Stories from the Skeleton

Behavioral Reconstruction in Human Osteology

Robert Jurmain

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Interpreting the Remains of the Past

A series edited by *Mary K. Sandford*, University of North Carolina–Greensboro

Volume 1
STORIES FROM THE SKELETON
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For

W. W. Howells and *T. Dale Stewart*

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INTRODUCTION TO THE SERIES

This series provides a forum for presenting innovative ideas and methods relative to our understanding of the human past. The concept developed during preparation of my edited work, *Investigations of Ancient Human Tissue: Chemical Analyses in Anthropology* (Gordon and Breach, 1993). While researching and writing that volume, I became acutely aware of the need for comprehensive and timely works focused on topics within the intersection of archaeology and physical anthropology.

That book examined the promise and pitfalls of using elemental and isotopic analyses in understanding past diets, nutritional patterns and disorders. Such topics, and the manner in which we elected to address them, influenced the scope and goals of the present series in several fundamental ways. The inauguration of these analytical techniques in anthropology signaled intensification of multi-disciplinary approaches. These techniques made their debut in anthropology during the 1970s and the decade itself was one of fervor and optimism, as students seized upon such new technologies in hopes of gaining a better and more accurate understanding of the human past.

The enthusiasm that marked the introduction of trace-element analysis in anthropology was tempered by recognition of the vast complications surrounding its use. Moreover, as with any method adopted from another discipline, most anthropologists simply lacked the training necessary for using the techniques or interpreting data. Remembering this as we prepared *Investigations of Ancient Human Tissue* some two decades later, we endeavored to contextualize our case studies with basic information on both theory and method, striving to make these techniques more accessible and understandable to a larger number of our colleagues.

The need for work with the requisite breadth to explore the reaches of a multi-disciplinary perspective, or the depth to probe the intricacies of a specialized technique, is even more compelling now. Indeed, what seemed to be quite extraordinary a mere twenty years ago has been far outpaced by innovations and discoveries of today. Scientific visualization and digital technology have revolutionized our ability to visually assess and quantify the objects of our investigations, while providing us with the means, through virtual technology, to share our latest findings with colleagues around the world. Advances

in biotechnology have expanded the bounds of our imagination; the ability of extract DNA from tissue may help us resolve issues emanating from such concerns as the history of disease to the origins of humankind. As we approach the new millennium, it is staggering to contemplate the matters we will be discussing, debating and endeavoring to understand in another twenty years. In providing a forum for cutting-edge ideas and techniques, it is my hope that this series will serve to both chronicle and propel our understanding of the human past.

Mary K. Sandford

PREFACE

While youth fosters enthusiasm as well as occasional intemperance, age—and with it, one hopes, experience—promotes reflection and reassessment. So it can be also with scientific inquiry. Certainly during my twenty-five years of research in paleopathology, I have often found it necessary to reflect upon much of my earlier work. Beginning at the Smithsonian Institution, my studies of osteoarthritis, especially, formed the primary thrust of my research in the 1970s.

At the Smithsonian, following the lead and encouragement of Donald Ortner, I began investigating patterns of degenerative disease in human skeletal populations. From his own provisional and innovative analysis of elbow osteoarthritis, Ortner suggested I further pursue a similar line of inquiry, most especially focusing on patterns of arthritic involvement in Inuit elbows. Ortner's direction proved to be quite inspired, as I did find what appeared to be clear patterning of elbow involvement, most notably as evidenced among the Inuit.

The primary hypothesis guiding most of my early investigations centered on establishing a link between *activity* and specific skeletal involvement of osteoarthritis. It seemed to me (and others) that the Inuit elbow evidence *proved* what has been termed the "stress hypothesis." Extrapolating from this empirical foundation, along with others, I postulated that much of OA patterning reflected activity; this assumption was rather easily extended to a variety of joints in a variety of populations.

None of this is to deny that activity cannot influence the onset of osteoarthritis; considerable skeletal and clinical evidence argues that in certain circumstances it can. Such observations have merit and are potentially informative, both to osteologists and clinicians. What was not generally critically assessed by osteologists like myself was the complexity concerning etiology of the condition. Further, this complex etiology limits considerably the capacity for osteologists to *predict* activity from the skeletal patterning of osteoarthritis. For me, however, it would take some time and further experience to recognize these limitations.

Sources of this experience were varied. First, from ongoing research on skeletal remains of Central California Indians I came to realize the patterns of degenerative involvement apparently did not conform simply to expectations of the stress hypothesis. Second, as other investigators, including students, friends and my spouse, attempted to apply methodologies and interpretations similar to my

own, I also grew more skeptical. It is perhaps easier to see equivocal components of certain research designs when they are attempted by others. Last, I had the good fortune to discuss such mutual concerns with all these colleagues, and their insights helped me immensely.

Beyond osteoarthritis, in more recent years I have also directed research on other types of skeletal conditions. In particular, the interpretation of traumatic injuries as seen in both human and great ape skeletons has become a focus of interest. Here, as well as with osteoarthritis, interpretations by me and colleagues have centered around potential behavioral influences. Most notably, the interpretations of "parry" fractures and craniofacial injuries have stimulated a critical evaluation, including reassessment of my own prior views.

In addition, a variety of other skeletal manifestations have fairly recently been proposed as "markers" of activity (e.g., stress fractures, enthesopathies and aspects of bone diaphyseal geometry). In order to provide a consistent and critical assessment of the value of these varied techniques and approaches, each is reviewed.

A basic perspective followed throughout these discussions is incorporation of *both* clinical and anthropological data. Both sources of information are necessary, since the bones alone do not (and cannot) provide an independent test of verification. We can never be sure solely from analysis of skeletal material what activities produce what osteological changes. If there is any confident means by which activity can ever be predicted from skeletal markers, such verification must first be demonstrated from controlled data sources.

Clinical data obviously are not perfect sources of information. Limitations arise not only because of sampling difficulties and imperfect research designs; most fundamentally, interpretations of osteoarthritis, enthesopathies and so forth are clouded because physiological (and environmental) processes leading to these conditions are complex. Indeed, this lesson is probably the most basic one to be learned from the rich literature produced by biomedical research. We all approach our investigations with an array of preconceptions, indeed, biases. Yet, if osteological interpretations are to achieve any real rigor, we must recognize these biases and seek to evaluate our data more critically. This is certainly a more difficult path to follow than one assuming simple relationships between activity and specific bone response. It is also a different path from the one hoped for in youth.

ACKNOWLEDGMENTS

In reality, this work reflects a considerable portion of my professional career in human osteology. Thus, there are a number of people to whom I am greatly indebted for assistance with research, access to collections, discussion, collegiality and friendship. The listing here is not comprehensive and I regret any omissions. It is both a rewarding and humbling experience to document the retinue of individuals who have contributed so fundamentally to one's life and work. And it is more than being able to stand on the shoulders of giants, although I certainly had that opportunity through the training and guidance of W. W. Howells, T. Dale Stewart and J. Lawrence Angel. Further support of students, friends, family and colleagues has made my professional life possible.

For assistance with my initial research on osteoarthritis at the Smithsonian Institution and Harvard I am indebted to Donald Ortner, Dale Stewart, Larry Angel, W. W. Howells, Jonathan Friedlaender, Henry McHenry, Jeff Froelich and Claudia Jurmain. My continued work on skeletal remains at San Jose State University has been possible because of the help of Lorna Pierce, Alan Leventhal, Tony Musladen, Viviana Bellifemine, Sandra Weldon, Rhonda Gillett, Muriel Maverick, Pat Rafter, Charlane Gross, David Calleri, Cynthia Arrington, Peggy Binns, and the staff of the photography laboratory, Instructional Resources Center. Bert Gerow at Stanford provided both assistance with research as well as access to skeletal material. The cooperation and collaborative support given by Rosemary Cambra, chairwoman of the Muwekma Ohlone Tribal Council, as well as other members of the Muwekma Ohlone Tribe, have made the osteological studies at San Jose State both possible and rewarding.

Analysis of the Nubian skeletal remains housed at the University of Colorado was made possible by the assistance and collaboration of Dennis van Gerven. Research on African ape materials was aided by M. E. Morbeck, Jane Goodall, Adrienne Zihlman, Betty and Harry Early, Derek Howlett (Powell-Cotton Museum), and Wim van Neer and Guy Teugels (Museé Royal de l'Afrique Centrale).

I am also grateful to numerous colleagues for sharing ideas and publications, including: Charlotte Roberts, Patricia Bridges, Nancy Lovell, Juliet Rogers, Allison Galloway, Clark Larsen, Bruce Rothschild, David Weaver and Robert Woods. Finally, my colleague and partner Lynn Kilgore has contributed intellectually and emotionally to most of

what is contained in this work. For what is good here, she can justly share in the credit. For what is not, the responsibility is mine alone.

In addition, this work has been further enhanced by the thoughtful comments of reviewers Donald Ortner and Charlotte Roberts. I am grateful to them for their efforts and adherence to high professional standards. I am indebted to the editor of this book series, Mary K. Sandford, who has provided her time as well as motivation and encouragement. Without her, this book would not have been initiated, to say nothing of completed. Editors Carol Hollander and Lauren Shulsky Orenstein have also been of tremendous help and encouragement in bringing the book to fruition. K.B.S.S. Sundaram shepherded the volume through production.

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Chapter 1

Introduction: Of Science and Stories

"So What?"
Anonymous

BACKGROUND

Every researcher labors with some expectation, one could even say illusion, that their results will mean something. Thus, it came as somewhat of a rude awakening when, a few years ago, I received the query of "so what" from a reviewer of an article on osteoarthritis I had submitted for publication. The article was eventually published, but this inquiry did prompt an immediate rejoinder and considerable further reflection. In fact, in many ways, this reflection has led directly to this book.

My own human skeletal research has focused on behavioral reconstructions, especially relating to my earlier work on osteoarthritis as well as more recent investigations of traumatic involvement. It is, however, my experience with osteoarthritis research which has, in recent years, most stimulated a reappraisal of the nature of behavioral reconstruction as it is practiced by human osteologists.

Why behavioral inferences are so commonly and so eagerly pursued in skeletal analysis is not difficult to fathom. Popular conceptions of what skeletal biologists ostensibly *can* learn from osteological remains usually center on such behavioral inferences. The wide popularity (and, also, misunderstanding) of forensic anthropological investigation has further led to similar unrealistic expectations of what inferences osteologists can reasonably make. Moreover, drawing behavioral inferences from "dry" skeletal materials, obviously, *is* fascinating, both to scientist and to the general public. Also shared with

forensic colleagues, there is the challenge of detective-like deduction as well as the motivation to "flesh-out" the individuals recovered from archaeological contexts, somehow to make them "real people." Thus, the intellectual compensations of pursuing behavioral reconstructions are considerable, but so too are the perils.

HISTORICAL PERSPECTIVES IN SKELETAL/BEHAVIORAL RESEARCH

In addition, it could be argued that the foundation of anthropology in the United States has further fostered a behaviorally oriented approach in osteology, and more generally, in physical anthropology. As usually a minority partner in a three-field (or four-field) general anthropology, physical anthropologists have been much encouraged to link osteological evidence with that obtained by our colleagues, be they archaeologists or cultural anthropologists. This is part of what has been termed the "biocultural perspective." Washburn succinctly stated the motivation for such an orientation: "If we would understand the process of human evolution, we need a modern dynamic biology and a deep appreciation of the history and functioning of culture. It is this necessity which gives all anthropology unity as a science" (1962: 13).

All this is well and good, and at a *general* level makes perfectly good sense. Ultimately, as anthropologists we wish to understand the human experience, as it has been shaped by human adaptation. Clearly, one cannot understand the biology of any organism without also understanding the environments to which it has adapted. Humans, and earlier hominids as well, obviously have adapted to a largely cultural environment. Yet, if one is to accept evidence of stone tools as a signature of early cultural behavior, then such cultural processes have been at work for at least 2.5 million years. Thus, to say that *Homo sapiens* shows biocultural adaptations is analogous to saying fish reflect aquatic adaptations. The statements are, of course, accurate, just not very informative.

What clearly is needed is *specificity*. That is, we wish to know what environmental factors influence human biology and how. These questions are, however, not as easy to address as are the broad glittering generalities. Over the last few decades, much of the paleoanthropological research of earlier hominids has attempted to wrestle with these issues, and so too with human osteological investigation of more recent populations.

A significant portion of human osteological research has focused on the reconstruction of certain behavioral attributes of earlier populations.

Some of this research has concerned dental variation, especially as it relates to dietary reconstruction. Similarly, much effort has been devoted to the investigation of chemical constituents of human bone, also aimed ultimately at dietary inference. This research, however, is not the focus here. Instead, this book reviews those techniques aimed at inferring *activity* from skeletal remains.

Assessing Activity

There is a fairly long history of such attempts by skeletal biologists, particularly by those who have come to be identified as paleopathologists. For example, in an early analysis of Nubian remains, Elliot Smith and Wood Jones (1910) drew behavioral inferences both from the patterning of osteoarthritic lesions as well as those resulting from trauma. In fact, the quality of documentation, especially as relating to traumatic involvement (Wood Jones, 1910), is still an excellent illustration of the necessary paleoepidemiological methodologies required in this type of research. Moreover, because these data remain so potentially informative, they have recently been reworked by Berger and Trinkaus (1995), and thus this research continues to provide useful data and to stimulate further investigation (see Chapter 6).

Probably the most central figure in the development of an explicit behaviorally oriented osteological perspective in the U.S. was J. Lawrence Angel. Angel's innovative perspectives have fundamentally influenced anthropological interpretations of osteoarthritis (1966; 1971); trauma (1976); enthesial reactions (1964; Angel et al., 1987; Kelley and Angel, 1987); parturition scars (1969; 1971; Angel et al., 1987), and Schmorl's nodes (1971; Angel et al., 1987). In fact, all of these topics are discussed in this book. Moreover, Angel's perspectives and enthusiasm provided a stimulus to many other researchers. Working with his colleague at the Smithsonian Institution, T. Dale Stewart, they initiated a remarkable series of skeletal studies and, in the process, launched an array of innovative techniques. This research was to have immediate derivative effects, for example, D.J. Ortner's investigation of osteoarthritis (1968). In fact, through the suggestion and direction of Don Ortner, and also influenced by Angel and Stewart at the Smithsonian, I began my own research on osteoarthritis.

As the balance of this book details, many others have followed in the footsteps of Angel, Stewart, and Ortner. Moreover, in a corresponding fashion, the influence of Calvin Wells (1963; 1964; 1965) in the U.K. also initiated a great interest in behaviorally oriented research, especially as concerning osteoarthritis.

CURRENT PERSPECTIVES

Both in the U.S. and the U.K. this interest has yet to wane, although it has recently been somewhat redirected. For example, at the 1997 American Association of Physical Anthropologists annual meeting numerous papers addressed a variety of skeletal "markers" of behavior/activity. Four of these contributions concerned trauma, all dealing with craniofacial injury and interpretations of interpersonal aggression. A fifth paper made a similar conclusion regarding cannibalism. Three papers dealt with activity inferences from bone geometric properties, one addressing nonhuman primates and another concerning behavioral reconstruction from late Paleolithic/Mesolithic contexts. Likewise, one contribution concerned Schmorl's nodes, two addressed skeletal asymmetries/handedness, one investigated squatting facets, and two others attempted to use osteoarthritic involvement to infer behavior. Indeed, with just two papers dealing specifically with osteoarthritis, there is an apparent trend away from using this once-popular approach as a grounds for behavioral inference (although a few other papers also incorporated degenerative lesions as part of a more "holistic" approach to such reconstructions).

The most obvious new emphasis for inferring activity clearly involves the use of "musculoskeletal markers," or, more specifically, enthesial reactions. In this recent national meeting, 11 papers focused on such evidence and did so specifically in an attempt to draw behavioral inferences, although one of these (Bridges, 1997), did report *negative* results. Much of this recent interest in enthesial reactions at the physical anthropology meeting was stimulated by a special symposium on this topic which included nine of these contributions.

Beyond the confines of professional conferences, it is quite apparent that such endeavors still attract much interest among osteologists. Given both the general public's eagerness to hear such stories as well as the biocultural emphasis of American anthropology (noted above), professional journals also commonly encourage such interpretations. A clear impression one gets from such an orientation is that evidence of osteoarthritis (and many other skeletal manifestations) is interesting only in so far as it helps reconstruct activity patterns in past groups. Unquestionably, my own earlier research helped fuel this unrealistic expectation. Moreover, it is an overly-limiting view, failing as it does to recognize the manifold other ways that skeletal data can contribute to understanding the biology of osteoarthritis and other significant conditions.

Despite the current fascination with enthesopathies, over the last two decades osteoarthritis has been the condition most frequently used

by osteologists to infer behavior. For example, in Kennedy's eclectic review of "skeletal markers of occupational stress" (1989), the majority of characteristics relate to some form of joint or peri-articular involvement. Likewise, in Merbs' landmark publication on "activity-induced pathology" (1983) most of the emphasis also was placed on osteoarthritic patterning and its utility in reconstructing activity from skeletal remains.

Following considerable enthusiasm during the 1970s and 1980s, some criticisms have been voiced over the past few years (e.g., Bridges, 1992; 1993; Jurmain, 1990a; 1991b). Perhaps, these critiques have had a inhibiting effect on certain aspects of research relating to osteoarthritis as a skeletal marker of activity. Other researchers are, understandably, not as convinced and continue to evaluate osteoarthritis for this purpose, but they usually do so with more caution than many of us exercised in past years! Certainly, these differing perspectives stimulated some lively and informative discourse in 1996 at a special symposium (as part of the Paleopathology Association annual meeting).

In fact, it would be a detriment for osteologists to turn away from investigation of osteoarthritis *simply* because its skeletal manifestations do not usually relate directly to an unambiguous behavioral etiology. There is an amazingly deep and rich clinical literature on osteoarthritis, which is the primary reason we are now more aware of its inherently complex etiopathogenesis.

This present review focuses somewhat more on osteoarthritis, both in terms of its clinical manifestations and osteological interpretations, than on those other skeletal conditions recently utilized to infer activity patterns. The rationale for this emphasis relates partly to my own experience, but is also linked to other considerations as well:

(1) Osteoarthritis is among the most common pathological condition found in human skeletal remains.
(2) As mentioned above, osteologists, to date, have used osteoarthritis more than other behavioral markers.
(3) The clinical literature is more detailed than for any other similar skeletally manifested condition, and a host of studies have dealt specifically with issues pertaining directly to osteological/behavioral interpretations.
(4) Osteoarthritis can then serve as a useful model for other emerging, and less well-tested, perspectives.

I would argue that osteological studies have contributed to a greater understanding of osteoarthritis, providing a complementary

perspective to that of clinical investigations. Indeed, as will be emphasized, the greatest potential for advances in understanding derive from collaborative research efforts, in which osteological data supplement those obtained from living subjects. This is a point well discussed by others (e.g., Buikstra and Cook, 1980) and has also been the central focus of the Paleopathology Association since its founding in 1973.

ORGANIZATION OF BOOK

Recognizing that osteologists have more consistently utilized osteoarthritis than other skeletal conditions to draw behavioral inferences, the next three chapters focus on osteoarthritis. Chapter 2 reviews clinical and osteological perspectives particularly relating to various hypotheses concerning the etiopathogenesis of the disease. In a sense, this chapter attempts to view these complex clinical data through the eyes of a skeletal biologist; the resulting approach thus emphasizes pathological processes affecting bone.

Chapter 3 reviews the occupational and sports literature on osteoarthritis. These data are clearly central to anthropologists, since they represent a direct clinical analogy to the types of activity-related inferences proposed by osteologists. Chapter 4 completes the discussion of osteoarthritis with a review specifically of anthropological perspectives. The focus here is on data derived from skeletal research, and the more explicit emphasis concerns how osteologists have utilized osteoarthritis patterning to reconstruct behavioral aspects of ancient societies.

In Chapter 5 the orientation shifts to a consideration of a range of other proposed osteological "markers" of activity. While a variety of skeletal manifestations including Schmorl's nodes, parturition scars, stress fractures (including spondylolysis), and auditory exostoses are discussed, the primary focus is on enthesopathies. As noted above, contemporary osteological research is emphasizing such enthesial reactions, and a review of this approach and its theoretical foundations thus seems prudent.

Chapter 6 addresses yet another commonly found skeletal condition, namely trauma. Behavioral interpretations of traumatic lesions have enjoyed a long history in anthropological research, and the osteological literature (as well as supporting clinical data) are reviewed. In particular, various proposals concerning the influence of interpersonal aggression affecting craniofacial involvement as well as "parry" fractures of the forearm are critically evaluated.

Probably the newest and potentially most innovative approach relating to behavioral reconstruction based upon skeletal material concerns analysis of bone geometry. Thus, Chapter 7 focuses on analysis of bone geometry, again emphasizing both theoretical bases and practical utility in osteological investigations.

Chapter 8 concludes the book with a synthesis of the varied perspectives presented in prior chapters. Moreover, this final chapter suggests how future methodological approaches can be refocused to avoid various problems inherent in current research.

OSTEOLOGY AS SCIENCE

Without some direct link between osteological indicators and well-documented morbid changes, skeletal biologists can only hypothesize in a theoretical vacuum. Relating bone changes only to *presumed* behavioral causation, and then assuming these very same bone "markers" corroborate said behavior, is obviously a circular piece of reasoning. Since osteologists usually lack any direct (and independent) evidence of behavioral attributes of earlier societies, they have occasionally sought other sources for such details. Most typically, this type of information has been extracted from ethnohistorical records. However, it should readily be apparent that this often-haphazard form of documentation is almost always inadequate to the task, as it lacks both the necessary precision and specificity. What osteologists then do (as exemplified by my own earlier assertions regarding Inuit osteoarthritis) is to selectively glean those bits from the ethnographic record that serve to "fit" a particular hypothesis. Such reasoning, however, is only slightly less circular than imaginative scenarios created solely from the skeletal evidence.

Another parallel line of evidence sometimes utilized for more recent archaeological contexts comes from historical accounts; unfortunately, these also are usually too imprecise to offer much in the way of rigorous insight. What such information clearly *cannot* usually do is provide an adequate means of independently testing the influence of behavior on skeletal response. For some recent historic contexts, there are, however, a few notable exceptions in which the quality of documentation has allowed a means of testing certain skeletal-behavioral relationships. In particular, the Spitalfields (Waldron and Cox, 1989) and the *Mary Rose* (Stirland, 1991; 1997) samples meet this standard.

Whatever their source of potential corroboratory evidence used to substantiate behavioral reconstructions, osteologists must always ask how accurate are these sources? Do they provide an independent test

(from that of the skeletal material) to verify either general or specific relationships?

Without answering these questions satisfactorily, there is no rigorous methodology of verification. Ultimately, the best documented sources derive from clinical contexts. As noted, osteologists investigating osteoarthritis have been well aware of the manifestly complex clinical evidence relating to this condition. Similarly, substantial clinical support has been provided for a few other behaviorally based interpretations of skeletal lesions. The most notable of these better-substantiated explanations is that by Merbs relating to spondylolysis (Merbs, 1989; 1996b).

It is important to remember, of course, that clinical perspectives are frequently limited in their applicability as well. For example, the same types of constraints related to inadequate sampling that handicap osteologists are also inherent to many clinical research designs. Moreover, there is oftentimes a similar tendency to rush to judgment, usually resulting in overly simplistic explanations of complex phenomena. This impulse is commonly fostered by attempts to explain medical conditions that are perceived as of major contemporary significance (e.g., AIDS therapies, environmental carcinogens, dietary influences on heart disease). Certainly, the way the popular press disseminates and exploits both current research findings and normal scientific debate further distorts the public's views and expectations of scientific investigation.

Given such obvious shared restrictions, there are, nevertheless, a variety of lessons skeletal biologists can learn from our clinical colleagues. Most notably, numerous methodologies frequently utilized in medical epidemiology are instructive. For example, the rigorous control of samples, standard statistical testing, and attention to potential confounding variables are all methodological approaches not as widely applied in paleopathology as they might be.

Beyond the success of providing a good behavioral explanation for spondylolysis, what of other contemporary assertions of presumptive behavioral correlates inferred from skeletal data? At present, none of these other areas of investigation has provided adequate independent confirmation. In some cases, as with enthesial reactions and bone geometric variation, clinical evidence is still quite limited (although some useful data do exist). Nevertheless, there has, to date, not been enough relevant clinical data presented by osteologists from which even to begin validating most specific behavioral hypotheses. Moreover, if the lessons provided by the last few decades of osteoarthritis research are any guide, those seeking such validation have a long way yet to go.

Thus, this is a book about human osteology, and, more specifically, it concerns the subfield of paleopathology. More to the point, this work raises issues about the *science* of paleopathology. Given, of course, that paleopathology is a scientific pursuit, it thus becomes a question of how paleopathologists can go about testing their assertions in a rational and rigorous manner.

Naturally, scientific endeavors cannot be practiced as if in an ideal world. Not all data sources are perfect or, necessarily, even particularly good. Frequently, we simply evaluate those skeletons that are available. Furthermore, it is obvious in paleopathology that not all hypotheses are testable, at least not in the classical scientific sense. This limitation, of course, should not inhibit us from making the *best* interpretation we can offer from the data available.

In many other applications within the natural sciences, wherein hypotheses are not amenable to complete testing, great insight has nevertheless been gained. Paleontology and astronomy, for instance, both have produced admirable bodies of data and theory, frequently without fully explicit testing criteria. What has proven successful in such scientific pursuits requires that: data are rigorously compared between studies; based on current knowledge, predictions are made; and subsequent studies are used to help verify (or discard) earlier hypotheses. Furthermore, an essential component in this, and in *any* other, type of scientific approach is that all possible alternative explanations must be considered. In this way, as future studies are performed to expand the database, various views can be fairly evaluated. Ultimately, then, the "best" explanation is attained.

Accordingly, it is not here argued that all paleopathology must be judged by idealized and generally unattainable standards. What is suggested, however, is that methodological limitations be more explicitly recognized. Equally important, alternative explanations need to be more honestly entertained.

In practice these basic but somewhat abstract scientific goals are not always easy to reach. The opportunities for and commensurate rewards of appealing interpretations are usually obvious; the inherent constraints frequently are not so easily, or willingly, recognized. Certainly, before the limitations can be overcome, they must first be confronted.

Galloway has pointedly emphasized some of these constraints, particularly the tendency to over-interpret certain aspects of skeletal-behavioral relationships:

> Our own biases and desires to focus on certain features and processes predispose us to accept those studies or portions of studies which support our

> viewpoint. The ability to read life histories in the bones is extremely attractive—it "gives voice" to the deceased and permits us to contribute a more rounded picture of the individual. We must always remember, though, that the voice which is spoken is through our mouths and there may be discrepancies between the life events and what we think we see (Galloway, 1995: 95).

If this book appears concerned with likely mistakes in interpretation, it is, no doubt because I have committed them myself. Moreover, if this work seems overly concerned with the temptation to over-interpret data, it is because it is a constant struggle not to do so in my own research.

This book, thus, is not offered as a judgmental treatise, identifying the failures of others. All those who do paleopathology are aware of the inherent obstacles. Here are offered some signposts to highlight many of the pitfalls, and, with some luck, a chance of avoiding them.

Chapter 2

Osteoarthritis: Clinical and Osteological Approaches

OA (osteoarthritis, degenerative joint disease) is a degenerative disease of the cartilage of joints. It is of diverse etiology and obscure pathogenesis (Mankin et al., 1986: 1132).

DEFINITION AND CLASSIFICATION

Because osteoarthritis is such a widespread and oftentimes debilitating condition, it has attracted intense scrutiny from a wide variety of clinicians. According to the most recently reported national health survey for the U.S., close to 40 million Americans are estimated to suffer from rheumatic complaints (the majority of which is at least loosely referable to osteoarthritis) (CDC, 1996a). As Hamerman (1989: 1322) emphasizes, "This prevalence and its costs—billions of dollars in medications, surgery, and days lost from work—account for the growing interest in uncovering the basic mechanisms by which this disease affects human joints."

While research interest has been intensive for almost a century, and the resulting accumulated data impressive, there is little in the way of consensus regarding the biology of osteoarthritis among clinical researchers. Indeed, there is even dispute regarding the proper name for the condition. Traditionally, in the United States it has been called "osteoarthritis" (OA), but this term was thought misleading by many (e.g., Bennett and Bauer, 1937), as it implied an inherently inflammatory condition. Among some contemporary researchers (Hough, 1993) the same criticism is still voiced, although others (Jasin, 1989) clearly recognize a significant inflammatory component.

As an alternative term, "degenerative joint disease" was suggested (Bennett et al., 1942), and this referent was widely used for a number of years. However, more recently, it too has been critiqued, since the term, "degenerative" is thought misleading and ill-defined (Mankin, 1982b), "implying a manifest, passive process associated with old age" (Dieppe, 1987: 16.76). Consequently, in the U.S. and the U.K., the preferred current terminology is once again "osteoarthritis," with the alternative term, "osteoarthrosis" used frequently on the continent (in the latter usage, modifying the "itis" suffix to de-emphasize inflammatory aspects). A variety of other terms have over the years also found their way into the literature, including: arthritis deformans, hypertrophic arthritis, traumatic arthritis, and for specific joints, gonarthrosis (knee) and coxarthrosis (hip). In this work, the term osteoarthritis (OA) will be used.

Among clinical workers, there is more general agreement concerning to which joints the term OA (or its synonyms) should be applied. Most researchers reserve the term for fully movable, diarthrodial, joints. Also called, "synovial joints," the articular surfaces are covered with hyaline cartilage and are contained within a joint cavity enclosed by a synovial membrane. Such joints include all the major articulations of the appendages (including those of the hands and feet) as well as the small interfacetal (also known as apophyseal or zygoapophyseal) joints of the spine.

However, OA is generally *not* the term preferred for the arthropathy associated with joints of the vertebral bodies. These slightly movable joints (amphiarthroses, also called syndesmoses) are separated by fibrocartilage as well as hyaline cartilage, the latter directly covering joint surfaces, and similar joint anatomy is also seen in the pubic symphysis and distal tibio-fibular joint.

The anatomy of the intervertebral disk joints is quite different from diarthrodial ones (Mankin and Radin, 1985). The fibrocartilage of the intervertebral disks differs from hyaline cartilage, as the former contains primarily Type I collagen (Mankin et al., 1986), and although these syndesmoses do show degenerative changes with aging, "Little is known of the process, however, except perhaps that facet joint OA and disk degeneration may be linked in some way" (Mankin et al., 1986: 1146). This association of apophyseal OA with disk disease has, in fact, also been well substantiated by osteological research (Merbs, 1983; Bridges, 1994; Kilgore, 1990). Regarding differences in synovial joints compared to intervertebral disk joints, Dieppe (1987) states that the clinical features are "quite different at the two sites," and he thus prefers the term spondylosis for the latter (to differentiate it from OA).

Commonly, in osteological studies, the conditions are kept distinct (both terminologically and in interpretation), and pathology of the intervertebral syndesmoses is thus usually termed "osteophytosis" or "vertebral osteophytosis (VOP)." This latter term will be used here.

Among clinicians there is, again, little consensus regarding an appropriate and general definition for OA. Hough (1993: 1699) describes OA as "an inherently non-inflammatory disorder," but Howell and Pelletier (1993: 1723) also emphasize a more dynamic series of secondary phenomena and define it "as a complex of interactive degradative and repair processes in cartilage, bone, and synovia, with secondary components of inflammation."

In efforts in the mid-1980s to reach greater consensus, two broad collaborative endeavors sought to define and characterize OA. In one attempt, a "workshop on the etiopathogenesis of osteoarthritis" (Mankin et al., 1986) with contributions by approximately 60 leading researchers, the following was offered by three of the participants (p. 1132):

> OA (osteoarthritis, degenerative joint disease) is a degenerative disease of the cartilage of joints. It is of diverse etiology and obscure pathogenesis... *Pathologically*, the disease is characterized by irregularly distributed loss of cartilage more frequent in areas of increased load, sclerosis of subchondral bone, subchondral cysts, marginal osteophytes, increased metaphyseal blood flow, and variable synovial inflammation." [Note: OA was, additionally, characterized according to clinical, histological, biomechanical, biochemical and therapeutic criteria.]

The reaction of the workshop participants as reported by Mankin and colleagues (1986: 1132) is informative, "The response to this definition was overwhelming and unanimous: no one cared much for it (some felt stronger)." On a more positive note, they, however, add, "On further reflection, this challenge to the definition of OA is a symptom of the need for further research that may clarify conceptions of the nature of the disorder" (Mankin et al., 1986: 1132).

The other attempt at a consensus definition was developed and published as part of a collaborative effort of 25 clinicians participating as part of the Subcommittee on Classification Criteria of Osteoarthritis (of the American Rheumatism Association):

> OA is defined as a heterogeneous group of conditions that lead to joint symptoms and signs which are associated with defective integrity of articular cartilage, in addition to related changes in the underlying bone and at the joint margins (Altman et al., 1986: 1039).

As reflected clearly in this last effort, many of the problems of definition, no doubt, reflect the variable causes and pathologic consequences

of what is subsumed under the clinical umbrella of "osteoarthritis." This depiction of the heterogeneity of the disease is also emphasized by Hough (1993) and Panush and Lane (1994). Paul Dieppe has perhaps stated the concept best in describing OA not as a single entity, but more of a "joint failure" "analogous to heart failure—the state of an organ which can result from a number of different diseases or physiological changes" (quoted in Mankin et al., 1986: 1132).

Dieppe in 1990 further elaborated this position,

> The term OA describes an abnormal state of a synovial joint. It is not a disease. Each of several different 'diseases' can trigger a reaction pattern leading to the characteristic features of the OA joint: focal loss of articular cartilage and hypertrophy of subchondral bone. Not surprisingly, attempts to define and classify OA as a single disease entity have not been helpful. (Dieppe, 1990: 262.)

From the above, it is clear from the outset that any discussion of osteoarthritis, other than the most superficial, requires consideration of a complex (and poorly understood) etiopathogenesis. In this and the following chapter the discussion will attempt to grapple with the complexities concerning the causation and patterning of OA. Osteologists must obviously not forget or gloss over these issues and resulting fundamental uncertainties which swirl around and obscure an understanding of OA. Osteological approaches that fail to do so are destined for failure, if not also some embarrassment in the eyes of our clinical colleagues.

Classification of OA

As with definitions, a variety of classifications has been proposed, although here perhaps with some wider agreement. Most commonly, the condition is divided most basically into primary and secondary forms. Primary OA (also referred to as "idiopathic OA") is recognized, "when it occurs in the absence of any underlying predisposing factor" (Moskowitz, 1993: 1735).

It is somewhat less than reassuring that this most basic form of OA (and the form by far most commonly studied by osteologists) is only defined by negative criteria. This point is further reinforced by the Subcommittee of the American Rheumatism Association, which characterizes primary OA as "of unknown origin" (Altman et al., 1986).

By contrast, secondary disease is "related to a known medical condition or event" (Altman et al., 1986). Such predisposing conditions could include acute trauma (Figure 2-1); inflammatory disease;

Figure 2-1 Secondary osteoarthritis. Right proximal femur. Kulubnarti, Sudanese Nubia; female, 50+years. The severe degeneration of the head of the femur is most likely secondary to trauma and aseptic necrosis. Photograph by the author.

metabolic, endocrine, or neuropathic problems; congenital malformations; or other arthropathies of still-unknown etiology, such as diffuse idiopathic skeletal hyperostosis (DISH) or calcium pyrophosphate deposition disease (CPPD). An often-cited classification (as developed by the American Rheumatism Association Subcommittee) is shown in Table 2-1.

Primary OA, as shown, is further divided into localized entities as contrasted with a manifestation, generalized osteoarthritis (GOA),

Table 2-1. A classification of OA

I. Idiopathic
  A. Localized
    1. Hands: e.g., Heberden's and Bouchard's nodes (nodal), erosive interphalangeal arthritis (non-nodal), schaphometacarpal, scapho-trapezoidal
    2. Feet: e.g., hallux valgus, hallux rigidus, contracted toes (hammer toes), talonavicular
    3. Knee
      a. Medial compartment
      b. Lateral compartment
      c. Patellofemoral compartment (e.g., chondromalacia)
    4. Hip
      a. Eccentric (superior)
      b. Concentric (axial, medial)
      c. Diffuse (coxae senilis)
    5. Spine (particularly cervical and lumbar)
      a. Apophyseal
      b. Intervertebral (disk)
      c. Spondylosis (osteophytes)
      d. Ligamentous (hyperostosis—or DISH)
  B. Generalized: includes 3 or more areas listed above
    1. Small (peripheral) and spine
    2. Large (central) and spine
    3. Mixed (peripheral and central) and spine

II. Secondary
  A. Post-traumatic
  B. Congenital or developmental diseases
    1. Localized
      a. Hip diseases: e.g., Legg-Calve-Perthes, congenital hip dislocation, slipped capital femoral epiphysis, shallow acetabulum
      b. Mechanical and local factors: e.g., obesity (?), unequal lower extremity length, extreme valgus/varus deformity, hypermobility syndromes, scoliosis
    2. Generalized
      a. Bone dysplasias: e.g., epiphyseal dysplasia, spondylo-apophyseal dysplasia
      b. Metabolic diseases: e.g., hemochomotosis, ochronosis, Gaucher's disease, hemoglobinopathy, Ehlers-Danlos disease
  C. Calcium deposition disease
    1. calcium pyrophosphate deposition disease
    2. apatite arthropathy
    3. Destructive arthropathy (shoulder, knee)

Table 2-1. *(Continued)*

D. Other bone and joint disorders: e.g., avascular necrosis, rheumatoid arthritis, septic arthritis, Paget's disease, osteopetrosis, osteochondritis

E. Other diseases
   1. Endocrine diseases: e.g., diabetes mellitus, acromegaly, hypothyroidism, hypoparathyroidism
   2. Neuropathic arthropathy (Charcot joints)
   3. Miscellaneous: e.g., frostbite, Kashin-Beck Disease, Caisson disease

(Altman et al., 1986: 1040).

which includes more widespread, polyarticular, involvement. GOA was argued forcefully as a distinct entity by Stecher (1940; 1959), emphasizing the polyarticular involvement of Heberden's nodes in distal interphalangeal joints of the hands (DIPs). The development of Heberden's nodes, that is, small bony and cartilaginous exostoses of the finger DIPs, was central in Stecher's description of GOA as well as later characterizations of the syndrome. Following intensive investigation by Kellgren and Lawrence (Kellgren et al., 1964), the definition of GOA was further refined to include a polyarthritis involving three or more joints (encompassing DIPs of hands, but also extended to other joints including 1st carpo-metacarpal, 1st tarso-metatarsal, vertebral apophyseal, knee, and hip joints).

Further, GOA was subdivided into nodal (i.e., with Heberden's nodes) and non-nodal variants. The generalized nodal expression of OA, particularly, is thought to stand out as a distinct entity with pronounced hand involvement (and especially as seen in white females) (Dieppe and Watt, 1985). Early on (Stecher, 1959) pointed out the strong familial association of GOA and even suggested a possible Mendelian mode of inheritance may underlie it. Several epidemiological studies have further confirmed this polyarticular manifestation, and some very tentative evidence suggests it may be tied to mutations (ultimately producing polymorphism) in the locus coding for Type II collagen (Silman and Hochberg, 1993). Moskowitz (1993) concurred that the nodal form of GOA appears to be inherited as a Mendelian trait, but argued that the non-nodal forms appear more polygenic in pattern. While seemingly a distinct entity, "which implies some systemic factor of cartilage vulnerability" (Peyron, 1986: 17), the differences between GOA and more localized manifestations may not be as obvious as once believed. For example, in one study of severe hip and knee disease, requiring surgical replacement, 62% of patients showed polyarticular involvement indicative of GOA (Cooke, 1983).

Nevertheless, recent studies have further confirmed a clear polyarticular pattern in various populations as well as suggesting strong genetic influences. Waldron (1997) found in a skeletal population from 18th/19th Century London a marked correlation of multiple site hand involvement with OA of the knee. A similar result was reported in a contemporary group from Australia (Cicuttini et al., 1997), here correlating hand OA with involvement of the tiobiofemoral compartment of the knee (but *not* the patellofemoral joint). In fact, these authors concluded that there may well be different etiological agents influencing the two compartments of the knee joint.

Biochemical and endocrine factors may also play a differential role in polyarticular OA, as compared to more localized manifestations of the disease. Dequeker et al. (1997) found increased levels of growth factors and hypermineralization in individuals with generalized OA.

Further emphasizing the blurred lines in differentiating subsets of OA, another well-known classification (Mankin et al., 1986) placed *all* traumatic variants (acute and microtraumata from "occupation" and "sports") as secondary conditions. Osteologists who assume they are clearly focused on primary disease may thus be accurate, but perhaps only to the point of which classification is employed, and frequently ignoring many of the unavoidable ambiguities. Indeed, Moscowitz (1993) pointed out that the distinction (primary/secondary) may be artificial, and many causes of so-called primary disease may well result from predisposing conditions (especially as seen in the hip; see below).

Another interesting wrinkle for osteologists is apparent in the subsets listed in Table 1 and as further refined by Altman (1991). As seen, the subsets of primary/idiopathic (localized) OA include hands, feet, knee, hip, spine (particularly cervical and lumbar segments), as well as "other sites" (shoulder, TMJ, sacroiliac, ankle, wrist, acromioclavicular). The elbow, however, does not receive even a mention. As will be discussed in Chapter 4, the elbow has probably figured more prominently in behavioral reconstructions by osteologists than any other joint.

The relative silence concerning elbow disease by most clinicians is, no doubt, explained by the very low prevalence of elbow involvement in contemporary populations. Dieppe (1990) lists the most commonly affected joints as those of the hand (particularly DIPs), base of the thumb, knee, hip, and apophyseal joints of the cervical and lumbar spine. Panush and Lane (1994) provide much the same order of joints most frequently affected, but they also specifically mention the proximal interphalangeal joints (PIPs) of the hand (second only to DIPs) and list hip involvement prior to that of the knee. Further

epidemiological patterns of OA involvement will be discussed below, but the elbow is rarely mentioned and almost never emphasized in studies of contemporary populations.

NORMAL JOINT ANATOMY AND FUNCTION

While OA is a very common condition, with a moderate to high prevalence in all contemporary populations thus far sampled, it must be remembered that it is not an inevitable condition (even in older individuals). In fact, most people, even those who work hard most of their lives, never experience symptoms or show signs of OA. In a normally functioning joint the surfaces move against each other with remarkably little friction, with friction resistance measured in experimental studies of animals as low as 0.002 (indicating friction resistance in cartilage twice as low as rubber on steel and 10 times lower than an ice skate on ice) (Mankin and Radin, 1985).

The articular cartilage achieves this nearly friction-free state by releasing water under load (under normal conditions the cartilage is hydrated, freely exchanging water with the surrounding synovial fluid). The cartilage surfaces maintain this superior accommodation even under markedly varying stress conditions by employing two different types of lubrication. When exposed to high loads, a hydrostatic mode is used, creating a fluid film separating the surfaces; under lower loads, lubricating glycoproteins keep the surfaces from touching in a form of boundary lubrication (Mankin and Radin, 1985). This dynamic interaction is facilitated by action of the synovial fluid, which is well adapted for both secretory and resorptive function. Thus, joint physiology should be viewed as dynamic, and, as discussed below, capable of considerable repair. Nevertheless, metabolic activity is usually quite restrained, as indicated by a joint's hypothermic environment (temperatures vary from 90.3 to 94.3 degrees) (McKeag, 1992).

Joint integrity is further enhanced by the anatomical structures of the joint providing stability through:

(1) close-fitting joint congruity (e.g., the ankle; alternatively, gliding joints like the shoulder provide much less stability)
(2) ligaments, some of which are contained within or merge with the joint capsule (others are more discrete)
(3) fibrous joint capsule consisting of dense connective tissue, covering the entire joint area and firmly attached to the bone
(4) muscles

(5) fibrocartilaginous partitions, e.g., menisci (particularly seen in the knee, TMJ, and sternoclavicular joints, but less consistently in acromioclavicular and distal radio-ulnar joints).

The tissues most centrally involved in osteoarthritic changes are the articular cartilage and subchondral bone. Articular cartilage has been the primary focus of most clinicians, as this tissue shows the most consistent and obvious gross osteoarthritic pathological changes. Because clinical approaches mostly emphasize articular cartilage changes in the pathogenesis of OA, such changes will be discussed first. However, the subchondral bone is obviously of major import to osteological researchers, and, in fact, several clinicians have also focused attention on degenerative aspects of this tissue. Specific components of bone involvement in OA will be discussed below.

Articular Cartilage

In its normally functioning state, articular cartilage must retain its dimensional stability; that is, it needs to be able to deform and elastically respond under potentially high loads (Mankin et al., 1986). As noted above, it is able to accomplish this by releasing water (when loaded), and it can compress up to 40% of its depth (Mankin and Radin, 1985). In fact, the deformation of cartilage is what permits under normal circumstances such excellent joint congruity, being thinner in better fitting joints and thicker in less well-fitting ones (Mankin and Radin, 1985). At no point in its life cycle, however, is the cartilage very thick, ranging in thickness from 2 to 5 mm (Mankin and Radin, 1985; Dieppe, 1987), including the articular cartilage of the patellofemoral joint, which is the thickest in the body (Mankin et al., 1986).

Articular cartilage is a highly unusual connective tissue, being avascular, alymphatic, aneural, and quite hypocellular (Mankin and Radin, 1985; McKeag, 1992). By volume, the tissue is composed of:

water	68–78%
collagen fibers	15%
proteoglycan ground substance	10%
cells (chrondrocytes)	<5%

(Mankin et al., 1986; Dieppe, 1987; McKeag, 1992)

More than 90% of the collagen is Type II (Hamerman, 1989), which is distinct from the collagen found in bone or skin (Mankin and Radin, 1985). At a fairly gross level (under low magnification), the cartilage can be viewed in four zones characterized primarily by differing shapes and orientations of the cells: (1) the tangential (gliding)