osteotomy or joint distraction to unload damaged areas of the joint [19]. Pain and disability require a biopsychosocial and holistic approach to the multiple problems of older people. As with any chronic disease, management of OA is complex and needs individualizing [20].

The problem we have is that very few people are actively researching OA, because the old concept of it as a boring age-related, degenerative disease persists. My plea to the next generation of rheumatologists is to take up the exciting challenge of OA.

**Acknowledgements**

Many of the ideas presented in this article have come from discussions I have had, and articles I have written [9, 10, 11, 20], with two specialists in the field of OA research: Ken Brandt and Eric Radin. I thank them for their friendship, generosity and scholarship.

**Disclosure statement:** The author has declared no conflicts of interest.

**Paul Dieppe**

1Peninsula Medical School, Universities of Exeter and Plymouth

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