

**IDENTIFICATION AND INTERPRETATION
OF JOINT DISEASE IN PALEOPATHOLOGY
AND FORENSIC ANTHROPOLOGY**

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PREFACE

Joint disease is an important focus in the fields of paleopathology and forensic anthropology because it represents a significant proportion of the pathological lesions identified in skeletal remains. Thus, lesions indicative of joint disease form a crucial component of the construction of osteobiographies and offer insights into individual life histories and ancient lifeways. Our aims in this guide to the identification and interpretation of joint disease are (1) to identify the diagnostic criteria that are relevant to investigations of joint disease in dry and macerated bone specimens; (2) to differentiate between various disease forms; and (3) to highlight contentious issues, such as the antiquity of rheumatoid arthritis and the implications of the prevalence and severity of joint disease for reconstructing the behaviours of past peoples and recently deceased individuals. We advocate the use of unambiguous terminology and hence discuss descriptive terms and illustrate how the use of colloquial or otherwise inappropriate terms can lead to errors of interpretation. And, since many users of this manual will not be specialists in rheumatology or osteology, we include a glossary that defines some of the specialist terminology that is in use currently.

Joint disease causes proliferative and/or erosive bony lesions that preferentially, but not exclusively, affect the synovial joints of the body (Appendix 1). Hence, this manual emphasizes diseases that affect the synovial joints. We use the term arthritis to refer to both inflammatory and non-inflammatory arthropathies, in keeping with the terminology in current use, but readers will find that some scholars, particularly outside of North America, prefer the term arthrosis when referring to forms of joint disease in which inflammation is not the principal feature.

In the sections that follow, we review the pathogenesis, disease process, anatomical distribution, and diagnosis of osteoarthritis; multifocal erosive arthropathies (i.e., rheumatoid arthritis and the seronegative

arthropathies); the less common diseases of synovial joints, including gout, juvenile idiopathic arthritis, and septic arthritis; and conditions affecting the non-synovial joints of the spine such as spinal osteophytosis, degenerative disc disease, Schmorl's nodes, and the seronegative spondyloarthropathies.

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Chapter I

OSTEOARTHRITIS

Although the first use of the term osteoarthritis occurred in 1889, as a degenerative disease, osteoarthritis (OA) has been differentiated in the medical community only since the nineteenth century, when a non-inflammatory arthritis of the elderly was distinguished from inflammatory arthropathies (Dieppe, 2008; Rogers & Waldron, 1995; Sokoloff, 1969).

There is no universally accepted term for the disease referred to in this manual as OA. The terms degenerative arthritis, degenerative arthrosis, and degenerative joint disease emphasize the progressive decline in quality of the joint(s), although Waldron (2009:27; 2012) argues against these terms because the joint changes in dry bone represent a reparative process. The most commonly used terms are osteoarthritis and osteoarthrosis. Osteoarthrosis is the preferred term for some, as inflammation is not a major characteristic of the disease, but the existence of an inflammatory component validates both terms (Resnick, 2002a; Weiss & Jurmain, 2007). Other scholars have argued that since the disease originates in cartilage rather than bone, and only eventually results in the failure of a synovial joint, its pathogenesis is more properly described by the terms chondroarthritis or chondroarthrosis (Hunter & Eckstein, 2009; Resnick, 2002a); these terms are used rarely in the paleopathological literature, however. Given the current uncertainty with regards to pathogenesis, etiology, and classification, current labelling practices may inadvertently include more than one disease under this rubric (Weiss & Jurmain, 2007).

PATHOGENESIS AND DISEASE PROCESS

The condition is characterized mainly by deterioration of the bone and cartilage of one or more joints. Usually, a progressive wearing down of apposing joint surfaces results in distortion of joint position, and produces pain, swelling, and stiffness in the living individual.

The typical age for the onset of OA is during middle or old age, and hence the disease was previously regarded as an unavoidable consequence of the aging process. Researchers assumed that, given their poor vascularization, **chondrocytes** had minimal repair capacity (Iannone & Lapadula, 2003; Sokoloff, 1969). Thus, in that model, OA is seen as a straightforward “wear and tear” consequence of repeated mechanical stress (Iannone & Lapadula, 2003). Current research, however, indicates that the pathogenesis of OA is a great deal more complex. Chondrocytes are, in fact, metabolically active, and their metabolism is actually enhanced in the early stages of OA (Iannone & Lapadula, 2003; Sokoloff, 1969). A breakdown of **extracellular matrix** (due to the activity of various proteins and hormones) causes an initial inflammation that encourages damage (Arden & Cooper, 2006; Berenbaum, 2008; Iannone & Lapadula, 2003). Essentially, the pathogenesis of OA appears to be a multifactorial loss of homeostasis at the cellular level (Hunter & Eckstein, 2009). This is exacerbated by increased friction in aged joints, as well as **hypertrophic** maturation of some chondrocytes (Berenbaum, 2008).

In addition to the intrinsic factors described above, four extrinsic factors can influence the development of OA: systemic risk, localized biomechanics (e.g., joint loading), genetic predisposition (i.e., some individuals are predisposed to hypertrophic bone response), and environment (Berenbaum, 2008; Brown et al., 2008; Ortner, 2003; Rogers et al., 1997; Waldron, 2009). The complexity of the relationships among its causative factors thus leads to great variability in the expression of the disease. Mechanical factors plus individual and populational systemic factors all interact to produce individual manifestations of disease. When combined with variation in research methodology and diagnosis, inter-population comparisons are fraught with difficulty. Thus, qualitative trends, rather than quantitative conclusions, seem to best describe observations of the expression and prevalence of the disease (Waldron, 2012).

It is not clear if cartilaginous change always precedes subchondral change. On one hand, repeated microfractures could modify the biomechanical properties of cartilage and the growth of bone, resulting in

the development of **osteophytes** and **osteosclerosis** (Berenbaum, 2008), two of the most commonly observed bony responses in OA. By contrast, although Dequeker and co-workers (1996) support a subchondral reaction as the initiating cause of OA, they suggest that it results from general change and slowing of turnover rather than microtrauma.

The cartilage progression of OA occurs in three stages. First, microcracks appear and chondrocytes proliferate. Second, the microcracks deepen and produce fissures, causing cartilage to fragment into the articular cavity, which exposes subchondral bone. Third, the subchondral bone develops microcysts, or **erosions**. Synovial inflammation occurs at this stage, along with sclerosis of the subchondral bone and formation of osteophytes, which may represent attempted repair of the joint (Berenbaum, 2008; Waldron, 2009). Osteophytes may provide a broader load bearing area that stabilizes the joint, although they may, alternatively, impede function of the joint (Hunter & Eckstein, 2009; Ortner, 2003; Ortner & Putschar, 1985; Rogers & Waldron, 1995; Sharma, 2003).

If enough cartilage is lost, the adjacent bones come into direct contact with each other, leading to abrasion of the subchondral surfaces. This causes osteoclastic resorption and the death of osteocytes in deeply eroded areas. In addition, **eburnation** of the sclerotic surface through mechanical attrition occurs at the point of maximum mechanical loading (Ortner, 2003; Ortner & Putschar, 1985; Rogers & Waldron, 1995). Some researchers believe this process occurs after years of initial degenerative and proliferative cartilage change (Ortner & Putschar, 1985), while others posit that bony and cartilaginous changes might occur simultaneously (Iannone & Lapadula, 2003). The new bone and fibrous or hyaline cartilage forms at the joint margins and subchondral plate. This new material gradually comes to echo a normal joint, albeit with significant bone formation in the underlying marrow and reorganization of internal epiphyseal architecture. Bone proliferates marginally in areas that are subject to the least compressive force. Unfortunately, the extent of the correlation between osteophyte development and erosion of the articular surface or eburnation is not clear (Ortner, 2003; Sokoloff, 1969).

CLINICAL DIAGNOSIS

The principal clinical symptom of OA disorders is pain. This includes use-related pain as well as a relatively short-lasting stiffness of joints after