

Osteoarthritis and Cartilage



Review

Q4 Defining the osteoarthritis patient: back to the future

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SUMMARY

The history of osteoarthritis (OA) is important because it can help broaden our perspective on past and present controversies. The naming of OA, beginning with Heberden's nodes, is itself a fascinating story. According to Albert Hoffa, R. Llewellyn Jones and Archibald Edward Garrod, the name OA was introduced in the mid-nineteenth century by surgeon Richard von Volkmann who distinguished it from rheumatoid arthritis and gout. Others preferred the terms 'chronical rheumatism', 'senile arthritis', 'hypertrophic arthritis' or 'arthritis deformans'. A similar narrative applies to the concept of OA affecting the whole joint vs the 'wear-and-tear' hypothesis, inflammation and the role of the central nervous system (CNS). In the late nineteenth and early twentieth centuries, the Garrods (father and son) and Hermann Senator argued that OA was a whole joint disease, and that inflammation played a major role in its progression. Garrod Jnr and John Spender also linked OA to a neurogenic lesion 'outside the joint'. The remaining twentieth century was no less dynamic, with major advances in basic science, diagnostics, treatments, surgical interventions and technologies. Today, OA is characterized as a multi-disease with inflammation, immune and CNS dysfunction playing central roles in whole joint damage, injury progression, pain and disability. In the current 'omics' era (genomics, proteomics and metabolomics), we owe a great debt to past physicians and surgeons who dared to think 'outside-the-box' to explain and treat OA. Over 130 years later, despite these developments, we still don't fully understand the unravelling complexities of OA, and we still don't have a cure.

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The disease called chronical rheumatism, which often passes under the general name of rheumatism and is sometimes supposed to be the gout, is in reality a very different distemper from the genuine gout and from the acute rheumatism, and ought to be carefully distinguished from both.

William Heberden (1816)¹, Appendix, p 414

Osteoarthritis: prevalence and major gaps

Osteoarthritis (OA) is one of the most prevalent and disabling chronic degenerative diseases and some predict a 7-fold increase by 2030². It affects over 250 million people worldwide, and impacts more than half of the over 65 population, with higher rates of disability in woman than men^{2,3}. Like many chronic diseases, the incidence of OA continues to rise from an ageing population, rapid rise in obesity, unhealthy dietary patterns, physical inactivity and chronic stress^{3–7}. There are many gaps in our understanding of OA, including the underlying drivers of its evolving pathophysiology, inflammation and the role of the central nervous system (CNS)^{2,3}. The aim of this narrative is to focus on some key characteristics that define the OA patient from a historical perspective to the present, and examine how many of the ongoing controversies were raised over 130 years ago. We also discuss the historical importance of a whole body systems-based approach to OA, which we believe is a key consideration for potential targets for future therapies.

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What's in a name? The fight for OA's clinical and pathological independence

It is not to be expected that there should be agreement about the definition of anything until there is agreement about the thing itself.

John Stuart Mill (1806–1873)

In 1816, Heberden clearly understood that a distinction was necessary to describe his “*digitorum nodi*” from gout and rheumatoid arthritis (RA) (Fig. 1, see first quote above), however, he left it to others to formulate a name. It is often reported that the designation OA was introduced in 1890 by Sir Archibald Edward Garrod in his famous *A Treatise of Rheumatism and Rheumatoid Arthritis*⁸. However, Garrod placed the name ‘oste-arthritis’ seventh on a list of eleven, and stated a preference for ‘arthritis deformans’⁹ p 234-5. The term OA appears to have originated earlier, in the mid-1850s, by one of the fathers of orthopaedic surgery, Richard von Volkmann (1830–89)¹⁰. This new information was not recognized until the turn of the twentieth century when von Volkmann's pioneering studies were revisited independently by Albert Hoffa (1859–1907), R. Llewellyn Jones and Garrod himself^{11–13}. All three maintained that von Volkmann clearly differentiated OA lesions anatomically and pathologically

from RA^{11,12}, and showed that the initial changes in RA began in the synovial membrane, with cartilage deterioration being affected secondarily¹². According to Jones, von Volkmann's work underwent “an almost total eclipse” as the field was largely dominated by Charcot and Trastour's monograph (1853), which maintained that OA and RA were different ‘grades’ of the same entity, ‘arthritis deformans’^{12,14}. Charcot and Trastour's interpretation was endorsed and widely publicised by the authority Rudolph Virchow in his *Cellular Pathology* (1858) (Fig. 1). In the fullness of time, however, von Volkmann's sharp anatomic and pathologic distinctions were validated by other physicians and surgeons around the world including Hoffa, Hueter, Samaran, Waldmann, and Wollenberg¹¹.

On the question of designation, Benedek also points out that the term OA may have been proposed in 1863 by the Nomenclature Committee established by the Royal College of Physicians of London¹⁵, which Garrod apparently referred to in his 1890 treatise. As a consequence of time to reach consensus for differentiating OA's primary bony/cartilagenous (osteo-) defect with secondary synovium complications from RA (infective and other variants), OA was not widely recognised as a distinct entity until the mid twentieth century^{13,14} (Fig. 1). Up until that time, ‘arthritis deformans’ remained the preferred term, with a number of less common names including *hypertrophic arthritis*, *chronic articular rheumatism*, *nodular rheumatism* and *senile arthritis*¹⁴.

Brief History of Osteoarthritis from 1800s to the present

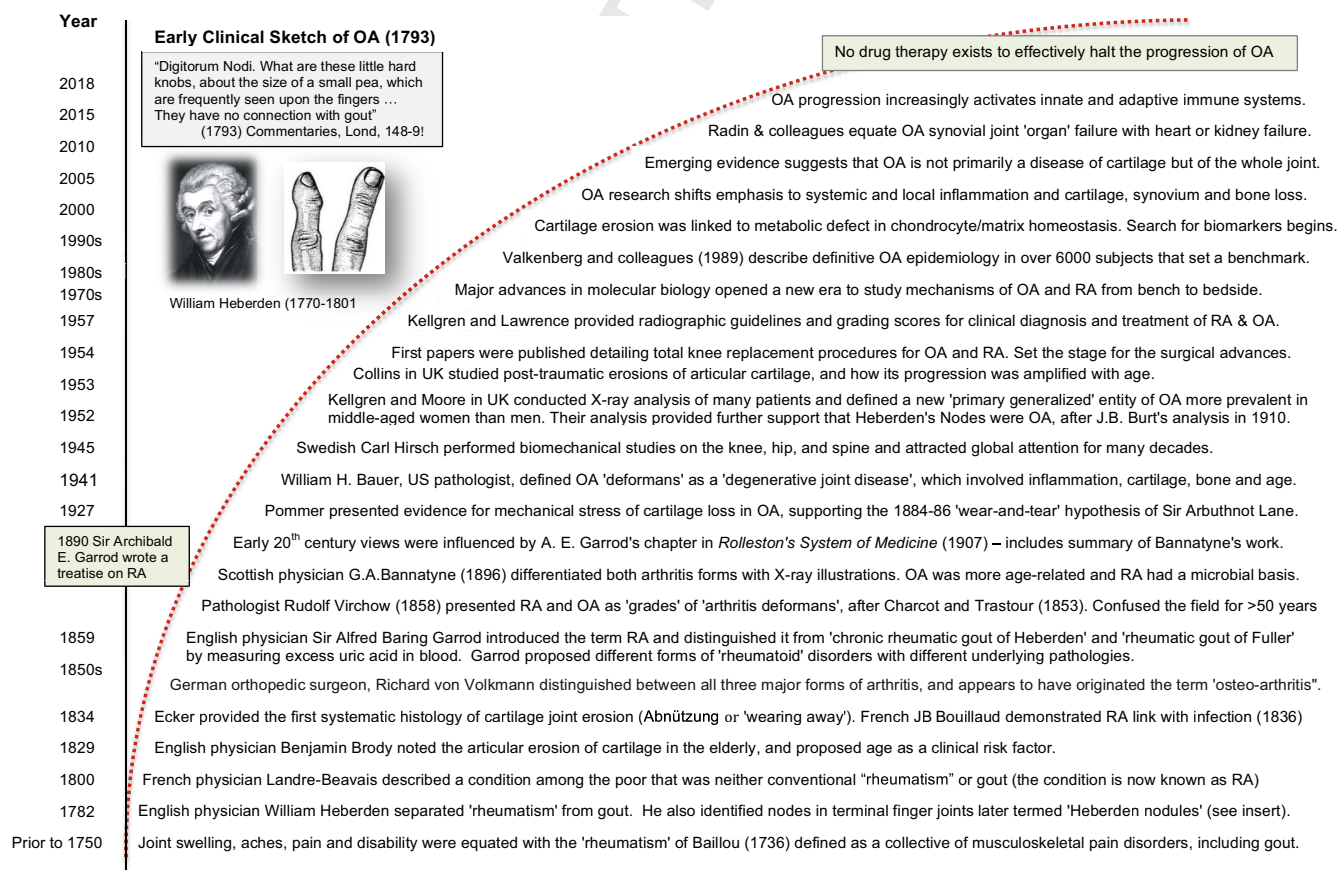


Fig. 1. Brief history of osteoarthritis (OA) showing the rise of its clinical and pathological independence from rheumatoid arthritis (RA) and gout. History provides a rich record of events as new knowledge and methods are developed, and a window into how active and insightful many of the early academic discussions on arthritis were in relation to the present. Despite extraordinary advances over the past 200 years, we still don't fully understand the underlying aetiology of OA and we still don't have a cure (see text).

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