

**A PALEOPATHOLOGICAL SURVEY OF ANCIENT PERUVIAN CRANIA
HOUSED AT THE PEABODY MUSEUM OF ARCHAEOLOGY AND
ETHNOLOGY AT HARVARD UNIVERSITY, CAMBRIDGE,
MASSACHUSETTS: A SPECIAL EMPHASIS ON SCURVY**

by

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A Thesis Submitted to the Faculty of

The Dorothy F. Schmidt College of Arts and Letters

In Partial fulfillment of the Requirements for the Degree of

Master of Arts

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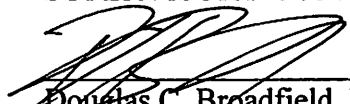
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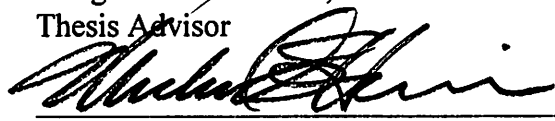
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This thesis was prepared under the direction of the candidate's thesis advisor, Dr. Douglas C. Broadfield, Department of Anthropology, and has been approved by the members of his supervisory committee. It was submitted to the faculty of the Dorothy F. Schmidt College of Arts and Letters and was accepted in partial fulfillment of the requirements for the degree of Master of Arts.

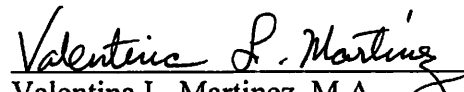
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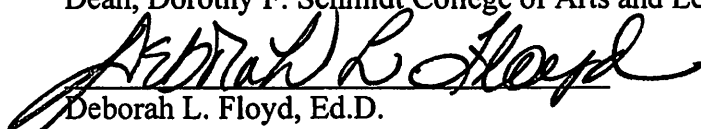
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ABSTRACT

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Title: A Paleopathological Survey of Ancient Peruvian Crania Housed at the Peabody Museum of Archaeology and Ethnology at Harvard University, Cambridge, Massachusetts: A Special Emphasis on Scurvy

Institution: Florida Atlantic University

Thesis Advisor: Dr. Douglas C. Broadfield

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This thesis is a paleopathological survey of ancient Peruvian crania housed at the Peabody Museum of Archaeology and Ethnology at Harvard University, Cambridge, Massachusetts. Chapter one discusses the significance of this research, work prior to this thesis's formulation, and defines paleopathological and bioarchaeological terms relevant to this thesis. Chapter two presents this thesis's materials and methods. Of the 196 Peruvian crania in this study sample, 11 case studies are presented. Chapter three reports a case of probable scurvy and likely anemia comorbidity. This case study is accompanied by a critical analysis and review of the literature surrounding scurvy, a detailed macroscopic examination, and a rigorous differential diagnosis process. Chapter four offers cases representing pseudopathology, hematopoietic disease, infectious disease, joint disease, neoplastic disease, trauma, and trauma-induced disease. Chapter five presents a summary of this thesis.

DEDICATION

I dedicate this work to family and friends, past and present. Particularly, in order of passing, I dedicate this thesis to the memories of Tony A. Chamoun, George A. Chamoun, Zeina Maalouf, and Yvette Chamoun.

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CHAPTER 1: INTRODUCTION

Thesis Nature, Purpose, and Significance

This work is a paleopathological survey of ancient Peruvian crania (n=196) housed at the Peabody Museum of Archaeology and Ethnology at Harvard University, Cambridge, Massachusetts (henceforth, Peabody). I do not present analyses of all these crania. Rather, I present 11 case studies of paleopathological interest.

I present cases of interest for several reasons. The individuals in my study are from various Peruvian spatial and temporal origins. A great number of these individuals, however, have no known contextual data other than their being Peruvian and archaeological. Such a paucity of contextual information is a common issue in museum collections, especially since some collections are personal donations that were not rigorously and meticulously excavated. So, any bioculturally meaningful population or community level study would be invalid. This paucity of contextual data also precludes investigating individual social identity and social disease experience. Further, there are crania whose remains are fragmentary, which in instances hinders the recognition of pathological states. And, not all cases display "remarkable" pathologies in the clinical sense of the term.

Although some paleopathologists view case studies as outdated and limited in the information they convey (for a discussion of this issue, see Mays 2012; Powell and Cook 2012:215-217), case studies serve to illumine disease distribution in the distant past. They

also add to the diagnostic potential of certain conditions. In other words, case studies offer unique opportunities for detailed, thorough examination of diseases and disease symptoms that might otherwise go unnoticed, are previously unreported, or are scantily documented. As such, case studies are well suited for drawing attention to skeletal signs of comorbidity, and therefore can heighten future researches' sensitivity to identifying such conditions. Moreover, as Powell and Cook (2012:217) argue, "disdain from attempting appropriate differential diagnoses, which could expand our understanding, [will] voluntarily limit the scope of our reconstructions of life in the past, to no real purpose." In these senses, case studies are invaluable to paleopathologists.

This text's case studies represent metabolic, hematopoietic, infectious, joint, neoplastic, trauma and trauma-induced diseases, and pseudopathology. Of these case studies is an individual with what I argue to be probable scurvy (vitamin C deficiency) and anemia comorbidity (*Chapter 3*). I devote considerable attention to this case. My reason for doing so is two-fold: (1) In the English language literature, there are very few published reports on scurvy in Peruvian human juvenile remains from antiquity. I know of only three (i.e., Klaus 2014a, Melikian and Waldron 2003; Ortner et al. 1999). And, (2) to my knowledge, this case study is the first to document potential scurvy and anemia comorbidity in Peruvian juvenile remains in the English language literature. Thus, expanding on and adding to scurvy's diagnostic potential in ancient Peruvian remains is a needed endeavor, as is recognizing signs of anemia comorbidity.

For this case, I present a critical review and analysis of the literature on juvenile scurvy, provide a detailed macroscopic examination, discuss differential diagnosis candidates, and construct a differential diagnosis flow-chart that reflects epidemiological

data and skeletal pathologies of most likely cause. In paleopathology, reported differential diagnosis flow-charts are few (e.g., Buikstra 1976). I construct one in this thesis as, in part, a response to calls for heightened scientific rigor—especially as it relates to juvenile scurvy diagnosis (e.g., Crandall and Klaus 2014).

Work Prior to This Thesis's Formulation

In the interest of scientific integrity and justifying the nature of my study sample, I am compelled to comment on this thesis's beginnings. This thesis did not begin as a paleopathological survey. In my Peabody study sample, I had proposed to macroscopically search for cases of mucocutaneous leishmaniasis (ML), explore social disease experience (when cultural and mortuary contextual data were available), and develop preliminary standard methods for quantifying leishmaniasis disease progression in osteological material. The significance of such an endeavor rested in the fact that leishmaniasis is an under investigated disease in antiquity.

This sample was selected based on clinical epidemiological data, current distribution of *Leishmania brasiliensis* (most common causative agent of New World ML [Jeronimo et al. 2006; Lucas et al. 1998; Murray 2009; Urbano et al. 2011]), and spatial origins of paleopathologically identified cases. Despite this sample selection process, no *convincing* cases of ML are noted. The possibility of not identifying ML was considered and planned for prior to osteological data collection. Consequently, my thesis is a series of case studies of paleopathological interest from my Peabody study sample.

I need note there are at least four reasonable possibilities for this paucity of macroscopic evidence concerning ML: (1) Some individuals (or at least one) in this

sample had ML but died before this disease manifested osteologically, as ML may take up to 20 years to present with skeletal involvement (Costa et al. 2009). (2) Some individuals (or at least one) in this sample had ML, but the osseous changes present at time of death were not advanced enough (and partially due to this lack of advancement, perhaps too non-specific) to be recognized as leishmanial in origin. (3) Some individuals (or at least one) in this sample had ML, but the fragmentary nature of their cranial remains precluded its identification. And, (4) the individuals in this sample simply did not have ML.

Terms and Definitions

There are certain disciplinary and multi-disciplinary terms I find necessary and unavoidable. But, because of the multiple ways these terms have been used throughout paleopathology's history (including the pathological conditions they have been associated with), these very terms may engender confusion in lieu of descriptive lucidity. For instance, while largely not the case at present, porotic hyperostosis was essentially synonymous with anemia (Buikstra 2010:402). Additionally, certain terms are ambiguous in that they may refer to a specific or broad range of criteria. For example, Scheuer and Black (2000:468-469) note *juvenile* may refer to any individual who is physiologically not an adult. Or, in some continental European countries, it may refer to that age range after first and second permanent molar emergence (7-14 years) and until spheno-occipital synchondrosis closure (about 22 years). (The cited authors indicate that these numeric ages for second molar emergence and spheno-occipital synchondrosis closure are too late.) For these reasons, it is necessary to comment on and define terms that have the

potential to elide intended meanings and understandings. Below, in alphabetical order, I briefly flesh out the terms relevant to this text.

Cribra Orbitalia

Presently, *cribra orbitalia* is the term widely applied to orbital lesions that are osteolytic (porous), osteoforming (hypertrophic), or a combination of the two (porous, hypertrophic). Porotic hyperostosis of the orbit, hyperostosis spongiosa orbitae (spongy hyperostosis of the orbit), and usura orbitae are synonymous with *cribra orbitalia* (Angel 1967; Steinbock 1976). I am well aware of the literature's (past) tendency to almost ubiquitously posit iron-deficiency anemia as the cause of *cribra orbitalia* in a given study. To clarify, I do not disavow that iron-deficiency anemia may engender or be associated with orbital lesions (see, e.g., Britton et al. 1960; Mcilvaine 2013; Oxenham and Cavill 2010; Stuart-Macadam 1985, 1987a,b, 1989a,b, 1991, 1992; Wapler et al. 2004). However, *solely* using dry bone to grossly diagnose iron-deficiency anemia from *cribra orbitalia* is an exercise in drawing conclusions beyond the data. *Cribra orbitalia* does not have an unvarying, specific etiology; nor is it a distinct disease entity. Rather, it is a non-specific symptom hypothetically and materially associated with many bone-impacting conditions (e.g., among many others, Blom et al. 2005; Brickley and Ives 2008; Buckley 2000; Carli-Thiele and Schultz 1997; Larsen 1997; Ortner 2003; Schultz 2001; Steinbock 1976; Vradenburg 2001; Walker et al. 2009; Wapler et al. 2004).

Disease

Disease is on the one hand “physiological failure. This is the fact of disease itself, recognized, in the burial record, by characteristic lesions in human tissues” (Fay 2006:190). On the other hand, disease is conceptual, and is referred to as disease culture (Fay 2006:190). That is, disease is influenced by sickness ideologies. And, as such, “the processes of naming, grouping and explaining diseases are culturally contingent and do not transcend time and space” (Fay 2006:190). Unless otherwise noted, this text employs the former.

Disability

Garland-Thomson (2002) defines disability as a body being deemed deviant. However, this deviance is not a product of the body in-and-of itself. Rather, this deviance is sequela to the contextually specific meaning ascribed to a body.

Impairment

Impairment is a physical, functional restriction. But, it needs to be noted that an impairment in the archaeological record cannot be equated with the experience of a person with a similar impairment today (Cross 2007). That is, “the meaning and effect of impairment is culturally constructed, what may be regarded as incapacitating today may not always have been so regarded” (Cross 2007:187).

Infantile Cortical Hyperostosis (Caffey’s Disease)

While noting infantile cortical hyperostosis (ICH) is of uncertain etiology, some paleopathologists and bioarchaeologists seem to favor the hypothesis that a virus causes

ICH (e.g., Bourbou 2010, 2013). Lewis and Gowland (2009:49) note ICH has been used in the paleopathological literature as an all-encompassing term for widespread lesions on infant remains. Until further research into ICH's skeletal manifestations is conducted, they suggest it may be more "prudent" to regard ICH as descriptive of a set of lesions, similar to cribra orbitalia and porotic hyperostosis. Here, however, I recognize ICH as a distinct clinical disease entity characterized by subperiosteal bone formation, with recent evidence suggesting it is autosomally inherited (Guerin et al. 2012; Kamoun-Goldrat and le Merrer 2008; Mahalingam et al. 2013; Nemec et al. 2012; Nistala et al. 2014; Shandilya et al. 2013). Whatever the triggering agent is, in part, genetic influences seem to determine this disease's appearance (Jaffe 1975:282).

Juvenile

In this text, *juvenile* is used to refer to an age unit of analysis of any individual who is *physiologically* not an adult, where physiological age is "the sequence of physical changes associated with human growth, maturity, and senescence" (Sofaer 2011:286). Because the adult body is the standard (i.e., normative) body in biomedicine (Geller 2009) and biomedicine informs paleopathological and bioarchaeological investigations (Geller 2005, 2008, 2009), I explicitly reject employing the term *sub(-)adult*. Either wittingly or unwittingly, labeling a body as *sub(-)adult* implies these bodies are subnormal (i.e., deviant), even in the most "objective" settings. To do so is to impose contemporary Western cultural understandings of age on temporally and spatially distant bodies. Indeed, *juvenile* is beginning to replace *sub(-)adult* in the literature (Scheuer and Black 2000:469).

Porotic Hyperostosis

Alternative names include osteoporosis symmetrica, cribra cranii, and spongy hyperostosis. Angel (1966, 1967) used porotic hyperostosis (PH) specifically to signal instances of diploic expansion in conjunction with porous and hypertrophic lesions of the cranium. He considered these porous, hypertrophic lesions *in association with* diploic expansion to be indicative of anemia. Scholars later deployed this term in instances for which there was no evidence of diploic expansion (Bauder 2009; Ortner 2010:xviii). Consequently and problematically, diagnoses of anemia based on purely porous or porous, hypertrophic lesions of the cranium are abundant in the literature (Ortner 2003, 2010). Similar to cribra orbitalia (see above), I do not consider porotic hyperostosis (PH) a distinct disease entity. Nor do I consider PH a synonym for anemia. I regard PH as a non-specific symptom. This is in accordance with present osteological practice. Ortner's definition, which is adopted in this text, accurately represents this symptom's name. Ortner (2003:55, 2010:xviii) defines PH as pathological porous, hypertrophic lesions of the skull vault—with anemia being implicated only in the presence of marrow hyperplasia.

CHAPTER 2: MATERIALS AND METHODS

In skeletal paleopathology the basic method is observation. [Ortner 1992:6]

Out of nearly 1300 Peruvian crania available for study, I selected 196 (see *Appendix A* for Peabody object numbers; bolded numbers indicate individuals presented in this thesis). These crania are housed at the Peabody Museum of Archaeology and Ethnology at Harvard University, Cambridge, Massachusetts. As mentioned above, this selection largely reflects geographic proximity to ML occurrence from clinical epidemiological data, current distribution of *L. brasiliensis*, and geographic proximity to paleopathologically diagnosed cases of ML. Data collection took place at Peabody's Osteology Department during the dates of January 6-17, 2014. I have yet to unearth detailed reports on diseases in these crania (although see Newman 1947:17-18, 42-43). Past works on Peabody's collection of Peruvian crania seem to have focused on cranial shaping, trephination, and assessing "race."

Along lines of age, this study sample comprises 171 adults, 24 juveniles, and 1 of unknown age. With regard to sex, there are 104 males, 63 females, and 29 of unknown sex. Peabody's Osteology Department records provided sex, general age categories (e.g., adult, child, infant), and these remains' contexts. In cases where I provide numeric age estimates, my methods of assessment are given in the individual case study reports.

Skeletal inventories are coded following Buikstra and Ubelaker's (1994) system. A code of "1" indicates an element is greater than 75% complete; "2," that 25 to 75% of a

given element is present; and "3," that less than 25% of a given element is present. In Buikstra and Ubelaker's (1994) system, leaving a field blank indicates that an element is missing. I opt to record "0" for a missing element instead. This is done to avoid future confusion in data analysis. In other words, I would not have to ask myself: "Is this field blank because a skeletal element is missing? Or, is it blank because I forgot to fill in that field?" (see Stodder 2012 on data recording issues). In cases where additional modification of this coding system felt needed, the modification and its parameters are defined in the individual case study reports.

Cribra orbitalia (porous and/or hypertrophic lesions of the orbital roof) is graded following Stuart-Macadam's (1991) classificatory system, which is based on Nathan and Haas (1966). "0" indicates "normal" bone structure; "1," capillary-like impressions; "2," scattered fine foramina; "3," large and small isolated foramina; "4," foramina linking into a trabecular structure; "5," trabecular structure outgrowth from outer table surface. Orbital roof sector definitions also follow Stuart-Macadam (1991). It is significant to reiterate that I do not imply iron-deficiency anemia in recording cribra orbitalia. Porotic hyperostosis and dental pathologies are graded following Steckel et al.'s (2005:13-14 and 16-18, respectively) criteria. For cribra orbitalia, porotic hyperostosis, and dental pathologies, in the presence of mixed lesion grades, the most "severe" form is recorded and a "mixed" state is noted. All of these lesions are accompanied by precise, unambiguous, and clear pathological description.

For all other pathologies, I opt for a descriptive paleopathology (see Mann and Hunt 2012:11). Pathologies observed on individuals are discussed and explained in the individual case studies. I use Ortner's (2003:49-51) outline as a guide for

paleopathological description and employ his procedures for gross skeletal differential diagnosis. Ortner's procedures and methods are as follows. (1) Describe all skeletal elements present; (2) thoroughly describe the presence, location, and morphology of observed pathology while using at the very least a hand lens; (3) determine whether these pathological conditions are antemortem or postmortem. Distinguishing between antemortem and postmortem skeletal changes is extremely difficult. Typically, however, antemortem pathologies are smooth and rounded along lesion edges and postmortem ones are jagged, irregular, and sharp. Antemortem pathologies tend to show signs of osteoblastic repair. "This is usually expressed by a rounding of destroyed edges or the proliferation of reactive bone at the margins of the lesion" (Ortner 2003:46). It needs to be noted that myeloma is an exception to this rule. One of the best ways to distinguish between antemortem and postmortem changes is bone coloration (Pinhasi and Bourbou 2008).

Elements affected by postmortem changes, however, will vary in coloration when compared to other elements, while antemortem and perimortem alterations will present coloration that is the same as the rest of the skeleton (Pinhasi and Bourbou 2008:34). (4) If a pathological condition is determined as antemortem, its etiology must be reconstructed via the use of unambiguous terminology, precise description of atypical distribution and location, and a descriptive summary of observed features.

Moreover, in the absence of molecular techniques, CT scanning (computerized tomography), and radiological analyses, as is the case in my research, accurate diagnosis of diseases in skeletal remains is dependent upon considering anatomic specificity, ecological and geographical restrictions, and epidemiological data. (Artistic renderings

and ethnohistoric accounts may also be useful indicators, but caution is necessary.) In this work, the assumption is that the disease manifestations that occur today are probably similar to those that occurred in the past (Buikstra 2010:398) (although see Ragsdale 1996).

CHAPTER 3: FACING SCURVY HEAD-ON. A CASE OF PROBABLE SCURVY AND LIKELY ANEMIA COMORBIDITY IN A PERUVIAN JUVENILE'S CRANIAL REMAINS

Introduction

Peabody Object Number 80-61-30/23985.0 (henceforth, Individual 23985) (Figure 1) is an archaeological Peruvian juvenile represented only by skull remains. I asked about this individual's precise temporal and spatial origins as well as potentially associated mortuary materials. Jane Rousseau, Collections Steward in the Peabody's Osteology Collections Division, told me this information is unfortunately unknown (verbal conversation with the author, January 17, 2014). I have yet to unearth any previously published paleopathological reports on this individual; nor have I found any further information on cultural or mortuary context. However, after sleuthing through *Reports of the Peabody* and corresponding object numbers, it seems that this individual was accessioned in 1880 as part of Dr. W. S. Bigelow's donation of materials from "ancient graves" in Peru (Putnam 1881:10, 35-36). Peabody's online records place "ancient graves" as an archaeological site. Rather than a specific archaeological site, my readings of *Reports of the Peabody* suggest that "ancient graves" is a general descriptor.

I argue this individual exhibits lesions consistent with probable scurvy (vitamin C deficiency) and likely anemia comorbidity. The salience of this case report rests in the fact that it reflects, to my knowledge, the first Peruvian juvenile in the English language

literature reported to display evidence of scurvy and anemia comorbidity. Additionally, in the English language literature, there are very few publications concerning juvenile scurvy from Peru. As such, this chapter gives Individual 23985 considerable attention.

Figure 1: Individual 23985 in anterior view. 80-61-30/23985.0, *Courtesy of the Peabody Museum of Archaeology and Ethnology, Harvard University.*



Demographic Data

As noted above, Individual 23985 is an archaeological Peruvian juvenile of unknown precise temporal and spatial origins (Peabody records). Because assessing sex from juvenile remains is tenuous, no attempt was made. This is in accordance with standard osteological practice (Mahoney-Swales and Nystrom 2009:32).

Age Assessment

Consulting Buikstra and Ubelaker's (1994:51, figure 24) dental aging diagram, Individual 23985's dental age is approximately 6 years \pm 2 years based on mandibular tooth eruption. This age estimate is consistent with the *London Atlas*. Consulting the *London Atlas*, which presents stages of tooth eruption through alveolar bone, this individual is between 5.5 and 6.5 years (AlQahtani et al. 2010:485, figure 6). I recognize there are caveats with using dental eruption to assess age. Dental eruption varies along lines of genetics, sex, ancestry, nutrition, and disease states (Lewis 2007; Scheuer and Black 2000). For instance, rickets may delay dental eruption (Lewis 2007:122; Schuurs 2013) and scurvy may result in premature dental shedding (Schuurs 2013). (For a discussion of various diseases delaying or accelerating dental eruption, the reader is referred to Schuurs [2013]. Schuurs's text is a clinical one. So, care must be taken in distinguishing dental eruption from alveolar bone [typical of archaeological settings] and from gums [typical of clinical settings] [Saunders 2008; Scheuer and Black 2000]).

I also recognize assessing age from dental formation is preferred to dental eruption in its accuracy (Lewis 2007:38; Saunders 2008:126; Scheuer and Black 2000). However, here, dental eruption is used because of its ease and speed in application (Scheuer and Black 2000). (For methods in assessing dental formation and reviews on that literature, the reader is referred to Buikstra and Ubelaker 1994; Lewis 2007; Moorrees et al. 1963a,b; Saunders 2008; Saunders and Spence 1986; Schaefer et al. 2009; Scheuer and Black 2000; Smith 1991. For caveats on relying on dental formation in age assessment, the reader is referred to Halcrow et al. 2007; Phillips and van Wyk Kotze 2009.)

Maxillary tooth eruption and cranial suture appearance at multiple sites are consistent with this age estimate (given signs of cranial shaping in this case).

Skeletal Analyses and Scurvy

Juvenile scurvy is not uncharted territory in the bioarchaeological and paleopathological literature (Klaus 2014a). However, only recently were criteria proposed for identifying juvenile scurvy in cranial remains. In the below sections, I flesh-out these proposed criteria, present controversies surrounding these criteria, provide evidence supporting these criteria, and discuss potential cases of Peruvian juvenile scurvy.

Proposed Scorbutic Lesions in Juvenile Crania

In a series of papers, drawing on skeletal remains and pathophysiological theory, Ortner and various colleagues established criteria for assessing scurvy in juvenile crania (i.e., Ortner 1984; Ortner and Ericksen 1997; Ortner et al. 1999; Ortner et al. 2001). These criteria may be summarized as “bilateral and symmetrical porosity of cortical bone...The most common of these bilateral and symmetrical lesions are those associated with the greater wing of the sphenoid” (Brown and Ortner 2011:205). Pathological sphenoid bone porosity is considered a diagnostic, though not necessary, criterion (see Brickley and Ives 2008:57, table 4.2). In this context, porosity is “a localized condition in which fine holes, typically less than 1 mm in diameter, penetrate a compact bone surface” (Ortner et al. 2001:344).

These cranial lesions are proposed to result from mechanical stress associated with chewing and blood vessel hemorrhage (Roberts and Manchester 2005:235-236). (Brown and Ortner [2011] and Ortner and Ericksen [1997] provide detailed theoretical explanations for the pathogenesis of these lesions.) Other skeletal reports thoroughly document scorbutic changes in juvenile skulls (e.g., among others, Brickley and Ives 2006, 2008; Bourbou 2014; Brown and Ortner 2011; Halcrow et al. 2014; Klaus 2014a; Mahoney-Swales and Nystrom 2009; Mays 2008a; Ortner 1984; Ortner 2003; Ortner and Ericksen 1997; Ortner et al. 1999; Ortner et al. 2001; Stark 2009, 2014; Stuart-Macadam 1989a). In Table 1, I summarize documented macroscopic lesions of juvenile cranial remains thought to result from or be associated with scurvy.

Table 1: Macroscopic changes associated with scorbutic juvenile skulls from the paleopathological and bioarchaeological literature.

<i>Sites affected</i>	<i>Skeletal pathology</i>	<i>References</i>
Alveolars	Porosity on surrounding region of alveolar bone; Alveolar processes and sockets are undoubtedly affected by scurvy. However, due to dental eruption and growth, porosity is a “normal” occurrence at these sites. Clear baselines for “normal” or pathological porosity at these sites are yet to be established. However, for porosity to be considered pathological, it should extend well beyond the alveolar process surrounding an erupting molar. New bone formation on alveolar sockets.	Brickley and Ives 2006; Halcrow et al. 2014; Ortner 1984; Ortner et al. 1999
Endocranium	Pathological branching vessel impression (sometimes including bone growth) (i.e., “branched lysis”). Active and healed bone plaques are compatible with a reaction to chronic hemorrhage. Parietal, occipital, temporal: pathological woven bone on internal squama (bilateral or unilateral)	Brown and Ortner 2011; Geber and Murphy 2012; Lewis 2004; Mays 2008a:180
Frontal	Cortical porosity; Vascular impression lesions (usually with some cortical porosity); New bone formation; Boss enlargement	Brickley and Ives 2006; Brown and Ortner 2011; Halcrow et al. 2014; Klaus 2014a; Ortner et al. 1999
Mandible	Medial surface porosity of coronoid process at insertion of temporal muscle; Porosity of pterygoid fovea; Porosity	Brickley and Ives 2006; Brown and

Table 1 CONT.

	surrounding mandibular foramen associated with pterygoid muscle; Porosity on labial surfaces, lingual surfaces, and ramus.	Ortner 2011; Ortner et al. 1999
Maxillae	Pathological (usually bilateral) porosity of posterior maxilla, infraorbital foramen, and to a lesser extent on inferior bone of nasal aperture. Hypertrophic (usually bilateral) lesions have also been noted.	Brickley and Ives 2006; Ortner 1984; Ortner and Erickson 1997; Ortner et al. 1999
Occipital	The most typical site for porous cranial vault lesions is in the lambdoid region. Cases coupled with hyperostosis are not uncommon. Vascular impressions (usually with some porosity); Multifocal hypertrophic bone formation; Porosity at <i>pars basilaris</i>	Brown and Ortner 2011; Klaus 2014a; Ortner et al. 1999
Orbits	Usually bilateral cribra orbitalia with no evidence of marrow hyperplasia; (Marrow hyperplasia would be indicative of anemia.) Lesions may be porous or hypertrophic (i.e., bone formation). Orbital roof lesions in association with other lesions of the skull provide support for scurvy. Porous lesions on the lateral orbit have been noted.	Brickley 2000; Crandall 2014; Lewis 2007; Ortner 1984; Ortner and Erickson 1997; Ortner et al. 1999; Stark 2014
Palatines	Denser porosity found in the anterior palate extends markedly onto the posterior palate. ("Normally," palatal porosity makes a u-shaped arc and is denser in the anterior portion. Increased porosity density and porosity extending to the posterior palate are considered pathological.)	Ortner et al. 1999
Parietals	Vascular impression lesions (usually with some porosity); Boss enlargement (Parrot's swelling); Multifocal hypertrophic bone formation; Cortical porosity	Brown and Ortner 2011; Ortner and Erickson 1997; Klaus 2014a
Sphenoids	Greater wing (superior and inferior) bilateral porous lesions (and sometimes hypertrophic) occurring in overwhelming association with cribra orbitalia. This pathology is probably virtually pathognomonic for scurvy. The greater wing of the sphenoid typically has vascular channels passing through it. But, in cases of scurvy, cortex-penetrating pores are smaller and greater in number. Disorganized spicule bone formation on the lesser wings and sphenoid body; Bilateral porosity (hypertrophy also possible) at lesser wings, foramen rotundum, and pterygoid fossae	Brickley and Ives 2006:168; Geber and Murphy 2012; Klaus 2014a; Ortner 1984; Ortner and Erickson 1997; Ortner et al. 1999; Ortner et al. 2001
Temporals	Bilateral dense porosity penetrating the cortex and continuing to the greater wing of the sphenoid has been noted in association with the overlying temporalis muscle. (Porous lesions less than 1 mm in diameter.)	Ortner 1984; Brown and Ortner 2011
Zygomatics (malars)	Pathological porosity of internal (posterior) and/or lateral aspect(s).	Armstrong et al. 2014; Brown and

Table 1 CONT.

Ortner 2011; Ortner
et al. 1999; Stark
2014

Comments

"Bilateral and symmetrical porosity of cortical bone are characteristic. The most common of these bilateral and symmetrical lesions are those associated with the greater wing of the sphenoid. This type and distribution of abnormalities is unlikely to occur in other blood disorders" (Brown and Ortner 2011:205). Porosity penetrates outer cortex (Bourbou 2014; Klaus 2014a,b). New bone formation may be fairly unorganized, patchy, and sclerotic (needs differentiation from infection) (Klaus 2014b:24). If hypertrophy occurs, bone formation will be superficial to compact bone (Bourbou 2014). Possible remnants of calcified hemorrhages alongside periosteal reaction/hypertrophy have been identified in association with bleeding on dry bone (Kozłowski and Krajewska 2012).

The recognition of these lesions informs a growing corpus of literature on juvenile scurvy in antiquity. Recently (June 2014), for instance, the esteemed *International Journal of Paleopathology* published a special issue on scurvy covering a broad set of research questions, honoring Ortner's contributions on this metabolic disease.

Bones of Contention

When it comes to diagnosing scurvy, however, special journal issues and tantalizing research questions are not free of opposition. Ortner and coworkers infer these diagnostic criteria using archaeological skeletal materials, historical medical reports, and a working knowledge of anatomy and pathology—not documented clinical cases of scurvy (Ortner et al. 1999). As such, some scholars have a bone to pick with these criteria, and understandably so. I respond to two papers, Rothschild 2013 and Melikian and Waldron 2003, which offer reasonable critiques on employing the criteria presented above to diagnose scurvy in juvenile crania. In assessing these papers, to paraphrase Geller's (2009) remarks in a critical analysis of sex, gender, and heteronormativity in bioarchaeology, my aim is not to be contrary for the sake of being contrary; nor is it to

make ad hominem attacks. Rather, my purpose is to address an unavoidable and welcome series of legitimate concerns.

Rothschild 2013

In characterizing bone “surface discontinuity” through epi-illumination, Rothschild (2013) studies an array of skeletal specimens with known pathological conditions. In so doing, he offers misgivings on two lesions that bioarchaeological and paleopathological reports have associated with juvenile scurvy. (He does not present scorbutic specimens.) These two lesions are sphenoid bone porosity and cribra orbitalia.

Rothschild 2013: Sphenoid Bone Porosity and Scurvy

Rothschild asserts,

Ortner et al. (1999) speculated that sphenoid porosity identifies scurvy, suggesting that bleeding at the sites of muscle insertion is responsible. However, that would require evidence of enthesitis. This evidence is lacking. The collar or rim component of the attributed Sharpey fiber attachments has not been recognized at sites of sphenoid surface discontinuity and therefore, falsifies that hypothesis. Further, Melikian and Waldron (2003) documented the lack of sphenoid porosity among individuals with clinically documented scurvy. That issue should be considered resolved: The presence of sphenoid porosity cannot be utilized to identify scurvy. [2013:587]

Here, at least four points of rebuttable (or consideration) are warranted.

(1) To the premise that Ortner et al. speculate sphenoid porosity identifies scurvy, suggesting bleeding at masticatory muscle sites is responsible for this porosity: Ortner et al. (1999) do argue masticatory stress and capillary hemorrhage in scurvy engender sphenoid porosity—though they do not speculate. They construct a theoretically plausible

inference grounded in anatomy and clinical understandings of scurvy pathogenesis (see Ortner and Ericksen 1997). This inference is in accordance with current understandings of angitis, osteoclastic and osteoblastic responses to focal hemorrhage, and the ways in which hematomas impact the periosteal envelope (Crandall and Klaus 2014:4).

Moreover, as Rothschild (2013) himself argues, because it is a relatively imprecise descriptor and is employed multi-disciplinarily, the term *porosity* has intended or unintended multivocal effects. To this end, Ortner and coworkers (Ortner 1984; Ortner and Ericksen 1997; Ortner et al. 1999; Ortner et al. 2001) qualify this porosity through scanning electron microscopy. This porosity is pathological, cortical, usually less than 1 millimeter in diameter, and truly penetrative (i.e., floor of lesion not visible)—sometimes presenting with hypertrophy (Ortner et al. 1999:323-325; Ortner et al. 2001:344-346). This porosity on the surface of the greater wing of the sphenoid bone is not to be confused with this bone’s “normal” foramina, nor with “normal” porous appositional bone in children. This porosity is further qualified as *virtually* pathognomonic when found *in association with* other lesions attributable to masticatory stress and spontaneous hemorrhage. In other words, these pathological observations of the sphenoid bone in isolation do not argue for a diagnosis of scurvy—all the lesions (Table 1) taken together do (Brickley and Ives 2006, 2008; Ortner et al. 1999).

(2) To the premise that evidence of enthesitis is lacking: While I am inclined to sympathize with this premise, Rothschild’s appeal to readily observable enthesitis (i.e., enthesal inflammation or irritation) might be premature and perhaps in need of complication. For Rothschild (2013:586, emphasis added), “discontinuity [i.e., pore or

hole] with elevated rims appears to be *required* for recognition of hematomas and for *enthesitis*.” In a study of arthritis in gorillas, Rothschild and Rühli (2005:210) note enthesitis may take the form of new bone formation. To the latter take on enthesitis, a clinical case re-diagnosed as juvenile scurvy, possibly by Thomas Barlow himself, exhibits hypertrophic bone formation bilaterally on the greater wings of the sphenoid bones (Ortner 2011:7; cf., Melikian and Waldron 2003:210; Ortner 2003:386). To the former, although greatly improved, methodologies in recording and identifying enthesal changes are works in progress (see, e.g., Havelková et al. 2011; Havelková et al. 2013; Henderson 2009a,b,c, 2013; Henderson et al. 2013; Jurmain et al. 2012; Jurmain and Villotte 2010; Mariotti et al. 2004, 2007; Mariotti et al. 2009; Santos et al. 2011; Schlecht 2012; Villotte and Knüsel 2013). In part, this is due to the dynamic structures of entheses, which are only as of late becoming unraveled in the clinical literature and even more recently being recognized in bioarchaeology.

In humans, entheses constitution is dependent upon (among other factors) anatomical site (Hems and Tillmann 2000; Villotte and Knüsel 2013) and age of individual (Claudepierre and Voisin 2005; Henderson 2009b,c, 2013; Henderson et al. 2013; Jurmain et al. 2012; Villotte and Knüsel 2013). For instance, a study of masticatory muscles in adult human cadavers reveals it is more common to find a mixture of enthesal structures in a given attachment zone than to find entheses with purely cartilaginous, periosteal, or bone insertions (Hems and Tillmann 2000:208). This pattern contrasts with the relatively uniform structure in a given attachment zone of trunk and limb entheses (Hems and Tillmann 2000:205) (although see Benjamin et al. 2002 and Benjamin et al. 2006 who argue limb entheses do not have a uniform structure. Rather,

they are part fibrocartilagenous and part fibrous, with the fibrous area in the most distal part of the enthesis.)

Thus, skull fibrous entheses possess a mixture of fibrocartilaginous and fibrous structures (Benjamin et al. 2002; Hems and Tillman 2000). Fibrous entheses (e.g., temporal muscle, masseter muscle, periodontal ligament [Hems and Tillmann 2000; Henderson 2009b,c]) tend to be more resistant to overuse injuries than fibrocartilaginous entheses (Claudepierre and Voison 2005; Jurmain et al. 2012). Periosteal fibrous entheses leave smooth marks on bone and bony fibrous entheses roughened, raised marks (Henderson 2009c; cf., Benjamin et al. 2002). But, precisely when are these roughened, raised marks considered pathological and not “normal” variations? This question remains unresolved (Henderson 2009a,c). Fibrous entheses may be periosteal but become bony with age (Benjamin et al. 2002). The specific age at which this transition occurs is unclear (Henderson 2009b; cf., Benjamin et al. 2002). Some fibrous entheses remain periosteal into old age (Hems and Tillmann 2000).

Enthesal changes, like so many bony responses, are either osteolytic or osteoforming (Mariotti et al. 2004; Jurmain et al. 2012), with respective nuances there-of (Mariotti et al. 2004). Differences along lines of anatomical site, age, stimulus, and other factors result in these nuanced responses (Schlecht 2012). The association of fibrous enthesal changes with periostitis or other diseases is largely unclear (Claudepierre and Voison 2005:32; Henderson 2013:67) (although see Henderson 2009b for possible fibrous enthesal changes in Paget’s disease and in hypertrophic osteoarthropathy).

Here, it bears reiterating that Ortner and colleagues studied juvenile crania. Unfortunately, there is a dearth of data on human juvenile cranial entheses and fibrous

enthesees in general. Anthropological studies of enthesal changes often exclude juveniles from their samples due to confounding factors like age and growth (see, e.g., Milella et al. 2012; Schlecht 2012; Schrader 2012; Steen 2003; Weiss 2010). Even if, however, Rothschild's (2013) appeal to enthesitis (necessarily identified by elevated rim components, as he defines it) holds true, it is necessary to consider that pathological, truly penetrative porosities of the greater wing of the sphenoid bone may be of a not-so-severe scorbutic state, prolonged scorbutic state, or more recent traumatic origin (see, e.g., Wood et al. 1992 on the Osteological Paradox), and therefore lack "elevated rim components." In other words, the osteo-immuno-pathophysiological response at those sites might not be stimulated enough. And, Lewis (2000) notes that juvenile Sharpey's fibres are less numerous and shorter than in adults. Thus, this feature might impact the presence of readily observable elevated rim components on the sphenoid bone. But, again, the constitution of juvenile cranial entheses remains unclear. Another confounding factor in cases considered to represent scurvy is researchers documenting truly penetrative porosities without noting edge morphology (see section 3.b). Given this paucity of information and the complex nature of entheses, assuming presence of elevated rim components as evidence of enthesitis on the sphenoid bone, especially of human juveniles, might be premature until additional insights are brought to the fore.

(It is interesting to note that six cranial specimens of possible scurvy [two adults, one late adolescent, remainder of unspecified age] exhibit "combed hair" lesions [striations] corresponding to the orientation of the temporalis muscle on the parietal bones [Cargill 2014]. Given these striations' orientations, two possibilities are noted: [1] These lesions are sequela to temporal fascia fibers of the temporalis muscle pulling on

galea aponeurotica [Cargill 2014]. And, [2] these lesions are sequela to cranial angiogenesis, a feature thought to be the primary cause of porosity in scorbutic archaeological remains [Cargill 2014]. It should also be noted that Cargill considers the two adult cases as representing residual osteological effects of scurvy, which Megan Brickley considers doubtful [Cargill 2014].)

(3) To the premise that the collar or rim component of the attributed Sharpey fiber attachments has not been recognized at sites of sphenoid surface discontinuity and therefore, falsifies the scurvy-sphenoid porosity hypothesis: It is unclear if this premise is in reference to findings in Rothschild's (2013) study or to other scholars' anatomical studies and scurvy diagnoses. I separately address the possibility of his statement being in reference to his study and to previous work. (Again, Rothschild's study is not centered on scorbutic manifestations; nor does he present scorbutic specimens. His aim is to characterize bone surface discontinuity in general.)

(3.a) If premise "3" is in reference to Rothschild's own study: His sphenoid discontinuity specimens are from Yucatan black howler monkeys (*Alouatta pigra*), North American beavers (*Castor canadensis*), and mountain beavers (*Aplodontia rufa*). However, the only image of sphenoid discontinuity presented is of *Pongo pygmeus* (Bornean orangutan) (2013:584, figure 2f). The depicted *Pongo* discontinuity does not match Ortner et al.'s (1999) definition of porosity. Non-penetrative furrows are depicted. In terms of the above-mentioned specimens, precise etiologies for these sphenoid discontinuities are not specified, though vascular origins are suggested. However, he

(2013:585) does note that “sphenoid ‘porosity’ is present in the form of small circular perforations in *Alouatta pigra*. Circular holes of variable size are noted in *Aplodontia rufa* sphenoids, surrounded by halos and associated with channels. Vascular channels are present in *Castor canadensis* sphenoid’s.”

I do not disavow the utility of studying non-human animal anatomy and pathological responses as a proxy for human ones. But, it is necessary to reiterate that entheses vary in constitution depending on anatomical site, age, and animal represented (see above and Schlecht 2012). These lines of variability potentially impact observable osseous changes (Schlecht 2012). Human skeletons undergo a much longer period of maturation, which might impact degrees of bony response to environmental forces (Schlecht 2012). (I need note there is debate concerning the definition of *Sharpey fiber* and which entheses possess Sharpey fibers [Benjamin et al. 2002; Benjamin et al. 2006; François et al. 2001]. Some researchers hold that fibrocartilaginous entheses possess Sharpey fibers, while others maintain that Sharpey fibers are only present in fibrous entheses [Benjamin et al. 2002; Claudepierre and Voisin 2005; François et al. 2001; Henderson 2009b]. François et al. [2001:256] argue Sharpey fibers are “scanty” when serving to attach periosteum. Additionally, not all muscles attach to bone via tendons and not all tendons possess entheses [Benjamin et al. 2002]. “There are many muscles that attach to relatively large areas of the skeleton by ‘fleshy’ fibres, a few tendons that link one region of a muscle to another and others that are simply present on the surface of a muscle as aponeuroses [e.g., *galea aponeurotica*] that enable one muscle to glide over another” [Benjamin et al. 2002:932].)

(3.b) If premise “3” is in reference to previous works: It bears mentioning that studies on adult humans note “Sharpey fibers” at the inferior temporal surface (lateral pterygoid muscle) and infratemporal crest (sphenomandibular bundle of temporal muscle) of the sphenoid bone’s greater wing (Hems and Tillmann 2000; Palomari et al. 2013). With individual variability, a study notes additional *Temporalis m.* origins at the temporal surface of the zygomatic bone and ectocranial surface of the greater wing of the sphenoid bone (Elazab et al. 2006:242). (Fibers used in mastication at the sphenoid bone’s pterygoid plates are worth noting [Hems and Tillmann 2000].)

Additionally, reports (the present one included) document pathological, truly penetrative porosities of the sphenoid bone without noting their edge morphology. This fact may reflect a deficit in recording and reporting procedures and current knowledge on entheses rather than a lack of evidence on the remains themselves (see also factors outlined in 2).

(4) To the premise appealing to Melikian and Waldron 2003: Melikian and Waldron’s (2003:209) sample of clinically documented scorbutic cases (all juveniles) included only four individuals. But, they do note that one of these four exhibited hypertrophic lesions on the greater wing of the sphenoid bone (cf., Ortner 2003:386; Ortner 2011:7). Further, while maintaining sphenoid bone porosity at the greater wing is virtually pathognomonic of scurvy when found in association with other lesions consistent with masticatory stress, Ortner et al. (2001:348) wisely point out the possibility “that scurvy can affect the skeleton without necessarily involving the greater wing of the sphenoid.” It bears mentioning that virtually pathognomonic is not the same as

pathognomonic. (By no means do I claim Rothschild [2013] or Melikian and Waldron [2003] confuse the two.) Virtually pathognomonic indicates a given symptom possesses a considerable but not unique specificity to a certain disease. Both pathognomonic and virtually pathognomonic do not mean that a given symptom will necessarily be present in a given disease.

A more detailed treatment of Melikian and Waldron's (2003) work is provided in the section, *Melikian and Waldron 2003*.

Rothschild 2013: Cribra Orbitalia and Scurvy

With regard to cribra orbitalia and scurvy, Rothschild claims,

The phenomena in cribra orbitalia is [*sic*] quite different, represented by very irregular disruption and side channels with new bone formation, similar to images reported by Brickley and Ives (2006), with or without full penetration. While [*sic*] they speculated that the orbital roof findings in 18–19th century infants from the St. Martin's cemetery in England were caused by scurvy, which seems an unlikely explanation in wild-caught monkeys. [2013:586]

He goes on to appeal to Melikian and Waldron (2003:210), who remark that the pathologies they observe in their clinically documented cases are not similar to archaeological material. (Again, the section *Melikian and Waldron 2003* provides a more detailed treatment of that work.) Rothschild (2013:586) then states that archaeological remains diagnosed as scorbutic are suspect and that cribra orbitalia's etiology is uncertain.

I agree that cribra orbitalia's etiology is uncertain. That is to say, cribra orbitalia must be considered a non-specific stress indicator, associated with multiple diseases and may have more than one etiology. On a gross, macroscopic level, I would advance that cribra orbitalia in isolation is of little diagnostic value. While Brickley and Ives

(2006:166-168) do attempt to better characterize orbital lesion morphology in their study and suggest that the orbital lesions they observe are due to scurvy, they do not take cribra orbitalia in isolation as indicative of this disease. Rather, the constellation of lesions observed in their study argues for a diagnosis of scurvy (Brickley and Ives 2006:171).

Whether or not scurvy causes cribra orbitalia *directly* is uncertain. For example, scurvy is well known to co-occur with anemia. Thus, we might ask, is anemia responsible for cribra orbitalia in such a case? Is vitamin C deficiency the culprit? Both interacting together? Either way, ocular health is in part dependent upon vitamin C (Semba 2007). And, that cribra orbitalia occurs in association with scurvy is unequivocal. Vitamin C-deficient monkeys are documented to present orbital pitting (Steinbock 1976:246, 256). Ortner (2003:385) notes that Fraenkel's autopsies of scorbutic humans reveal orbital porous, hypertrophic bone.

Additionally, scurvy may potentiate subperiosteal orbital hemorrhages, which is a site of rapid physiologic growth (Semba 2007). Hemorrhage is most frequent at the orbital plate of the frontal bone, with the left eye more susceptible than the right (Semba 2007; cf., Barlow 1883:176; Still 1915:114). Subperiosteal orbital hemorrhage in scurvy (Barlow 1894:1032; Jaffe 1975:455; Sloan et al. 1999; Snow 1905; Still 1915:114; Verma et al. 2007) would in principle lead to new bone formation (Brickley and Ives 2008; Brown and Ortner 2011; Steinbock 1976:256).

Melikian and Waldron 2003

Now, I devote exclusive attention to Melikian and Waldron's study, as they too offer reasonable misgivings on diagnosing scurvy from juvenile cranial remains.

One hundred and twenty-three “subadults” constitute Melikian and Waldron’s (2003:208) study. These 123 are comprised of 104 archaeological British individuals and 19 archaeological Peruvian individuals (Melikian and Waldron 2003:208). In addition to these 123 individuals, they examined four clinically documented cases that served as positive controls (Melikian and Waldron 2003:209). Of the archaeological individuals, Melikian and Waldron (2003:210) identify seven (four British, three Peruvian) possible cases of scurvy. Following Ortner and coworkers’ definition of porosity (see above), these identifications are based on concomitant pathological porosity of the greater wing of the sphenoid bone, cranial vault, and orbital roof. The four British juveniles are the only possible scurvy cases to possess associated postcranial remains. The postcranial remains exhibit no grossly visible, macroscopic scorbutic pathologies, though radiology was not performed (Melikian and Waldron 2003:211). (For a discussion on diagnostic imaging criteria of long bones in scurvy, the reader is referred to Brickley and Ives 2008; Greenfield 1975; Jaffe 1975; Murray and Jacobson 1977a:670-673; Stark 2009, 2014; Taylor et al. 2010.)

Melikian and Waldron essentially offer two critiques. The first is:

[In this study,] there were no changes in the post-cranial skeleton which would be consistent with the diagnosis [of scurvy], including periostitis or alterations in the morphology of the metaphyses of long bones. If the cranial appearances are to be assumed to be caused by scurvy then it seems essential that typical post-cranial appearances should also be present. [2003:211]

Postcranial pathological changes occurring with cranial ones would greatly support a scurvy diagnosis. But, differences in (individual) symptoms and symptom presentation times should be noted (see, e.g., Popovich et al. 2009:408-411; Stark and Garvie-Lok 2011). Ocular complication in scurvy might be the first symptom (Snow

1905:100). Scorbatic living individuals have exhibited ocular complications (and sanious nasal discharge) with their limbs not being affected at all (Barlow 1883:176). From his own cases, Barlow (1894:1032) notes orbital hemorrhage occurring before limb symptom presentation, and in some of those cases the limb symptoms are slight—“great local tenderness, much irritability, but little swelling.” Interestingly, a 48-year-old woman presented with gingival hypertrophy as the sole gross manifestation of her scorbutic state (Li et al. 2008). Another interesting case involves a 5-year-old scorbutic boy who did not display the classic radiographic indicators of scurvy, though he did present with gelatinous marrow transformation, anemia, and limb pain and tenderness (Brennan et al. 2012). Differences in lesion resolution times further confound matters (see, e.g., Stark 2009:227-228). For instance, in a post-scorbutic state, we might expect to see remnant porotic cranial lesions but simultaneously note macroscopic limb lesion resolution (with some possible radiological exceptions) (Stark 2014:23). It is not clear if the individuals with associated postcranial remains in Melikian and Waldron’s (2003) work exhibit healed/healing lesions or active ones.

The second critique is:

The changes which we have seen in the [archaeological] skulls that we have examined do not conform in any respect to those found in the skulls of clinical cases... and their relationship to scurvy must be questionable, as must those described by Ortner and his colleagues... The diagnosis [of scurvy] cannot be supported, however, if the appearances do not conform to those seen in known cases of the disease which plainly those described here and elsewhere do not. It is, of course, entirely possible that less florid changes may accompany sub-clinical or mild cases of vitamin C deficiency and that these may be similar (or identical) to those seen here; without clinical confirmation, however, making a firm diagnosis is a leap of faith rather than a nosological certainty. [2003:211-212]

To be sure, the tacit assumption in paleopathology is that today’s diseases are similar to those of the past (Buikstra 2010:398). Clinically documented cases (of either

curated remains or contemporary living patients) exhibiting lesions similar to those described by Ortner and his coworkers would certainly lend credence to their inferences (Ortner 2003:390-391). I agree that diagnoses of skeletal diseases in the archaeological record necessarily require biomedical knowledge. But, an over-reliance on biomedicine to make a diagnosis is problematic in the following senses:

- (1) The number of available clinically documented infantile scurvy dry bone specimens for study is few (Mays 2008b:225).
- (2) Curated clinical cases are likely to be far more severe manifestations of disease than typically encountered archaeologically (Brickley and Ives 2006:171; Mays 2008b:225).
- (3) Not all the clinical diagnoses in museum collections would be acceptable today (due to past criteria employed or misdiagnoses) (Brickley and Ives 2006, 2008:11). And, many probably represent comorbidities rather than the one-disease-diagnosis usually offered (Brickley and Ives 2008:11). Other nutritional deficiencies may inhibit skeletal scorbutic manifestations (Agnew et al. 2008). For example, rickets and scurvy may co-occur but it is unclear which condition masks the other (Ortner 2003:385).
- (4) No other disease readily explains the concomitant lesion presence and distribution Ortner and coworkers observe (Mays 2008b:225). Additionally, these pathological changes are hinted at in early medical literature (e.g., Barlow 1883:169, 171) (Stark and Garvie-Lok 2011).
- (5) Although bone deposition of the skull vault is a known occurrence in juvenile scurvy, in contemporary living cases, cranial radiographs are usually avoided

(Stark and Garvie-Lok 2011). Moreover, with regard to the more subtle proposed changes in juvenile scurvy (e.g., sphenoid bone porosity), waiting on evidence from contemporary clinical case reports of living patients is futile. Radiographs in life are not sensitive enough to detect bone surface porosity (Rothschild 2013:581).

- (6) One of Melikian and Waldron's clinical cases is actually a very nice example of what Ortner and colleagues infer (Ortner 2011:7), displaying hypertrophic lesions of the greater wing of the sphenoid bone (see also above subsection, (4) *To the premise appealing to Melikian and Waldron 2003*).

If skeletal analyses are to advance, careful and reasonable inferences must be made (Ortner 2003:111), just like in any other scientific discipline.

Reconciling Evidence

As gleaned from above, in the context of juvenile scurvy, some skeletal analysts view the proposed scorbutic cranial changes (especially sphenoid bone pathologies) as presenting an aporia of sorts. There comes a stage in which theoretically plausible explanations require physical evidentiary grounding. The commentary to follow presents these evidences.

Roberts (1987) presents a case of a British juvenile (100 B.C. to 43 A.D.) exhibiting lesions consistent with those Ortner (1984) describes. (Ortner [1984] documents a series of pathological porosities related to masticatory stress and hemorrhage in an Alaskan juvenile skull from the modern time period. He attributes these changes to scurvy.) Roberts (1987) also conducts a radiographic analysis on her case,

noting arrested growth lines in long bones. Taking cranial and postcranial lesions together, she attributes these changes to scurvy. In their North American study sample, in association with their proposed suite of cranial lesions, Ortner et al. (2001) do identify macroscopic postcranial changes consistent with juvenile scurvy. Others also note postcranial changes consistent with scurvy co-occurring with the proposed cranial criteria (e.g., among others, Bourbou 2014; Brickley and Ives 2006; Brown and Ortner 2011; Buckley et al. 2014; Crandall 2014; Geber and Murphy 2012; Klaus 2014a; Lewis 2010; Lovász et al. 2013; Mahoney-Swales and Nystrom 2009; Mays 2008a; Ortner 2003; Wrobel 2014).

Stark's (2009) study is perhaps the most convincing in lending physical, evidentiary legitimacy to Ortner and coworkers' criteria. In his study of juvenile scurvy from ancient Greece, clinically accepted radiographic indicators are correlated to the proposed scorbutic macroscopic cranial changes. I find it significant to note that these radiographic indicators are assessed in consultation with Dr. Bhargava, an associate professor of radiology and diagnostic imaging at the University of Alberta (Stark 2009:164). In total, Stark (2009) shows that 21% (3/14) of cases in his study sample exhibit a co-occurrence of compelling cranial macroscopic criteria (including pathological porosity at the greater wing of the sphenoid bone) and compelling clinically accepted radiographic indicators of scurvy. He considers these cases as "strong" potential cases of scurvy. Stark (2009) also finds individuals expressing lesions consistent with Ortner and coworkers' criteria, but lack compelling radiographic evidence for scurvy. This finding includes individuals without and with sufficiently preserved postcranial remains for investigation. Others in his sample exhibit radiographic evidence of scurvy

but without compelling macroscopic criteria. This finding includes individuals with and without sufficiently preserved cranial remains for investigation.

Stark (2009) argues that his findings of clinically accepted radiographic indicators co-occurring with Ortner and coworkers' criteria lend strong evidence in support of the latter's criteria. Stark (2009) notes the overall variability in radiographic and grossly observable lesions in his scorbutic individuals may be attributed to scurvy severity or differences in timing of manifestations. Both possibilities require further clinical and archaeological investigation (Stark 2009). Infection co-occurrence may also be a confounding factor (Stark 2009).

Peruvian Cases of Scurvy from Antiquity

There are very few published reports in the English language literature on scurvy in archaeological Peruvian human remains (see Table 2, which is found on page 37). I am aware of only three (i.e., Klaus 2014a; Melikian and Waldron 2003; Ortner et al. 1999). Only one of these reports notes skeletal evidence of scurvy possibly co-occurring with another disease. Klaus (2014a) suggests one of his cases displays scurvy and rickets comorbidity. He believes this individual is the first documented Andean case to express this particular comorbidity.

Other works on Peruvian remains mention the possibility of scurvy in passing. In a discussion of various diseases in ancient Peru, Cabieses (1979:33) briefly reviews “spongy hyperostosis” (i.e., porotic hyperostosis) and notes the literature's ascribing anemia to these lesions. (A diagnosis of anemia based on porotic hyperostosis is typical of that time in paleopathology's history.) However, citing famines and ritualized fasting

as causative factors of vitamin C deficiency, he believes these lesions are attributable to scurvy (Cabieses 1979:35). Cabieses (1979:34) also provides a skull photograph in top surface view of an individual from the Huaura culture, commenting that the “spongy hyperostosis” depicted is probably due to healed scurvy. No other evidence is provided.

Because earlier reports do not rely on presently established criteria and largely lack detailed, precise, lucid lesion descriptions, they should be regarded as tenuous. It should also be noted that until the publication of Ortner and coworkers’ criteria, skull lesions (i.e., porotic hyperostosis and cribra orbitalia) were almost ubiquitously attributed to anemia. While anemia may have played a role in these lesions’ developments, earlier reports citing anemia as the sole cause (or a cause at all) of these lesions in many individuals presents an arguably considerable possibility for many misdiagnoses. These misdiagnoses might under represent readily observable lesions attributable to scurvy in Peruvian remains.

Skeletal Inventory

Noticing the skeletal alterations of this individual’s partially mummified skull, I asked about the availability of Individual 23985’s postcranial remains for possible examination. I was informed that the only elements present were those of the skull. Nevertheless, skull preservation is good. The skull is complete (see Table 3, which is found on page 38) and the teeth are partially present (Figures 2 and 3).

Table 2: Summary of juvenile scurvy reports on Peruvian human remains in the paleopathological and bioarchaeological literature.

<i>Reference</i>	<i>Sample size (age, sex)</i>	<i>Total cases (age, sex)</i>	<i>Remains’ current location</i>	<i>Remains’ spatial origins</i>	<i>Remains’ temporal origins</i>	<i>Method of scurvy diagnosis</i>
Klaus 2014a ¹	641 (J, S?)	5 (J, S?)	Not specified	Various sites within Lambayeque Valley Complex	Various dates from A.D. 900— 1750.	Macroscopic examination of cranial and postcranial remains
Melikian and Waldron 2003 ²	19 (J, S?)	3 (J, S?)	Natural History Museum in London	Provenance unsecure	Provenance unsecure	Macroscopic examination of cranial remains
Ortner et al. 1999 ³ (see also Ortner 2003:390 and Zuckerman et al. 2014)	363 (J, S?)	38 (J, S?)	National Museum of Natural History, Smithsonian Institution	Caudivilla; Junin; Chicama; Libertad; Various sites from Lima	Various dates from 2000 BC— Before 1530 AD	Macroscopic examination of cranial remains and SEM

J = Juvenile; S? = Sex not specified; SEM = Scanning electron microscopy

¹ Klaus considers these cases to reflect a diagnosis of probable scurvy. One case exhibits skeletal signs consistent with scurvy and rickets comorbidity.

² Melikian and Waldron consider these cases to reflect a diagnosis of possible scurvy and offer misgivings on diagnosing scurvy from juvenile cranial remains. The authors studied an additional 104 British “subadult” remains, noting 4 possible cases of scurvy in that sample. They also examined 4 clinically documented cases of scurvy.

³ Ortner et al. consider these cases to reflect probable scurvy.

Table 3: Skull inventory of Individual 23985.¹

<i>Element</i>	<i>Score</i> ²
Frontal	1
Parietal, right	1
Parietal, left	1
Occipital	2
Temporal, right	1
Temporal, left	1
TMJ surface, right	1
TMJ surface, left	1
Sphenoid, greater wing of, right	1
Sphenoid, greater wing of, left	1
Zygomatic (malar), right	S/O
Zygomatic (malar), left	S/O (but posterior surface and superior-lateral surface visible and 100% present)
Maxilla, right	S/O (but alveolars visible and 100% present)
Maxilla, left	S/O (but alveolars visible and 100% present)
Palatine proper, right	1
Palatine proper, left	1
Mandible	1

¹After Buikstra and Ubelaker's (1994) criteria but with modifications.

²"0," element absent (in Buikstra and Ubelaker's [1994] work, "blank" indicates element is absent); "1," >75% present; "2," 25-75% present; "3," <25% present; "S/O," soft tissue greatly obstructs visibility of skeletal element.

Figure 2: Maxilla and maxillary teeth of Individual 23985. Note the presence of soft tissue, (pathological?) bony reactivity (black arrow), dental carries, dental calculus, non-erupted tooth (white arrow), and chipped tooth (chipping is of uncertain etiology) (arrow-head). 80-61-30/23985.0, *Courtesy of the Peabody Museum of Archaeology and Ethnology, Harvard University.*

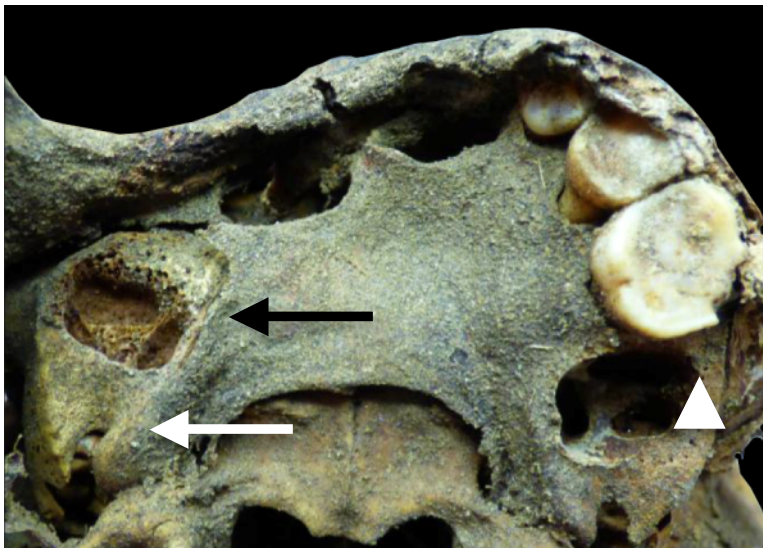


Figure 3: Mandible and mandibular teeth of Individual 23985. **a:** Mandible in anterior view. **b:** Mandible in posterior-top view. 80-61-30/23985.0, *Courtesy of the Peabody Museum of Archaeology and Ethnology, Harvard University.*

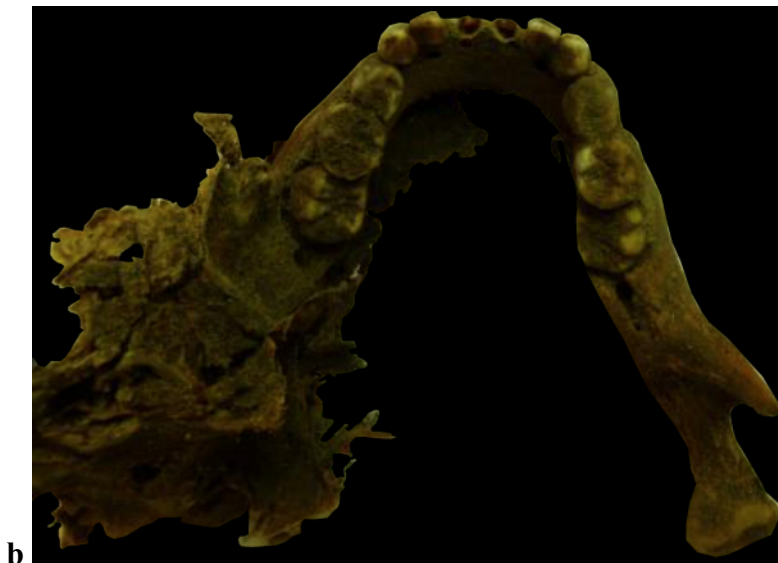
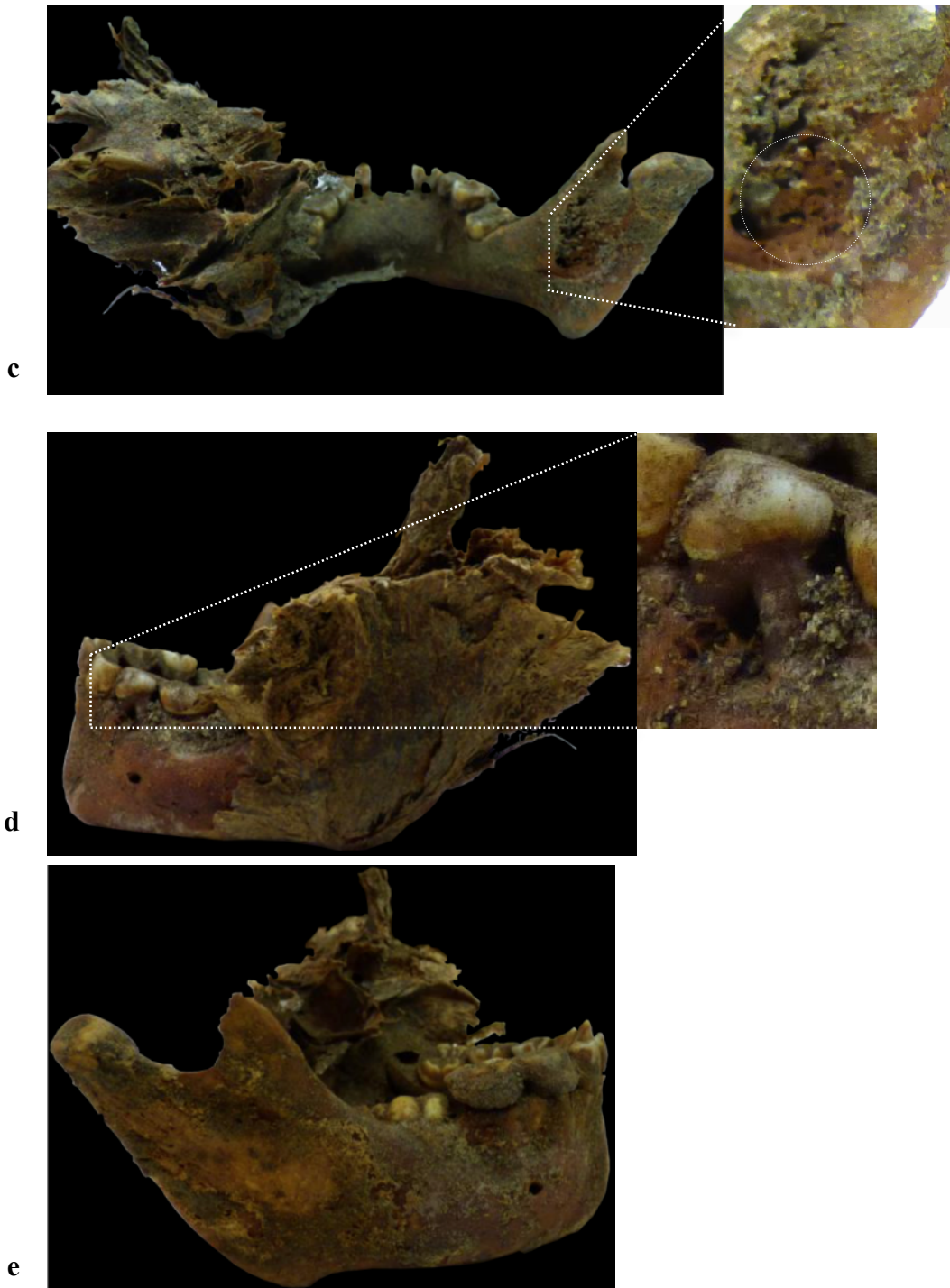


Figure 3 (Continued): Mandible and mandibular teeth of Individual 23985. **c:** Posterior view; note porosity posterior to mandibular foramen (encircled). **d:** Left lateral view; note exposed roots of first deciduous molar. **e:** Right lateral view; note dental calculus. 80-61-30/23985.0, Courtesy of the Peabody Museum of Archaeology and Ethnology, Harvard University.



Results: Macroscopic Examination

Table 4 provides a summary of the distribution of the pathologies in Individual 23985. Detailed lesion descriptions by cranial region are provided below.

Table 4: Summary of lesion distribution in Individual 23985.

<i>Anatomical site</i>	<i>Pathological feature¹</i>
Alveolars	P?
Endocranium	P
Frontal	P
Parietal, right	P
Parietal, left	P
Occipital	P
Orbit, right	N/O
Orbit, left	P
Temporal, right	P
Temporal, left	P
Sphenoid, greater wing of, right	P
Sphenoid, greater wing of, left	P
Zygomatic (malar), right	N/O
Zygomatic (malar), left	P
Maxilla (excluding alveolars), right	N/O
Maxilla (excluding alveolars), left	N/O
Palatine proper, right	A
Palatine proper, left	A
Mandible (excluding alveolars)	P

¹ “A” = pathological feature absent; “P” = pathological feature present; “P?” = possible pathological feature present; “N/O” = not observable due to soft tissue obstructing visibility.

Pathologies of the Cranial Vault (Frontal Bone, Parietal Bones, and Occipital Bone)

Frontal Bone and Orbits

In the region just above the right superior orbital margin, there is pathological cortical porosity in the form of small isolated foramina. Just above glabella, giving a thickened appearance, an appositional porous bone mass superficial to the original cortex

is present (Figure 4a). Two prominent, deep pathological vascular impression lesions, which run parallel to each other and are surrounded by small pathological cortical porosities, feed into this mass superiorly. Areas of this bony mass show remodeled bone on the mass's inferior portions and superior-right-lateral portions. Unremodeled and transitional remodeling sections are found on the mass's superior and left-lateral portions. The clearly defined, finely raised edge of the bony mass, which is inferior and lateral to the left vascular impression lesion, indicates bone formation rather than pure porosity.

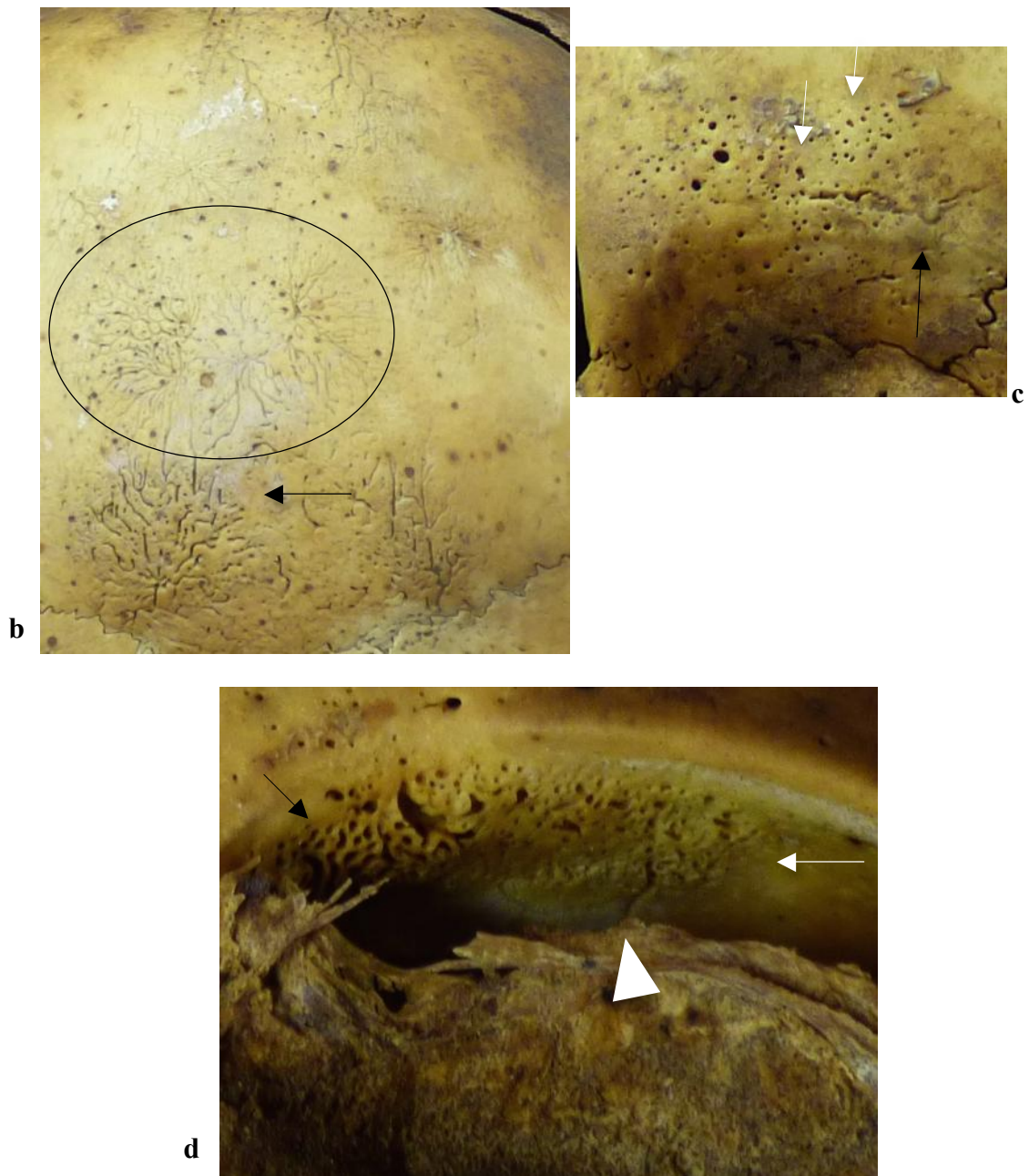
Isolated vascular impression lesions, vascular impression lesions possessing a star-shape (stellate pattern), and areas of vascular impression lesion anastomosis are found throughout the top surface of the frontal bone (Figure 4b). Some vascular impressions are surrounded by pathological porosities. Just anterior to and left of bregma, but not crossing the coronal suture, one of these vascular, stellate impression lesions is slightly raised with pathological porosity (raised lesion measures 12.3 millimeters along the sagittal plane and 14.1 millimeters along the coronal plane). This raised lesion is morphologically less pronounced than the raised vascular lesions found on the parietal bones (see *Parietal Bones* section). This lesser degree might indicate this lesion is in either an earlier formation phase or a healing phase. The left antero-lateral aspect of the frontal bone, just superior to the greater wing of the sphenoid bone, presents a regional cluster of pathological cortical porosity that extends to the superio-lateral orbital margin (Figure 4c). These porosities are a mixture of small, large, and coalesced foramina. Further, here, the irregular surface suggests remodeling (i.e., healing). Due to soft tissue obstructing visibility, the same region on the right side could not be observed. The outer table of the frontal bone's surface is intact (i.e., not obliterated).

The right orbit is entirely covered in soft tissue, precluding examination. The left orbit is partially covered in soft tissue but the orbital roof is available for inspection. Following Stuart-Macadam's (1991:109) classificatory system and criteria (*Chapter 2*), the left orbital roof presents with "type 5" cribra orbitalia (Figure 4d). In the antero-medial sector (sector definitions follow Stuart-Macadam [1991:109]), lesions bulge into the orbital cavity with foramina linking into a trabecular structure. The compact bone of this bulging lesion has essentially been transformed into trabecular bone (Ortner 1992). I consider this feature of the orbital lesions definite diploic hypertrophy—a raised bony surface (either of the superior vault or of the orbits) in association with enlarged and coalesced pores, which suggests marrow hyperplasia (Wilczak 2011:58). Thick deposits of new porous bone are present in the antero-intermediate sector. There is also a pronounced vessel channel feeding into the new bone formation in the antero-intermediate sector.

Figure 4: Individual 23985 frontal bone and orbital pathologies. **a:** Pathology just above glabella; note the deep vascular impression lesions with surrounding porosity (black arrows) feeding into appositional mass that is superficial to cortex, areas of unremodeled/transitional bone (white arrows), and areas of remodeled bone (white arrow-heads). 80-61-30/23985.0, *Courtesy of the Peabody Museum of Archaeology and Ethnology, Harvard University.*



Figure 4 (Continued): Individual 23985 frontal bone and orbital pathologies. **b:** Top surface of skull; note vascular impression lesions, vascular impression lesions possessing a star-shape pattern, raised impression lesion (arrow), and vascular impression lesion anastomoses (encircled) on skull's top surface. **c:** Coalesced (white arrows) and isolated porosities and irregular surface (black arrow) of left anterior-lateral frontal aspect just above greater wing of sphenoid bone extending to superio-lateral orbital margin. **d:** Cribra orbitalia of left orbit; note bulging trabecular structure (black arrow), thick deposits of new bone (white arrow), and channel (white arrow-head). 80-61-30/23985.0, *Courtesy of the Peabody Museum of Archaeology and Ethnology, Harvard University.*



Parietal Bones

On the left parietal ectocranial surface, posterior to the coronal suture, just above the sphenosquamosal suture and squamous suture, there is pathological cortical porosity (Figure 5a). On the left parietal bone, there are vascular impression lesions exhibiting a mixture of features just posterior-left-lateral to bregma and running parallel to the sagittal suture (Figure 5c). These mixed features are deep, flattened, raised, branching, connected, and star-shaped (see Figure 5c in which I indicate these different features). These vascular impression lesions are accompanied by surrounding porosity. There are two raised vascular-stellate pattern lesions with surrounding porosity along, but never crossing, the sagittal suture. The antero-superior raised lesion measures 21.5 millimeters along the coronal plane and 18.2 millimeters along the sagittal plane. The directly postero-inferior raised lesion measures 18.8 millimeters along the sagittal plane and 14 millimeters along the coronal plane.

The posterior aspect of the left parietal bone, along and toward the inferior portion of the lambdoid suture, exhibits severe porous, hypertrophic lesions (Figure 5d and e). On the superior portion of the posterior left parietal bone along the lambdoid suture and posterior sagittal suture, sclerotic patches (appearing as superficial to the original cortex) are present (Figure 5d and e). (Comparative images suggest these patches might be calcified hematomas [see, e.g., Kozłowski and Krajewska 2012].) Endocranially, a basal skull fracture reveals pitted lesions (Lewis 2004) of the inner table of the left parietal bone (Figure 5g).

On the right parietal bone's top ectocranial surface, there are vascular impression lesions accompanied by surrounding porosity parallel to the sagittal suture (Figure 5c).

These vascular impression lesions, similar to those on the left parietal, exhibit a mixture of features including deep, flattened, raised, branching, connected, and star-shaped. There is one raised vascular impression lesion just posterior-right-lateral to bregma and parallel to the sagittal suture (measures 19.1 millimeters along coronal plane and 16.3 millimeters along sagittal plane). None of these lesions cross the sagittal suture. These aforementioned vascular impression lesions are approximately symmetrical in location to those on the left parietal bone (see above) but are generally less pronounced. (See also *Occipital Bone* section.)

Further, on the right parietal ectocranial surface, posterior to the coronal suture, just above the sphenosquamosal suture and squamous suture, there is pathological cortical porosity, which is symmetrical to the observed porosity of the same region on the left parietal bone (Figure 5b). The posterior aspect of the right parietal bone, along and toward the inferior portion of the lambdoid suture, exhibits porous, hypertrophic lesions (Figure 5d and f). These features are approximately symmetrical to those observed on the left parietal bone. Superiorly, on the posterior portion of the right parietal bone, along the lambdoid suture and posterior sagittal suture, there are sclerotic patches, appearing as superficial to the original cortex (Figure 5d). (Comparative images suggest that these patches might be calcified hematomas [see, e.g., Kozłowski and Krajewska 2012].) Inferiorly and to the right of this patch, pathological small and large isolated cortical porosities are present (Figure 5d and f). Antero-superior to these porosities are deep vascular impressions (Figure 5d and f).

Occipital Bone

This bone exhibits pathological widespread (and relatively dense) porosities, especially just below lambda and stretching to the right aspect of the lambdoid suture (Figure 5d). These porosities are a mixture of large, small, and coalesced porosities. There are also rounded furrows. Along the left and right aspects of the lambdoid suture, there are porous hypertrophic diffuse lesions. These lesions cross those sites and thus engage the left and right parietal bones as well. There are also sclerotic patches along the left-lateral aspect of the lambdoid suture. The inferior patches are superficial to porous, hypertrophic lesions. These sclerotic patches are possibly calcified hematomas (see, e.g., Kozłowski and Krajewska 2012). Basally, there is a fracture thought to be a result of post-mortem damage (Figure 6a). The edges of the fracture are jagged and non-uniform in coloration. This fracture reveals a potentially slightly thickened diploë and a patch of endocranial fibrous bone deposition superficial to the inner table (Figure 6b). It is significant to note that the occipital bone is usually thick in this area and diploë thickness measurements were not taken at time of investigation.

Figure 5: Individual 23985 parietal bone pathologies. **a:** Left-lateral parietal bone showing porosity. **b:** Right-lateral parietal bone showing porosity. 80-61-30/23985.0, *Courtesy of the Peabody Museum of Archaeology and Ethnology, Harvard University.*

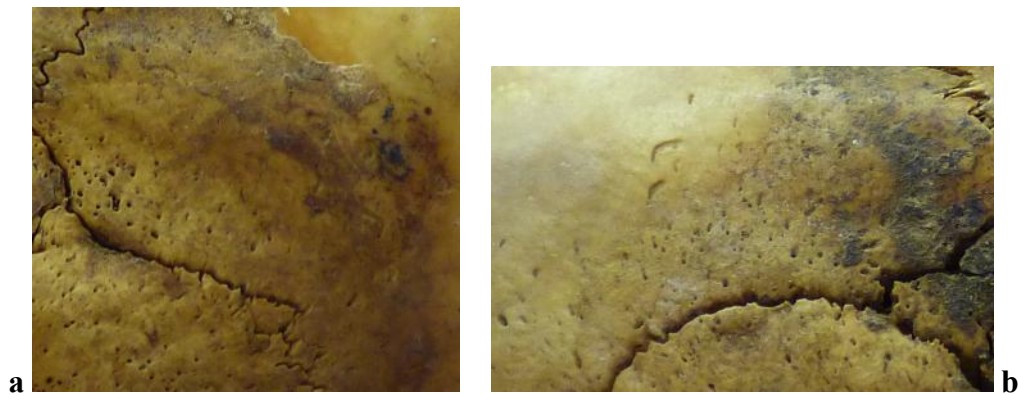
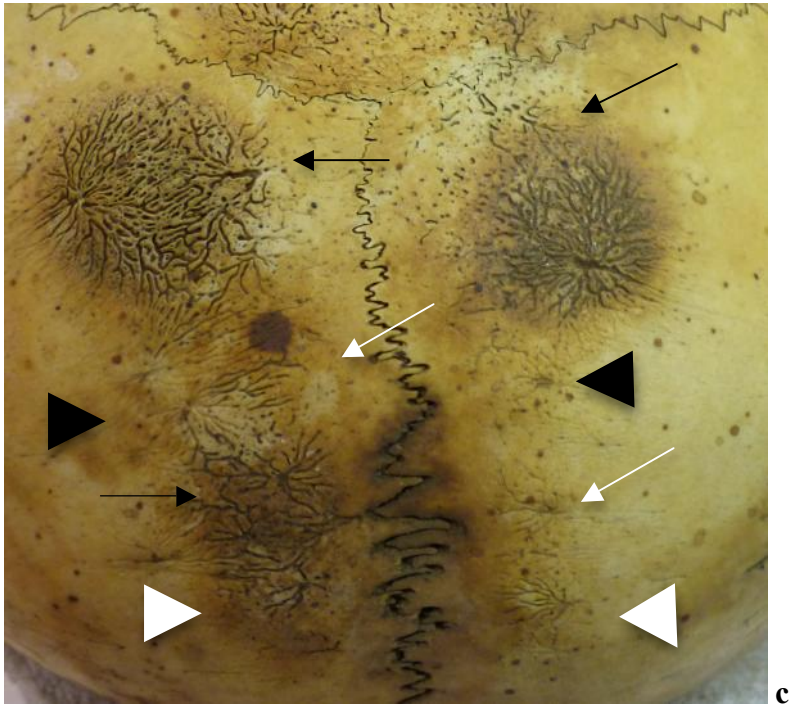


Figure 5 (Continued): Individual 23985 parietal bone pathologies. **c:** Top surface view of both parietal bones showing a mixture of vascular impression lesions with surrounding porosity; note the raised, deep lesions with a stellate appearance (black arrows), deep but non-raised lesions (white arrow), flat but non-deep lesions (black arrow-heads), and anastomosed lesions (white arrow-heads). **d:** Cranium in posterior view; note involvement of parietal bones and occipital bone. 80-61-30/23985.0, *Courtesy of the Peabody Museum of Archaeology and Ethnology, Harvard University.*



c



d

Figure 5 (Continued): Individual 23985 parietal bone pathologies. **e:** Close-up of left parietal bone in lateral-posterior view showing porous, hypertrophic lesions with engagement of occipital bone. **f:** Close-up of right parietal bone in lateral-posterior view showing porous, hypertrophic lesions with engagement of occipital bone. **g:** Pitted endocranial lesions of left parietal bone. 80-61-30/23985.0, *Courtesy of the Peabody Museum of Archaeology and Ethnology, Harvard University.*

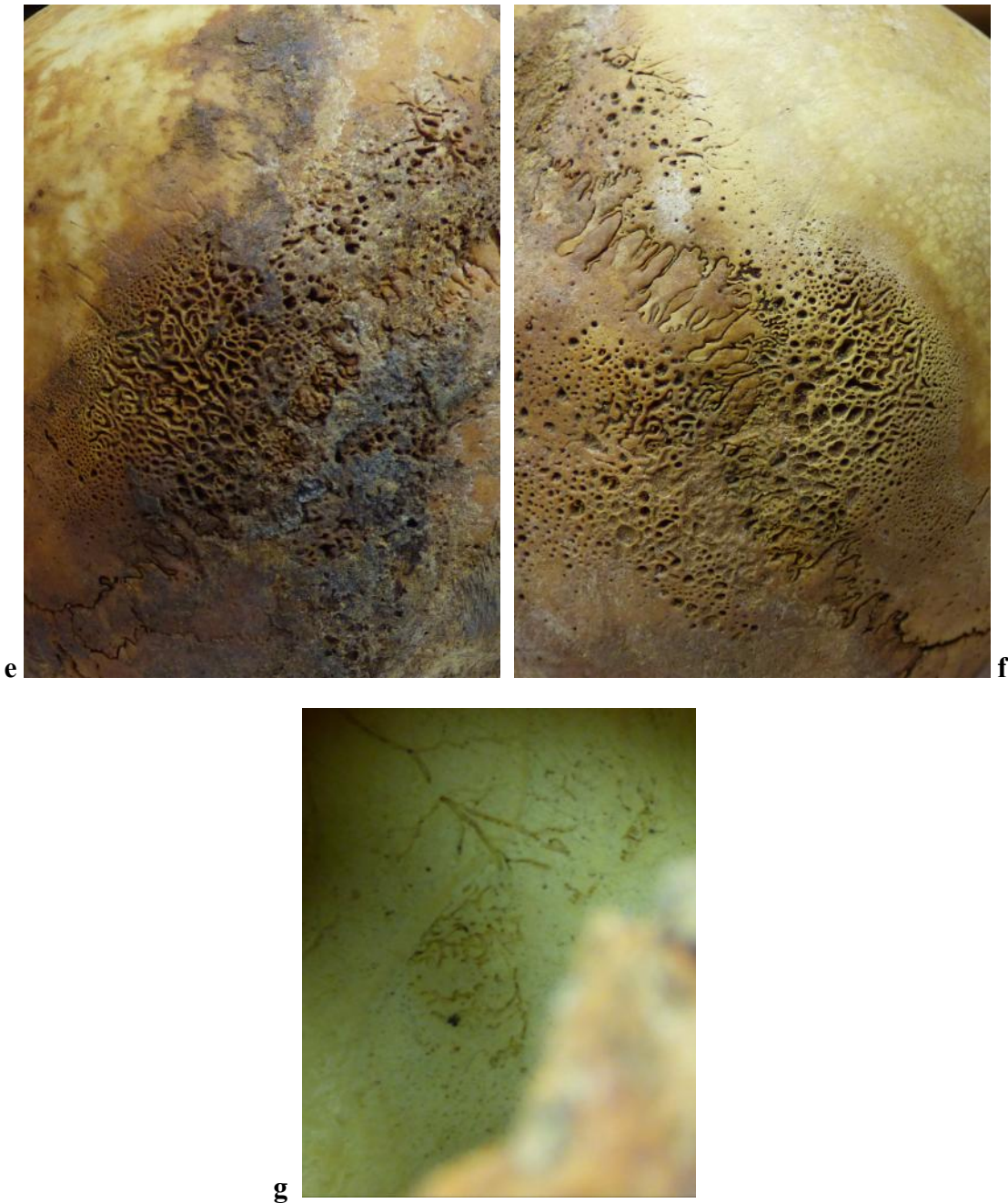
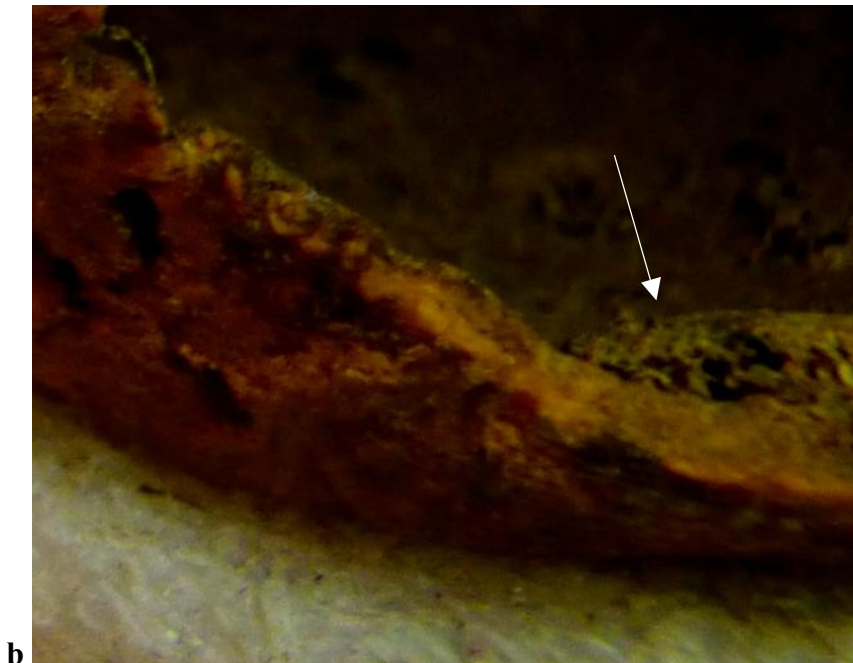
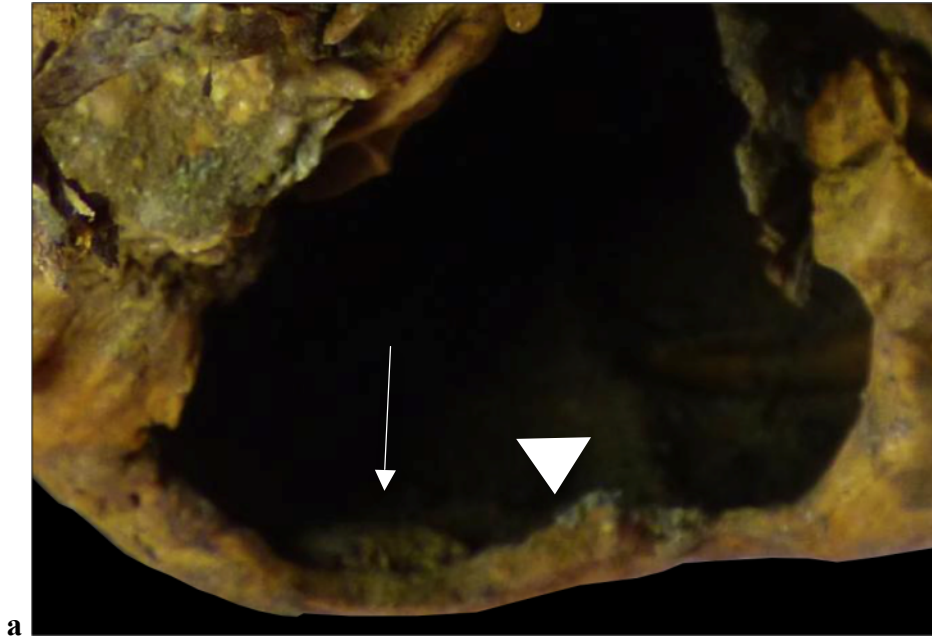


Figure 6: Individual 23985 occipital bone fracture. **a:** Slightly thickened diploe? (arrow-head) and fibrous bone deposition (arrow). **b:** Endocranial fibrous bone deposition (arrow) close-up. 80-61-30/23985.0, *Courtesy of the Peabody Museum of Archaeology and Ethnology, Harvard University.*



Pathologies of the Cranial Face and Mandible

Zygomatic (Malar) Bones

Bilaterally, these bones are covered in soft tissue and could not be examined closely and carefully. However, it needs to be noted that the left zygomatic bone's frontal process, temporal process, and portions of the posterior surface are observable. In these areas that are exposed, porosity and furrowing is visible (not to be confused with the typically occurring zygomatic foramina.)

Maxillae Bones and Maxillary Teeth (Figure 2)

The maxillae bones are largely covered in soft tissue and could not be examined. However, the posterior right and left alveolar margins are exposed and exhibit porosity. While alveolar bone porosity is implicated in scurvy, no baseline for assessing porosity as pathological in juvenile alveolar bone is established (Ortner et al. 1999). Whether or not this porosity is pathological could not be determined; but the poor dental health in this individual suggests that a pathological process might be present. There are three teeth erupted from alveolar bone in the left quadrant and one visibly unerupted in a molar crypt in the right and left quadrants. Dental health is poor, with the presence of dental calculus and caries. In the right quadrant, an alveolar socket exhibiting (pathological?) bony reactivity and porosity is present.

Distinguishing between pathological changes of alveolar sockets and “normal” alveolar socket porosity in juveniles is problematic. However, comparative images of “normal” controls in Ortner's (2002, slide no. 142-390) research slide collection suggest a pathological process in this case, possibly resulting in antemortem tooth loss.

Mandible and Mandibular Teeth (Figure 3)

The medial and lateral surfaces of the left mandibular ramus, coronoid process, and mandibular condyle are covered in soft tissue and could not be examined. Soft tissue covers the lateral surface of the right mandibular ramus. Light, isolated pinhole porosities are visible on the mental eminence, the right mandibular coronoid process, the right mandibular condyle, external mandibular body, and alveolar margins. Posterior to the right mandibular foramen, there is pathological cortical porosity. There are five erupted teeth from alveolar bone in the left quadrant and five for the right quadrant. Both central incisors are absent. Both first deciduous molar roots are exposed, with evidence of alveolar reactivity. Enlarged gubernacular canals of all anterior teeth are present, suggesting an eruptive process of successional teeth. Dental caries and dental calculus are present.

Pathologies of the Cranial Base (Including Entire Temporal and Sphenoid Bones)

Sphenoid Bones

Due to the presence of soft tissue, the right and left greater wings were partially unobservable to different degrees. However, on the greater wings, pathological truly penetrative cortical porosity was still well observed bilaterally (Figure 7a and b).

Temporal Bones

Bilaterally and symmetrically, light pathological porosity covers the surface of the temporal bone (Figure 8a and b). However, bilaterally and symmetrically, porosity is especially severe in the region encircling the postglenoid process, suprameatal crest, just

inferior to the suprameatal crest, and supramastoid crest (Figure 7a and b). The porosity seen here is clearly pathological in density, spread, and larger than expected size.

Figure 7: Individual 23985 greater wing of sphenoid bone pathologies. **a:** Right-lateral view of porosities (white arrows); note non-pathological foramen (arrow-head). **b:** Left-lateral view of porosities (white arrows); note non-pathological foramina (black arrows). 80-61-30/23985.0, *Courtesy of the Peabody Museum of Archaeology and Ethnology, Harvard University.*

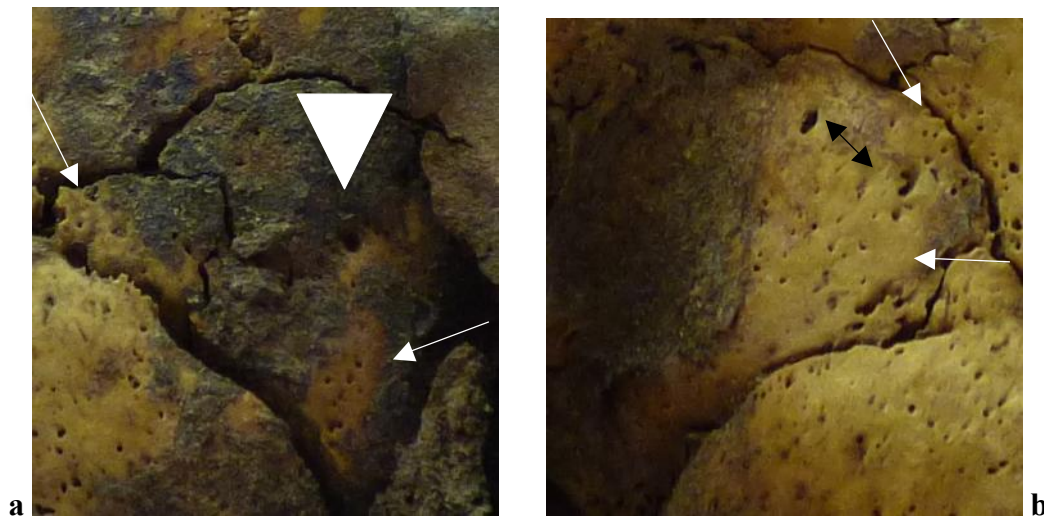
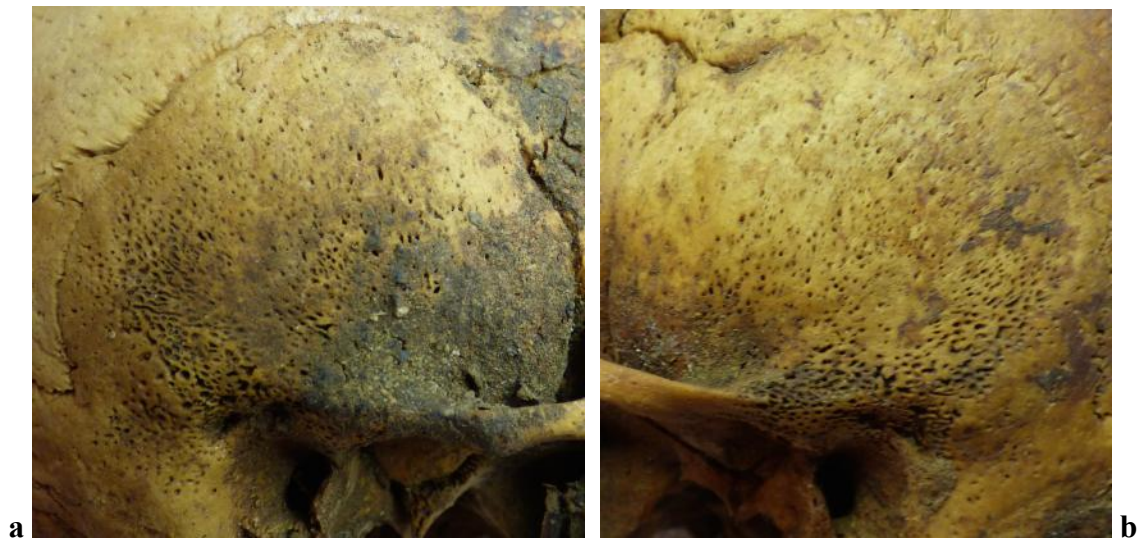


Figure 8: Individual 23985 temporal bone pathologies.. **a:** Right-lateral view showing porosity. **b:** Left-lateral view showing porosity. 80-61-30/23985.0, *Courtesy of the Peabody Museum of Archaeology and Ethnology, Harvard University.*



Additional Comments

There are circular brown and black stains of various sizes (mostly small) throughout the skull's surface. The raised vascular impression lesions exhibit brown staining as well. Given the proposed disease, it is conceivable, though I do not push this line of inquiry, that these are possibly hematogenous stains (e.g., Maat 2004). At present, this speculation cannot be confirmed. Alternatively, these stains might be a result of post-mortem taphonomic processes. Wormian bones are present along the left and right aspects of the lambdoid suture.

Differential Diagnosis Candidates¹

In this differential diagnosis, I consider anemias, cortical hyperostosis (infantile and prenatal onset), infectious diseases, rickets, and scurvy. In my estimate, this list reflects conditions that would most likely complicate diagnosis in this case. In these conditions, symmetrical porous and porous, hypertrophic cranial lesions are documented common variables. In general, Individual 23985's pathological changes can be described as such. More or less, be it in the clinical, paleopathological, or bioarchaeological literature, these conditions seem to be considered together in suspected cases of scurvy. Additionally or alternatively, scurvy is considered as a diagnostic possibility when the above respective conditions are suspected. By no means do I consider this list exhaustive. I welcome scholars' future additions to this list.

The following commentary concerns these conditions' clinical aspects and documented archaeological and contemporary skeletal manifestations. With regard to gross osseous manifestations found in these candidate diagnoses, I focus only on lesions

of the skull. Postcranial changes are not presented here, as they would not aid in the present case's differential diagnosis. (No postcranial remains are associated with Individual 23985.) Table 5 (p. 74) provides a summary of these diseases' etiologies, pathogeneses, epidemiological data, and direct, associated, or suggested skull changes.

Anemia

Anemia is hemoglobin concentration below established cut-off levels (Roberts and Manchester 2005:226; Stuart-Macadam 1989a, 1991; WHO and UNICEF 2004). Alternatively, anemia may be defined as “a condition in which the number of red blood cells (and consequently their oxygen-carrying capacity) is insufficient to meet the body's physiologic needs” (WHO 2011:1; see also Roberts and Manchester 2005:226). When speaking of anemia in a general sense, it is perhaps best to consider anemia a symptom rather than a disease entity. That is to say, anemia is an indicator of a disease state (Kale and Aftab 2012:75). In this sense, then, anemia is (loosely) analogous to pyrexia (fever), in which multiple diseases may induce this presentation. When we speak in more specific terms (e.g., Fanconi's anemia, Cooley's anemia, sickle-cell anemia), then we may think of anemia as a disease entity.

In this case, for the following reasons, I consider anemias broadly: (1) There are hundreds of anemias, some 200 hemolytic anemias alone (Walker et al. 2009). Discussing these different anemias in clinical detail is well beyond the scope of this work (for a starting point, see Silverberg's [2012] text, *Anemia*). (2) Not all anemias (recognizably) affect bone (Ortner 2003:363). (3) There is a lack of information concerning cultural and precise geographic context. This point is significant in that there

are genetic anemias that might not have been present in the New World prior to colonialization (Cabieses 1979; Ortner 2003). Further, nutritional anemias will be, at least in part, dependent on cultural context (e.g., prescribed rules on the frequency of consuming certain foodstuffs). (4) This case study's only method is gross dry-bone observation of cranial remains. (5) These two latter facts, "3" and "4," make it extremely difficult (perhaps almost impossible) to arrive at a specific anemia in a differential diagnosis process, as anemias most often encountered in archaeological contexts present similar cranial lesions (Ortner 2003).

Clinical Aspects

Based on pathogenesis, anemias may be broadly classified into the following categories: Blood loss anemias, hemolytic anemias, hemoglobinopathies, and hypoproliferative anemias (DeRossi and Raghavendra 2003:131, table 1). Depending on the anemia in question, there are predilections along lines of sex, age, ancestry, socio-economic status, and current disease state (Silverberg 2012).

In clinical settings, signs and symptoms of anemia may present an aporia of sorts. For instance, the cranial changes in anemia may not correspond to the severity of the anemia, especially if the anemia has developed gradually (e.g., in iron-deficiency anemia) (Stuart-Macadam 1989a). In the past, severe osseous signs occurred perhaps because of an advanced anemic state or comorbidity with other diseases (e.g., microbial diseases) and deficiencies (Ortner 2003:369; Stuart-Macadam 1989a). In patients with iron-deficiency anemia, cranial changes are most observed in non-adults (Stuart-Macadam 1989a). From the clinical literature, cranial changes in anemias include marrow

hyperplasia, hyperostosis and “cranial osteoporosis” (particularly frontal and parietal bossing), “hair on end” trabeculation, occipital depression, and obliteration of the outer table (, et al. 1960; Gould and McAfee 1958; Greenfield 1975; Jaffe 1975; Murray and Jacobson 1977b; Stuart-Macadam 1987a,b, 1989a,b). The most commonly affected cranial bones are the frontal (especially at orbital roof) and parietals, with the occipital rarely affected (Gould and McAfee 1958; Stuart-Macadam 1989a:215) (although see Stuart-Macadam 1989a:217, 1991:102). In iron-deficiency anemia, severity in bony changes varies even in individuals of the same age or with the same anemia severity (Stuart-Macadam 1989a,b). Whichever the bone-impacting anemia, the most commonly associated variable appears to be marrow hyperplasia (Ortner 2003).

Identifying Anemia

Diploic hypertrophy—indicated by a raised bony surface of the superior vault or orbits with enlarged and coalesced pores—is suggestive of marrow hyperplasia, and therefore anemia (Wilczak 2011:58). Crania possessing fractures may readily reveal pathologically thickened diploë, indicating marrow hyperplasia. Outer table obliteration or thinning may also suggest marrow hyperplasia (Ortner 2003). Ortner (2003:364, 2010:xviii-xix) holds that a diagnosis of anemia based solely on cranial dry-bone macroscopic examination necessarily requires evidence of marrow hyperplasia, not just porosity at the cranial vault and/or orbits. However, a lack of this evidence does not suggest an individual did not have anemia. Nor does it suggest that a given individual was never anemic. (Marrow hyperplasia might not be readily identified in earlier stages [Armstrong et al. 2014:12] or in advanced healing/healed stages). It is to suggest that this

feature is important in justifying an anemia diagnosis when cranial dry-bones are the sole line of evidence. It should be noted, however, that marrow hyperplasia does not necessarily implicate anemia, as this phenomenon has been well documented in polycythemia (blood cell count above “normal” levels) (Aufderheide and Rodríguez-Martín 2006; Gould and McAfee 1958; Steinbock 1976:219, table X). Some scholars have suggested that anemia might produce (or at least be associated with) endocranial lesions (Lewis 2004). Some genetic anemias may produce orbital bone infarctions (McNab 2014). (For genetic anemia examples from the bioarchaeological and paleopathological literature, the reader is referred to Angel 1966; Ascenzi et al. 1991; Hershkovitz et al. 1997; Lewis 2012; Tayles 1996.)

Cortical Hyperostosis, Infantile and Prenatal Onset

Clinical Aspects

Caffey’s Disease did refer to infantile cortical hyperostosis (ICH) and prenatal onset cortical hyperostosis (PCH), as they were considered variants of the same entity. However, in a recent article, Nemec et al. (2012) suggest ICH and PCH are clinically and radiologically two distinct entities. To stress PCH is a separate disease, Nemec and colleagues suggest PCH’s alternate name ought to be Caffey’s Dysplasia and ICH’s remain Caffey’s Disease. While multiple hypotheses concerning etiology exist, recent evidence suggests ICH and PCH are autosomally inherited (Drinkwater et al. 1997; Guerin et al. 2012; Kamoun-Goldrat and le Merrer 2008; Kamoun-Goldrat et al. 2008; Mahalingam et al. 2013; Nemec et al. 2012; Nistala et al. 2014; Shandilya et al. 2013).

Clinically, ICH is a usually self-limiting inflammatory disease of rare occurrence (although see Greenfield 1975:389, who says it is “not uncommon” and Jaffe 1975:282, who considers it “not rare”), with onset at a postnatal age of before 5 months (Antoniades et al. 1995; Greenfield 1975; Guerin et al. 2012; Jaffe 1975; Kamoun-Goldrat and le Merrer 2008; Shandilya et al. 2013). In a localized area (often the mandible, shoulder girdle, or limb), pain, swelling, and inflammation characterize this condition’s symptoms (Antoniades et al. 1995). New bone formation and cortical thickening underlying soft tissue swelling is a hallmark (Guerin et al. 2012; Mahalingam et al. 2013; Shandilya et al. 2013). PCH is rare, potentially lethal, lacks clinical inflammation, and is characterized as either severe or mild (Drinkwater et al. 1997; Guerin et al. 2012; Kamoun-Goldrat et al. 2008; Nemec et al. 2012). The severe prenatal variant is onset before 35 gestational weeks and the mild variant after 35 gestational weeks (Kamoun-Goldrat and le Merrer 2008; Shandilya et al. 2013).

Three in every 1,000 infants less than 5 months of age are affected (Gorlin et al. 2001; Kamoun-Goldrat and le Merrer 2008; Lewis and Gowland 2009). (This frequency is reported prior to ICH and PCH being considered distinct diseases. As such, it might reflect combined ICH and PCH frequencies.) ICH and PCH exhibit no identified predilection for ethnic group, social class, or sex (Jaffe 1975; Kamoun-Goldrat and le Merrer 2008; Shandilya et al. 2013). In the so-called familial form, onset tends to be at 6 to 8 weeks of age and is present at birth in 24% of cases (Kamoun-Goldrat and le Merrer 2008). In the so-called sporadic form, age of onset tends to be at 9 to 11 weeks and the mandible is involved much more often than in the familial form (Kamoun-Goldrat and le

Merrer 2008; Shandilya et al. 2013). No cases have presented onset after 5 months of age (Kamoun-Goldrat and le Merrer 2008).

As noted above, ICH tends to be self-limiting. A typical time span for resolution is 3 to 30 months since onset (Gorlin et al. 2001). During healing, subperiosteal new bone formation and underlying cortex homogenize through remodeling and present as “normal” (Antoniades et al. 1995; Jaffe 1975; Shandilya et al. 2013). Further, “as is common under all circumstances in which new bone is deposited on pre-existing cortical bone, the original cortical bone yields by becoming increasingly porous” (Jaffe 1975:284). It should be noted that successive exacerbations (relapses) do occur with eventual clearance in the second and third years of life (Antoniades et al. 1995; Drinkwater et al. 1997; Guerin et al. 2012; Kamoun-Goldrat and le Merrer 2008; Thometz and DiRaimondo 1996). “Delayed” cases are probably ones that were initially mild and went undetected (Blank 1975). In spontaneous regressive courses, dental macroscopic and microscopic “abnormalities” as a result of compromised dentin formation have been suggested; but this line of inquiry requires further study (Kamoun-Goldrat and le Merrer 2008).

Chronic courses with remission and relapse displaying residual hyperostotic effects up to early childhood and occasionally adulthood are noted (Blank 1975; Jaffe 1975; Kamoun-Goldrat and le Merrer 2008; Murray and Jacobson 1977b; Shandilya et al. 2013). In severe cases, which are exceedingly rare, this chronic and recurrent course has been associated with considerable crippling of extremities and muscular and motor delayed development (Kamoun-Goldrat and le Merrer 2008).

Clinically Documented Lesions

In PCH, lesions are usually symmetrical and diffuse (polyostotic) (Kamoun-Goldrat et al. 2008; Nemec et al. 2012). With regard to the skull, PCH is typified by symmetrical mandibular hyperostosis, skull base hyperostosis, and orbital roof density (Kamoun-Goldrat et al. 2008; Nemec et al. 2012). Macrocephaly with pressure-induced skull “deformability” is noted (Kamoun-Goldrat et al. 2008), as are brachycephaly, wormian bones, poorly mineralized calvaria, and “abnormally” open sutures (Nemec et al. 2012).

In ICH, lesions are usually (but not always) asymmetrical and multifocal (Nemec et al. 2012). This classic form may present as polyostotic or monostotic (Antoniades et al. 1995; Jaffe 1975; Shandilya et al. 2013). When the skull flat bones are involved, the most common sites are the mandible, parietal bones, and frontal bone (Antoniades et al. 1995). Sclerosis of flat bones is a known occurrence (Greenfield 1975:390). However, the calvarium is affected in only 3% of cases (Gould and McAfee 1958). Nasal bones may also be involved (Shandilya et al. 2013:1197; Sheppard and Pressman 1988:111).

In clinical cases, mandibular involvement, which is usually bilateral, is a diagnostic criterion (Shandilya et al. 2013) considered virtually pathognomonic (Kamoun-Goldrat and le Merrer 2008). Approximately 70-80% of cases show mandibular involvement (especially affecting the angle, body, and ascending ramus [Gorlin et al. 2001; Sedano et al. 1977]) in isolation or with hyperostosis of other elements (Kamoun-Goldrat and le Merrer 2008; Lewis and Gowland 2009; Sedano et al. 1977; Shandilya et al. 2013; Wong and Cheng 2008). Kamoun-Goldrat and le Merrer’s (2008) clinical case report documents an unaffected cranial vault with mandibular

hyperostosis. Shandilya et al. (2013) report on a case with mandibular involvement as the only osseous symptom (see also Jaffe 1975). One case report of “delayed” ICH notes unilateral mandibular involvement, a unilaterally enlarged hard palate, and unilateral thickening and sclerosis of the maxilla and zygomatic bone but with no signs of malocclusion (Antoniades et al. 1995). Reports also detail small mandibles with malocclusion (Blank 1975).

Uncommonly, several reports have noted lytic areas on the cranial vault. In addition to mandibular hyperostosis, Neuhauser (1970:58-59) reports lytic “serpiginous and rounded areas” of the frontal, parietal, and occipital bones. Later, these areas appeared “normal.” Boyd et al. (1972) reports on a 3 month old girl who had periorbital swelling, bilateral patchy lytic lesions on the frontal bone, and early mandibular hyperostosis. At 7 months of age, the lytic lesions disappeared, and by 10 months of age some resolution was noted in lower face appearance.

Clinical studies report anemia, exophthalmos, pallor, and other conditions as complications (Boyd et al. 1972; Gorlin et al. 2001; Jaffe 1975; Mahalingam et al. 2013; Sheppard and Pressman 1988; Thometz and DiRaimondo 1996; Wong and Cheng 2008).

Bones from the Past

In the bioarchaeological and paleopathological literature, the identification of Caffey's disease is sparse (Bourbou 2010; Lewis 2007). Below, I focus on skull lesions in four selected possible cases of ICH from the literature. Though I do not discuss them here, postcranial pathological changes are present in all the following cases.

In a 7.5-8.5 years old individual, Bourbou (2010, 2013) notes porotic hyperostosis on the right parietal bone and periosteal reaction as woven bone on both mandibular rami. In a 1.5 years old individual, Lewis and Gowland (2009) note healing and active new bone lesions and hypertrophy on both orbits, frontal bone, occipital bone, and both parietals. Further, there is a stellate arrangement of new bone formation on some areas of the parietal bones and frontal bone. The mandibular body exhibits inflammatory pitting and new bone formation. Both greater wings of the sphenoid bone also exhibit pitting. Pathological changes of the cranial vault are "not uniform, with the left orbit and frontal being thickened" (p. 43). Endocranially, vascular woven bone is present near the cruciate eminence. There is also a crest of lamellar bone on the frontal bone. According to the authors, these lesions are indicative of scurvy, but microscopic examination and the severity of the lesions renders scurvy not the most likely cause of these lesions (p. 47, 50). They provide the possibility that this individual also suffered from scurvy (p. 49).

In a 10-18 month old individual, Rogers and Waldron (1988) note an asymmetrical, generalized periosteal reaction throughout the skeleton and thickening of some of the cortices. The mandible is indeed involved, showing asymmetrical periosteal reaction. Radiographs show the periosteal new bone is not separated from the cortex. They also report on a 1-year-old individual with a layer of new bone ectocranially, "which had a fine honeycomb appearance. The distribution was patchy, with the parietals being most severely affected" (p. 9). I am not aware of any diagnosed cases of PCH in the bioarchaeological and paleopathological literature.

Infectious Diseases

Information on epidemiology, mechanisms of pathogenesis, and pathologies is dependent upon (among other factors) the infectious agent in question. There are far too many microbial diseases to discuss in the allotted space. So, I frame my discussion broadly. I do, however, highlight similar cranial lesions that might be encountered in scurvy or infectious diseases.

General Character of Cranial Lesions Resulting from Infection

Cranial osteomyelitis is rare in children, but would not be unexpected in cases of cranial trauma (Ortner 2003). However, infants and children constitute most cases of osteomyelitis (Buckley 2000). Cloacae, involucra, and sequestra characterize osteomyelitis (Aufderheide and Rodríguez-Martín 2006; Ortner 2003; Waldron 2009). Fine pitting, longitudinal striation lesions, and periosteal new bone formation superficial to the original cortex characterize periostitis (Mensforth et al. 1978; Roberts and Manchester 2005:172). Periostitis is most commonly encountered on the tibia (Roberts and Manchester 2005:172-173). Documented cranial periostitis, however, is greatly lacking in the literature (Klaus 2014b:20) (although see Mensforth et al. 1978). It should be noted that periostitis does not necessarily implicate a response to infection, although such is often implied (Minozzi et al. 2012; Ortner 2003; Roberts and Manchester 2005:172).

Similar Cranial Lesions in Infectious Disease and Scurvy

The star-shaped impression lesions associated with scurvy might be confused with caries sicca scarring found in treponemal disease (Klaus 2014b:20). However, caries

sicca scarring would possess a central necrotic focus and sclerotic margins (Klaus 2014b:20). In cases of treponematosi, one might also look for mulberry molars and Hutchinson's teeth (Klaus 2014a,b; Ortner 2003; Rogers and Waldron 1989). Parietal bossing (Parrot's swelling) is also found in treponemal disease (Ortner 2003). Endocranial lesions have been associated with infectious diseases inducing meningitis, including tuberculosis (TB) (Hershkovitz et al. 2002; Lewis 2004; Ortner 2003). Approximately 0.1% of skeletal TB (which is ~5% of TB cases) involves the cranium (Roberts and Buikstra 2003). Cranial involvement in TB is more common in juveniles than in adults (Klaus et al. 2010). Porotic hyperostosis and cribra orbitalia may be found in cases of infection (Vradenberg 2001). Unorganized and patchy bone formation may also be found in infectious disease and would require differentiation between scurvy, rickets, anemia, and typical growth (Brickley and Ives 2008:103, table 5.5; Klaus 2014b).

Rickets

Rickets is "a disease of a growing child in which there is failure of mineralization of the growth plate and osteoid matrix, resulting most commonly from conditions that cause chronically low concentrations of calcium and phosphate in extracellular fluid" (Mughal 2011:291). Read another way, this disorder of growing children results from impaired hypertrophic cell apoptosis and delayed growth plate mineralization (Mughal 2011:291). In bioarchaeology and paleopathology, rickets is reserved for the "juvenile form" of this disease and osteomalacia for the "adult form" (Brickley and Ives 2008; Ortner 2003). This distinction is also seen in clinical works (e.g., Jaffe 1975:381; Rosenberg 1999:1228). But, in the strict clinical sense of the term, children may develop

osteomalacia (excessive osteoid tissue accumulation resulting from defective osteoid tissue mineralization throughout the skeleton) (Brickley and Ives 2008:91; Glorieux et al. 1998:764). Here, I adopt bioarchaeological and paleopathological convention. I reserve *osteomalacia* for this disease in adults and *rickets* encompasses clinically defined juvenile rickets and juvenile osteomalacia.

Clinical Aspects

Rickets most commonly results from vitamin D deficiency (Holick 2006:2063). This deficiency prevents calcium deposition in developing cartilage and in newly formed bone osteoid, hindering bone mineralization (Brickley and Ives 2008:90). This deficiency may be sequela to inadequate sunlight to skin exposure or inadequate dietary intake—either because of prescribed dietary practices (e.g., dietary fads) or stratified access and use of vitamin D-rich foodstuffs. Vitamin D deficiency heightens risk of infection, type 2 diabetes, atherosclerosis, and neurodegenerative diseases (Chesney 2010; Kassi et al. 2013; Mezza et al. 2012; Riek et al. 2014; Schlögl and Holick 2014). Today, in instances of simple vitamin D deficiency, those most at risk are children, pregnant women, the superannuated, and those with inadequate sunlight to skin exposure (Al-Atawi et al. 2009; Brickley and Ives 2008; Holick 2006; Schlögl and Holick 2014; Veselka et al. 2013). However, because these “simple” cases result from inadequate dietary intake or inadequate sun exposure, any individual may develop vitamin D deficiency. Data indicate 90% of vitamin D in humans is endogenously synthesized from sunlight-skin exposure (Mughal 2011). Diseases, practices, or environments that reduce the possibility of

sunlight exposure put individuals at risk for acquiring rickets (Al-Atawi et al. 2009; Brickley and Ives 2008; Holick 2006).

There are individuals who are born with the inability (or a restricted ability) to absorb vitamin D (Brickley and Ives 2008; Glorieux et al. 1998). Some genetically inherited forms of rickets manifest more severely in males than in females (Glorieux et al. 1998). (For a paleopathological example of possible inherited rickets, the reader is referred to Blondiaux et al. 2002.) Moreover, because vitamin D is a fat-soluble molecule, vitamin D can get trapped in an enlarged pool of subcutaneous fat tissue, reducing its bioavailability (Mezza et al. 2012). Metabolic disturbances such as increased parathyroid hormone and decreased calcium concentrations correspond to vitamin D deficiency (Mezza et al. 2012).

Thus, rickets may take several forms. These forms may be categorized as calcipenic or phosphopenic (see Glorieux et al. 1998:765, table 26-4). (For a more detailed clinical picture of rickets, the reader is referred to Glorieux et al. 1998:764-777; Holick 2006; Jaffe 1975:381-447; Mughal 2011.) Nonetheless, the skeletal manifestations of these multiple forms are similar to those in simple vitamin D deficiency (Ortner 2003:393; cf., Glorieux et al. 1998).

Cranial Changes

Rachitic and scorbutic cranial changes are difficult to differentiate, especially since there is a great deal of lesion overlap and these diseases may co-occur. Rachitic cranial changes are usually ultra-fine deposits of periosteal porous bone, particularly in the squamous portion of the occipital bone and lateral parietal bones (Klaus 2014a). One of the earliest signs of rickets is the deposition of osteoid on the external table of the

skull, mimicking porotic hyperostosis (Giuffra et al. 2013). Active cases of rickets exhibit cortical bone porosity (Mays et al. 2006). However, porosity found in rickets does not usually penetrate the underlying cortex, as this porosity is sequela to osteoid mineralization failure (Giuffra et al. 2013). Taken together, skull vault porosity, orbital roof porosity, and mandibular ramus bending (medial/posterior) and porosity are considered diagnostic of rickets (Mays et al. 2006; cf., Ortner and Mays 1998). However, in isolation, these lesions are non-specific (Brickley 2000; Mays et al. 2006).

Porosity in the glabella region is documented, as is woven bone deposition on the outer table of the skull vault (Brickley and Ives 2008; Mays et al. 2006, figure 1; Ortner 2003; Ortner and Mays 1998). Zygomatic and temporal bone porosity and thickening are noted features (Ortner and Mays 1998). More pronounced changes, such as large pores and spicular bone formation, might be related to rapid growth of an individual (Brickley and Ives 2008; Mays et al. 2006). Craniotabes (flattening of the parietal and occipital bones), frontal and parietal bossing, delayed cranial suture and fontanel closure, craniosynostosis as a secondary feature, square-shaped crania, thinning or thickening of cranium, and enamel hypoplasias are documented in rickets as well (Bauder 2009:138; Berrizbeitia 1992:13; Brickley and Ives 2008; Castilla et al. 2014; Gładkowska–Rzeczycka 2001; Gould and McAfee 1958:636; Lewis 2007; Stuart-Macadam 1989a). In rickets, the sphenoid bone is typically spared, and rickets pathogenesis precludes truly penetrative porosities of this bone (Klaus 2014b). Endocranially, fibrous bone deposition may occur (Giuffra et al. 2013; Lewis 2004; Ortner and Mays 1998). This feature requires differentiation from typical deposition seen during growth (Brickley and Ives 2008).

In their elite Italian sample, Giuffra et al. (2013) note the following cranial changes that are associated with rachitic postcranial pathologies: Increase in biparietal diameter, frontal bossing, endocranial changes consistent with rickets hydrocephalus, widely separated coronal suture (especially in the bregma region), particularly severe porosity around the parieto-temporal sutures, mandibular ramus bending, new bone formation on the mandible, new bone formation on the maxilla and hard palate, skull vault porosity, orbital porosity, and ectocranial and endocranial bone deposition. Case Med 40.45, who exhibits porosity at the sphenoid bone, is considered a less convincing case of rickets and congenital scurvy is offered as a diagnostic option.

Scurvy

Here, I provide a brief overview of scurvy's clinical aspects and clinically documented cranial changes, which are largely based upon Thomas Barlow's classical characterization of this disease (Jaffe 1975; Pop-Jordanova et al. 2008; cf., Barlow 1883, 1894).

Clinical Aspects

Scurvy refers to a disease state resulting from vitamin C (ascorbic acid) deficiency. Vitamin C is a significant co-factor in collagen formation (and thus osteoid matrix formation), carnitine synthesis (necessary for muscle function), ocular health, and osteodentin formation (Fain 2005; Jen and Yan 2010; Popovich et al. 2009; Semba 2007). (For a review of vitamin C's biological significance, see Iqbal et al. 2004; Packer and Fuchs 1997.) This deficiency disease is induced by not consuming enough ascorbic acid.

Therefore, scurvy can occur at any age and in any sex. However, today, children, the poor, those engaging in diet fads and religiously prescribed diets, alcoholics, smokers, and the superannuated are at greatest risk (Barlow 1894; Burk and Molodow 2007; Fain 2005; Heymann 2007; Ho et al. 2007). Some recent data suggest there are genetic predispositions to possessing low levels of vitamin C (Halcrow et al. 2014:69; cf., Delanghe et al. 2007). These predispositions, of course, are distinct from the human species-wide incapacity to endogenously synthesize vitamin C.

When vitamin C levels are deficient, the body may undergo multi-system disease (Li et al. 2008). One source indicates children require at least 15 mg of vitamin C daily to avoid clinical and subclinical presentation of ascorbic acid deficiency (Popovich et al. 2009). (Official dose recommendations vary by country and health organization.) Symptom presentation corresponds to scurvy duration and severity (Brennan et al. 2012). Scorbutic symptoms include, but are not limited to, weakness, pallor, heightened irritability, capillary fragility, hemarthrosis, pseudovasculitis, proptosis (and subperiosteal orbital hemorrhages), premature dental shedding, gingival hemorrhaging and hypertrophy, petechial hemorrhages, multi-focal subperiosteal hemorrhages pulling the periosteal sheath off growing bone, edema, ecchymosis, impaired wound healing, scorbutic rosary, pseudoparalysis, and limb pain and swelling (Ahuja and Karande 2002; Barlow 1883, 1894; Besbes et al. 2010; Clemetson 2002; Fain 2005; Grau 2002; Grewar 1965; Halligan et al. 2005; Heymann 2007; McNab 2014; Mertens and Gertner 2011; Pimentel 2003; Schuurs 2013; Sloan et al. 1999; Stark 2009; Still 1915; Verma et al. 2007). Approximately 80% of scurvy cases display musculoskeletal manifestations (Fain 2005:125).

Complications in scurvy include, but are not limited to, cardiovascular disease, intestinal complication, increased risk of infection, nutritional comorbidities, periodontal disease, and anemia (Ahuja and Karande 2002; Aguirre and May 2008; Clemetson 2002; Fain 2005; Grewar 1965; Halligan et al. 2005; Larralde et al. 2007; Mettier and Chew 1932; Olmedo et al. 2006; Staudte et al. 2005). Anemia is present in roughly 75% of patients (Ho et al. 2007), and may be persistent and result from iron-deficiency, blood loss, and/or other dietary deficiencies (Brown 1955; Burk and Molodow 2007; Cohen and Paeglow 2001; Ho et al. 2007; Larralde et al. 2007). Indeed, anemia is a laboratory hallmark in scurvy cases (Burk and Molodow 2007; Larralde et al. 2007:196). Anemia degree corresponds to scurvy severity and duration (Larralde et al. 2007:196). Rickets and scurvy commonly co-occur as well (Hess 1920:110; Pimentel 2003:331, table 1). If untreated, scurvy is fatal (Halligan et al. 2005). Sudden death has been documented in infantile and adult cases (Grewar 1965; Larralde et al. 2007).

Cranial Lesions from the Clinical Literature

Scorbutic cranial changes documented in the paleopathological and bioarchaeological literature are presented in Table 1 and will not be duplicated. Rather, here, I present skull changes from the clinical literature. (As noted earlier, sphenoid bone porosity is not reported in the clinical literature [Noordin et al. 2012].)

Parietal swelling is a documented feature of scurvy (Barlow 1883, 1894; Pop-Jordanova et al. 2008). Fraenkel's autopsies reveal porous, hypertrophic bone of the orbital roof (Armelagos et al. 2014; Ortner 2003) and maxillary and mandibular involvement (Ortner 2003:385). Porotic hyperostosis is also noted in individuals known

to have had scurvy in life, as is marrow hyperplasia secondary to anemia (Noordin et al. 2012). Facial bones may present swelling (Barlow 1894:1029; cf. Barlow 1883). From autopsies, the cranium may show subperiosteal hemorrhages in the vicinity of parietal bone porosity (Barlow 1894:1030; cf., Barlow 1883). Autopsies also reveal parietal and frontal bone bossing (Barlow 1883). The occipital bone is an affected site (Barlow 1883). Radiography demonstrates cranial osteoporosis (Gould and McAfee 1958). Unless an individual is edentulous, gum hemorrhages are to be expected, as is resorption of alveolar bone and subsequent dental shedding (particularly incisors of juveniles) (Barlow 1894; Grewar 1965; Li et al. 2008:427; Ortner 2003; Schuurs 2013).

Table 5: Summary of differential diagnosis candidates for suspected cases of juvenile scurvy in cranial remains.

<i>Disease</i>	<i>Etiology</i>	<i>Pathogenesis /mechanism</i>	<i>Direct, associated, and suggested skull changes</i>	<i>Epidemiology</i>	<i>Comments</i>
Anemias ¹	Varies	Bone changes sequela to bone marrow expansion	<p>Porotic hyperostosis and/or cribra orbitalia <i>in association with marrow hyperplasia</i></p> <p>Diploic hypertrophy suggests marrow hyperplasia.</p> <p>Diploë hyperplasia in calvarium with <i>vertical spicules</i> of bone bridging the inner and outer tables ("hair on end appearance")</p> <p>Prominent bossing of frontal and parietal bones; Facial bones usually spared; pneumatization of paranasal sinuses usually not delayed; flattening or depression of the occiput; thinning of the outer table; thickened diploë; endocranial lesions possible</p>	<p>Dependent on the type of anemia. Nutrition-based anemias (e.g., iron deficiency and folate deficiency) are common and can occur at any age, though juveniles and elderly patients at increased risk.</p> <p>Diet fads and religious practices may also contribute to these anemias. These anemias are also dependent upon individual disease states, particularly those that result in hemorrhagic episodes (such as in scurvy).</p> <p>Some genetically inherited anemias restricted to the Old World prior to European contact and are now found in New and Old World populations.</p>	<p>(1) Evidence of marrow hyperplasia necessary for diagnosis</p> <p>(2) Association between anemia severity and skeletal manifestation is poor.</p> <p>(3) Sphenoid bone porosity would not occur. In principle, because little marrow is found here, it is an unlikely place for marrow hyperplasia in children.</p>

Table 5 CONT.

<p>Cortical hyperostosis, infantile (ICH) and prenatal onset (PCH)²</p>	<p>Unclear but autosomal inheritance suspected</p>	<p>Unclear (but see Nistala et al. 2014 for a proposed mechanism and implicated molecular players in ICH)</p> <p>Considered inflammatory and collagenopathic</p>	<p><i>ICH:</i></p> <p>Porotic hyperostosis, cribra orbitalia</p> <p>Monostotic or (usually) polyostotic</p> <p>When skull flat bones are involved, parietal bones, frontal bone, and mandible are common sites. (But, cranial bones are considered rarely involved.) Nasal bones are considered rarely involved.</p> <p>Ca. 80% of cases display mandibular involvement in the form of hyperostosis, particularly on the mandibular body, ramus, and angle. Mandibular involvement may be unilateral or bilateral, symmetrical or asymmetrical; but usually bilateral and asymmetrical.</p> <p>Lytic lesions of the parietal, frontal, and occipital bones are rarely documented.</p> <p>In a possible case of ICH from the bioarchaeological literature, Lewis</p>	<p><i>ICH:</i></p> <p>Rare occurrence (but see Greenfield 1975:390 and Jaffe 1975:284)</p> <p>Disease frequency of 3 per 1,000 (This frequency might reflect ICH and PCH frequency.)</p> <p>Primarily occurs from infancy until 5 months of age (but see <i>Comments</i> column, “2”).</p> <p><i>PCH:</i></p> <p>Rare. The severe prenatal variant is onset before 35 gestational weeks and the mild variant after 35 gestational weeks.</p>	<p><i>ICH:</i></p> <p>(1) In clinical cases, resulting facial and mandibular asymmetry may require surgical intervention.</p> <p>(2) Usually, resolves on its own without treatment within 1 year of onset. But, “delayed,” recurrent (not uncommon with usual clearance in second and third years of life), and chronic (up to early childhood and adulthood) are noted.</p> <p>(3) Anemia, exophthalmos, and other conditions as complications.</p> <p>(4) In spontaneous regressive courses, dental macroscopic and microscopic “abnormalities” as a result of compromised dentin formation have been suggested; but this line of inquiry requires further study.</p> <p>(5) Radiographic symptoms lag behind clinical presentation.</p>
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Table 5 CONT.

			<p>and Gowland (2009) note associated pitting of the greater wing of the sphenoid bone that might be due to scurvy comorbidity.</p> <p><i>PCH:</i> Usually symmetrical and diffuse—most common skull lesions include symmetrical mandibular hyperostosis, skull base hyperostosis, orbital roof density. Atypically patent sutures.</p>		
Infectious diseases ³	Varies	Varies	<p>Porotic hyperostosis; cribra orbitalia; presence of involucra, clocae, and sequestra (i.e., osteomyelitis); periostitis (rare?);</p> <p>Endocranial lesions; stellate-pattern lesions with necrotic focus; unorganized and patchy bone formation</p>	Varies	<p>(1) Skull uncommon site for infection, unless there is evidence of trauma (either due to physical interactions or disease state).</p> <p>(2) Skull rarely affected in TB—juveniles display skull involvement more often than adults.</p>
Rickets ⁴	Most commonly, Vitamin D deficiency due to poor nutrition and inadequate sun exposure	Prevention of calcium deposition; Poor calcification	<p>Porotic hyperostosis; cribra orbitalia; vascular-like impression lesions; Endocranial lesions possible; endocranial lesions; temporal porosity; palatal hypertrophy;</p>	<p>Highest frequency is between 6 months and 2 years of age. Few new cases develop after 4 years of age</p> <p>But, because most commonly due</p>	<p>(1) Sphenoid porosity unlikely</p> <p>(2) Irregular, spiculated bone formation requires differentiation between typical growth, scurvy, infection, and anemia.</p>

Table 5 CONT.

			maxillary hypertrophy; large porosities in severe cases; irregular, spicular bone in severe cases; square-shaped head; craniotabes; delayed fontanel and suture closure; bending of mandibular ramus	to inadequate sun to skin exposure or poor diet, rickets may occur at any juvenile age (note that in this text, osteomalacia reserved for adults).	
Scurvy ⁵	Vitamin C deficiency	Hematogenous. Poor matrix formation—contrast with poor calcification in rickets.	See Table 1	At any age, but usually at ~9 months. Occurs in that individual with poor vitamin C intake due to food sources or food preparation.	(1) Anemia and proptosis are sequelae and diagnostically consistent with scurvy. (2) Sphenoid bone porosity not explicitly documented in clinical literature, but is pathophysiologically consistent.

¹Blom et al. 2005; Britton et al. 1960; Klaus 2014a,b; Lewis 2004, 2012; Mcilvaine 2013; Ortner 2003; Ortner and Ericksen 1997; Ortner et al. 1999; Ortner et al. 2001; Stuart-Macadam 1987a,b, 1989, 1991, 1992; Wapler et al. 2004; Walker et al. 2009; Oxenham and Cavill 2010; Wilczak 2011:58

²Antoniades et al. 1995; Blank 1975; Kamoun-Goldrat and le Merrer 2008; Kamoun-Goldrat et al. 2008; Lewis and Gowland 2009; Mahaligam et al. 2012; Neuhauser 1970; Nemec et al. 2012; Nistala et al. 2014; Rogers and Waldron 1988; Shandilya et al. 2013; Sheppard and Pressman 1988; Thometz and DiRaimondo 1996; Wong and Cheng 2008

³Hershkovitz et al. 2002; Klaus 2014a,b; Klaus et al. 2010; Lewis 2004; Mensforth et al. 1978; Minozzi et al. 2012; Ortner 2003; Roberts and Buikstra 2003; Roberts and Manchester 2005

⁴Brickley and Ives 2008; Giuffra et al. 2013; Klaus 2014a,b; Lewis 2004; Mays et al. 2006; Ortner 2003; Ortner and Mays 1998

⁵Clemetson 2002; Ho et al. 2007; Noordn et al. 2012; Ortner 2003; Ortner and Ericksen 1997; Verma et al. 2007

Differential Diagnosis

A descriptive differential diagnosis and differential diagnosis flow-chart are provided below. These two subsections are meant to complement each other and should be read as such.

Descriptive Differential Diagnosis

Anemia

As outlined above (see *Differential Diagnosis Candidates* section and Table 5), when based solely upon gross dry-bone observation, evidence of marrow hyperplasia is necessary to justify an anemia diagnosis. The antero-medial sector of the left orbital roof of Individual 23985 displays diploic hypertrophy (Figure 4d; cf., Halcrow et al. 2014:66, figure 7a), which suggests marrow hyperplasia (Wilczak 2011:58) and therefore anemia. That is, there is a bulging bony surface (not originating from additional bone apposition) with enlarged and coalescent porosities. (These criteria for identifying diploic hypertrophy are only applicable to the superior cranial vault and orbits [Wilczak 2011:58].) While marrow hyperplasia speaks to the likely presence of anemia (Ortner 2012), it would be imprudent to offer a specific anemia as a diagnosis in this case. Otherwise, by-and-large, Individual 23985's cranial outer tables are not obliterated. Additionally, the observed sphenoid bone porosity is unlikely to result from anemia, as little marrow is found there in children (Klaus 2014b).

Cortical Hyperostosis, Infantile (ICH) and Prenatal Onset (PCH)

On epidemiological and demographic grounds, PCH is eliminated from this differential diagnosis.

New bone formation underlying regions of soft-tissue swelling does characterize ICH (Guerin et al. 2012), much like certain cases of scurvy. Bilateral temporal and masseter muscle swelling is documented in the literature on ICH (e.g., Shandilya et al. 2013). Although bilateral, these swellings tend to be asymmetrical (Shandilya et al. 2013). Additionally, approximately 80% of ICH cases exhibit mandibular hyperostosis (see Table 5). In contrast, Individual 23985's lesions can be largely characterized as bilateral and symmetrical. While mandibular porosity is noted in this case, there are no signs of hyperostosis. Because Individual 23985's dental age is approximately 6-years-old and the majority of ICH cases resolve in the first year or two of life, ICH is an unlikely diagnosis. ICH is eliminated from this differential diagnosis based on epidemiological and demographic grounds (see Table 5), lack of mandibular hyperostosis in this case, and lack of lesion asymmetry in this case (see Nemec et al. 2012, table 2).

Infectious Diseases

The larger porosities of the occipital bone located below lambda (Figure 5d), as well as the overall patterning of lesions on the occipital bone, are very similar to those one would expect to see in cases of infectious disease. The bone formation superficial to the cortex on both parietal bones (Figure 5e,f), which engage the occipital bone bilaterally and symmetrically, could be consistent with an infectious process. However, these above lesions may be found in other kinds of diseases (Ortner 2003), and therefore only indicate a pathological (probably inflammatory) process. No involucra, sequestra,

nor cloacae are noted. Nor are there any lytic lesions with sclerotic margins that would be consistent with infection. The raised vascular impression lesions of the frontal and parietal bones could be confused for stellate scarring found in treponemal disease (Klaus 2014b). However, neither necrotic foci nor sclerotic margins are noted for these lesions (see Klaus 2014b).

In the molars available for inspection, there are no instances of Mulberry molars, which would be indicative of congenital syphilis (see Ortner 2003; Rogers and Waldron 1989). The only incisors present are the two lateral mandibular incisors, which do not reflect Hutchinson's teeth. By-and-large, the lesions found in Individual 23985 correspond to muscle attachment sites. This correspondence would not be an expected occurrence in chronic infectious diseases (Klaus 2014b:20).

Rickets

Individual 23985's "square-shaped" head (see Figure 5d) might correspond to rachitic manifestation. While there is little contextual data associated with this individual, it is necessary to recall that Individual 23985 is of ancient Peruvian origin. Thus, it is possible that this head shape represents cranial shaping practices, which are common in Peruvian antiquity.

The porosities found in rachitic new bone depositions tend to be very fine (Ortner 2003). Only in severe cases are large porosities and spicule bone formation documented occurrences (Mays et al. 2006). The pronounced pathological changes in this individual do suggest a more extreme case. The larger porosities of the occipital bone noted earlier could, therefore, be attributed to rickets. Rickets, similar to scurvy, may also engender

new bone deposition on glabella. However, the non-ultra fine porosities found in the bone depositions, the truly penetrative porosities found throughout the skull (in contrast to the “pseudoporosity” found in rickets [Giuffra et al. 2013]), and these lesions’ overall correspondence to muscle attachment sites involved in chewing argue against a rickets diagnosis. On the occipital bone, however, there are instances of furrowing. But, these furrows appear to be a result of hypertrophic bone formation rather than poor matrix mineralization. The observed pathological sphenoid bone porosity is unlikely to result from rickets. Rickets typically spares the sphenoid bone and rickets pathogenesis precludes truly penetrative porosities of this bone (Klaus 2014b). Moreover, fontanel closure is not delayed. No mandibular bending is noted. Ultimately, however, rickets comorbidity cannot be ruled out.

Scurvy

All the lesions in this case, less the marrow hyperplasia evinced in the left orbit, are most consistent with scurvy (see Table 1). The adjacent deposits of new bone in the left orbit (Figure 4d) are consistent with a bony response to subperiosteal orbital hemorrhage (Brickley and Ives 2008). The new porous bone deposition on glabella (Figure 4a) is a documented occurrence in scurvy (Ortner 2003). The bilateral and symmetrical bone porosities of the temporal bone correspond nicely to masticatory stress associated hemorrhage at temporal muscle attachment sites (Ortner and Erickson 1997). The ectocranial vascular impression lesions, which are similar in appearance to Mays’s (2008a:180) endocranial “branched lysis” lesions, are rarely documented in cases of scurvy (Klaus 2014a). Although rarely documented (e.g., Brown and Ortner 2011; Klaus

2014a), these lesions are entirely consistent with scurvy pathogenesis (see Brown and Ortner 2011:204). The hypertrophic, porous lesions found on both parietal bones with engagement of the occipital bone (Figure 5e,f) indicate a severe inflammatory process. These lesions pose no discrepancies to a diagnosis of scurvy.

Mandibular involvement at the various locations in this case is also documented in the literature on scurvy (see Table 1). Notably, if scorbutic in nature, the observed porosity surrounding the mandibular foramen (Figure 3c) may be in relation to medial pterygoid muscle contraction inducing hemorrhage of the inferior alveolar artery and its branches (Brown and Ortner 2011:203).

In this case, I consider the porosities at the greater wing of the sphenoid bone to be pathological (Figure 7). They are smaller and more numerous than would be expected in a non-pathological state (see Ortner et al. 2001). Additionally, the porosities seen here do not appear to enter at an oblique angle, supporting the conclusion that they are pathological (Mays 2008a:179). In *association with* all the above lesions, the observed truly penetrative bilateral porosities of the greater wings of the sphenoid bones serve as virtually pathognomonic scorbutic indicators (see *Skeletal Analyses and Scurvy* section).

Additional Comments

Earlier (p. 78), I noted that the overall patterning and morphology of the ectocranial occipital bone porosities could be expected in cases of infectious disease. I also noted that these lesions in isolation simply indicate a pathological, probably inflammatory process. Ortner (2003:375) himself indicates that scorbutic bony reactions “could easily be confused with those of anemia or infectious disease.” It is interesting to

note, however, that the overall patterning and morphology of these occipital bone porosities (although larger) nicely match those found in images of diagnosed scurvy cases (Ortner et al. 1999:326, figure 6; see Ortner 2002, slide no. 141-402 for a larger image).

The absent maxillary and mandibular incisors could be attributable to premature scorbutic dental shedding (Ortner 2003). They could also, however, be attributable to typical growth patterns. The alveolar margins do display porosity, but the presence of soft tissue precludes assessing these porosities as pathological. To this case's potentially pathological molar socket (Figure 2), comparative images of "normal" controls in Ortner's (2002, slide no. 142-390) research slide collection suggest a pathological process, possibly resulting in antemortem tooth loss. Problematically, alveolar porosity in juveniles is often too common to serve as a pathological indicator (Stark 2014:21, table 2). Additionally, although premature dental shedding is possible in scurvy (Schuurs 2013), it is nearly impossible to confirm or reject this phenomenon as scorbutic in paleopathological cases (Stark 2014:21, table 2).

The observed pitted endocranial lesion (Figure 5g) and endocranial fibrous bone deposition (Figure 6b) may occur in scurvy, other conditions (e.g., rickets, anemia, infectious diseases), or in cases of scurvy comorbidity (Brown and Ortner 2011; HersHKovitz et al. 2002; Lewis 2004, 2007:141-143; Mays 2008a; Mensforth et al. 1978; Schultz 2001:123, table 3). In particular, endocranial fibrous bone deposition has also been associated with typical growth (Brickley and Ives 2008:103, table 5.5; Lewis 2007:143).

Comparing images (Kozłowski and Krajewska 2012), the noted ectocranial sclerotic patches (appearing as superficial to the original cortex) might be calcified hematomas (see *Results: Macroscopic Examination* section).

Differential Diagnosis Flow-Chart

Differentiation between skeletal symptoms of these diseases requires a comprehensive model for bony expression of pathology including attributes of lesion form and distribution, as well as epidemiological information. [Buikstra 1976:357]

Although Buikstra's comments are specifically in regard to diagnosing tuberculosis in her study sample, her statements are equally applicable here.

In the past, diagnostic models were common in the literature (see Powell and Cook 2012 for a discussion). Currently, however, diagnostic models, particularly differential diagnosis flow-charts, are few in the paleopathological and bioarchaeological literature (for an example, see Buikstra 1976:359, figure 7). It is perhaps true that no flow-chart can fully cover the range of a given disease's manifestations, from its extreme subtleties to its extremely pronounced markers. Diagnostic models also may not adequately reflect variables influencing disease presentation, such as comorbidities, duration of disease state, and changes in infectious disease manifestations brought on by host-pathogen coevolution (Holloway et al. 2011; Lewis 2000, 2007; Powell and Cook 2012; Ortner 2003; Ragsdale and Lehmer 2012). To be sure, diagnostic models have their pitfalls and will likely change as new information concerning manifestations of various diseases comes to light (Powell and Cook 2012).

However, I would advance that differential diagnosis flow-charts and tables serve as useful heuristic devices. They highlight the "big picture," so to speak, which might

otherwise be neglected when focusing on minute details of pathologies. Moreover, these charts and tables speak to the necessary scientific rigor implored in the literature (e.g., Crandall and Klaus 2014)—precisely because, if constructed properly, they provide a well-organized device that effectively communicates current knowledge about epidemiological, anatomical, and pathological data in relation to appropriate diagnostic options.

Figure 9 presents a differential diagnosis flow-chart that acts as an important diagram in facilitating this case’s diagnosis. This diagram should be understood as complementary to the information presented in the *Differential Diagnosis Candidates* and *Descriptive Differential Diagnosis* sections. Neither approach, however, supersedes the other. The data I present in this figure reflect the epidemiological, pathological, and anatomical data presented in Table 5, albeit in truncated form. Buikstra’s (1976:359, figure 7) flow-chart acts as a model for this diagram’s construction. Admittedly, the presented flow-chart will need future refinement. I welcome scholars’ future additions, suggestions, and modification to this diagram.

As can be seen from Figure 9, the most compatible diagnosis is anemia and scurvy comorbidity. This diagram and the discussion presented throughout this chapter support a diagnosis of probable scurvy and likely anemia comorbidity.

Summary and Concluding Thoughts

This chapter has presented a critical review and analysis of the literature surrounding scurvy, a detailed macroscopic examination following Ortner's (2003:49-51) guidelines for pathological description and diagnosis, a rigorous consideration of differential diagnosis candidates, and a rigorous differential diagnosis process following Buikstra (1976) as a guide for constructing differential diagnosis flow-charts and tables. Based on the presented differential diagnosis flow-chart and discussion throughout this chapter, I conclude Individual 23985 displays lesions consistent with probable scurvy and likely anemia comorbidity.

The bilateral and symmetrical porous and hypertrophic lesions found throughout this individual's skull largely correspond to muscle attachment sites. In conjunction with these lesions, the truly penetrative and pathological porosities at the greater wings of the sphenoid bones serve as virtually pathognomonic scorbutic markers. The marrow hyperplasia, suggested by diploic hypertrophy in the left orbital roof, speaks to the likely, although not certain, presence of anemia (Ortner 2012). The described cranial lesions in this case fulfill diagnostic criteria presented in other works (see Brickley and Ives 2008:57, table 4.2) and are in accordance with the paleopathological literature on scurvy (see Table 1) and anemia (see Table 5). While additional conditions could be present, we can only report on the osseous lesions that indicate the presence of a given disease(s) of most probable cause.

Paleopathologists have emphasized the importance of considering comorbidities (e.g., Brickley and Ives 2006, 2008; Ortner 2003). However, works have often reported scurvy and anemia separately (White et al. 2006:39). These conditions' co-occurrence in the same individual should not be surprising (White et al. 2006:39). Anemia is present in

roughly 75% of scurvy patients (Ho et al. 2007), and may be persistent and result from iron-deficiency, blood loss, and/or other dietary deficiencies (Brown 1955; Burk and Molodow 2007; Cohen and Paeglow 2001; Ho et al. 2007; Larralde et al. 2007). Indeed, anemia is a laboratory hallmark in scurvy cases (Burk and Molodow 2007; Larralde et al. 2007:196).

This case study is significant in that there are few publications concerning Peruvian juvenile scurvy in the English language literature. Additionally, I believe this is the first Peruvian juvenile case in the English language literature reported to display evidence of scurvy and anemia comorbidity. Moreover, this case study offers a differential diagnosis flow-chart (Figure 9) and table (Table 5) that other scholars may modify and use in suspected cases of juvenile scurvy in cranial remains. In so doing, I hope to have added a line of inquiry to the literature that offers the potential for rigorous diagnosis of scurvy in juvenile crania. However, this model does require evidentiary supplementation from the literature (e.g., Table 1 and the *Differential Diagnosis Candidates* and *Descriptive Differential Diagnosis* sections) (Buikstra 1976), as no model should be assumed to fully encompass all pathological possibilities (Powell and Cook 2012).

As scholars have noted, “a thorough differential diagnosis is essential in any paleopathological assessment” (Ragsdale and Lehmer 2012:241). Indeed, a more general diagnosis (e.g., metabolic disease) is often more correct than a specific one (e.g., scurvy) (Ortner 2012; Ragsdale and Lehmer 2012; Waldron 2009). And, a specific diagnosis should only be reached in the presence of appropriate evidence (Waldron 2009). This evidence is dependent upon thorough, detailed, and clear documentation of pathological

lesions (Ortner 2012) and a rigorous consideration of appropriate diagnostic options (e.g., Buikstra 1976). This chapter reflects these criteria.

Notes

1. Some comments on two diseases not presented in this differential diagnosis are warranted. These two conditions are hypertrophic osteoarthropathy (HOA) and hypervitaminosis A.

HOA comes in three variants—primary (inherited), secondary (acquired), and cranio-osteopathopathy (COA) (primary HOA without pachydermia) (Dabir et al. 2007). The secondary form is the most common, the primary form a rare entity, and COA rarer still (Chen et al. 2012). HOA spares muscle attachment sites (Klaus 2014b; cf., Ortner 2003:354).

Primary HOA is most commonly onset in adolescence at the earliest (Klaus 2014b) and its development is gradual (Jajić et al. 2001). In a clinical sample of 76 individuals ranging in age from 18 to 64 years (mean age 43), Jajić and Jajić (1998) note periosteal reaction on the whole surface of the calvaria. They note periosteal reaction of short and flat bones in 14 individuals (18.5%), though no frequency is reported for cranial involvement specifically. The periosteal reaction in flat and short bones is not as pronounced as that of long bones (Jajić and Jajić 1998).

In acquired HOA, the skull is usually spared, except for the facial bones (Aufderheide and Rodríguez-Martín 2006). When HOA does affect the skull, other than finding associated cribra orbitalia and porotic hyperostosis (Masson et al. 2013), the inner table is most commonly affected (Klaus 2014b). In a clinical sample, 42% of cases (20 individuals) showed skull involvement—most commonly of the maxillae and mandible

(Ali et al. 1980). This involvement's precise osseous markers are unclear. Typically, secondary HOA is not onset prior to adolescence (Klaus 2014b). Indeed, acquired HOA most often occurs in adults and is associated with various malignancies, including lung carcinoma (Chen et al. 2012)—although juvenile cases are reported (e.g., Varan et al. 2000). Thus, one might search for evidence of malignant disease in assessing secondary HOA status.

In a literature search, Dabir et al. (2007) identified only 28 clinically documented cases of COA. COA displays no significant predilection along lines of sex (Dabir et al. 2007). The age range for these 28 cases is 3 months to 24 years old and the mean age is 3 years old (Dabir et al. 2007). One case documents a case of COA in an 11.5-year-old girl displaying patent cranial sutures, patent anterior and posterior fontanel, and facial asymmetry (Dabir et al. 2007). These patent sutures and fontanel are a result of poor neurocranium ossification (Dabir et al. 2007; see also O'Connell et al. 2004). Wormian bones are documented as well (Chen et al. 2012).

Vitamin A is rapidly absorbed and slow to clear (Binkley and Krueger 2000). An intake of greater than 75,000 units of vitamin A daily over a 6 month period would almost certainly induces hypervitaminosis A (Jaffe 1975:468). Acute hypervitaminosis A may be induced if greater than 100,000 units of vitamin A are consumed (Heird 2004). This is an exceptionally high amount of vitamin A. Today, vitamin A toxicity is usually a result of consuming a great deal of vitamin A supplements or vitamin A containing supplements over an extended period of time (Rothenberg et al. 2007). Vitamin A toxicity presents with skull changes similar to those in rickets, but skull involvement is rarely observed (Gould and McAfee 1958). However, craniotabes is reported (Pennes et

al. 1984) and is considered common (Heird 2004:181). Increased cranial pressure is documented (Heird 2004:181). The mandible is typically spared (Buckley 2000; Jaffe 1975). Cranial suture widening, cranial elongation, and hyperostosis of the temporal and occipital bones can occur (Buckley 2000). Lesions tend to be bilateral and symmetrical. Bone resorption may also occur (Binkley and Krueger 2000). Hypervitaminosis A is demonstrated to cause anemia (Perrotta et al. 2002).

HOA (in any of its variants) and hypervitaminosis A are poor diagnostic options. Although cranial changes are documented in these conditions, cranial changes are rare occurrences. On epidemiological grounds, primary and secondary HOA poorly match this case, as Individual 23985's dental age is approximately 6-years-old. The extreme rarity of HOA and its variants also argue against this condition as a diagnostic option. Based on clinical literature searches (Dabir et al. 2007), COA is exceptionally rare. Not only this, the documented cranial changes (patent cranial sutures and fontanelles well after typical closure times) are not present in this case; nor is there facial asymmetry. Moreover, the lesions in this case correspond nicely to muscle attachment sites, which would not be expected in HOA.

While naturally occurring vitamin A toxicity is not unheard of (e.g., Rothenberg et al. 2007:1267; Walker et al. 1982), it does seem like an exceptionally unlikely scenario in this case given the amount of vitamin A that would need to be consumed. Additionally, craniotabes (flattening of the occipital and/or parietal bones [Bauder 2009:138-139]), a common cranial change in hypervitaminosis A when the skull is involved, is not noted here.

This is not to suggest these conditions were necessarily absent in this case. Examination of postcranial remains would be needed to effectively rule out these conditions. But, the purpose of a differential diagnosis process is to arrive at a most probable cause(s) provided that there is appropriate evidence for a diagnosis. These two diseases do not seem to reflect most probable causes.

CHAPTER 4: NEED A HEAD EXAM?

Introduction

In *Chapter 3*, for reasons already stated, I devoted considerable attention to Individual 23985's case of metabolic disease. Here, however, I offer a less in-depth treatment of the individuals to follow. Although not nearly as in-depth as the preceding chapter, I certainly do not imply that the individuals presented here are any less significant to this thesis.

Ragsdale and Lehmer (2012) caution it is often wiser to attribute the pathologies we observe to general disease categories (e.g., infectious disease) rather than specific ones (e.g., tuberculosis). The cases to follow are grouped into general categories. If sufficient evidence is present, a specific diagnosis within that general disease category is offered (Ortner 2012). Based on this thesis's sample, this chapter offers a survey of individuals displaying pseudopathology, hematopoietic disease, infectious disease, neoplastic disease, joint disease, and trauma and trauma-induced disease.

Pseudopathology

In some instances, pseudopathologies are fairly straightforward to identify. Discoloration relative to the rest of the remains, particularly sharp and jagged edges, unmistakable weathering effects, or certain fracture angles tend to betray an alteration's status as postmortem (Buikstra and Ubelaker 1994). Often times, however, distinguishing

between antemortem and postmortem changes is a difficult task (Ortner 2003). Some changes may produce (near) identical changes to those seen in periosteal reactivity (see Wells 1967). Or, true pathological changes may not present with typical antemortem features (e.g., multiple myeloma) (Ortner 2003:46). It should also be noted that the occurrence of postmortem changes does not negate the possibility of antemortem pathology co-presence (Ortner 2003:46). For instance, “post-mortem changes produce ‘pseudo-tumors’ but they may also *obscure to varying degrees changes resulting from the presence of true tumors*” (Brothwell 1967:321). Below, I present a case of pseudopathology that more or less resembles syphilitic changes, probably as a result of insect activity. But, first, a brief discussion on insect activity is warranted.

Insect Activity

Various authors have discussed the effects of insect activity on skeletal remains, noting this activity’s potential to “mimic” pathologies and result in misdiagnoses (e.g., Aufderheide and Rodríguez-Martín 2006; Buikstra and Ubelaker 1994:97; Ortner 2003; Wells 1967). One well-known example is Lortet’s (1907:212, figure 1) “evidence” of syphilis in an ancient Egyptian female cranium (cf., Aufderheide and Rodríguez-Martín 2006:16; Moodie 1923:116; Wells 1967:10). Concerning Lortet and his “evidence” of syphilis in that cranium, Smith (1909:77) writes, “l’auteur est mon distingué ami, M. le professeur Lortet...dont l’autorité scientifique eût suffi à donner à son opinion grand poids, même si elle eût été insoutenable, comme c’est le cas.” However, Smith (1909) acknowledges Lortet’s accurate description of these “lesions” and their resemblance to syphilitic ones. Smith (1909) goes on to convincingly argue these “lesions” are a result of

postmortem damage, citing issues such as lesion non-uniform coloration and a lack of evidence concerning inflammatory reactions and necrotic foci. He concludes that beetles or beetle larvae produced these alterations.

Individual N4162 (Peabody Object Number 46-89-30/N4162.0)

Individual N4162 (Figure 10) provides an example of pseudopathology, probably due to insect activity.

Demographic Data

Individual N4162 is represented by a calvarium—there are no dental or postcranial remains. Following Meindl and Lovejoy’s (1985) criteria for “vault system” age assessment, this Individual’s composite score is 13; associated inter-decile range, 31-65 years-old; and associated mean age, 45.2 years-old. A narrower age range could not be determined. (In this case, the inferior sphenotemporal suture cannot be observed, which precludes employing the lateral-anterior system.) It is thus more prudent to report a conservative age category of adult. This individual’s prominent supraorbital margin, prominent glabella region, large mastoid process, and overall robusticity are consistent with a sex assessment of male (Buikstra and Ubelaker 1994) (although see Walker 1995 on issues of assessing sex from crania). These data are consistent with Peabody’s records, which indicate Individual N4162 is an adult male from the central coast of Peru.

“Pathological” Description

The ectocranial surface is covered in serpentine and circular erosive, lytic lesions with notched edges, essentially destroying the outer table in affected sites. These lesions are bilateral but not symmetrical (Figure 11). The endocranium reveals lesions of the same morphology (Figure 12), but their location does not precisely correspond to the outer table lesions. Lesions observable on the outer table do not involve the inner table, and those that involve the inner table do not involve the outer table. All these lesions possess white coloration, making their coloration and that of the rest of the calvarium incompatible. There are no signs of bony reactivity.

Much like Lortet's (1907) case study, Individual N4162's lesions resemble *caries sicca*, a pathognomonic marker of syphilis (Rogers and Waldron 1989:620-621). However, the non-uniform coloration of these lesions in relation to the rest of the calvarium and the non-existent bony reactivity indicate pseudopathology. There are no true pathological lesions in this case. These pseudopathologies are probably due to insect activity.

Figure 10: Individual N4162 in anterior view. Note white discoloration of pseudopathological lesions. 46-89-30/N4162.0, *Courtesy of the Peabody Museum of Archaeology and Ethnology, Harvard University.*



Figure 11: Top surface view of pseudopathological lesions depicting bilateral but asymmetrical nature. Although difficult to see in this image, the edges of the “lesions” are white. 46-89-30/N4162.0, *Courtesy of the Peabody Museum of Archaeology and Ethnology, Harvard University.*



Figure 12: Endocranial pseudopathological lesions. Note “lesion” discoloration. 46-89-30/N4162.0, *Courtesy of the Peabody Museum of Archaeology and Ethnology, Harvard University.*



Hematopoietic Disease

Anemia

Earlier, I addressed issues associated with diagnosing a specific anemia from dry-bone cranial remains alone (see pages 5, 56-57). I also presented the skeletal markers associated with or caused by anemia (pages 57-59 and Table 5), emphasizing the need for evidence of marrow hyperplasia. To eschew redundancy, these topics will not be

reproduced here other than in the form of a few remarks. Briefly, a diagnosis of likely anemia is warranted in the presence of marrow hyperplasia, indicated by diploic hypertrophy. Outer table obliteration or thinning, a potential consequence of marrow expansion, is also suggestive of anemia. It is nearly impossible to determine a specific anemia (e.g., iron-deficiency anemia) from macroscopic examination of dry-bone cranial remains alone.

Below, I present two potential cases of anemia, which are suggested by evidence of marrow hyperplasia.

Individual N8641 (Peabody Object Number 59-27-30/N8641.0)

This Individual is an archaeological Peruvian juvenile of unsecure provenance (Peabody records). Active type 4 cribra orbitalia (Stuart-Macadam 1991) is present bilaterally in both orbital roofs. Possible (Figure 13) and likely (Figure 14) evidence of marrow hyperplasia is suggested by the slightly raised bony surface affecting the orbital roof with concomitant presence of large and coalescent foramina. Bilaterally, the orbital surface of the sphenoid bones present active type 2 cribra orbitalia (Stuart-Macadam 1991). Type 2 porotic hyperostosis (Steckel et al. 2005) is present on the frontal bone, both parietal bones, and occipital bone.

Figure 13: Individual N8641 right orbit cribra orbitalia with *possible* evidence of marrow hyperplasia. 59-27-30/N8641.0, *Courtesy of the Peabody Museum of Archaeology and Ethnology, Harvard University.*

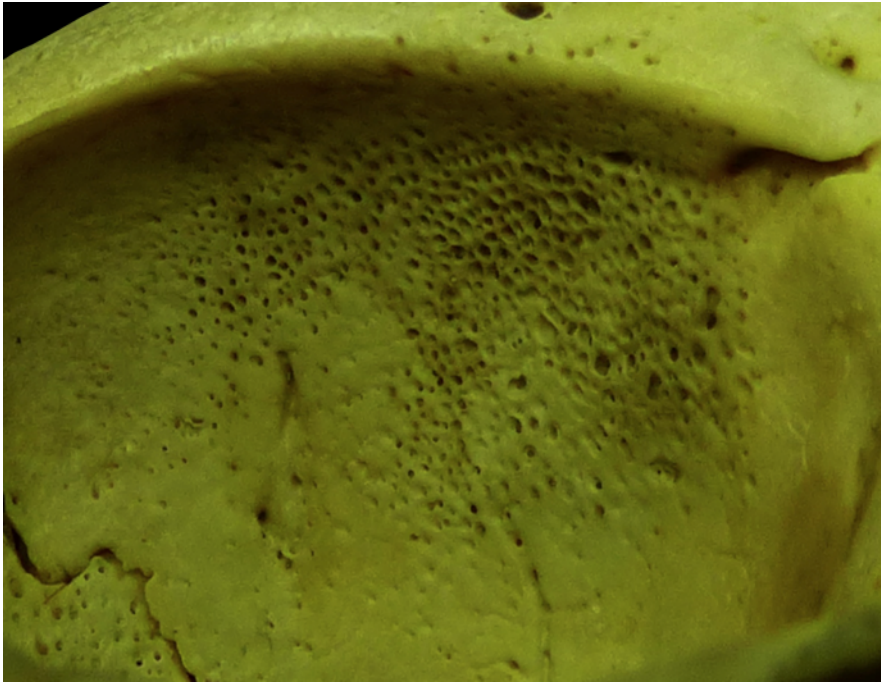
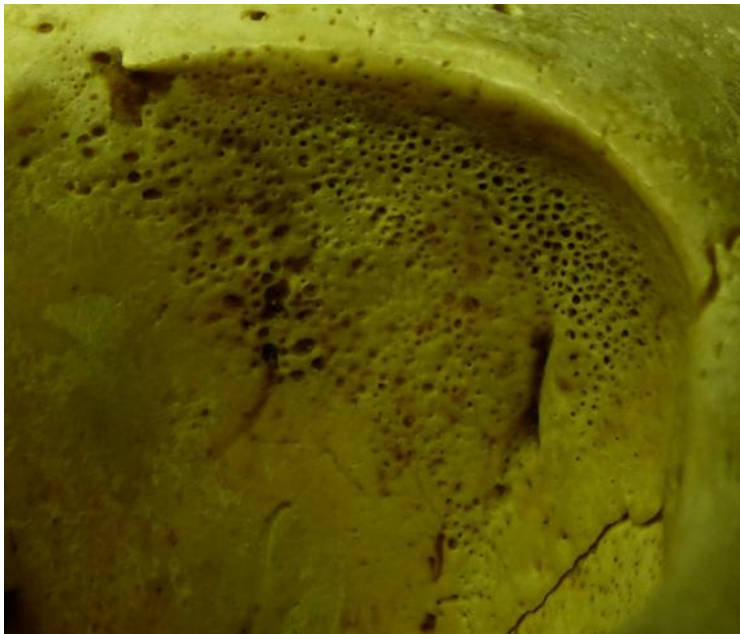


Figure 14: Individual N8641 left orbit cribra orbitalia with *likely* evidence of marrow hyperplasia. 59-27-30/N8641.0, *Courtesy of the Peabody Museum of Archaeology and Ethnology, Harvard University.*



Individual 23998 (Peabody Object Number 80-61-30/23998.0)

This individual is an archaeological Peruvian juvenile (Peabody records). (Peabody records indicate this individual is from a site named “ancient graves.” However, my readings of *Reports of the Peabody* suggest that “ancient graves” is a general descriptor, not a specific archaeological site.)

Individual 23998’s right orbit exhibits a mixture of types 3 and 5 cribra orbitalia (Stuart-Macadam 1991). This individual’s right orbital roof displays a slightly raised surface with large and coalescent porosities, suggestive of anemia. This raised portion comes at the expense of the outer table and is located in the orbit’s antero-lateral sector (Figure 15). This individual’s left orbital roof, displays type 3 (borderline type 4) cribra orbitalia with some evidence of coalescent porosities (Figure 16).

Figure 15: Individual 23998 right orbit cribra orbitalia. Note definite diploic hypertrophy. 80-61-30/23998.0, *Courtesy of the Peabody Museum of Archaeology and Ethnology, Harvard University.*

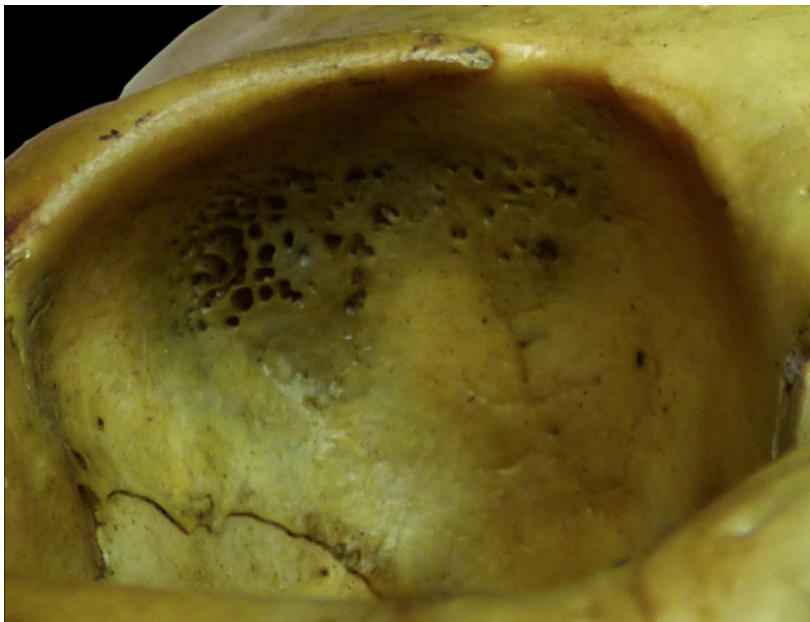


Figure 16: Individual 23998 left orbit cribra orbitalia. Note possible diploic hypertrophy (i.e., barely noticeable raised surface with some evidence of coalescent porosities). 80-61-30/23998.0, *Courtesy of the Peabody Museum of Archaeology and Ethnology, Harvard University.*



Infectious Disease

General characteristics of infectious disease in bone have already been presented and will not be duplicated here (see *Chapter 3's Differential Diagnosis Candidates*). For comprehensive clinical considerations of infectious diseases, the reader is referred to Jaffe (1975) and Murray (2009). For comprehensive paleopathological considerations of infectious diseases, the reader is referred to Aufderheide and Rodríguez-Martín (2006), Ortner (2003), and Roberts and Manchester (2005).

Individual N8434 (Peabody Object Number 56-42-30/N8434.0)

Individual N8434 (Figure 17) is an adult Peruvian female of unsecure provenance (Peabody records).

Figure 17: Individual N8434 in anterior view. 56-42-30/N8434.0, *Courtesy of the Peabody Museum of Archaeology and Ethnology, Harvard University.*



Description

The right aspect of the frontal bone, in the position of the right superior orbital margin and encroaching onto the glabellar region, exhibits a postmortem break. This is evinced by the white discoloration on the medial surface of the right orbital margin.

The large lesion stretching from the frontal to both parietal bones exhibits a mixture of osteoblastic and osteoclastic responses (Figure 18). This lesion's hole measures 54.1 millimeters along the coronal plane and 69.7 millimeters. This lesion's margins are irregular (spiculated), "notched," and expanded. This lesion necrotized the outer table, diploe, and inner table. The outer table is more necrotized than the diploe, and the diploe more than the inner table. This feature is particularly noticeable on the

most posterior portion of the lesion. This lesion lacks the projecting long and thin spicule bone formation (“sunburst” pattern) that characterizes some cancerous lesions (Figure 18) (Aufderheide and Rodríguez-Martín 2006:179). Instead, the osteoblastic activity at the lesion’s margins is short and thick.

Both of the parietal bones present with vascular-like scarring characteristic of infection (Figure 19). These bones exhibit hypervascularity in the form of small but diffuse porous lesions and periosteal reactivity. There is one prominently depressed lesion on the right parietal bone, which indicates healing (Figure 20) (Erdal 2006:18). This depression’s size is 28.3 millimeters along the sagittal plane and 19.7 millimeters along the coronal plane. There are also signs of sequestration on the right parietal bone (Figure 20). There are two depressed lesions on the right parietal bone (Figure 21), which indicate healing (Erdal 2006:18). The inferior depressed lesion measures 26.7 millimeters along the sagittal plane, 19 millimeters along the coronal plane. The superior depressed lesion measures 18.4 millimeters along the coronal plane and 18.4 millimeters along the sagittal plane. Overall, necrotic bone and a “lumpy” surface characterize the parietal bones.

There are two fistulae (similar in size) just anterior and lateral to the right spheno-temporal suture (Figure 22a). There are two fistulae (one larger than the other) just anterior and lateral to the left spheno-temporal suture (Figure 22b).

The nasal spine, nasal floor, nasal walls, and vomer are not compromised. The occipital bone is covered in soft tissue and could not be examined. The maxilla exhibits signs of periodontal disease. No teeth are present. The mandible is not present.

Figure 18: Frontal bone lesion detail. Note that outer table is necrotized more than diploë and inner table (arrow). Also note the shortness and thickness of the bony reactivities. 56-42-30/N8434.0, *Courtesy of the Peabody Museum of Archaeology and Ethnology, Harvard University.*

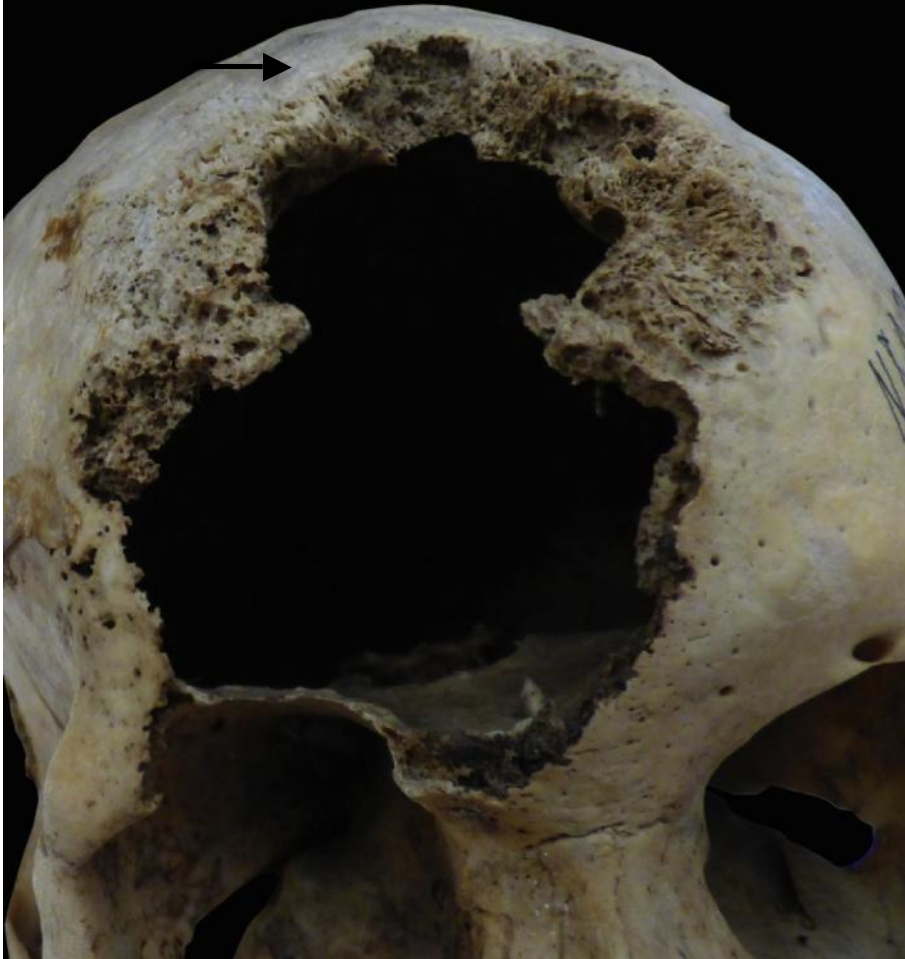


Figure 19: Parietal bone lesions in top surface view. Note necroses (white arrows), scarring (black arrows), diffuse porosity, and “lumpy” character. 56-42-30/N8434.0, *Courtesy of the Peabody Museum of Archaeology and Ethnology, Harvard University.*

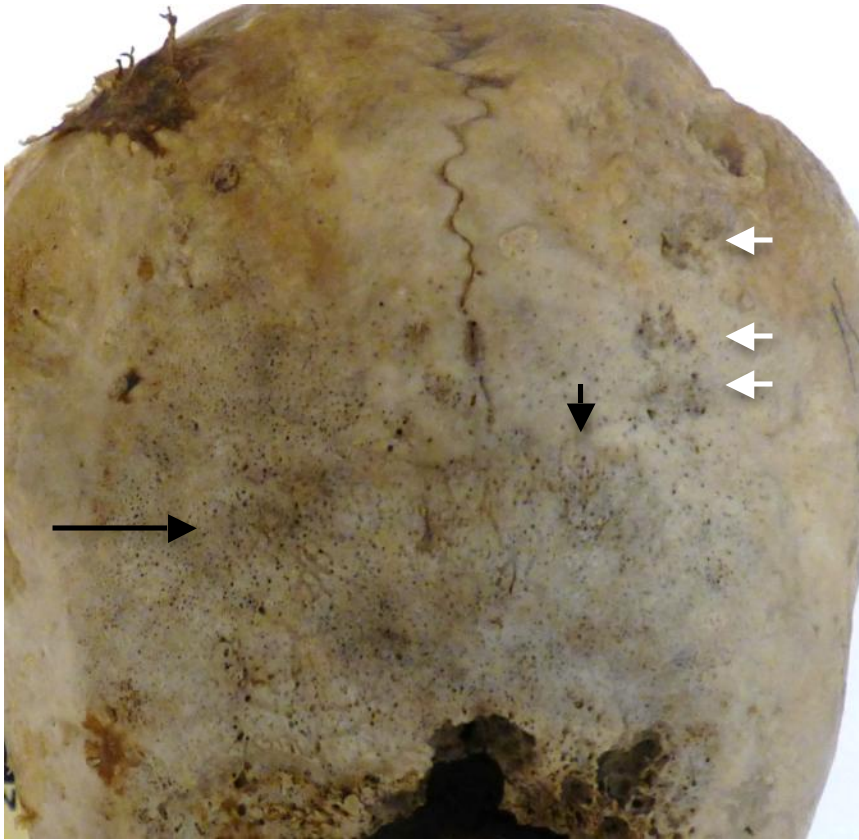


Figure 20: Left parietal bone pathologies. Note the prominent depressed lesion indicative of healing and sequestrum (arrow). 56-42-30/N8434.0, *Courtesy of the Peabody Museum of Archaeology and Ethnology, Harvard University.*

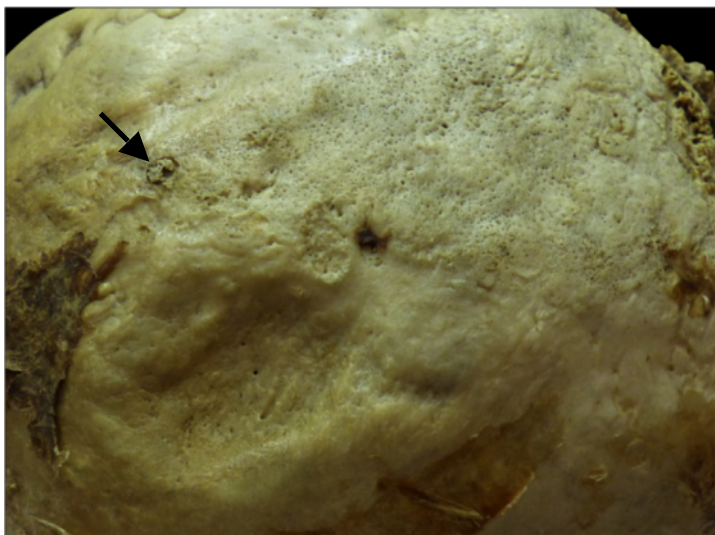


Figure 21: Right parietal bone pathologies. Note the two prominent depressed lesions indicative of healing. 56-42-30/N8434.0, *Courtesy of the Peabody Museum of Archaeology and Ethnology, Harvard University.*



Figure 22: Fistulae. a: Fistulae in right lateral view. b: Fistulae in left lateral view. Note the small sequestrum (arrow). 56-42-30/N8434.0, *Courtesy of the Peabody Museum of Archaeology and Ethnology, Harvard University.*



Diagnosis

The adage, “better a broader diagnosis than a misdiagnosis” (Marques et al. 2013), is applied in this case—especially in the absence of radiography, postcranial remains, and unequivocal pathognomonic markers (e.g., classic carries sicca) (see Ragsdale and Lehmer 2012 on employing broad disease categories). A diagnosis of probable infectious disease is offered. The possibility that the large lesion on the frontal

bone is a result of cancerous processes should be considered. This large lesion's mixed osteoblastic and osteoclastic activity, and notched margins can be associated with cancerous conditions (e.g., Assis and Codinha 2009; Kaufmann et al. 1997; Marks and Hamilton 2007; Ortner 2003). These features, however, can equally be attributed to infectious processes (Ortner 2003).

The “sunburst” pattern found in some cancerous conditions is not observed. In this case, the large lesion's bony reactivity is short and thick—contra long and thin spicule formation found in some cancers (Aufderheide and Rodríguez-Martín 2006:179). While “lumps” are found in tumors, they are also found in cases of infection (Ortner 2003). The “lumpy” appearance of the cranium in this case is more consistent with a flux of active and healing responses rather than uncontrolled growth. This is particularly noted in the pronounced depression lesions described above. The fistulae might be confused with small lytic lesions in, for example, multiple myeloma. However, the infectious character of the parietal bones argues for these small lesions being fistulae resulting from infection, as does the presence of a small sequestrum (Figure 22b). Evidence of sequestra, depressed lesions, short and thick reactivity of the large frontal bone lesion, presence of scarring due to healing, and fistulae argue for a diagnosis of infectious disease rather than a cancerous condition.

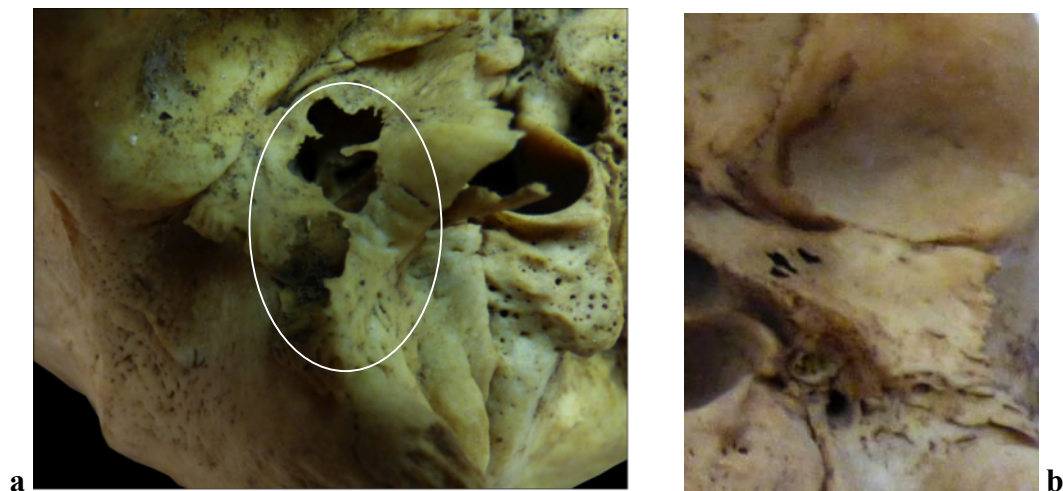
Individual 472 (Peabody Object Number 67-11-30/472.0)

Individual 472 is an archaeological adult Peruvian male from Pachicamac (Peabody records). This individual displays a possible case of otitis media (middle ear

infection). Otitis media may be caused by various microbial agents and may result in mastoiditis (Flohr and Schultz 2009).

Individual 472 presents with possible bilateral otitis media. The right tympanic plate is severely perforated inferiorly and exhibits spicular bone formation (Figure 23a) (see Mann and Hunt 2012). The lateral portion of the right tympanic plate also displays osteoclastic activity (Figure 23a). The left tympanic plate is less severely affected, displaying three small lytic lesions (Figure 23b). This individual also presented with type 2 (borderline type 3) (Stuart-Macadam 1991) healed bilateral cribra orbitalia.

Figure 23: Individual 472. **a:** Right tympanic plate perforation and spicule bone formation (encircled); **b:** Left tympanic plate perforation as three small lytic lesions. 67-11-30/472.0, *Courtesy of the Peabody Museum of Archaeology and Ethnology, Harvard University.*



Neoplastic Disease

By definition, a neoplasm (new growth) is an uncontrolled growth of tissue cells, which may be either benign or malignant in nature (Roberts and Manchester 2005:252).

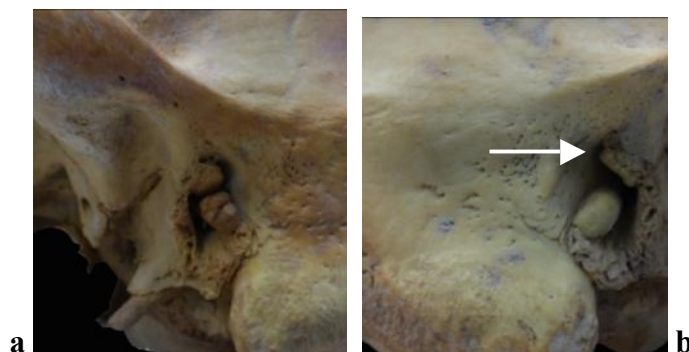
Benign neoplasms are generally comprised of differentiated cells, localized, and

considered clinically insignificant in comparison to malignant neoplasms (i.e., cancer), which are generally comprised of poorly differentiated cells that may affect other parts of the body through blood and/or lymphatic vessels (Ortner 2003:503; Roberts and Manchester 2005:252-253).

While Roberts and Manchester (2005) discuss auditory exostoses in their chapter on joint diseases, I follow Ortner's (2003) classificatory system in that I consider auditory exostoses a neoplastic condition.

As an example of auditory exostoses (a benign neoplasm), I turn to Individual N4147 (Peabody object number 46-89-30/N4147.0)—an archaeological adult Peruvian male from Peru's central coast (Peabody records). This individual has two auditory exostoses in each auditory meatus (Figure 24).

Figure 24: Individual N4147. **a:** Two exostoses in left auditory meatus. **b:** Two exostoses in right auditory meatus. Note that one is a smaller, incipient exostosis (arrow). 46-89-30/N4147.0, *Courtesy of the Peabody Museum of Archaeology and Ethnology, Harvard University.*



Joint Disease

This section focuses on temporomandibular joint disease (TMJD). The temporomandibular joint may become compromised as a result of—among other causes—infection, trauma, degenerative osteoarthritis (associated with old age),

rheumatoid arthritis, and metastatic malignancies (Langsjoen 2006:399-400). Below, I present two cases of temporomandibular joint disease.

Individual N8431 (Peabody object number 56-42-30/N8431.0) (Figure 25a) presents a severe case of TMJD. This individual is an archaeological adult Peruvian female (Peabody records). Cranial suture obliteration at multiple sites suggests this individual is superannuated. Pathologically, this individual presents with an edentulous jaw, mandibular asymmetry, repositioning of the right mental foramen, and temporomandibular joint ankylosis (Figure 25a,b). This last feature is considered especially incapacitating in TMJD (Figure 25b) (Langsjoen 2006:400).

Figure 25: Individual N8431. **a:** In anterior view. Note mandibular asymmetry, repositioning of mental foramen, and edentulous jaw. **b:** Temporomandibular ankylosis (squared). 56-42-30/N8431.0, *Courtesy of the Peabody Museum of Archaeology and Ethnology, Harvard University.*



Individual 59650 (Peabody object number 16-8-30/59650.0) is an archaeological Peruvian adult male from Samanka (Peabody records). This individual exhibits bilateral temporomandibular osteoarthritis in the form of mandibular fossa pitting (Figure 26).

Figure 26: Individual 59650. Note pitting of both temporal bones' TMJ surface. *16-8-30/59650.0, Courtesy of the Peabody Museum of Archaeology and Ethnology, Harvard University.*



Trauma and Trauma-Induced Disease

The case studies presented below exhibit signs of trauma and/or disease possibly during or following a physiologically traumatic event.

Individual N3919 (Peabody Object Number 41-93-30/N3919.0)

I turn to Individual N3919 (Figure 27) for an example of trauma. This individual is an archaeological Peruvian adult male from Ollantaytambo, Cuzco region.

Description of Trauma

This individual displays multiple craniofacial fractures and lesions. Beginning on the frontal bone, there is a large healed depression lesion (Figure 27, 28). This lesion is

concordant with this individual's trauma-induced cranio-facial asymmetry—in which the left orbit superciliary arch is positioned below the expected glabellar horizontal axis such that the left orbital walls are expanded laterally (Figure 27). On the left superciliary arch, just medial and posterior to the frontozygomatic suture, in association with this large depression lesion, there is a healed fracture line (Figure 28). The left orbital roof presents remodeled fracture lines. At the center of this remodeling, there is an open lesion that displays light osteoblastic activity (Figure 29). Lateral to the left infraorbital foramen, there is an osteolytic lesion, exposing the maxillary sinus (Figure 30). This lesion measures 19.3 millimeters in height, 16.5 millimeters in width. There is bony reactivity in the form of spicular growths on the medial side as well as superior portion of the lesion. At the lateral-inferior portion of this lesion, there is a possibly healed fracture line (Figure 30).

On the medial aspect of the right orbito-nasal margin, there is what appears to be a sclerotic bony growth projecting into the orbital cavity (Figure 31). The inferior aspects of the nasal bones exhibit healed fracture lines (Figure 31). This feature is not to be confused with typical nasomaxillary suture morphology.

The nasal septum is deviated to the left. There is also a healed trephination hole on the left parietal bone. Dental and maxillary alveolar bone pathologies are present in the forms of alveolar bone recession, antemortem tooth loss, dental caries, periapical abscesses, and dental chipping. There is no associated mandible.

Figure 27: Individual N3919 in anterior view. 41-93-30/N3919.0, *Courtesy of the Peabody Museum of Archaeology and Ethnology, Harvard University.*



Figure 28: Healed depression lesion and healed fracture line (arrow). 41-93-30/N3919.0, *Courtesy of the Peabody Museum of Archaeology and Ethnology, Harvard University.*

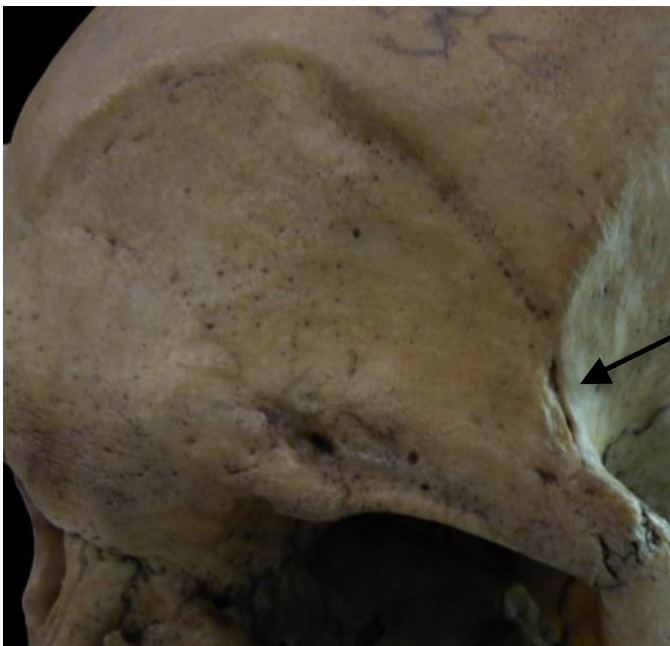


Figure 29: Healed orbital fracture (black arrow) and osteoblastic activity (white arrows). 41-93-30/N3919.0, Courtesy of the Peabody Museum of Archaeology and Ethnology, Harvard University.

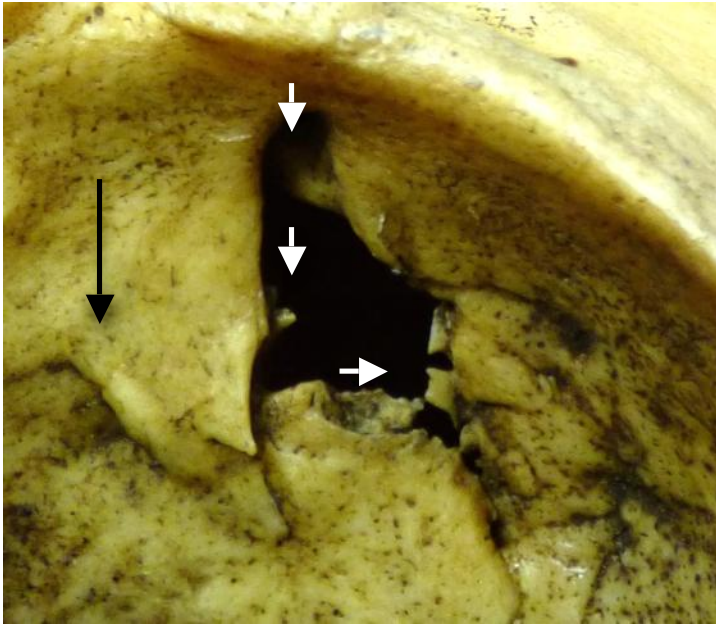


Figure 30: Osteolytic lesion. Note healed fracture line (arrow-head), spicule bone formation (encircled), and periapical abscesses (arrows). 41-93-30/N3919.0, Courtesy of the Peabody Museum of Archaeology and Ethnology, Harvard University.

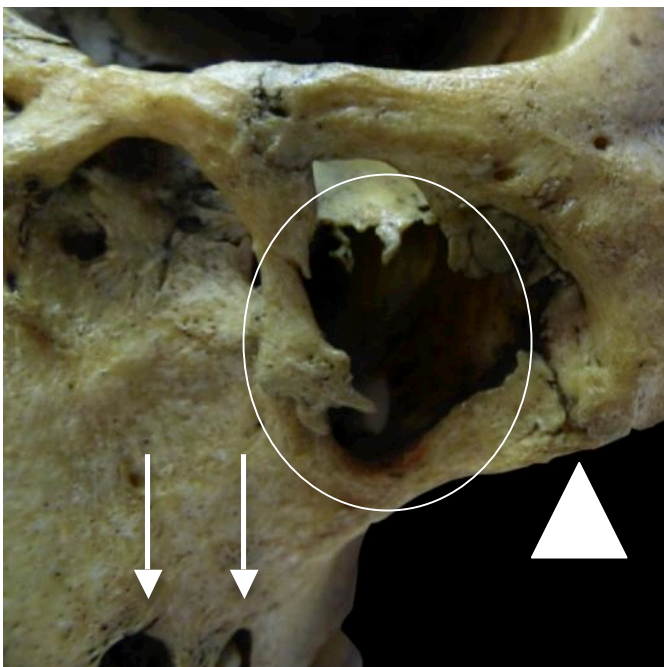


Figure 31: Nasal lesions detail. Note bony projection (white arrow) and fracture healing with remodeling (encircled), which is not to be confused with nasomaxillary sutures. 41-93-30/N3919.0, *Courtesy of the Peabody Museum of Archaeology and Ethnology, Harvard University.*



Individual N7945 (Peabody Object Number: 56-42-30/N7945.0)

This Individual (Figure 32) is an archaeological Peruvian adult, probable female from Cushashica (Peabody records).

Description

There are probable trephination marks on the left and right parietal bones (Figure 33). These marks surround a large hole encompassing both the left and right parietal bones in the position of the vertex (Figure 33). This hole measures 48.7 millimeters along the sagittal plane and 31.6 millimeters along the coronal plane. Bone necrosis and sequestra are present throughout the top surface of both parietal bones (Figure 33).

There are signs of extensive remodeling on the left parietal bone in a semi-circumferential pattern (Figure 34). This feature is characterized by a well-defined groove whose superior margins exhibit osteoclastic activity on the edges (which are sharply defined) of the outer table. The right parietal bone also exhibits a semi-circumferential

pattern of bony activity. However, areas of sequestra that are surrounded by necrotic tissue characterize this activity (Figure 33). And, this semi-circumferential pattern is smaller than that of the left parietal bone. These two patterns of the right and left parietal bones meet at the above-described large hole's most anterior portion and at the large hole's most posterior portion on the sagittal suture. However, this posterior meeting of the two semi-circumferential patterns' posterior aspects produces the appearance of an approximately 120-degree angle outline (Figure 35). There is also a fracture on the posterior aspect of the right maxillary sinus, which measures 8 millimeters in width and 9 millimeters in height. This fracture is probably postmortem damage (Figure 36).

Diagnosis

The trephination marks and their proximity to necrotic bony responses are clear. Possibly, an infectious process took place either during or following trephination. It is interesting to note that the described semi-circumferential grooves, particularly of the left parietal bone, are similar to osseous responses produced in instances of blows to the head or scalping (respectively, for comparative images, see Ortner 2003:130, figure 8-17 and Ortner 2003:168, figure 8-76). Scalping, accidental or intentional, is not well documented in Peruvian contexts (see Toyne 2011 for possible examples). In the absence of postcranial remains and contextual data, *by no means* do I offer a specific physiologically traumatic etiology for these osseous responses. However, the proximity of necrotic tissue, sequestra, and remodeling to signs of physiological trauma (i.e., trephination marks) argues for an infectious process during or after a possible physiologically traumatic event, whatever that possible event(s) may be.

Figure 32: Individual N7945 in anterior view. 56-42-30/N7945.0, *Courtesy of the Peabody Museum of Archaeology and Ethnology, Harvard University.*

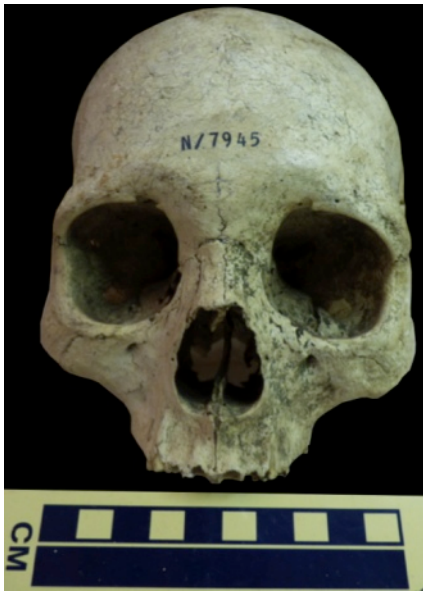


Figure 33: Lesion on vertex. Note cut/trephination marks (arrows), necrotic tissue, and sequestra. 56-42-30/N7945.0, *Courtesy of the Peabody Museum of Archaeology and Ethnology, Harvard University.*

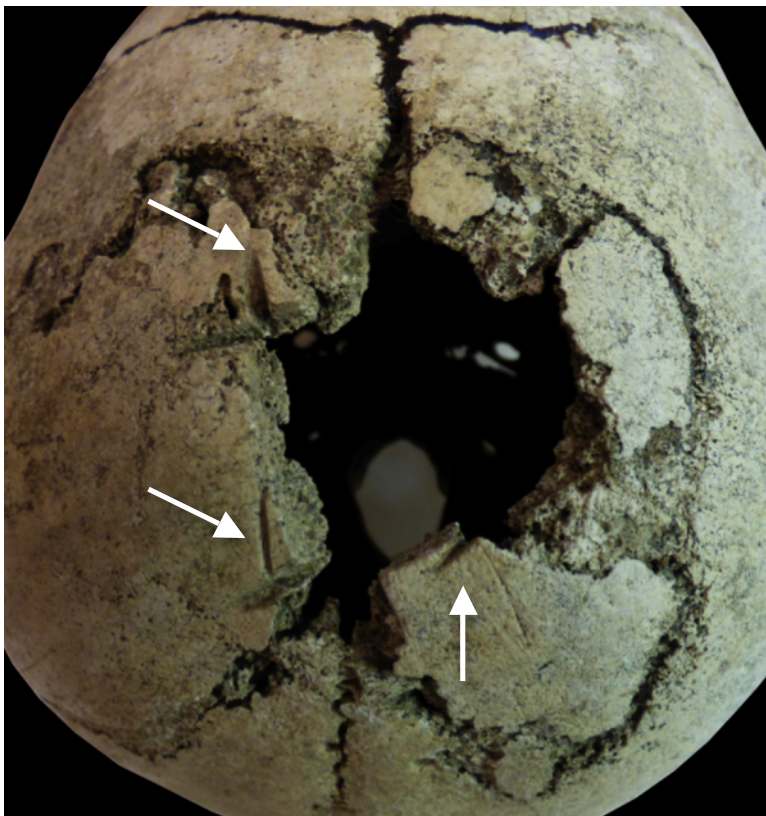


Figure 34: Right parietal bone remodeling detail in a semi-circumferential pattern with a well-defined groove. Note also possible scrape mark (black arrow). 56-42-30/N7945.0, *Courtesy of the Peabody Museum of Archaeology and Ethnology, Harvard University.*

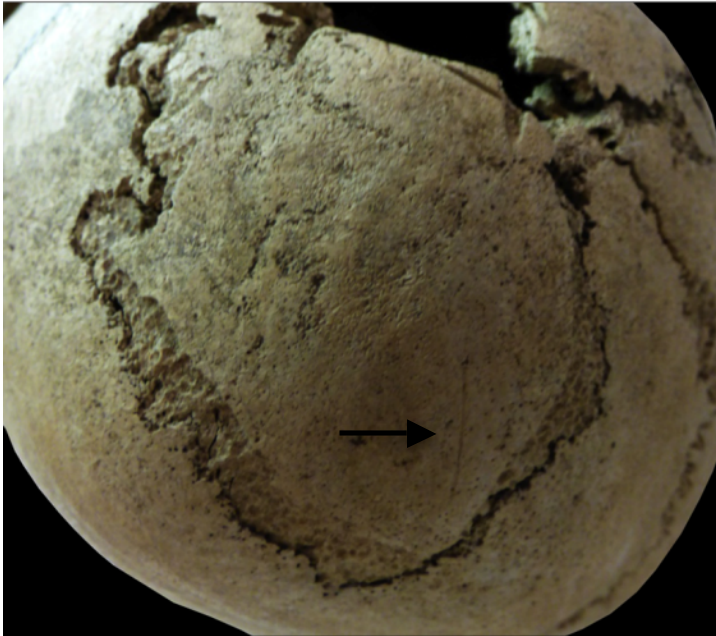


Figure 35: Individual N7945 in posterior view. Note the approximately 120-degree angle created at the posterior meeting of the vertex lesion. 56-42-30/N7945.0, *Courtesy of the Peabody Museum of Archaeology and Ethnology, Harvard University.*

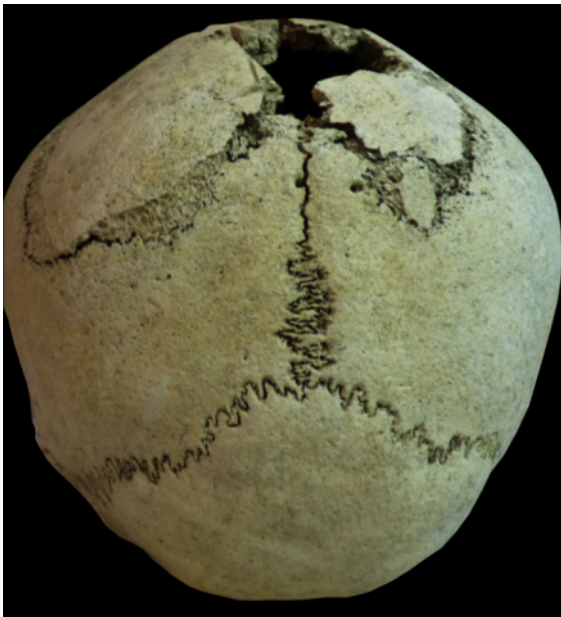


Figure 36: Right-posterior maxillary fracture exposing maxillary sinus. Fracture is probably postmortem. 56-42-30/N7945.0, *Courtesy of the Peabody Museum of Archaeology and Ethnology, Harvard University.*



Summary

This chapter has provided case studies representing pseudopathology, hematopoietic disease, infectious disease, neoplastic disease, joint disease, and trauma and trauma-induced disease. Individual N4162 is a case of pseudopathology, probably due to insect activity. Individual's N8641 and 23998 exhibit cases of possible and likely anemia, respectively. Individual's N8434 and 472 reflect cases of probable cranial osteomyelitis and possible bilateral otitis media, respectively. A case of benign neoplasms is represented in Individual N4147 in the form of auditory exostoses. Severe TMJ osteoarthritis in the form of temporomandibular ankylosis is present in Individual N8431. A less severe manifestation of TMJD is displayed in Individual 59650. Individual's N3919 and N7945 are cases of trauma-induced craniofacial asymmetry and trauma-induced infection, respectively.

CHAPTER 5: THESIS SUMMARY

In this thesis, I presented 11 case studies of paleopathological significance. I discussed cases representing pseudopathology, hematopoietic diseases, joint diseases, infectious diseases, neoplastic diseases, and trauma and traum-induced diseases. In other words, I offered diagnoses based on broad disease categories and only presented specific diagnoses in the light of appropriate evidence. I also indicated my level of confidence in attributing a diagnosis by stating whether a proposed condition is “possible,” “likely,” or “probable.” These aspects have been called for in the paleopathological literature (e.g., Ortner 2011, 2012; Ragsdale and Lehmer 2012).

I gave considerable attention to Individual 23985. This case’s significance rests in the fact that there are few publications on Peruvian juvenile scurvy from antiquity in the English literature. This individual, to my knowledge, offers the first documented Peruvian juvenile case in the English language literature that displays signs of scurvy and anemia comorbidity. I provided a critical review and analysis of the literature surrounding juvenile scurvy. As a response to calls for heightened rigor in diagnosing cases of scurvy, I offered a detailed macroscopic examination and differential diagnosis process. This process entailed constructing a differential diagnosis table and flow-chart. This table and flow-chart may be used and modified by future scholars in the diagnosis of suspected cases of juvenile scurvy.

Further, I recognized that diagnostic models have their limitations. However, I also advanced that such models serve as useful heuristic devices, allowing for an effective tool that communicates anatomical, pathological, and epidemiological data. Through an approach that combined a descriptive differential diagnosis and a diagnostic model, I diagnosed Individual 23985 with probable scurvy and likely anemia comorbidity. I stressed, however, that this case's diagnostic description and model construction were equally important in arriving at Individual 23985's diagnosis. Moreover, this case highlighted the detailed descriptions required to identify signs of disease comorbidity. Ultimately, this case study added to the literature on Peruvian juvenile scurvy. And, it is hoped that the process undertaken to do so enriched paleopathological diagnostic approaches.

APPENDICES

Appendix A: Peabody Object Numbers. Individuals Presented in This Thesis are in Bold Font.

11-1-30/58588.0	46-77-30/N4229.0	46-89-30/N4162.0
14-4-30/59402.0	46-77-30/N4230.0	46-89-30/N4163.0
14-4-30/59418.0	46-89-30/N4135.0	46-89-30/N4164.0
14-4-30/59434.0	46-89-30/N4136.0	46-89-30/N4165.0
14-4-30/59436.0	46-89-30/N4137.0	46-89-30/N4166.0
16-5-30/59393.0	46-89-30/N4138.0	46-89-30/N4167.0
16-5-30/60798.0	46-89-30/N4139.0	46-89-30/N4168.0
16-8-30/59650.0	46-89-30/N4140.0	46-89-30/N4170.0
16-8-30/59651.0	46-89-30/N4141.0	46-89-30/N4172.0
16-8-30/59652.0	46-89-30/N4142.0	46-89-30/N4173.0
16-8-30/59654.0	46-89-30/N4143.0	46-89-30/N4174.0
16-8-30/59655.0	46-89-30/N4145.0	46-89-30/N4175.0
16-8-30/59656.0	46-89-30/N4146.0	46-89-30/N4176.0
35-33-30/N1300.0	46-89-30/N4147.0	46-89-30/N4177.0
35-33-30/N1301.0	46-89-30/N4148.0	46-89-30/N4178.0
35-33-30/N1305.0	46-89-30/N4149.0	46-89-30/N4179.0
38-28-30/N3970.0	46-89-30/N4150.0	46-89-30/N4180.0
40-28-30/N3620.0	46-89-30/N4151.0	46-89-30/N4181.0
40-28-30/N3621.0	46-89-30/N4152.0	46-89-30/N4182.0
40-28-30/N3622.0	46-89-30/N4153.0	46-89-30/N4183.0
41-93-30/N3919.0	46-89-30/N4154.0	46-89-30/N4184.0
46-77-30/N4220.0	46-89-30/N4158.0	46-89-30/N4185.0
46-77-30/N4223.0	46-89-30/N4159.0	46-89-30/N4186.0
46-77-30/N4224.0	46-89-30/N4160.0	46-89-30/N4187.0
46-77-30/N4226.0	46-89-30/N4161.0	46-89-30/N4188.0

46-89-30/N4189.0	56-42-30/N8372.0	67-11-30/435.0
46-89-30/N4190.0	56-42-30/N8373.0	67-11-30/436.0
46-89-30/N4191.0	56-42-30/N8375.0	67-11-30/437.0
46-89-30/N4199.0	56-42-30/N8376.0	67-11-30/451.0
46-89-30/N4211.0	56-42-30/N8377.0	67-11-30/452.0
46-89-30/N4212.0	56-42-30/N8428.0	67-11-30/453.0
46-89-30/N4214.0	56-42-30/N8431.0	67-11-30/454.0
46-89-30/N4218.2	56-42-30/N8432.0	67-11-30/455.0
50-70-30/N7460.0	56-42-30/N8433.0	67-11-30/456.0
50-70-30/N7463.0	56-42-30/N8434.0	67-11-30/457.0
50-70-30/N7465.0	56-42-30/N8435.0	67-11-30/458.0
51-23-30/N7338.0	56-42-30/N8436.0	67-11-30/459.0
56-42-30/N7941.0	56-42-30/N8437.0	67-11-30/460.0
56-42-30/N7942.0	59-27-30/N8628.0	67-11-30/461.0
56-42-30/N7943.0	59-27-30/N8629.0	67-11-30/462.0
56-42-30/N7944.0	59-27-30/N8630.0	67-11-30/463.0
56-42-30/N7945.0	59-27-30/N8633.0	67-11-30/464.0
56-42-30/N7946.0	59-27-30/N8634.0	67-11-30/465.0
56-42-30/N7947.0	59-27-30/N8635.0	67-11-30/466.0
56-42-30/N7948.0	59-27-30/N8636.0	67-11-30/467.0
56-42-30/N7949.0	59-27-30/N8637.0	67-11-30/468.0
56-42-30/N7951.0	59-27-30/N8638.0	67-11-30/469.0
56-42-30/N7952.0	59-27-30/N8639.0	67-11-30/470.0
56-42-30/N7953.0	59-27-30/N8640.0	67-11-30/471.0
56-42-30/N8161.0	59-27-30/N8641.0	67-11-30/472.0
56-42-30/N8162.0	59-27-30/N8642.0	67-11-30/473.0
56-42-30/N8370.0	67-11-30/432.0	67-11-30/474.0
56-42-30/N8371.0	67-11-30/434.0	67-11-30/500.0

67-11-30/501.0

67-11-30/502.0

67-11-30/505.0

67-32-30/699.0

75-20-30/8853.0

75-20-30/8855.0

75-20-30/8856.0

75-20-30/8857.0

80-25-30/23195.0

80-25-30/23196.0

80-61-30/23985.0

80-61-30/23986.0

80-61-30/23987.0

80-61-30/23988.0

80-61-30/23989.0

80-61-30/23990.0

80-61-30/23991.0

80-61-30/23992.0

80-61-30/23993.0

80-61-30/23994.0

80-61-30/23995.0

80-61-30/23996.0

80-61-30/23997.0

80-61-30/23998.0

80-61-30/58801.0

968-9-30/N8930.0

968-9-30/N8931.0

968-9-30/N8932.0

Appendix B: Case Study Summaries (In Order of Case Discussion in This Thesis)

Individual 23985

Recorder: Tony J. Chamoun

Date Of Data Collection: January 16, 2014

Peabody Object No.: 80-61-30/23985.0

MNI: 1

Inventory: Cranium: Complete; Mandible: Complete; Dental: Partial

Provenience: “Ancient graves,” Peru, South America

Age/Criteria: Juvenile of approximately 6 years/Mandibular tooth eruption following Buikstra and Ubelaker (1994:51, figure 24) and AlQahtani et al. (2010:485, figure 6)

Sex/Criteria: ?/Not attempted

Lesion Summary: Cribra orbitalia and new bone formation of left orbit; Bilateral and symmetrical porous, hypertrophic lesions; Bilateral and symmetrical cortical porosities throughout skull, including greater wings of both sphenoid bones; New porous woven bone formation on frontal; Vascular impression lesions throughout top surface of skull; Endocranial pitted lesions; Post-mortem fracture of occipital bone; Pathological? maxillary alveolar socket reactivity; Maxillary alveolar porosity; Mandibular porous lesions; Mandibular alveolar porosity; Dental caries and calculus of mandibular and maxillary teeth

Other Observations: Bone and soft tissue; Wormian bones along lambdoid suture; Hematogenous staining?; Calcified hemorrhages?

Diagnosis: Metabolic disease—probable scurvy and likely anemia comorbidity

Individual N4162

Recorder: Tony J. Chamoun

Date Of Data Collection: January 9, 2014

Peabody Object No.: 46-89-30/N4162.0

MNI: 1

Inventory: Cranium: Partial; Mandible: Absent; Dental: Absent

Provenience: Li-15, central coast, Peru, South America

Age/Criteria: Adult/Peabody records and consistent with Meindl and Lovejoy (1985)

Sex/Criteria: Male/Peabody records and consistent with cranial features

Lesion Summary: Ectocranial (resembling *caries sicca*) and endocranial “lesions” with white edges and margins.

Other Observations: N/A

Diagnosis: Pseudopathology, probably due to insect activity.

Individual N8641

Recorder: Tony J. Chamoun

Date Of Data Collection: January 14, 2014

Peabody Object No.: 59-27-30/N8641.0

MNI: 1

Inventory: Cranium: Complete; Mandible: Absent; Dental: Partial

Provenience: Peru, South America

Age/Criteria: Juvenile/Peabody records

Sex/Criteria: ?/Not attempted

Lesion Summary: Active type 4 cribra orbitalia (Stuart-Macadam 1991) in both orbital roofs. Evidence of marrow hyperplasia. Active type 2 cribra orbitalia on sphenoid bone orbital surface, bilaterally (Stuart-Macadam 1991). Type 2 porotic hyperostosis (Steckel et al. 2005) on the frontal bone, both parietal bones, and occipital bone.

Other Observations: “Normal” greater wing of sphenoid bone porosity; Pathological temporal bone porosity bilaterally?

Diagnosis: Hematopoietic disease—likely presence of anemia

Individual 23998

Recorder: Tony J. Chamoun

Date Of Data Collection: January 16, 2014

Peabody Object No.: 80-61-30/23998.0

MNI: 1

Inventory: Cranium: Complete; Mandible: Present; Dental: Partial

Provenience: “Ancient Graves,” Peru, South America

Age/Criteria: Juvenile/Peabody records

Sex/Criteria: ?/Not attempted

Lesion Summary: Mixture of types 3 and 5 cribra orbitalia in right orbit (Stuart-Macadam 1991). Evidence of marrow hyperplasia. Left orbital roof type 3 (borderline type 4) cribra orbitalia.

Other Observations: Cranial shaping; Palatine torus

Diagnosis: Hematopoietic disease—likely presence of anemia

Individual N8434

Recorder: Tony J. Chamoun

Date Of Data Collection: January 14, 2014

Peabody Object No.: 56-42-30/N8434.0

MNI: 1

Inventory: Cranium: Complete; Mandible: Absent; Dental: Absent

Provenience: Peru, South America

Age/Criteria: Adult/Peabody records

Sex/Criteria: Female/Peabody records

Lesion Summary: Postmortem break on portion of frontal. Antemortem large lesion stretching from frontal to both parietal bones has mixture of osteoblastic and osteoclastic responses. Lesion’s hole measures 54.1 millimeters along coronal plane and 69.7 millimeters along sagittal plane. Margins irregular (spiculated), “notched,” and expanded. Reactivity short and thick. Lesion necrotized outer table, diploe, and inner table. Both parietal bones present vascular-like scarring characteristic of infection and small but diffuse porous lesions and periosteal reactivity. Depressed lesion on parietal bone indicates healing-- 28.3 millimeters along the sagittal plane and 19.7 millimeters along the coronal plane. Signs of sequestration on right parietal bone. Two depressed lesions on the right parietal

bone--indicate healing-- inferior lesion measures 26.7 millimeters along sagittal plane, 19 millimeters along coronal plane. The superior depressed lesion: 18.4 millimeters along coronal plane and 18.4 millimeters along sagittal plane. Overall, necrotic bone and a “lumpy” surface characterize the parietal bones. Presence of fistulae.

Other Observations: Periodontal disease; Soft-tissue preservation

Diagnosis: Infectious disease—cranial osteomyelitis (i.e., specific etiology unknown)

Individual 472

Recorder: Tony J. Chamoun

Date Of Data Collection: January 15, 2014

Peabody Object No.: 67-11-30/472.0

MNI: 1

Inventory: Cranium: Complete; Mandible: Absent; Dental: Partial (one tooth, chipped)

Provenience: Pachicamac, Peru, South America

Age/Criteria: Adult/Peabody records

Sex/Criteria: Male/Peabody records

Lesion Summary: Right tympanic plate severely perforated inferiorly and exhibits spicular bone formation. The lateral portion of the right tympanic plate also displays osteoclastic activity. The left tympanic plate is less severely affected, displaying three small lytic lesions. Type 1 (Stuart-Macadam 1991) healed bilateral cribra orbitalia.

Other Observations: Periodontal disease

Diagnosis: Infectious disease—possible bilateral otitis media

Individual N4147

Recorder: Tony J. Chamoun

Date Of Data Collection: January 8, 2014

Peabody Object No.: 46-89-30/N4147.0

MNI: 1

Inventory: Cranium: Complete; Mandible: Complete; Dental: Partial

Provenience: Li-29, central coast, Peru, South America

Age/Criteria: Adult/Peabody records

Sex/Criteria: Male/Peabody records

Lesion Summary: Two auditory exostoses on each side. Porotic hyperostosis level 2 (Steckel et al. 2005) of cranial vault. Pitting of both temporal's TMJ surface (level 2); Dental caries and calculus.

Other Observations: Mandible stained green; cranium orange and purple hue

Diagnosis: Neoplastic disease—benign—auditory exostoses; Porotic hyperostosis uncertain etiology; TMJ disease—pitting of TMJ surfaces

Individual N8431

Recorder: Tony J. Chamoun

Date Of Data Collection: January 14, 2014

Peabody Object No.: 56-42-30/N8431.0

MNI: 1

Inventory: Cranium: Partial; Mandible: Complete; Dental: Absent

Provenience: Viscas Yauyos, Peru, South America

Age/Criteria: Adult/Peabody records

Sex/Criteria: Female/Peabody records

Lesion Summary: Temporomandibular ankylosis, mandibular asymmetry, repositioning of mental foramen. Light occipital porosity (level 2, Steckel et al. 2005)

Other Observations: Facial bone fracture (maxilla and nasals absent postmortem, ethmoid sinus exposed). Occipital-parietal fracture (postmortem—white discoloration—fracture lines)

Diagnosis: Joint disease—TMJD—temporomandibular ankylosis

Individual 59650

Recorder: Tony J. Chamoun

Date Of Data Collection: January 6, 2014

Peabody Object No.: 16-8-30/59650.0

MNI: 1

Inventory: Cranium: Complete; Mandible: Absent; Dental: Partial

Provenience: Samanka, Peru, South America

Age/Criteria: Adult/Peabody records

Sex/Criteria: Male/Peabody records

Lesion Summary: TMJ surface pitting; Porotic hyperostosis level 2, healed (Steckel et al. 2005)

Other Observations: Lambdoidal wormian bones

Diagnosis: Joint disease—TMJD—TMJ surface pitting

Individual N3919

Recorder: Tony J. Chamoun

Date Of Data Collection: January 6, 2014

Peabody Object No.: 41-93-30/N3919.0

MNI: 1

Inventory: Cranium: Complete; Mandible: Absent; Dental: Partial

Provenience: Puncuyiluna/Ollantaytambo, Quispicanchi Province, Cuzco Region, Peru, South America

Age/Criteria: Adult/Peabody records

Sex/Criteria: Male/Peabody records

Lesion Summary: Craniofacial asymmetry; Depressed healed lesion on frontal bone; multiple healed craniofacial fractures; lytic maxillo-zygomatic lesion with spicule bone formation (lesion measures: 19.3 millimeters in height, 16.5 millimeters wide); periodontal disease

Other Observations: N/A

Diagnosis: Trauma

Individual N7945

Recorder: Tony J. Chamoun

Date Of Data Collection: January 10, 2014

Peabody Object No.: 56-42-30/N7945.0

MNI: 1

Inventory: Cranium: Complete; Mandible: Absent; Dental: Partial (multiple fractures)

Provenience: Cushashica, Cuzco Region?, Peru, South America

Age/Criteria: Adult/Peabody records

Sex/Criteria: Female/Peabody records

Lesion Summary: Probable trephination marks on the left and right parietal bones. Marks surround a large hole encompassing both the left and right parietal bones in the position of the vertex. Hole measures 48.7 millimeters along sagittal plane and 31.6 millimeters along coronal plane. Bone necrosis and sequestra throughout top surface of both parietal bones. Extensive remodeling on left parietal bone in semi-circumferential pattern—well-defined groove whose superior margins exhibit osteoclastic activity on the edges (sharply defined) of outer table. Right parietal bone also exhibits a semi-circumferential pattern of bony activity—areas of sequestra surrounded by necrotic tissue. This semi-circumferential pattern is smaller than that of the left parietal bone. These two patterns of the right and left parietal bones meet at the large hole's most anterior portion and at the large hole's most posterior portion on the sagittal suture. Posterior meeting of the two semi-circumferential patterns' posterior aspects produces the appearance of an approximately 120-degree angle outline. Postmortem? fracture on posterior aspect of right maxillary sinus—8 millimeters in width and 9 millimeters in height.

Other Observations: Weathering effects

Diagnosis: Trauma and infection

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