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Evidence for Disease and Trauma in Crania from the Late Neolithic Site of Algar do Bom Santo, Portugal.

by

Shauna Catherine McGarvey 

A thesis submitted to the Faculty of Graduate Studies and Research in partial fulfillment of the requirements for the degree of Master of Arts

Department of Anthropology

Edmonton, Alberta

Spring, 2002

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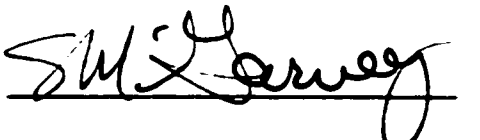

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Faculty of Graduate Studies and Research

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Dr. Owen Beattie

Dr Andrzej Weber

Dr. Jim Russell

FOR TWO WOMEN,

PATTI & HOLLY

THANK YOU

ABSTRACT

Differential diagnosis with its emphasis on description, concern for diagnostic features, consideration of the literature and process of elimination is hypothesized to be an effective approach to the analysis of commingled remains. This thesis presents the descriptive information and differential diagnoses of observed features interpreted to be lesions in crania from the Late Neolithic site of Algar do Bom Santo (ABS), Portugal (c. 4500 BP). ABS is a cave 'ossuary' with the commingled and fragmentary remains of at least 121 individuals. The cranium was chosen for analysis because it is an identifiable unit of the individual and further a number of crania with unusual changes reminiscent of trephination were noted during excavations. Nineteen individual crania (MNI 24) were examined for the purposes of this thesis; a further two crania were examined outside of the main sample. The analysis will show that differential diagnosis is a moderately successful tool for identifying and categorizing the diseases of the crania from ABS. Prevalences of specific and general disease categories were calculated from the results of the differential diagnoses and the possible interpretations regarding the ABS population are provided. Evidence for blunt force trauma, osteoarthritis, cribra orbitalia/porotic hyperostosis and infection provides a foundation for understanding the health status of the ABS population. Cutmarks and two possible trephinations on the crania from outside the sample provide evidence of cultural modification of the skeleton.

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CHAPTER 1

INTRODUCTION

Diagnosis is by far the greatest problem (in palaeopathology)

Brothwell (1961:13)

1.1 Introduction

This thesis will present the descriptive information and differential diagnoses of observed features interpreted to be lesions in crania from the Late Neolithic site of Algar do Bom Santo, Portugal. Algar do Bom Santo (ABS), is an 'ossuary' cave with the collectively deposited remains of at least 121 individuals (Duarte 1998:110)[1]. Partial excavation and survey of the site suggests that both primary and secondary mortuary behaviours were practiced resulting in considerable commingling of the skeletal material. Taphonomic processes such as intentional alteration of the mortuary space and skeletal remains, animal activity, human trampling, and tectonic activity have also resulted in fragmentation and further disassociation of the remains.

Given the commingled nature of these deposits, it was recognized that it would be virtually impossible to associate the cranial and post-cranial material. For the purposes of this study, cranial remains were chosen as the unit of analysis because it was believed to provide a manageable subset of the population through which individuals could be recognized (i.e. one cranium = one individual). Further, during the 1995 field season, unusual bone features were identified on a number of crania still in situ, including several with possible lesions exhibiting characteristics consistent with trephination. For these reasons, research was directed towards the palaeopathology of the cranial remains with the express purpose of identifying the etiology of the osseous changes and possible trephinations. The difficulty of determining the etiology/pathogenesis of abnormal bone from the sole consultation of cranial material does not preclude a methodical approach to its description and attempted identification (Waldron 1987b). Differential diagnosis with its emphasis on the descriptive process, particular concern for diagnostic features, consideration of the clinical literature and process of elimination is hypothesized to be both an effective and instructive approach to the analysis of this material (Palkovich 1987).

It is the primary goal of this thesis to address the question: what disease(s), cultural process(es), taphonomic process(es) and their interrelationships are responsible for the osseous changes observed on the cranial material from ABS. The specific research objectives include

1. Description, differential diagnosis and attempted classification of diseased crania.
2. Recognition of the presence or absence of trephination from ABS.
3. Calculation of the prevalence of identified disease categories.

---

[1] A MNI of 121 is based upon observations of the surface deposits and includes both adults and juveniles. See Duarte (1998) for the process used for estimating MNI.

Finally, the broad objective of this thesis, in which the specific goals are couched, is the evaluation of differential diagnosis and its role in assessing fragmentary and commingled skeletal remains from ABS.

The focus on cranial material was chosen with specific questions in mind. It does not represent a failure to recognize that the examination of isolated bones outside of the context of the both the skeleton and the population limits the ability of the researcher to classify the origins of the condition and make broader statements regarding the biocultural impact of disease on the population (Blau 2001:174; Rogers and Waldron 1989:624; Rothschild and Martin 1993:7; Striland 1991: 43). The particular challenge of dealing with fragmentary and commingled remains, in addition to the above mentioned goal of determining the etiology of the abnormal crania necessitates this approach. Integration of the diagnostic data into a population context will be attempted in Chapter five, although as I will discuss, the population profile appears limited. As well, further avenues of research will be suggested that integrate the cranial data back into the skeleton and the population.

1.2 Context of the Project

Cemeteries and mortuary structures including burial caves and dolmens are the most common type of archaeological site for the Portuguese Late Neolithic (c.4500 – 3200 b.p[2]) (Duarte 1993; Kalb 1996; Lubell et al. 1994; Oosterbeek 1997; Zhilao 1993; Whittle 1994). Despite this, there is a dearth of information regarding the skeletal biology and palaeopathology of Neolithic populations in Portugal. Until recently, the prevailing interest in Portuguese archaeology was the reconstruction of culture histories and artifact typologies (Lubell et al. 1994; Zilhão 1993). In addition, weak heritage legislation and poor curation resulted in the creation of enormous collections of unanalyzed human remains (Lubell et al., 1994). Among the first to systematically investigate previously and newly excavated human skeletal remains were M. Jackes, D. Lubell and C. Meiklejohn (Jackes and Lubell 1988:13; Lubell 1984:7). Their research into the biocultural implications of the Mesolithic-Neolithic transition marked a shift from a predominantly culture-history perspective, towards a largely multidisciplinary approach concerned with bioarchaeological questions (Lubell 1984; Lubell et al. 1994). However, palaeopathological research is almost non-existent in Portugal (Duarte 1993; see Riquet 1972 for the exception). In recent years, a few studies derived from the modern skeletal sample curated at Coimbra (Cunha 1995; Santos 1995; Santos and Roberts 2001) as well as two reports on trauma from the Medieval Reconquest period (Cuhan and Silva 1997; Santos et al. 1998) have been published. Pathological analysis of prehistoric material is confined to dental pathology (e.g.Duarte 1993; Frayer 1987) one report by Riquet (1972), and a few isolated references to palaeopathology within archaeological site reports (Jackes and Lubell 1992; Leitão et al. 1987). For the most part, interest in the skeletal material was directed largely at cranial morphology to demonstrate the biological connection between the modern Portuguese and prehistoric populations (Hodder 1991).

ABS is among the first of the late Neolithic mortuary sites in Portugal to be excavated with bioarchaeological questions in mind. This thesis is part of a larger research project that includes ABS and Gruta de Fetirea II in which osteoarchaeological questions are the focus. Research regarding the mortuary practices of the population and

[2] Dates are reported in this thesis as they appear in the original publications.

taphonomic processes of the site are currently in progress. As noted, with the exception of Riquet's work (1972), the work on dental pathology (Duarte 1993; Frayer 1987; Lubell et al. 1994) and information contained in a number of unpublished documents, little is known of Portuguese palaeopathology. It is hoped that this thesis will provide some of the needed groundwork for further palaeopathological research at ABS and throughout Portugal.

1.3 Palaeopathology

Palaeopathology identifies as its specific research objectives: 1) the identification of disease and trauma in human archaeological remains; 2) concern for the origin and evolution of disease; and 3) the evaluation of the impact of disease upon human populations through time and space (Miller et al. 1996). The history and development of palaeopathology as a discipline mirrors these objectives. The work of early researchers, primarily anatomists and medical doctors, was focused upon the identification and description of "interesting" and unusual cases (Aufderheide and Rodriguez-Martin 1998; Ubelaker 1982; Buikstra and Cook 1980). From this, palaeopathology developed into specific attempts to identify the origin and evolution of diseases such as syphilis and small pox (Aufderheide and Rodriguez-Martin 1998). In both instances, much of the concern was placed upon the recognition of the specific disease process in the individual. The population was of interest only in that it *contained* individuals. The evolution of processual archaeology ostensibly turned palaeopathology's focus from the identification of disease to the interpretation of its role within a past community. Diagnosis, due to its inherent inadequacies and limitations upon interpretation became passé (Waldron 1994; Buikstra and Cook 1980).

The current focus in palaeopathology of placing evidence for disease within a biocultural and populational context has not obscured the fact that the diagnosis of abnormal bone change continues as one of the discipline's most significant challenges (Ortner 1991 1994; Ubelaker 1982; Waldron 1994). Most publications and standard texts on the subject advocate a biocultural approach in which the various biological and cultural systems are integrated into a populational and environmental context, over that approach which focuses upon the individual (i.e. the more clinically based approach) (Bush and Zvelebil 1991; Meiklejohn and Zvelebil 1991; Ortner 1991). The fact remains, however, that most palaeopathologists continue to be compelled to diagnose lesions (Miller et al. 1996) at the same time as being repelled by the diagnostic inadequacies (Buikstra and Cook 1980). The problem of diagnosis and the connected problem of description continue in the literature's theoretical and methodological concerns (Ortner 1991, 1994). While some advocate the development of "population-based characterizations" through broad disease classifications (Buikstra and Ubelaker 1994:107), debates range from those who feel that they can identify the most obscure to the "diagnostic nihilists of palaeopathology" who chose to avoid the process as far as possible (Rothschild and Rothschild 1995b: 1402; Waldron 1994:39).

Ortner and Aufderheide (1991:1-2) advocate that among the major theoretical, technological, and epidemiological problems under consideration, the most urgent is a careful review of the methods of description and classification currently at use in palaeopathology. Fundamental to exploring questions regarding the biocultural implications of disease, is the careful description of the bony response to external

stressors (e.g. trauma, diet, cultural practices), and internal factors (e.g. congenital malformations, malabsorption of nutrients). A descriptive methodology with particular attention to using non-ambiguous terminology, identifying the predominant osseous activity, the patterning of lesions, the distribution within the population and, the archaeological and biocultural context, facilitates the ability of the palaeopathologist to interpret lesions at the level desired (specific or general), draw comparisons between populations and allow for individual interpretation.

Trauma, osteoarthritis, and porotic hyperostosis/cribra orbitalia benefit from being among the most commonly observed and best-documented disorders in palaeopathology (Buikstra and Ubelaker 1994; Miller et. al. 1996; Roberts and Manchester 1995; Rogers and Waldron 1994; Rogers et al. 1987; Stuart-Macadam 1992). Relative to other disease categories, the development (or attempted development) of operational criteria and standardized descriptive methodologies has facilitated their identification, enhanced biocultural interpretation and improved the applicability of cross-populational comparisons (Frayer 1997; Lovell 1997a; Roberts 1991; Rogers and Waldron 1995; Stuart Macadam 1992; Waldron 1994). Biocultural interpretation of other disorders, such as neoplasia, infectious disease and congenital/ developmental abnormalities lag far behind.

Our ability to diagnose disease in antiquity is limited to the small fraction of diseases and traumatic conditions that manifest osseous change, and the even smaller percentage of those that will be severe or chronic enough to exhibit observable differences. In addition, bone can only change in a limited number of ways, therefore, most diseases that affect the skeleton present non-specific, non-diagnostic lesions (Wood et al. 1992:344). Finally, the affects of taphonomy, burial environment and excavation can further obfuscate the bony change. Therefore, the palaeopathologist must maintain realistic expectations, and anticipate that it will not always be able to identify the etiology of a specific lesion (Aufderheide and Rodriguez-Martin 1998:12; Ortner 1991).

While many researchers restrain from presenting definitive diagnoses, the identification/classification of disease must be attempted before the integration of the data into a larger context can proceed (Waldron 1994:28). The differential diagnosis is a tool used in modern clinical and palaeopathological studies to interpret descriptive data (symptoms or bone changes), and establish a list of alternative conditions consistent with the observed information. Through the process of gradual elimination, the list of possibilities is narrowed until, theoretically, a diagnosis is achieved (Roberts and Manchester 1994: 6; Waldron 1994:28-29). In palaeopathological studies, the identification of a list of diagnostic possibilities from which there are several competing conclusions may be the final result and the only realistic conclusion.

1.4 Overview

The following chapters provide the background information and the results of the palaeopathological analysis performed in the summer of 1997. Chapter 2 provides description of ABS and the known information regarding burial practices within the context of the Late Neolithic of the Portuguese Estremadura. Chapter 3 describes the skeletal material used in this study and outlines the methods of description, differential diagnosis and calculations of disease prevalence. Chapter 4 provides the descriptions of the individual specimens and their differential diagnosis. Chapter 5 is a broad review of

the biological and cultural processes observed on the ABS crania, as well as their possible interpretations. Chapter 6 will evaluate the role of differential diagnosis in the context of this thesis and present the conclusions. Appendix 2 is a comprehensive, though succinct review of the major biological and cultural processes that affect the skull. Emphasis is on the type of osseous reaction that is observed on dry bone.

CHAPTER 2

CULTURE HISTORY & BURIAL CONTEXT

2.1 Introduction

Algar do Bom Santo is a Late Neolithic cave cemetery located in the Portuguese Estremadura on the eastern slope of Montejunto (Figure 2.1; Plate 1). Discovered by spelunkers in late 1993, the extensive cave system contains the remains of over 121 individuals (Duarte 1998: 110). Four subsequent field seasons in 1994, 1995, 1997 and 2001[1] revealed one of the most important mortuary caves in Western Europe.

2.2 Paleoenvironmental Setting

The Portuguese Estremadura is a large limestone massif that occupies most of central Portugal, bounded by the Mondego River basin to the north, mountains to the east, the lower Tagus to the south and the Atlantic Ocean to the west (Marks et al. 1994:55). According to Ribeiro and colleagues (cited in Marks et al. 1994:55), climatic, eustatic, karstic and tectonic processes have had a considerable, if poorly understood impact on the landscape. The Estremadura contains numerous karstic caves and is known for a large number of mortuary sites (Oosterbeek 1997).

Montejunto is located 80 km north of Lisbon. ABS sits on the southern exposure of the mountain at approximately 350 m above sea level (Duarte 1998:107) (Figure 2.1). The site is not directly associated with the Tagus or its primary estuaries. Although Montejunto is presently 20 km east (as the crow flies) of the Atlantic coast, data from other areas of Portugal suggest significant reductions in coastline over the last 10 000 years.

The environmental setting of present-day central Portugal largely reflects the major environmental shifts of the early and middle Holocene (Geddes 1986:763; Lubell and Jackes 1988:235). A sediment core taken north of the Tagus River at Serra da Estrela (elevation 1600m) reflects the last 9000 years of palaeoenvironmental history. It provides evidence for pine forests in its earliest segments (Janssen and Woldringh 1981; Van Den Brink and Janssen 1985); pine forests are generally associated with cool, moist climates typical to the end of the last glacial period and early Holocene. By 8300 bp, the pine is replaced by predominantly oak and birch vegetation, indicating open woodlands and a drier, warmer climate (Janssen and Woldringh 1981). The core indicates that this type of vegetation continues up until 2700 bp, at which point the effects of agricultural and pastoral activities can be identified (Van Den Brink and Janssen 1985; Janssen and Woldringh 1981). Another core, taken from the Tagus river basin provides further evidence for a decrease in arboreal pollen between the period of 6500 BP to 2700 BP. In addition, the effects of cultivation and animal domestication are evident at 2700 BP. The Flandrian transgression resulted in the Atlantic Ocean retreating approximately 30km over the last 10 000 years (Lubell and Jackes 1987:2). It is hypothesized that the marine transgression precipitated the initial change from a cool, moist climate, to dry, open scrub vegetation that was further enhanced by the escalation of agricultural activities (Geddes 1986). According to Marks et al. (1994:56), the Estremadura of today is "ecologically balanced" between Mediterranean and Atlantic plant species and demonstrates a "complex mosaic of environmental niches".

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[1] Skeletal data from the 1997 and 2001 field seasons are not included in this thesis.

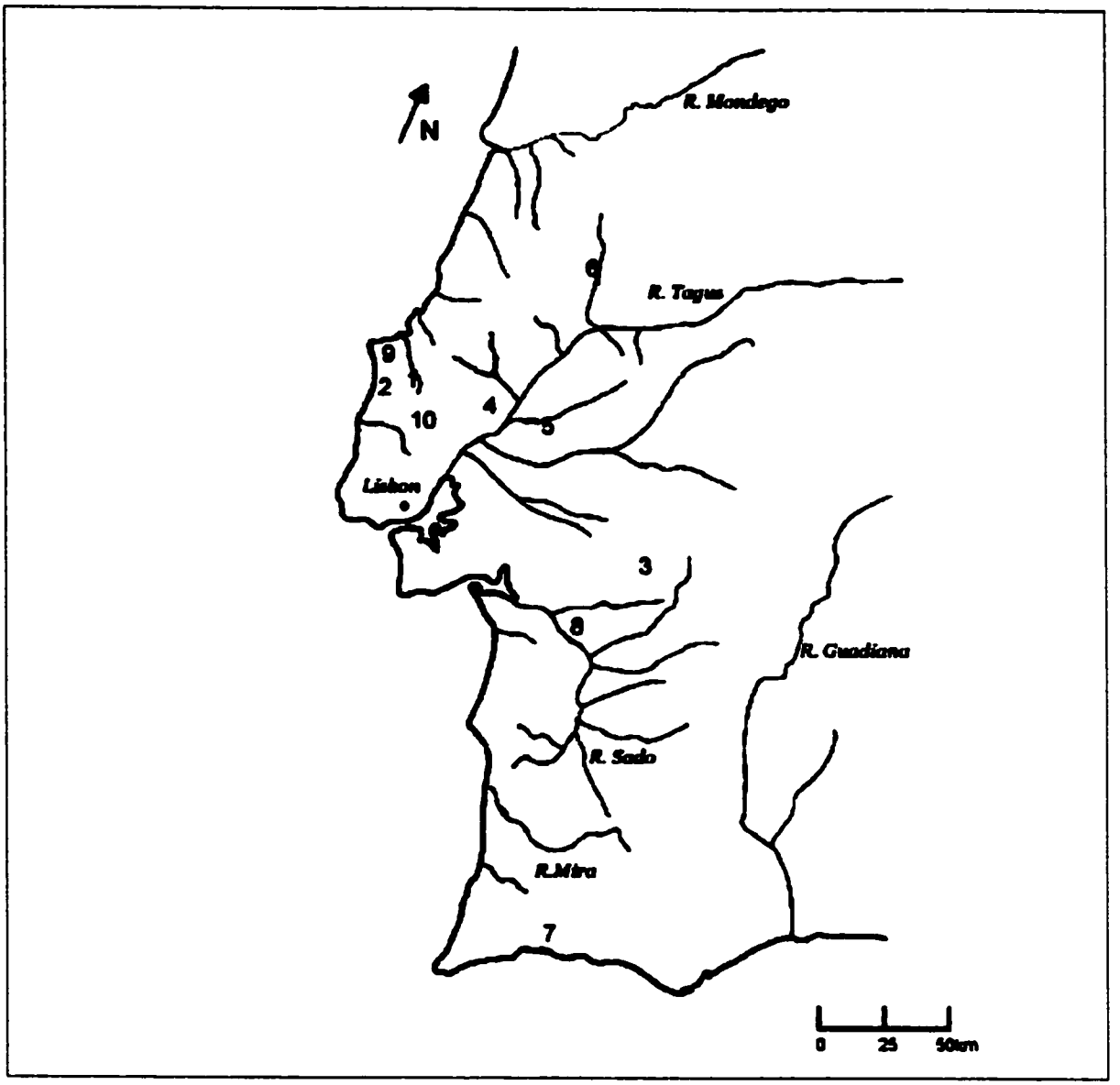

Figure 2.1 Archaeological sites mentioned in text (adapted from Duarte, 1997).
1. Algar do Bom Santo; 2. Gruta da Feteira; 3. Gruta do Escoural; 4. Gruta do Lugar do Cantos; 5. Muge shellmiddens; 6. Gruta do Caldeirão; 7. Algarão da Goldra; 8. Sado shellmiddens; 9. Grotte da Furninha; 10. Zambujal.

2.3 Culture History

Recent research into the Holocene history of Portugal has largely focused on the Mesolithic-Neolithic transition. The significance of the shift from subsistence foraging to agricultural and pastoral life ways has engaged the interest of academics for the last thirty years (Binford 1968; Cohen and Armelagos 1984; Larsen 1995; Martin et al. 1985; Meikeljohn and Zvelebil 1991). The highly visible Mesolithic shell middens and the Early Neolithic open-air cave and rock shelter sites have resulted in a *relatively* clearer

picture of the nature of the communities that straddle this shift[2]. This compares with the contrasting dearth of Middle to Late Neolithic open-air settlement sites. The predominance of collective cave cemeteries and megalithic monuments and the associated reliance on artifacts from these periods has contributed to the biased focus of prehistoric research in Portugal (Barnett 1992; Lubell and Jackes 1988; Lubell et al. 1994). While the lack of open-air sites such as habitation or resource extraction sites limits our palaeoeconomic understanding of the Late Neolithic to a significant degree, the opportunity to explore the biological and social aspects of ABS is exceptional. The bioarchaeological focus of this thesis and other research at ABS will contribute to our understanding of this period beyond simple artifact typologies and culture histories.

While there is literature on the Mesolithic and Neolithic in Portugal, it is almost exclusively published in Portuguese and therefore not readily accessible. Further, it is not the intent of this chapter to provide an exhaustive review of Portuguese archaeology; rather, what follows should serve to place ABS within its prehistoric context. Issues surrounding the introduction of the Neolithic are highly complex and much debated, not only in Portugal but also throughout the Mediterranean and Europe. Zilhão's (1993), detailed analysis of the stratigraphic, radiocarbon and taphonomic data from Portugal and the western Mediterranean within the context of several models is the foundation for the following discussion.

2.3.1 Mesolithic (~ 10 000 BP – 6800BP)

The Mesolithic period in Portugal dates from the end of the last glacial period, c. 10 000 years ago, until the gradual and regional appearance of a Neolithic way of life (agriculture and domesticated species) after 6800 BP. Shell-middens are among the most excavated site types for the Mesolithic Period (Barrett 1995; Zilhão 1993). Early and Middle Mesolithic middens comprised primarily of shellfish and other aquatic resources are concentrated along the coast in what appear to be temporary. In contrast, the Late Mesolithic (7500-6000 BP) sites of Portugal are clustered along the Tagus, Mira and Sado estuaries, suggesting a pattern of prolonged settlement in estuarine environments (Zilhão 1993). Throughout the Mesolithic, primary, individual burials within the midden or occupation site appear to be the standard mortuary practice (Lubell and Jackes 1988:245).

The Muge shell-middens (Moita do Sebastião, Cabeço da Arruda, Cabeço da Amoreira) located in central Portugal, along the Tagus system, indicate possible year-round occupation with emphasis on certain seasonal activities. Large mounds, prolonged habitation features (pits, post holes), and the deliberate inclusion of hundreds of human burials in the shell middens seem to confirm that the sites were occupied over extended periods of time (Lubell and Jackes 1988; Whittle 1996; Zilhão 1993). Specialized camps, contemporaneous with the larger sites suggest that regional or seasonal resource extraction occurred in the areas surrounding the Tagus system. Faunal data from the Muge sites suggest that the extraction of estuarine resources was the primary mode of subsistence, while stable isotope analysis of human skeletal remains and the presence of

[2]I use the term "relatively" because while the Mesolithic-Neolithic transition is better understood than the following periods, there is still much that is not known. Until recently, research focused predominantly on artifact typologies and culture-histories. For a good example of this increasingly clearer picture please consult: Lubell , Jackes and Meiklejohn (1985); Lubell and Jackes (1988), Lubell et al. (1994).

terrestrial fauna indicate a diet that included significant amounts of land-based resources (Lubell and Jackes 1988:235; Lubell et al. 1994). According to Zilhão (1993:13), similar patterns emerge from the sites associated with the Mira and Sado river systems, suggesting the possibility of three separate social groups with a common adaptation.

2.3.2 Early Neolithic (ca. 6800 BP)

Current thinking suggests that, similar to sites in Spain and France, the Neolithic in Portugal begins c. 6800 - 6200 BP (Barnett 1992: 83; Zilhão, 1993:5-6). Most archaeologists agree that the Early Neolithic in central Portugal can be identified by the presence of "classic cardial wares" (Zilhão 1993:28, Barnett 1990:859), domesticated animals (primarily ovicaprids) and ground stone artifacts (Gilman 1992:296). While open-air habitation (e.g. Vale Pincel and Vale Vistoso) and resource extraction sites are known for the Early Neolithic in Portugal, cave sites used for both short-term habitation and mortuary purposes are the more rigorously excavated and published (e.g. Gruta do Caldeirão) (Gilman 1992; Lubell 1988). Primary, individual interments in caves or rock shelters appear to be the characteristic burial pattern for the Early Neolithic (Oosterbeek 1997:71).

The first Early Neolithic communities in Portugal are found in the northern Estremadura, between and along the major river systems and appear to be contemporaneous with Mesolithic communities in southern Portugal (Zilhão 1993:36). The archaeological evidence suggests that these "Neolithic enclaves" (Zilhão 1993:27) were established in areas not occupied by forager communities. According to Zilhão, (1993:52) this, in addition to the relatively simultaneous appearance of a Neolithic industry across the western Mediterranean and central Portugal, suggests that the immigration of "small Neolithic seafaring groups" is responsible for the introduction of agro-pastoralism to the western Mediterranean[3]. Eventually, the established Neolithic populations expanded south, and then north replacing and absorbing extant hunter-gather groups. While the "Neolithic Package" is assumed to have included grains, the lack of habitation sites renders them invisible, resulting in a poorly understood agricultural component. Evidence for domesticated species linked to herding suggests that these populations were nomadic or semi-nomadic pastoralists. The Neolithic herders of central Portugal were likely more mobile than the relatively settled hunter-gatherers of the Late Mesolithic (Whittle 1996:304).

Gruta do Caldeirão, a small cave site in central Portugal dated c. 6700-5700 BP is one of the most important Early Neolithic sites in Portugal. While primarily a burial site, the presence of wild and domesticated fauna is possibly indicative of episodic, seasonal use of the cave (Zilhão 1993:26). Cardial pottery in the Early Neolithic components may be as old as 6800 BP and is definitely older than the averaged date of 6200 BP (human bone) that is usually associated with Caldeirão. According to Lubell and Jackes, (1988: 236) the faunal assemblage from the early Neolithic components of Caldeirão contains primarily wild species with a few domestic elements. Similar to ABS, archaeologists have not located Gruta do Caldeirão's associated settlement site.

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[3] As opposed to the "wave of advancement" or "indigenous development" models. Chapman (1985:153) argues that ceramic and lithic evidence suggest the continuity of Iberian populations from the Epipalaeolithic through to the Neolithic. Jackes et. al (1997a,b) feel that the biological evidence (DNA, skeletal and palaeodemographic) implies continuity between the Mesolithic and Neolithic populations.

Evidence for Neolithic subsistence strategies is limited to a very small collection of habitation sites (see Lubell et al. 1994:207). Stable isotope analysis from Gruta do Caldeirão and several other sites suggests that Early Neolithic subsistence underwent a substantive shift away from a diet based on aquatic and terrestrial resources towards a diet based primarily on resources of terrestrial origin (Lubell and Jackes 1988; Lubell et al. 1994). Faunal studies from Neolithic and Chalcolithic sites suggest that while domesticated species (pig, sheep/goat) make up an increasing proportion of the diet, the consumption of marine resources (*Mytilus* and *Patella*) continued.

2.3.3 Middle Neolithic (ca. 6000 BP)

The evidence from the western Mediterranean suggests that after the Early Neolithic, agriculture became widespread and intensified. According to Geddes (1986), there is a diversification of cultivated plant species and an increase in animal husbandry. In Portugal and the western Mediterranean, the widespread use of cardial impressed wares declines in the Middle Neolithic. Gradually, pottery styles became localized and increasingly undecorated (Barnett 1990; Guilaine 1979).

Individual internment in caves and rock shelters continued as a popular method of mortuary practice during the Middle Neolithic. One of the better-known sites from central Portugal is Gruta do Cadaval (5350± 50 BP - 5160±50 BP). The two-chambered cave includes the buried remains of two individuals (Oosterbeek 1997:71). Each of the burials made use of existing topographic features and were associated with grave goods, including pottery lithics and bone tools. Another site, Algarão da Goldra (ca 5000 BP) located in southern Portugal contained the disarticulated remains of seven individuals. The association of ash and charcoal, bone and stone artifacts, ceramic sherds and a complete incised bowl suggest that the human remains were included within the habitation site (Straus 1997:5). During the Middle Neolithic, megalithic monuments that include collective burials begin to appear inland from the coast. According to Oosterbeek (1997), the artifact assemblages from these sites are different from that of the individual cave burials.

2.3.4 Late Neolithic (ca. 4500 – 3200 BP)

Our knowledge of the Late Neolithic in the Iberian Peninsula is derived primarily from mortuary contexts in chambered tombs[4] and natural caves in Portugal and Catalonia (Gilman 1992:296). In light of the limited number of open-air sites dated to the Middle and Late Neolithic, chambered tombs and caves [5] are the most "tangible remains of Neolithic activity" (Oosterbeek 1997:70). The dearth of Late Neolithic settlement sites is attributed to a number of factors. First, caves appear to be the primary location of choice for funerary purposes. This combined with the high visibility of cave and rock shelters compared to that of open-air sites results in their relative dominance in the archaeological record (Straus 1997:2). Second, the mobile settlement patterns of Late Neolithic populations would not have left sizable deposits or significant structures on the landscape (Gilman 1992:297). Further, intense erosion, poor preservation of the grains, the lack of

[4] There is much debate regarding the origins and development of the chambered tombs. Megalithic cists, passage tombs, tholoi and rock-cut tombs appear to have been in used in Portugal from the Neolithic to the Bronze Age (Gilman 1992). See Chapman (1981) for an in-depth discussion on the evolution and distribution of these structures.
[5] See Straus (1997) and Oosterbeek (1997) for a discussion on the use(s) of caves in Portugal and the Iberian Peninsula.

systematic survey (Chapman 1985:164) and the accumulated destruction of settlement sites from centuries of agricultural production have inhibited our recognition of sites from this period (Oosterbeek 1997:70; Zilhão 1993; Chapman 1985). As a result, our understanding of palaeoeconomics of the Late Neolithic is limited (Oosterbeek 1997:70).

Similar to the previous period, the Neolithic package continues through the Late Neolithic with cardial pottery replaced by undecorated wares. In the burial caves of the Nabão Valley, regular artifact assemblages appear uniform in their composition, while there are differences in the assemblages of 'prestige' goods that may reflect status differences and social differentiation (Oosterbeek 1997:74). For example, the Gruta do Ossos assemblage contains flutes and Atlantic shell; Gruta do Morgado contains red deer bones while textile implements are exclusive to Gruta do Cadaval.

Collective cave burials became widespread in Portugal in the Late Neolithic (Oosterbeek 1997; Zilhão 1984). According to Oosterbeek (1997:74) in the Nabão Valley, burial caves tended to be clustered in small areas known as "*necropoleis*". This represents a change from earlier periods when there was no apparent pattern to the selection of burial caves. He hypothesizes that most of the communities' investment was placed on the necropoleis, while "settlements were small, (and) in the open air" (brackets mine) and that the community could "... move easily, within territories in which boundaries were defined by the necropolis" (1997: 77).

According to Lubell and Jackes the only reliable dietary evidence for the Neolithic comes from the previously discussed faunal and stable isotope data from the Early Neolithic site of Caldeirão (1988:236). Our understanding of Late Neolithic subsistence strategies is derived from the extension of the data from Early Neolithic and Chalcolithic sites. Faunal and stable isotope analysis from these sites suggests that subsistence appears to be largely based on terrestrial resources with marine resources constituting a small proportion of the diet (Lubell et al. 1994:208). Further research, most likely stable isotope analysis, is required to confirm this pattern for the Late Neolithic in Portugal.

2.3.5 Chalcolithic[5]

Copper and copper metallurgy in association with fortified villages and increasing social stratification provides a basic characterization of the Chalcolithic (Castro 1995:33; Chapman, 1986:156). The presence of well-excavated and highly visible fortified Chalcolithic villages such as Zambujal and Villa Nova Sao Pedro provide the basis for our current understanding of this period in Portugal. Copper was likely used as an item of prestige, decoration, and trade; there is no data to suggest that it replaced stone tools.

According to Gilman (1992: 298), there is an abundance of "deeply stratified, often fortified settlements" centered upon intensive agricultural exploitation and long-term settlement in southeast Spain and central Portugal (Gilman 1992:298). In the Tagus region, the archaeological assemblage from Zambujal, a Chalcolithic hill fort, includes variety of luxury goods including metal implements, fine pottery (beakers) and ivory combs as well as two chronologically distinct pottery types: the Vila Nova de São Pedro (VNSP) I (a patterned-burnished *copos*) and VNSP II, (Maritime Beaker). The presence

[5] The beginning of the Chalcolithic is dated to approximately 3000 BC in Portugal and Western Europe (Gilman 1992:298). The transition from Neolithic to Chalcolithic is not as dramatic as the transition from Mesolithic to Neolithic.

of luxuary goods suggests increasing social stratification and inequality. These items, in addition to the replacement of geometric points with bifacially flaked points further define the Chalcolithic. The use of chambered tombs and burial caves continue throughout the Chalcolithic. There is an eventual shift towards individual interments and away from collective burials (Geddes 1986; Oosterbeek 1997).

Faunal evidence from Lower and Middle Chalcolithic levels of Rotura, suggests that domesticated animals made up 64% of the vertebrate assemblage (Gautier and Lentacker, 1985, cited in Lubell and Jackes, 1988:236); and marine resources also apparently comprised a significant proportion of the diet. At Zambujal, a later Chalcolithic site, domesticated animals (suids) made up 87% of the vertebrate remains, while wild deer and marine mollusks provided the remainder (Dreisch and Boessneck, 1976: cited in Lubell and Jackes, 1988:236). The evidence suggests that while domesticated animals became increasingly important, wild and marine resources continued to play a significant role in the diets of Chalcolithic communities.

2.3.6 Summary

Our understanding of the Portuguese Late Neolithic is very limited. Evidence based on a small number of mortuary sites and an extension of analogies from the Early Neolithic and Chalcolithic suggests that while basic material cultural and dietary patterns continue (Lubell et. al. 1994), there is hypothesized intensification of agricultural and pastoral activities (Geddes 1986). The construction of megaliths and the use of caves/rockshelters as collective burials appear to reach their zenith in the Late Neolithic.

Mortuary complexes like ABS are the common type of archaeological sites for the Late Neolithic in Portugal (Jackes 1988; Lubell et. al. 1994). The number of mortuary sites notwithstanding, our understanding of the bioarchaeology of Neolithic populations is limited. Despite the availability of skeletal remains, little is known about the health status and subsistence strategies of the Late Neolithic populations of Portugal.

2.4 Algar do Bom Santo

Algar do Bom Santo is a limestone cave, comprised of 12 discrete rooms (Table 2.1). While our understanding of the cave structure and research into the site formation processes are currently under consideration, the cave appears to have formed largely by tectonic activity and geomorphic processes (Duarte 1998). The prehistoric population that used the site appears to be responsible for the construction of smaller spaces within the cave. They created discrete mortuary "niches" by modifying existing topographic structures such as (Sala B) (Duarte 1998). Almost no sedimentary deposition has resulted in mortuary remains close to, or on the surface (Plate 2). Mortuary deposits cover 285 m^2 of the cave's surface. The 1994, 1995 and 1997 excavations focused on Sala A and Sala B, the two contiguous rooms immediately accessible via the entrance (Figure 2.2). Mortuary deposits in Sala A, the larger of the two rooms, measure 36 m^2 while the deposits in Sala B measure 16 m^2. Of these two rooms a total of 19 m^2 were excavated over the three years. Sala C, directly below Sala A and B, provides access to the remaining nine rooms. The lower rooms of the cave were mapped and extensively surveyed during the previous excavations, and appear, at least superficially, to demonstrate similar patterns of deposition and distribution (Figure 2.3) (Duarte 1997).

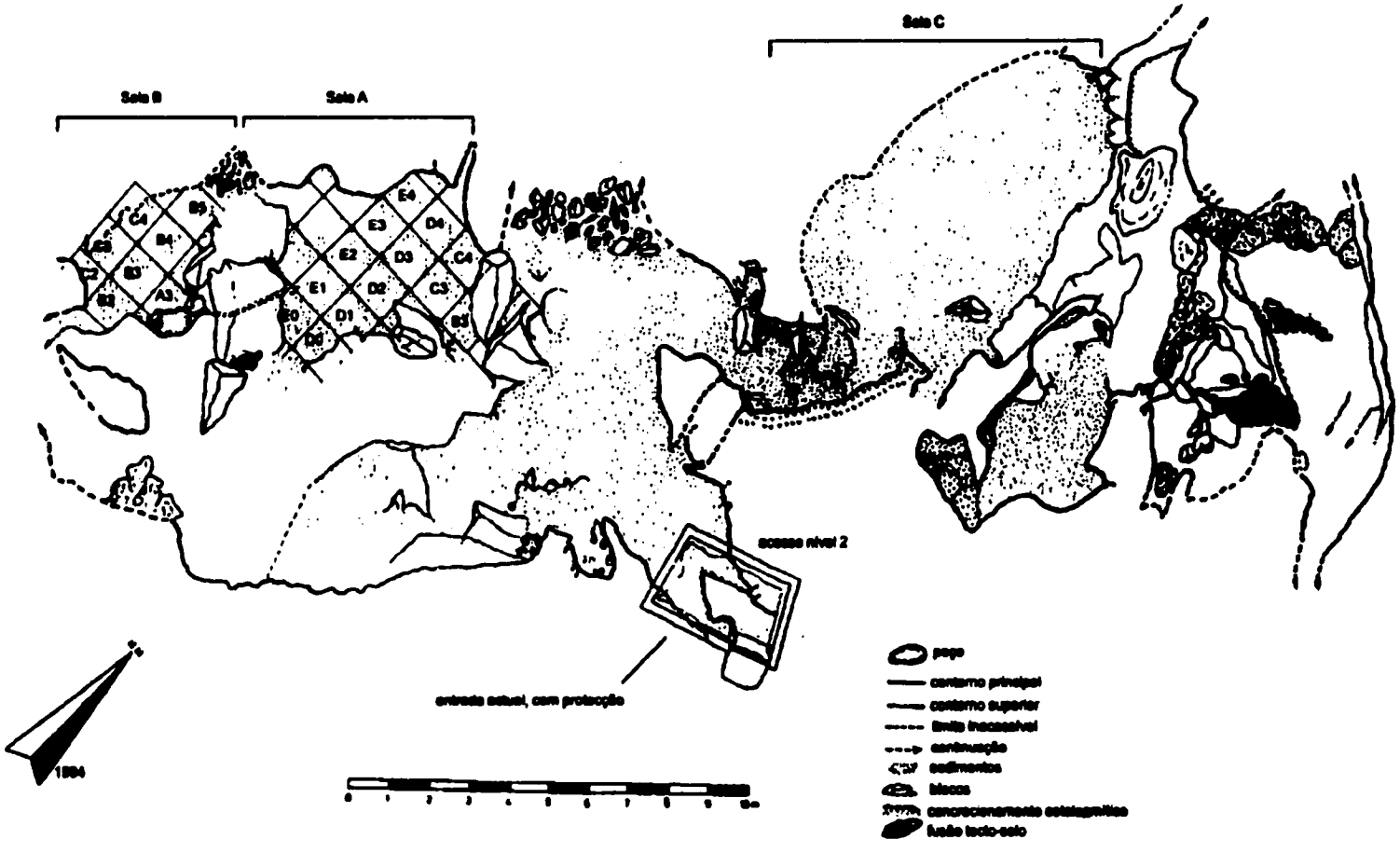

Figure. 2.2 Algar do Bom Santo, Floor plan of the upper level of the cave (adapted from the original from AESDA).

Table 2.1 Superficial funerary deposits, for Algar do Bom Santo

Room	Area in m^2
Sala A (Sete Cabeças)	36
Sala B (Concha)	16
Sala C (das Pegadas)	41
Area das Prateleiras	18
Sala das Pulseiras	42
Sala dos Ossos Queimados	25
Passagem	16
Sala da Caçador	26
Sob os ossos queimados	12
Sala Gémeas	47
Estreito	6
Total Area	285

Adapted from Duarte (1998:109).

Six radiocarbon dates were obtained from human bone samples from Sala's A, B, and C (Table 2.2). The mean age for all the samples from ABS is 4573 years BP. For Salas A and B the averaged age is 4531 years BP, while the averaged age for each room is 4445 and 4588 years BP respectively. According to Monge Soares, (cited in Duarte 1998:113) there is no significant difference between the dates for Sala A and B. Therefore, until further evidence and examination of the relationship between the radiocarbon dates, stratigraphy, taphonomy and material culture, these two rooms are considered archaeologically contemporaneous. Excavation of Sala A and B have not revealed deposits dating to earlier or later periods of occupation. The initial observations and radiocarbon dates suggest that the cave may have been used for approximately 500 years. The cave may have been visited a final time during the Medieval Period – closing some point after that.

There is an abundance of artifacts associated with the human remains at ABS. Observation of the surface deposits as well as the excavation of Sala A and B revealed approximately a 1:20 relationship between artifacts and human remains (Duarte personal communication). The material culture at ABS conforms to that found at other Late Neolithic cemeteries in Portugal. Flint blades and geometric blade tools are present, while polished stone axes and adzes made from anphibolite and schist are the most common artifacts within the mortuary complex (Duarte 1997:26; Plate 2). Items of personal adornment are also found in ABS. Stone and shell beads as well as 30 perforated *Trivia* shells were recovered from the site (Duarte 1997). Bracelets made from *Glycymeris* shell adorn the skeletal remains of several individuals still *in situ* (Duarte 1998). Pottery is an uncommon finding at ABS. A complete black, non-cardial (non-impressed) pot was recovered in the 1995 excavation. Other findings include small fragments of the non-impressed ware. Grave goods do not appear to be distributed within the cave as prestige items to distinguish particular individuals. While ABS dates to the end of the Late Neolithic/Early Chalcolithic, the artifact assemblage thus far recovered from the site is clearly Late Neolithic in nature. There is no evidence for copper metallurgy as would be expected in a burial of Chalcolithic origin. Finally, as stated above, cave cemeteries are frequently associated with the Neolithic in Portugal, particularly the Late Neolithic.

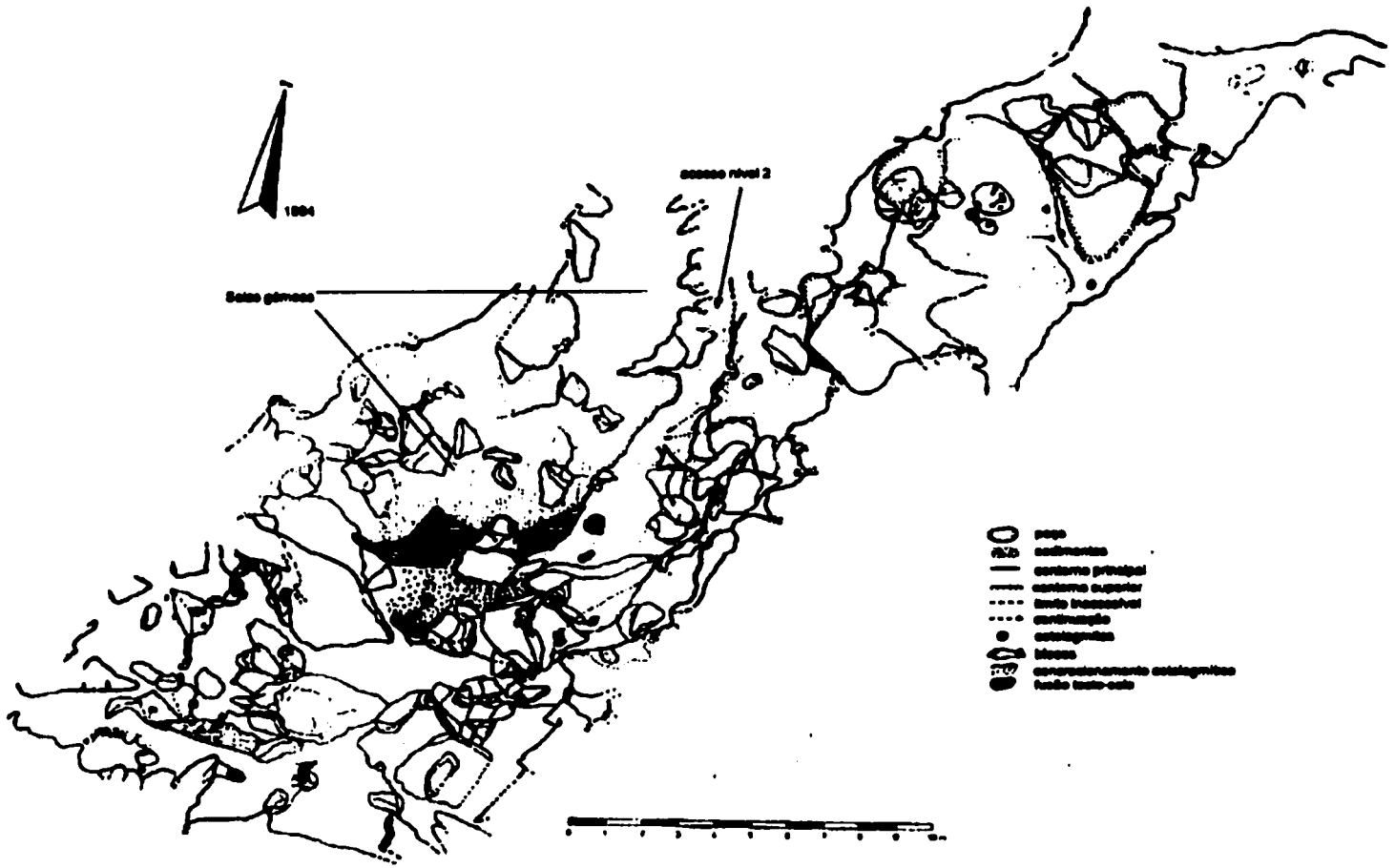

Figure 2.3 Algar do Bom Santo, Floor plan of the lower levels of the cave (adapted from the original from AESDA).

Table 2.2 Radiocarbon dates from Sala A, B and C, Algar do Bom Santo. All dates derived from human bone samples.

Sample Number	Provenance	Bone	*dC13* (‰)	Date BP	Calibrated BC	*Cal BC* (1 sigma)	*Cal BC* (2 sigma)
ICEN-1181	Surface: Sala A	Femur	-21.8	4030 ± 80	2563	2910-2140	3350-1750
OxA-5513	Sala A.2309	Femur	-19.6	4860 ± 100	3647	3760-3740	3930-3870
						3720-3620	3810-3490
OxA-5511	Surface: Sala B.1	Femur	-19.6	4705 ± 65	3505	3620-3580	3640-3350
					3412	3540-3370	
OxA-5512	Sala B.C2.27	Femur	-19.6	4630 ± 60	3367	3500-3420	3620-3590
						3380-3350	3530-3300
							3230-3180
							3170-3110
Beta-120047	Sala B.B3.394	Sternum	-20.7	4430 ± 50	3045	3100-2930	3325-2910
Beta 120048	Sala C	Cranium	-19.6	4780 ± 50	3625	3640-3515	3660-3495
					3565		3445-3380
					3540		

Adapted from Duarte (1998:113).

Questions regarding the highly complex nature of the mortuary behaviour and taphonomy are currently under exploration. Excavations in 1995 revealed initial evidence for both primary and secondary burial practices that clearly present difficulties for the analysis of the skeletal remains. Commingling of multiple individuals is evident in most instances. This makes the identification of individuals and the association of cranial and post-cranial remains difficult, if not impossible. The particular ramifications this has for palaeopathological and taphonomic research will be discussed further in Chapter 3, *Materials and Methods* and Chapter 5, *Results and Discussion.*

In some rooms, the deliberate sorting of skeletal elements is obvious. For example, in Sala C a discrete nucleus of approximately five crania can be distinguished from an adjacent focus of lower limb elements (Plate 3). In other areas of the cave the pattern of distribution and the subsequent effects of taphonomy and burial environment are ambiguous. According to Duarte (personal communication) both Sala A and B present evidence for secondary deposition. ABS is clearly a collective cemetery containing multiple skeletons that were interned as a group rather than as individuals; however, at this stage it is difficult to determine the relationship between the space, specific individuals, their parts and the collective.

2.4.1. Preservation

Caves can be the site of much taphonomic activity (human and animal) and geologic disturbance. In addition to using caves as living sites or resting places for the dead, humans have used caves for dumping garbage, stabling livestock, shelter, resource extraction, and recreation (Straus 1997). The deposition of archaeological material does not preclude use at a later date, and archaeologists must be careful when interpreting evidence of human activity. Animals also use caves as shelters or to den or hibernate. Their activities may disturb sedimentary deposits, introduce other animal remains or modify existing human remains. Most limestone caves are active karst systems subject to a host of ongoing changes. Water may disturb or deposit sediments, alter the cave environment and modify human material. Caves are also susceptible to rock falls that

may alter the internal structure of the cave and the relationship of archaeological deposits, disturb sediments and crush exposed material (Straus 1997:2).

Differential preservation of osteological materials is also evident throughout the mortuary complex at ABS. Secondary deposition is frequently associated with intentional disarticulation, breakage and defleshing which may result in a variety features of that require careful assessment (Hurlbut 2000:11). As stated previously, we do not yet understand the influence of the mortuary behavior on the skeletal material from ABS. In some instances, skeletal elements are intact with the cortical surfaces exceptionally well preserved, while in others there is severe fragmentation and considerable destruction of the bone. Calcite ($CaCO_3$) deposits are a significant feature in limestone caves, and ABS is no exception. Calcite deposits on bone ranges from small accumulations on individual elements to massive flows covering numerous bones. Diagenetic change at the mineral level of bone is very likely (Jackes 1988; Jackes et al. 2001; Karkanas 2000). At present, no active hydrological features exist in the cave; however, it is a moist environment with standing water in some locations and consistently high humidity. As a result, some bones are very soft and particularly susceptible to breakage during recovery, curation and analysis. Differential preservation and how it relates to various regions of the cave, geomorphic activity, specific tectonic events animal and insect activity and human alteration all remain to be explored.

2.5 Summary

A full understanding of the taphonomic processes of the site, mortuary behaviours of the community associated with ABS, and the biological profile of those interred awaits investigation. Further, little is known about the life ways of this Late Neolithic population. There are no known settlement sites in the region directly or indirectly associated with the cave. Two other caves used exclusively as "ossuaries" have been identified for Montejunto: Gruta das Fontainhas (4170 ±60 years BP) and Roche Forte II (4480 ±60 years BP) (Lubell and Jackes 1988: 234; 1994:202-203). Lubell and Jackes explain that Fontainhas was investigated in the late 19th-century and Roche Forte was tested in the mid-1980's. The skeletal materials were not examined for this project nor were further references to these sites found during the course of this research (Lubell and Jackes 1994:202-203).

The radiocarbon dates, Neolithic assemblage, domesticated caprid bone and the collective nature of the burial indicate that the population associated with ABS was of Late Neolithic origin. The bioarchaeological research from ABS will constitute an important source of information for furthering our understanding of this enigmatic period. Not only is palaeopathology a source of data for the health status of a population, it can provide a valuable window into past economic and cultural systems.

CHAPTER 3

MATERIALS AND METHODS

3.1 Introduction

The nature of the ABS cranial sample and methods of analysis are presented in this chapter. The materials section considers the calculation of the minimum number of individuals (MNI), sample size and composition, preservation and fragmentation, and the unit of analysis. The aspects of the methodology to be discussed in this chapter include, the methods of osteobiographical and palaeopathological analysis relevant to this study, the description of the process of differential diagnosis, and the use of descriptive statistics to reflect disease prevalence.

3.2 Materials

3.2.1 Nature of the Sample

As stated previously, ABS is a collective cave cemetery. In all likelihood, remains were deposited secondary to a primary burial or process at some other location. While there is no dearth of skeletal material from ABS, the precise nature of the mortuary practices and burial context is currently under consideration; therefore, an understanding of the burial (spatial) relationship of the commingled remains could not be utilized to decipher individual relationships. The inability to associate cranial with post-cranial remains presents two specific problems that must be considered. First, there are difficulties in reconstructing the palaeodemographic profile because age and sex estimates rely on the holistic assessment of cranial and post-cranial remains (Jackes 1988). Second, there are difficulties recognizing the abnormal processes because most diseases are not normally restricted to a single part of the skeleton nor are they uniformly distributed along the skeleton (Blau 2001; Ortner and Putschar 1984; Rogers and Waldron 1987; Rothschild and Martin 1993; Waldron 1994). The diagnosis of particular disease processes often relies upon evidence from diverse parts of the skeleton (Roberts and Manchester 1995); diagnostic changes in the post-cranial skeleton may elucidate ambiguous changes on the skull. These challenges were tempered with the realization that the analysis of all the excavated cranial and post-cranial material was too ambitious given the time and funding restrictions. Finally, in order to answer the questions regarding the etiology of the abnormal crania, it was decided to restrict the analysis to the cranial material. The potential biases generated by this approach will be considered in Chapter 5.

Calculating sample size from commingled remains is a difficult task. Waldron (1994:55) recommends calculating a minimum number of individuals (MNI) based on the most frequently occurring anatomical element. Using MNI and frequency of skeletal part methods adapted from Lyman (1994:101, 240-245), a total of 19 points from the left and right parietal bones, left and right temporal bones, frontal bone, occipital bone and mandible were counted to obtain the number of the most frequent anatomical feature. A MNI of 24 adults and seven juveniles was obtained from the left petrous portion of the temporal bone. Gregg and Gregg (1987:20), in their analysis of the Crow Creek Massacre, South Dakota, also found that the petrous portion was the most accurate index of population size in a commingled sample. According to Waldron (1994:55), although

MNI is a good indication of the size of the excavated population, it cannot be used as the denominator for calculating prevalence. He recommends using the number of all of one bone or the number of all of one joint in calculating prevalence. For this study, the articulated and associated bones of the cranium will represent the individual and provide the denominator for most calculations of prevalence.

For the purposes of this study, the cranium was considered the identifiable unit of the individual[1]. Complete crania have often been the focus of palaeopathological analyses where remains are commingled or poorly preserved (Papathanasiou et al. 2000; Webb 1995), preferentially curated (Bartel 1993; Walker 1989) or where there is a specific question to address from the crania (Jurmain and Bellifemine 1997). Not anticipated was the incomplete and fragmentary nature of much of the cranial material. As will be discussed below, this reality provided a particular challenge to the quantification process and the already difficult task of age and sex determination for the ABS sample.

The study sample, consisting of 19 adult crania was derived from the excavated material of Sala A (n=14) and Sala B (n=5) (Appendix one). The material from these rooms was pooled together because the radiocarbon dates suggest that they are contemporaneous. Also, the rooms appear to have similar modes of burial practice (Duarte 1998). The limitations of the palaeopathological sample often necessitates the combining of sub-units that may belong to different time intervals and include individuals who died several hundred years apart (Waldron 1994:21). Buikstra (1981) recommends lumping small samples together where temporally defensible, although careful consideration is required in the interpretation process. Accession numbers and the provenance for each skull are provided in Appendix one. For the purpose of this project, each cranium has been assigned a Case number (Case # 1 through Case # 19).

The possibility of trephination at ABS was the basis of much speculation. Under the justification of establishing the nature of the abnormal bone, two of the *in situ* crania noted to have trephination-like lesions were taken from the site for inclusion in this study at the end of the 1997 field season. Using the process of differential diagnosis, the two crania were considered in this project for evidence of pathology; however, they *were not* included as part of the main study sample. Although their relevance to the ABS population will be considered, the descriptive statistics do not include either of the crania. One skull was taken from the unexcavated Sala C, (cranium will be referred to as: Case A) and another from the partially excavated Sala A (cranium will be referred to as Case B). The radiocarbon dates presented in the previous chapter suggest that Salas A, B and C could be considered contemporaneous.

Only two individuals were identified from the juvenile material. They were not included in this study due to time restrictions and the difficulty of recognizing disease from the routinely fragmentary remains of subadults (Jackes 1988). However, as they can be aged, they will be considered as part of the population profile in Chapter five.

The identification of the presence or absence of trephination was one of the ultimate goals of this study; as a result, the unit of analysis was the individual cranium. The majority of individuals in this sample were represented by partially complete (50-90%) crania; however, several completely preserved crania (>90%) and incomplete crania (<50%) were also included (see Table 3.2). A cranium was considered complete if

[1] Because of the secondary and commingled nature of Neolithic burials, Jackes, (1988:144) uses a single element to represent the individual as her unit of study.

90% or more of the vault, face and base were present. For the purposes of this study, an associated mandible and *in situ* teeth were not required to assess the cranium as complete, and indeed, there were no instances in the current ABS sample of an associated mandible and cranium. A cranium was considered partially complete if 50-90% of the vault, face and base were present. In most instances, a partially complete cranium consisted of an articulated or refitted vault. The skull face and base were the most commonly missing portions. For example, in the study sample (n=19), out of a possible 38 maxillary bones and zygomatic bones only seven and six were counted respectively (18.4% and 15.8% of the number expected) (Table 3.3).

Similarly, the MNI represented by the occipital bone at lambda was 20/24 (83.3% of the number expected) while the MNI represented at the basi-occiput was 7/24 (29.2 % of the number expected). The reasons for this differential preservation of the vault, face and skull base are currently under investigation, but it is likely to have significant implications. First, the researcher will be limited in the ability to use the distribution of lesions on the cranium to identify pathological conditions. As well, the prevalence of disease in the cranio-facial and the basi-occipital regions will likely be underrepresented (Waldron 1987). Incomplete crania (<50%) were included if it could be demonstrated that none of the features overlapped with those of the identified partial and complete cranial material.

Table 3.1. Number of adult crania represented by degree of preservation.

Degree of Preservation	# N
Complete crania (>90%)	3
Partial crania (50-90%)	14
Incomplete crania (<50%)	2
Fragmentary cranial material*	>100**

*fragmentary and isolated material not included in sample
** may include extremely fragmentary juvenile remains

As the unit of analysis in this study is the individual cranium, fragmentary and isolated cranial remains were excluded from this analysis. This decision can be further justified by the limited ability of palaeopathologists to determine age and sex as well as the sheer impracticality of diagnosing and identifying disease from fragmentary and isolated remains (Rogers and Waldron 1989:624; Waldron 1987:58; Webb 1995:11). The exclusion of the fragmentary remains will likely impact the applicability of the data for comparative purposes; therefore, strong emphasis should not be placed upon the prevalence data presented in Chapter five. Further uncertainties regarding the representative nature of the sample, reliability of the age and sex estimation methods from the cranium, the secondary origin of the burial sample, and the reality that subadult remains are unusually underrepresented in Neolithic samples (and are not included in this study) (Jackes 1988:144) does not inspire confidence in the unbiased nature of the sample. For these reasons, full reconstruction of the ABS palaeo-demography must await further excavation and analysis of both the cranial and post-cranial material.

Table 3.2. Proportions of cranial bones recovered from 19 adult crania from Algar do Bom Santo.

Skeletal Element	Side	# Recovered	# / N Expected	%
Parietal	Left	18	18/19	94.7
	Right	15	15/19	78.9
	Total	33	33/38	86.8
Temporal	Left	11	11/19	52.6
	Right	7	7/19	36.8
	Total	18	18/38	47.4
Maxilla	Left	3	3/19	15.8
	Right	4	4/19	21.1
	Total	7	7/38	18.4
Zygomatic	Left	3	3/19	15.8
	Right	3	3/19	15.8
	Total	6	6/38	15.8
Frontal		15	15/19	78.9
Occipital		16	16/19	84.2
Mandible		0	0/19	0.0

Note: '# recovered' was obtained by counting those portions of the cranium that were complete (>90%), partially complete (50-90%) and incomplete (<50%). Unidentifiable fragments, and unsided portions of the cranium were not included.

3.3 Methods of Palaeopathological Analysis

After each individual was identified and the degree of completeness and preservation was recorded, detailed written descriptions and differential diagnoses were undertaken. This section describes the following methods used in the palaeopathological analysis: 1) sex determination; 2) age estimation; 3) description of pathological lesions 4) methods of differential diagnosis; and 5) calculation of prevalence.

3.3.1 Sex Determination

Sex determination was based upon the identification of the cranial morphological criteria outlined in Buikstra and Ubelaker (1994: 20) as well as those criteria established in Bass (1987) and Ubelaker (1989). The mastoid process, nuchal crest, supra-orbital margin and supra-orbital ridge/glabella were scored as '1' female, '2' probable female, '3' ambiguous sex, '4' probable male and '5' male. Those features that were missing or unobservable due to pathological intervention were scored as blank. The tendency of females to present gracile skulls, with subtle muscle marking, rounded orbits and a small foramen magnum, as well as the tendency of males to present large, robust skulls with well-marked muscle attachments, square orbits and a large foramen magnum was used to support or validate the sex assignment (Bass 1987; Stewart 1978; Ubelaker 1989).

Table 3.3. Sex and age distribution of the Algar do Bom Santo adult cranial remains. Probable females and males separated from females and males. Sala A and Sala B pooled.

Sex and Age	# /n	%	# /N	%	Sex and Age	# / n	%	# /N	%
Female					Probable Female				
Young Adult	0/2	0.0	0/19	0.0	Young Adult	1/6	16.7	1/19	5.3
Middle Adult	1/2	50.0	1/19	5.3	Middle Adult	0/6	0.0	1/19	0.0
Old Adult	0/2	0.0	0/19	0.0	Old Adult	2/6	33.3	2/19	10.5
Adult	1/2	50.0	1/19	5.3	Adult	3/6	50.0	3/19	15.8
Male					Probable Male				
Young Adult	1/3	33.3	1/19	5.3	Young Adult	1/2	50.0	1/19	5.3
Middle Adult	0/3	0.0	0/19	0.0	Middle Adult	1/2	50.0	1/19	5.3
Old Adult	2/3	66.7	2/19	10.5	Old Adult	0/2	0.0	0/19	0.0
Adult	0/3	33.3	0/19	0.0	Adult	0/2	0.0	0/19	0.0
Unknown									
Young Adults	0/6	0.0	0/19	0.0					
Middle Adults	0/6	0.0	0/19	0.0					
Old Adults	0/6	0.0	0/19	0.0					
Adult	6/6	100	6/19	31.6					
Totals									
Young Adults			3/19	15.8					
Middle Adults			2/19	10.5					
Old Adults			4/19	21.1					
Adults			10/19	52.6					

Note: '#' = number of individuals; n = sub sample; N = total sample; % = percentage of individuals within the sample or sub sample. (Adapted from Bartel 1993:44)

Cranial material that was ambiguous, fragmentary and poorly preserved, or whose lesions obscured the features was labeled as 'unknown'. Due to the incomplete nature of much of the material, there was often only one or two of the criteria available for observation. This resulted in much of the material earning the designation of "probable male" and "probable female". Further, for 31.6% of the sample, sex could not be reliably established and was labeled 'unknown'. This is expressed in Table 3.4 which presents the sex and age distribution for the entire sample. In Table 3.5 the sex and age distribution is presented with the probable males and females included under the male or female designation. Table 3.6 presents the sex and age distribution for Sala A and Sala B.

3.3.2 Age Estimation

Age-at-death was established using the ectocranial suture closure method described in Buikstra and Ubelaker (1994:36-38) and adapted from Meindl and Lovejoy (1985). Closure of the cranial sutures, like those of the post-cranium is positively correlated with increasing age. The utility of cranial age estimation is debated by those who feel that the applicability of methods developed on a single sample as well as individual and population variability reduces the reliability of the age estimations (Iscan and Loth 1989; Key et.al. 1994; Masset 1989). It is recommended that researchers use as many independent indicators of age as possible (Meindl and Lovejoy 1985:68; Ubelaker 1974:

51); however, where no additional criteria exist, estimation of age based upon the closure of the sutures may be the only alternative. Estimations of age based upon endocranial suture closure were not calculated because most endocranial suture sites were unobservable due to post mortem damage and calcite deposits. Similarly, estimation of age based upon tooth wear was not attempted given the small number of *in situ* teeth included in the sample (n=35 of a possible 304 maxillary teeth: x=11.5%) and the unsurprising fact that on those skulls with *in situ* teeth, the cranial sutures were comparatively intact and observable. While these methods did not contribute to the direct measurements of age, they were occasionally used to clarify estimations and provide an additional qualitative assessment.

A score of '0' open, '1' minimal closure, '2' significant closure and '3' complete obliteration were recorded from ten 1 cm sites on the lateral-anterior region (sphenofrontal, inferior sphenotemporal and superior sphenotemporal, pterion and midcoronal) and vault region (pterion, midcoronal, midlambdoid, lambda, obelion, anterior sagittal and bregma) of the cranium. For those sites that are paired, the left site was preferred, although where missing or unobservable the right side was considered acceptable. Key and colleagues (1994) found that there was no significant bias in closure towards either side in their application of Meindl and Lovejoy's methods to the Spitalfields sample. Those cranial sites that were missing or obscured by pathological lesions were scored as blank. Scores were compiled for each region and an interdecile age (eg.19-44) was obtained from which a mean age (e.g. x =34 years) was derived. According to Meindl and Lovejoy (1985:62), the lateral-anterior sites provide a smaller standard of deviation and interdecile range and therefore should be considered to provide the most reliable indication of age. This did not present a conflict to this analysis, because both the vault and lateral anterior estimates could be calculated for only one cranium (Case #5). This sample is comprised primarily of partially complete crania for which a significant proportion are missing one or more of the ectocranial suture sites. In addition, several of the crania exhibited lesions that obscured the estimation site. As a result, for 52.6% of the sample, a cranial age could not be assigned a more reliable age range than 'adult'.

Due to the large age-ranges provided by ectocranial suture closure, the mean ages of the crania were grouped into three age categories. 'Young Adult' represented those individuals between and including the ages of 20-34 years, 'Middle Adult' represented those individuals between the ages of 35-49 years and 'Old Adult' represented those individuals older than 50 years of the age (Buikstra and Ubelaker 1994:36). While Key and colleagues (1994), found that ectocranial suture closure rates differed between males and females, and that open suture sites can occur in individuals regardless of age, they suggest using broad age ranges to offset this problem. Tables 3.4 and 3.5 present the sex and age distribution for the entire sample while Table 3.6 presents the sex and age distribution for Sala A and Sala B.

Table 3.4. Sex and age distribution of the Algar do Bom Santo adult cranial remains. Sala A and Sala B pooled. Definite and probable sex estimations are also combined

Sex and Age	# / n	%	# / N	%
Female				
Young Adult	1/8	11.1	1/19	5.3
Middle Adult	1/8	11.1	1/19	5.3
Old Adult	2/8	22.2	2/19	10.5
Adult	4/8	50.0	4/19	21.1
Male				
Young Adult	2/5	40.0	2/19	10.5
Middle Adult	1/5	20.0	0/19	5.3
Old Adult	2/5	40.0	2/19	10.5
Adult	0/5	0.0	0/19	0.0
Unknown				
Young Adults	0/5	0.0	0/19	0.0
Middle Adults	0/5	0.0	0/19	0.0
Old Adults	0/5	0.0	0/19	0.0
Adult	6/6	100.0	6/19	31.6
Totals				
Young Adults			3/19	15.8
Middle Adults			2/19	10.5
Old Adults			4/19	21.1
Adults			10/19	52.6

Note: '#' = number of individuals; n = sub sample; N = total sample; % = percentage of individuals within the sample or sub sample. (Adapted from Bartel 1993:44)

3.3.3 Description of Pathological Lesions

Once sex and age were determined from the cranial material, burial and room location were noted. Further observations regarding burial practices (i.e. primary vs. secondary) were noted if available (Appendix one). The next step was to record the bones present and provide a description of the inventory and state of preservation for each cranium. Postmortem damage was identified and described in detail. Next, abnormal bone changes were noted and described using methodology based in part on Buikstra and Ubelaker (1994), Mann and Murphy (1990), Ortner and Putschar (1984), and Roberts (1991). Histological sections were taken from several of the crania for future research, although a recent study from Portugal suggests that the microstructure may be severely compromised (Jackes et al. 2001).

According to Ortner and Putschar, (1984:36) critical to all palaeopathological analysis is: 1) the use of unambiguous terminology; 2) careful and methodical description of the location, extent and distribution of the abnormal bone; and 3) careful and methodical description of the morphologic features of the abnormal bone. Studies that provide an opinion or diagnosis of a pathological specimen without first providing a detailed description of the lesion(s) limit the ability of the outsider to independently evaluate the diagnosis and compare populations (Buikstra and Cook 1980; Ortner 1991). Effectively, the ability to make statements regarding the role of disease in antiquity and discern "what it means" is limited by the descriptive process (Ortner 1991:5).

Using standardized terminology derived from Buikstra and Ubelaker (1994: 108, 177-181), detailed written descriptions regarding the distribution, location, size and

shape, quality of abnormal bone, margins, degree of healing, cross section and associated features were recorded under the heading 'Description of Abnormal Bone". When two or

Table 3.5. Age and sex distribution for Sala A and B

	Sala A				Sala B			
Sex and Age	# / n	%	# / N	%	# / n	%	# / N	%
Female								
Young Adult	1/7	14.3	1/14	7.1	1/1	100.0	1/5	20.0
Middle Adult	1/7	14.3	1/14	7.1	0/1	0.0	0/5	0.0
Old Adult	2/7	28.6	2/14	14.6	0/1	0.0	0/5	0.0
Adult	3/7	42.6	3/14	21.4	0/1	0.0	0/5	0.0
Male								
Young Adult	1/2	50.0	1/14	7.1	2/3	66.7	2/5	40.0
Middle Adult	0/2	0.0	0/14	0.0	0/3	0.0	0/5	0.0
Old Adult	1/2	50.0	1/14	7.1	1/3	3.3	1/5	20.0
Adult	0/2	50.0	1/14	0.0	0/3	0.0	0/5	0.0
Unknowns								
Young Adults	0/5	0.0	0/14	0.0	0/1	0.0	0/5	0.0
Middle Adults	0/5	0.0	0/14	0.0	0/1	0.0	0/5	0.0
Old Adults	0/5	0.0	0/14	0.0	0/1	0.0	0/5	0.0
Adult	5/5	100.0	5/14	35.7	1/1	100.0	1/5	25.0
Totals								
Young Adults			2/14	14.6			3/5	60.0
Middle Adults			1/14	7.1			0/5	0.0
Old Adults			3/14	21.4			1/5	20.0
Adults			9/14	64.3			1/5	20.0

Note: '#' = number of individuals; n = sub sample; N = total sample; % = percentage of individuals within the sample or sub sample. (Adapted from Bartel 1993:44)

more lesions were recognized as distinctive processes, they were each considered separately under the 'Description of Abnormal Bone' and 'Differential Diagnosis' headings. Lesions or features that were not directly associated with the primary disease process, but bear possible relationships, were considered as associated features.

The first step in the description of abnormal bone is to determine whether the bone presents a solitary lesion with a single focus, multiple lesions with more than one foci, diffuse lesion(s) without a specific focus or whether the bone tissue exhibits a normal quality but abnormal size and/or shape (Buikstra and Ubelaker 1994; Ortner and Putschar, 1984: 37). Lesions that are multiple, diffuse or abnormal in size and shape introduce a degree of complexity to the description process and special care must be given to the differentiation of different morbid processes

The "crucial descriptive parameter" that is recognized by Ortner and Putschar, (1984:37) is the location of the lesion(s). Different disease processes may predilect a specific bone, a specific location on the bone (e.g. diaphysis, articular; inner table/outer table), specific groups of bones (e.g. cranial vault, facial bones, vertebral column), and specific types of bone tissues or surfaces (e.g. long bone, flat bone, cortical/cancellous or periosteal/endosteal). Finally, abnormal bone may either present unilaterally or bilaterally on the skeleton. The patterning of osseous lesions is essential to the recognition of the abnormal condition on the skeleton (Ortner 1991; Rogers and Waldron 1989; Rothschild

and Rothschild, 1995b). The location of the lesion(s) on the bone was always provided with reference to specific landmarks or features. In this thesis, the distribution of lesions was considered with the cognizance that the complete skeleton was not available for analysis. Statements confirming the etiology of a disease process were not made if the post-cranial material was essential for the diagnosis.

The size, depth/height and shape of the lesion(s) are a basic descriptive parameter (Buikstra and Ubelaker 1994; Ortner and Putschar 1984). Size is an important morphological consideration that may provide the discriminatory evidence required in the differential diagnosis (e.g. button osteoma are usually smaller than 2cm (Capasso 1997). The shape of a lesion can also provide significant descriptive and pathogenic clues. For example, the shape of a depressed fracture can be indicative of the type of instrument involved (Buikstra and Ubelaker 1994:120; McGee 1991:149; Wells 1964:49). The size and depth/height of the lesion was measured using spreading and sliding calipers and, where relevant, the shape of the lesion was noted.

The fundamental basis for description of pathological lesions is the recognition of the predominant osseous activity: abnormal bone loss, abnormal gain, a combination of both abnormal bone loss and abnormal bone gain, and a change in bone contour, size or shape (Ortner and Putschar 1984:42). However, due to the nature of disease and bone interface, the recognition of a specific disease from a given lesion may not always be possible. Bone is a living and dynamic tissue that is continually remodeling to meet the biomechanical needs of the body (Mann and Murphy 1990:17; Steinbock 1979:9). In healthy bone, there is a balance between the activities of the cells responsible for bone resorption (osteoclasts) and the activities of the cells responsible for bone deposition (osteoblasts) (Mann and Murphy 1990:17; Ortner and Putschar, 1984:13; Roberts and Manchester 1995:5; Rogers and Waldron 1995:8 Steinbock 1976: 11).

The reaction of bone to a stressor can be divided into four categories that reflect an imbalance in the dynamic relationship of bone at the cellular level. First, abnormal bone loss resulting in generalized atrophy or resorptive lesions (also: osteolytic/ lytic/osteopenia) can be characterized by increased osteoclastic activity, reduced osteoblastic activity, as well as the failure of the organic matrix to mineralize properly. Second, abnormal bone gain resulting in hypertrophy or proliferate lesions (also: osteosclerotic/sclerotic) may be the result of increased osteoblastic activity or decreased osteoclastic activity. Third, changes in bone may be classified as a combination of both abnormal bone loss and abnormal bone gain. Finally, bone changes may be the result of abnormal shape or contours due to abnormalities and malformations during growth or pathogenic remodeling later in life (Ortner and Putschar 1984:37-40; Roberts and Manchester 1995:4; Rothschild and Martin 1993:11). The non-specific nature of most osseous conditions is directly related to the limited number of ways in which bone can change.

Observations of the margins are a critical component of the morphological description because they often present evidence for the speed and duration at which the lesion formed (Buikstra and Ubelaker 1994:117). It is along the margin that the interface of abnormal and normal osseous activity occurs (Madewell et al. 1981:719). Resorptive lesions with smooth borders, circumscribed with sclerotic bone tend to be indicative of a disease process that was slow and chronic. Resorptive lesions with sharp margins, no evidence of bony circumscription and an associated zone of lytic activity generally

represent disease processes at the other end of the spectrum (Ortner and Putschar 1984:38). Diagnostic evidence of healing is usually observed along the margins of a lesion (Buikstra and Ubelaker 1994). Morphological evidence of healing will differ depending upon the relationship of a number of potentially overlapping factors. These include, the type of osseous reaction or traumatic event, type of bone, chronic or acute nature of the disorder, secondary complications, duration of healing and the age of the individual (Merbs 1989; Ortner and Pustchar 1984:63).

Like the margins, the cross-section provides critical evidence regarding the abnormal process, duration and severity of the lesion. Proliferative lesions can occur on any osseous surface, however, when viewed via the cross-section the distinct features of the formation are revealed. These include, the type of deposited bone (i.e. fibrous/woven or lamellar) and its relationship to the periosteum/endosteum; similarly, the relationship of the abnormal formation to the cortex (surfical/remodeled; unilaminar/ multilaminar), the integrity of the surface (perforated/continuous) and the pattern of deposition (cauliflower vs. sunburst) (Buikstra and Ubelaker 1994; Ortner and Putschar 1984; Rothschild and Martin 1993) can be observed best in cross-section. Resorptive lesions are also observable along the cross-section. The extent of bone loss to the two tables and diploë of the cranium and the integrity of the inner and outer surfaces can be assessed. Finally, the nature of cancellous resorption and the relationship between the cortex and diploic table can be qualified (e.g. diploic expansion; thinned trabeculae) (Buikstra and Ubelaker 1994:115).

Given the limited number of avenues of bone change, more than one disease may manifest similar lesions, and conversely, a single disease may manifest more than one type of lesion (Roberts and Manchester 1995:6). Recognition of the predominant process (abnormal bone loss, abnormal bone gain, a mixture of both, or a change in size or shape) and its morphological consequences is the basis of pathological description (Mann and Murphy 1990:17). In this thesis, careful consideration was given to the morphological features observed at the margins and, where visible, cross-sections.

Associated features were considered as lesions or morphological features that may or may not be associated with the primary disease condition. For example, all of the cranial sutures on Case # 16 exhibit complete obliteration. It is not clear whether this is: a) related to the extensive remodeling of vault due to a combination of abnormal bone loss and bone formation, b) whether it is due to the advanced age of the individual, or c) whether the two are somehow related.

For the osseous lesions identified as porotic hyperostosis/cribra orbitalia, trauma and osteoarthritis, it was felt that the operational criteria for identification were reasonably clear (Buikstra and Ubelaker; Lovell 1997b; Rogers and Waldron 1995; Rogers et al. 1987; Stuart-Macadam 1987a,b, 1989b, 1991). For this reason, specimens that exhibit evidence of these conditions and fit the operational criteria described in Appendix two will not be differentially diagnosed here. If the lesions are ambiguous, then a differential diagnosis will be provided.

Lesions identified as porotic hyperostosis and cribra orbitalia were recorded using methods based upon Stuart-Macadam (1985: 392) and Walker (1986:349) (both are adapted from Nathan and Haas 1966). Evidence for porotic hyperostosis was noted and scored as: 'blank' - unobservable; 'absent' - no evidence of porotic hyperostosis; 'ectocranial porosis' – scattered fine foramina; 'medium' - large and small pores that

have linked to form a trabecular structure; and 'severe' – outgrowth in trabecular structure perpendicular to the normal contour of the cranial table. Cribra orbitalia was identified by scoring each orbit as: 'blank'- unobservable; 'absent' - no evidence of cribra orbitalia; 'light' - scattered fine foramina; 'medium' - combined small and large foramina that cluster together but are distinct; and 'severe' linked foramina, thickening and involvement of a substantial area (>1cm²). The degree of healing was recorded as 'active' or 'healed'.

Evidence for trauma was not scored. It is widely acknowledged that while there are a variety of methodological protocols for trauma analysis, there is little consensus on the descriptive standards (Lovell 1997a: 148). This is perhaps a result of differing samples, research questions and the level of analysis of most studies. In this project, cranial fractures were assessed according to the following criteria: 1) location; 2) size and shape; 3) morphology of the margin and the involvement of the various tables; 4) type of fracture (linear, depressed, concentric, comminuted, spider, ring or hinge); 5) type of force (blunt or sharp force); 6) associated bony reaction; and 7) timing/origin of the lesion (antemortem, perimortem and postmortem). Differential diagnosis was employed if the lesion was well-healed or presented ambiguous features inconsistent with trauma. Where possible, consideration was given to the amount of force involved in the traumatic event, the timing of multiple fractures, and the nature of the implement.

Evidence for temporomandibular joint (TMJ) osteoarthritis was recorded using methods based upon Richards (1988:1529) and to a lesser extent Richards and Brown (1981) and Hodges (1991). Evidence for arthritis was considered present if erosive lesions were noted on at least one of the surfaces of the condyls or mandibular fossae (Hodges 1991:369). The elements in question were scored as; 'blank' – unobservable; 'normal'; 'moderate' – localized areas of erosion or proliferation; and 'severe' – generalized proliferation and eburnation (Richards 1988:1529-131).

After the descriptive data were assembled, a record of the cranial material was made with black and white photographs while drawings and colour photographs were taken to augment the descriptive and diagnostic process. Unfortunately, due to laboratory inadequacies a majority of the black and white photographs provided unsatisfactory images. Therefore, most of the images included in this thesis are colour photographs. Using the detailed written descriptions and photographs the data were compared to the images and descriptions in the palaeopathological and clinical literature. This evidence was considered within the context of the site and the differential diagnosis was developed.

3.3.4 Differential Diagnosis

After each case was described and the postmortem damage identified, the descriptions and pictures of the crania were compared to the palaeopathological and clinical literature. Ortner and Putschar (1981), Roberts and Manchester (1995), Aufderheide and Rodríguez-Martín (1998) Mann and Murphy (1990), and Buikstra and Ubelaker (1994) and various articles provided the foundation for comparison along with consultation with colleagues. The clinical literature was also consulted, although care must be exercised when comparing the clinical descriptions, which are based on *in vivo* soft tissue and radiographic observations to the dry bone manifestations of disease. The location and pattern of the lesions, the characteristics of their morphology, and the

dominant osseous reaction were used to compile a list of competing alternatives (Roberts and Manchester 1995:6; Waldron 1994:28-29).

The diagnostic possibilities are compared once again to the literature. The biocultural context was also taken into consideration at this point in the differential diagnosis. Many of the disease conditions that were considered had specific etiologies that were differentially expressed according to the individual (age, sex, health status, biological affinity), environmental context (geography, ecology), and cultural factors (social status, cultural practices). These were evaluated when considering each possible condition. Most importantly, competing disease processes were excluded on the basis of inconsistent morphological features. It was possible to accept a diagnosis in the absence of pathonogmonic features (e.g. Case A, no diagnostic cutmarks on cranium with possible trephination) provided that it was not absolutely necessary for the diagnosis. Conversely, consistent features were almost always present to some degree in each diagnosis, even if it was eventually excluded. The significance of a feature to the lesion and the diagnosis must be carefully evaluated and considered according to its importance. The possibilities are also compared with each other to ensure that the most parsimonious explanation was favoured. On this basis, competing disease conditions were considered as excluded, accepted or neither excluded or accepted on the basis of the current information.

Once all the conditions were considered, the diagnostic possibilities were then arranged according to their respective likelihood and a final diagnosis was made. Ideally, after all the inconsistent diagnoses were differentially excluded, the one remaining possibility effectively explained the lesion; however, this was not always the case. There may be a certain degree of uncertainty due to postmortem damage, incomplete material, and unknown age and sex. In this case, the lesion was diagnosed as "Possible" or "Probable". If the specific disease could not be identified, an attempt was made to identify the "Disease Category" and the list of possibilities was given. If none of the disease processes considered sufficiently explained the lesions or abnormal bone then the cranium was classified as "Unknown" and the dominant osseous activity was listed (e.g. abnormal formation, abnormal bone loss; mixed; abnormal contour). Where possible, recommendations for future avenues of consideration that may lead to a diagnosis were provided.

3.3.5 Disease Prevalence

The proportion or prevalence[2] of each disease category was calculated for this thesis. Prevalence was also calculated for specific disorders: blunt force trauma, porotic hyperostosis/cribra orbitalia and osteoarthritis of the TMJ. Prevalence is the most common calculation used in palaeopathology and the most useful for samples derived over long time periods (Waldron 1994:47)

Assemblages of disarticulated skeletons and commingled individuals presents particular challenges for palaeodemographic and palaeoepidemiological profiles. Usually an MNI is calculated on the basis of the most frequent anatomical element; however, the MNI cannot be used to calculate disease frequencies, (i.e. cannot be used as the denominator in calculations of prevalence). For the purposes of this thesis, the prevalence of the disease categories for the total sample was calculated with the number of

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[2] Prevalence = $\frac{\text{Number of cases}}{\text{Total sample}}$

individuals affected by a specific disorder in the numerator and the 19 individual crania as the denominator (Waldron 1994:55). For example, the number of individuals in the ABS sample identified with depressed fractures is two. The prevalence is expressed as 2/19 (10.5%). The prevalence of disease for the entire sample was calculated for the different age and sex cohorts and the distribution of each disease was also calculated according to age and sex sub-samples. The prevalence of cribra orbitalia, otitis media/mastoiditis and osteoarthritis were also calculated using the total number of elements (orbital roofs, temporal bones) or joints (TMJ and atlanto-occipital) present as the denominator.

Prevalence statistics are provided to permit future comparisons of the ABS sample to other archaeological samples, but not for comparison with living populations (Cohen 1989; Wood et. al. 1992). Further, the prevalence of specific lesions in this sample is not intended to reflect the proportional mortality of the given condition associated with the recorded lesion (Wood et al. 1992).

CHAPTER 4

DESCRIPTION, DIFFERENTIAL DIAGNOSIS AND RESULTS

4.1 Introduction

The following are the descriptions and differential diagnoses of the 19 crania from the Algar do Bom Santo sample (Case #1 - Case #19). Also included are the two crania from outside the main sample (Case A and Case B). The osteobiographical details of the sex determination and age estimation are provided in Appendix three and a summary of burial location and osteobiographical information are included in Appendix one. Descriptions of the disorders included in each differential diagnosis are provided in Appendix one.

4.2 Case Description, Differential Diagnosis & Results

Case #1 Description of Cranium

Age: Adult

Sex: Probable male.

Articulated left and right parietal bones and a partially complete occipital bone missing the basi-occiput represent the partial cranium. Also present is a reconstructed frontal bone that is almost complete along the coronal margin, with postmortem breakage along the distal left and right margins of the suture. The right orbital roof is missing and the refitted left orbit and glabellar region exhibit extensive postmortem damage. A partially complete left temporal bone exhibits breakage along the squamous border and zygomatic process. The face is missing, but despite the damage, the vault is in good condition with some spalling (chipping of the surface) to the outer table.

Case #1 Pathology

A. Perforation of the Cranial Vault

Description of Abnormal Bone

There is a concentric fracture surrounding an area of depressed, comminuted bone with three radiating fracture lines on the lateral-posterior aspect of the left parietal boss, 49mm from the sagittal suture (Plate 4). The cranium is partially perforated, with portions of the outer table missing or displaced into the inner cavity. The fracture measures 20 mm in diameter medial-lateral and 13.9 mm in diameter superior-posterior. The ectocranial margins exhibit an inward bevel with much of the inner table displaced into the vault. The endocranial aspect is larger, measuring 24.8 mm, medial-laterally and 48.2 mm superior-posterior. The three linear fractures that radiate towards the depression measure 29.7 mm, 58.0 mm and 52.3 mm (superior to inferior line) (Gurdjian et al. 1950:313). The margins are very sharp with no evidence of sclerosis or bony reaction, indicating that the fracture was not in the process of healing at the time of death (Lovell 1997a; Merbs 1989; Walker 1989).

Differential Diagnosis

Completely perforated crania may result from multiple myeloma, tuberculosis, cranial dysostosis and trephination (Kauffmann et al. 1997; Ortner and Putschar 1984); however, all can be ruled out on the basis that they do not conform to the above

constellation of features. The careful description of the lesion reveals depressed comminuted bone, a concentric fracture, inwardly beveled margins, a well-defined ellipsoidal shape, radiating fracture lines and no evidence of bony reaction in the form of non-specific infection or bone remodeling (healing). The above morphological features are clearly consistent with the diagnosis of unhealed trauma as the result of a direct impact to the cranial vault (Berryman and Haun 1996; Hurlbut 2000; Lovell 1997a; Lambert, 1997; Walker 1989). The absence of healing suggests that the traumatic lesion was formed in the period immediately prior to or after death (Lovell 1997; Ortner and Putschar 1984). The following explanations are considered in this differential diagnosis: postmortem damage, perimortem trauma.

According to Lovell (1997a: 145), postmortem fractures can be recognized by the presence of smaller fragments, distinct colour contrasts, angular fractures, and the tendency of bone to shatter upon impact. While one of the comminuted pieces of the lesion presents whiter margins suggesting the damage is recent in nature, most of the lesion's margins are similar in colour to the cranial sutures. Because damage to dry bone characteristically results in angular fractures with sharp corners (Berryman and Haun 1996; Buikstra and Ubelaker 1994; Hurlbut 2000; Maples 1985; Ubelaker and Adams 1995), the presence of "round" edges suggests that the fracture formed when the bone was still fleshed and pliable. Additionally, the adherence of the small-comminuted fragments suggests that the periosteum and other soft tissues were intact upon impact (Ortner and Putschar 1984:72). The above evidence suggests that the depressed fracture formed in the perimortem period.

According to Roberts (1991:223), two types of head injury are common in human remains: blunt force and sharp force trauma. The internally beveled margin and the depressed and comminuted nature of the lesion are characteristic features of a blunt force impact to the skull. Furthermore, the force required to penetrate cranial bone usually results in blunt force fractures. As a result, sharp force trauma to the skull is rare (Polson 1965:131) and usually results in clean-cut elongated lesions that are V-shaped in cross-section (Stewart 1979:78). The speed of impact is an important consideration in the analysis of traumatic lesions. Low velocity impacts tend to result in single linear fractures, while a concentric and radiating fracture pattern along with inward displacement of the cranial tables suggests that a (relatively) high velocity impact was responsible (Lovell 1997:154-156). Finally, the shape of the traumatic lesion is often indicative of the implement involved (Merbs, 1989:175; Roberts and Manchester 1995:82). The lesion is identified as a depressed fracture resulting from a blunt force impact with an oval/triangular shaped object.

B. Porotic Hyperostosis and Cribra Orbitalia

Description of Abnormal Bone

Evidence for ectocranial porosis was identified on the frontal bone along the left temporal line and above the left and right orbits, in the area along the supraorbital torus towards the glabella. Under a 10x hand lens, cribra orbitalia lesions on the right orbit exhibit clear evidence of coalescing foramina, with slight evidence of thickening and rounded margins. Similarly, the supraorbital process presents very fine coalesced porosity and "labyrinth-like lesions" (Ortner and Putschar 1984: 262). The degree of porosity along the temporal line and the glabella can be characterized as ectocranial porosis

because it does not exhibit coalescence or thickening of the foramina (Nathan and Haas: 1966; Stuart-Macadam 1985; Walker 1986; Williams 1994). The porosity on the vault above the orbits was recorded as ectocranial porosis because the fine pores did not exhibit observable evidence of diploic thickening. Cribra orbitalia was recorded as 'medium'.

Case #2 Description of Cranium

Age: Middle adult

Sex: Probable male.

The cranium consists of an incomplete calvarium with a complete left parietal bone articulated to a partial right parietal, frontal bone and occipital bone. The occipital is missing the entire skull base below the inferior nuchal line and the frontal is broken below the frontal eminence. There is a small lateral fragment of the left orbital margin and roof. The cortical surface is in good condition despite significant postmortem breakage.

Case #2 Pathology

A. Lesion on the Left Parietal

Description of Abnormal Bone

There is a shallow, semi-circular depression on the left parietal 45.5 mm posterior to the coronal and 72.3 mm lateral the sagittal along the temporal line. The depression, measuring 17.2 mm in diameter is circumscribed by a sharp circular groove and a change in contour to the otherwise normal continuity of the vault (Plate 5). The mark is sharp and somewhat 'V' shaped in cross-section and circumscribes the inferior margin of the depression. Superior to the groove is a rim of dense, well-remodeled (sclerotic) bone and superior to this is an area of depressed bone. The cranial vault is not perforated and there is no apparent involvement of the inner table of the skull. The smooth nature of the depression and the rim of sclerotic bone adjacent to the circular line suggest that the lesion was well-healed and long standing.

Differential Diagnosis

The lesion on the left parietal bone of Case #2 is characterized by an unusual constellation of features. The change in the parietal contour is the key characteristic of the lesion. The differential diagnosis for Case #2 includes, button osteoma, trephination, healed sharp force trauma, and healed blunt-force trauma.

Button osteomas present dense, ivory-like projections of bone, most commonly on the frontal bone or parietals. The projections are small (<20mm), well organized and are frequently marked by a circular constriction along the periphery (Capasso 1997:615; Ortner and Putschar 1984:368). While the lesion is characterized by a rim of dense bone, marked by a sharp groove on the inferior margin, it is large and irregularly organized. Further, the lesion is associated with a shallow depression on its superior aspect. Finally, the peripheral constriction of the lesion is common to button osteomas and marks the extent of bony growth. In contrast, the groove is sharp, "V-shaped" in cross section and appears to be traumatic rather than biogenic in origin. On this basis, button osteoma is excluded from the differential diagnosis.

The location, size and shape of the lesion are reminiscent of an incomplete trephination. The sharp margin appears traumatic in origin and may represent a cut or

scraping mark considered diagnostic of the surgical procedure. However, the sharp nature of the inferior margin in comparison to the dense, well-remodeled rim and depression seems inconsistent. If the sharp inferior margin implies trephination then further evidence of the operative method would realistically be expected to accompany the lesion (Brothwell 1994; Campillo 1984); however, this is not the case. Further, the lesion lacks the external bevel commonly associated with trephination (Lisowski 1967:663). A more parsimonious explanation may lie in another cause. On the basis of the above description, there is sufficient evidence to exclude trephination from the differential diagnosis.

Sharp-force trauma also provides a possible explanation for the lesion on the left parietal bone. Elongated lesions that are V-shaped cross-sections are often associated with such trauma (Stewart 1979: 78). The sharp inferior margin, is reminiscent of such lesions, with the dense rim of bone on the margin of the depression possibly representing evidence of healing. Nevertheless, there are inconsistent features that argue against sharp-force trauma. The sharp margin of the lesion of Case #2 is semi-circular rather than elongated. This differs from the very small and/or linear lesions that represent most examples sharp-force trauma (Helpern 1934). Further, the degree of depression associated with the lesion suggests that the impact was substantial. If the force of impact from a sharp implement is great enough, blunt force trauma associated with linear and depressed fractures is frequently the result (Chandu Lal 1974; Lovell 1997a). Together, these features suggest that sharp-force trauma should be excluded from the differential diagnosis.

A depressed fracture as the result of blunt force trauma is an explanation for the lesion. While lacking the comminuted bone of the previous skull, Case #2 exhibits a significant depression in association with a rim of sclerotic bone. Well-remodeled bone can mask the type of fracture originally present (Grauer and Roberts 1996:534); three features suggest that an oblique impact with a blunt object is responsible for the possible fracture. First, the slight gash may represent the inferior margin where the deepest impact likely occurred. Second, the rim of sclerotic bone directly adjacent to the sharp margin may represent remodeled displaced bone. Finally, in the place of a superior margin there is an area of inbending that could reflect a compressive force applied to the vault (Berryman and Haun 1993). The shallow, depressed contour of the skull also suggests the possibility of a healed pond fracture (Knight 1991:166; Polson 1965:133). The lesion is well healed and substantially remodeled, indicating it formed well within the ante-mortem period.

While the sharp margin may be the un-remodeled portion of the anterior depression, is inconsistent with the diagnosis. While this may reflect differential healing, it is possible that the sharp groove is not associated with the blunt force impact. On the basis of this inconsistency, Case #2 is classified as a possible depressed fracture.

B. Porotic Hyperostosis

Description of Abnormal Bone

The bregmatic region of the frontal and parietals exhibits discernible pinprick sized pores identified by Williams (1994), as ectocranial porosis. The pores, which are rounded and appear to be healed and inactive, are distributed within a 40 mm radius around bregma. Neither of the orbits was available for observation. Porotic hyperostosis of the crania vault was recorded as ectocranial porosis.

C. Other

Description of Abnormal Bone

Sutural Complexity and Closure

The lambdoid suture(s) exhibit(s) exceptional complexity and interdigitation in comparison to the rest of the ABS sample. These sutures include at least 12 wormian bones, the largest, measuring 4 mm in length. The lambdoidal sutures can be divided into superior and inferior suture lines, within which the majority of the wormian bones are included. There is minimal closure along the entire suture. The inferior suture displays minimal closure on the medial portions with minimal to significant closure on the lateral portion. The superior suture exhibits significant closure on the lateral portion. The coronal suture was recorded as open at bregma on the frontal and parietal bones, but was recorded to exhibit significant to complete closure on the lateral ends. The sagittal suture, open at bregma, closes as it runs posteriorly. A palpable depression at the anterior-mid sagittal is associated with a completely obliterated posterior sagittal suture.

Case #3 Description of Cranium

Age: Adult

Sex: Undetermined sex.

The partial cranium consists of fragmentary left and right parietal bones, a frontal bone and an occipital bone. The fragmentary remains were associated and refitted during the curation. An incomplete frontal fragment consisting of a small segment of the right frontal body terminates just before the frontal crest due to postmortem damage. The frontal bone was glued to an incomplete right parietal that exhibits a well-remodeled portion of the coronal suture.

The posterior 2/3 of the left and right parietal bones are articulated along the sagittal suture. Lambda and the medial 2/3 of the lambdoidal suture are observable. Another small portion of the occipital bone adheres to the left lateral margin of the lambdoidal suture. This portion of the vault is refitted from four pieces broken in the postmortem interval. The cortical surfaces of both the inner and outer tables are in relatively good condition; however, the cortical surface exhibits slight flaking of the outer table and there are patches of calcite deposited on the bones. The cross section of the cranial vault is available for observation in several places.

Case #3 Pathology

A. Abnormal Bone Formation

Description of Abnormal Bone

The outer and inner tables of Case #3 do not exhibit observable changes in morphology; however, in cross-section more than 2/3rds of the trabecular tissue exhibits dramatic formation of abnormal bone (Plate 6). While abnormal bone formation characterizes the changes to the overall structure, there appears to be discrete differences in the individual bones. On a medially exposed cross-section of the frontal bone, the cortical surfaces of the inner and outer tables demonstrate exceptional expansion into the cancellous spaces where the individual trabeculae are small and indistinct (Plate 7). Similarly, a cross-section of the left parietal bone reveals an occluded diploic tissue with little or no individual diploë. A cross-section of the inferior right parietal is unusually

thick, exhibiting an expanded outer table and diploë. In comparison to the above sections where the cortical surfaces appear to be expanded, a laterally exposed cross-section of the frontal bone demonstrates greatly expanded diploë. A cross-section of the occipital bone reveals an enlarged layer of diploë, with the inner and outer tables of more or less normal proportions. The other observable cross-sections of the parietal bone and occipital bone are normal in appearance.

Overall, the abnormal formation is characterized by dense cortical tissue that is morphologically indistinguishable from the original cortex. The maximum medial cross-section of the frontal bone measures 10.3 mm; the maximum lateral cross-section measures 9.0 mm. The maximum cross-section of the right parietal bone measures 8.3 mm while the maximum left parietal bone measures 11.0 mm.

There are no observable changes on the ectocranial and endocranial surfaces that correspond to the changes along the cross-section. The margins of the trabeculae appear rounded and smooth along the medially exposed cross-section, while the laterally exposed margin has a larger proportion of sharp margined trabeculae. These changes should be considered modest and discrete. Both regions exhibit extensively remodeled cancellous spaces; however, the regional differences may reflect a condition that is in the process of differential development and/or healing.

All sutures exhibit advanced states of closure, with the lambdoidal and sagittal sutures completely obliterated. While advanced age is an obvious explanation, the direct association of the cranial sutures with the excessive bone formation and remodeling of the cranial tables must also be considered.

Differential Diagnosis

Case #3 exhibits extensive abnormal bone formation characterized predominantly by the expansion of cortical bone. The differential diagnosis includes, pycondosostosis, anemia, hyperostosis frontalis interna, Paget's disease, acromegaly, osteopetrosis and normal variation.

Pycnodysostosis is a congenital disorder of early childhood that results in increased bone densities. In pycnodysostosis the sutures remain open and the fontanels separated into adulthood (Ortner and Putschar 1984:342; Revell 1986:61). Case #3 clearly exhibits closed and obliterated sutures with not retention of the fontenalles; therefore, pycnodysostosis can be easily excluded from the differential diagnosis.

Hyperostotic cranial changes related to anemia can be quickly ruled out because marrow hyperplasia results in expansion and thickening of the cancellous spaces. The densely occluded diploic bone could not have accommodated the expansive marrow characteristic of anemia (Garrow and James 1993). Additionally, most anemias are associated with increased vascularity of the cortical surface observed as hyperostosis (Aufderheide and Rodríguez-Martín 1998:349; Ortner and Putschar 1984:43; Stuart-Macadam 1985:392:). While porosity is a non-specific response to a number of conditions (osteoporosis, Paget's, infection), most researchers associate vault porosity with anemia in the absence of other evidence. Porosity is notably absent from the cortical surface of Case #3.

Cranial changes characteristic of hyperostosis frontalis interna include a bumpy, rugose surface with many tumor-like swellings on the endocranial surface (Barber, Watt and Rogers 1997; Revell 1986; Roberts and Manchester 1995; Zimmerman and Kelley 1982). This contrasts with the smooth endocranial surface of the specimen and the

restricted nature of the abnormal bone formation to the cross-section. In cross-section, hyperostosis frontalis interna presents proliferative cancellous bone and only a thin inner table of cortical bone (Aufderheide and Rodríguez-Martín 1998; Hershovitz et al. 1999). Case #3 does exhibit minor cancellous expansion on the lateral portions, however the principal osseous change involves expansion of the cortical tables into the diploë. On this basis, hyperostosis frontalis interna can also be excluded from the differential diagnosis.

Later stages of Paget's disease characterized by unchecked osteoblastic activity should be considered. The enlarged nature of the cranial tables as well as the probable advanced age of Case #3 is consistent with Paget's disease. However, in Paget's disease, the proliferative new bone is characterized as abnormal fibrous bone with a spongy appearance. This contrasts with the thickened, but regular appearance of the cortical tables in Case #3. Similarly, in Paget's the proliferation of the cancellous bone onto the surface of the skull results in a patchy "pumice' or "cotton-wool' appearance (Aufderheide and Rodríguez-Martín 1998:415; Barnes and Peel 1990:132; Olmsted 1981:707). Again, this contrasts with Case #3, which exhibits a normal endocranial and ectocranial surface.

Acromegaly occasionally results in periosteal bone deposition and enlargement of the cranial vault (Ortner and Putschar 1984). A massive, prognathic mandible and bony build up on the maxilla and mandible leading to separation of the teeth are indicative of acromegaly (Aufderheide and Rodríguez-Martín 1998:327; Ortner and Putschar 1984:300; Zimmerman and Kelley 1982:63). Because of the fragmentary nature of this specimen, acromegaly cannot be definitively ruled out; however, the absolute rarity of the condition makes it a very unlikely explanation.

Osteopetrosis is a rare congenital/developmental abnormality that is characterized by the inhibition of the osteoclastic activity resulting in densely formed abnormal bone (Ortner and Putschar 1984:340). Infantile osteopetrosis results in extensive hypertrophic development of the skull base and vault; however, most individuals do not survive into adulthood. As Case #3 is clearly an adult, this is inconsistent with the infantile abnormality. Osteopetrosis tarda is a milder form frequently survived into adulthood, however changes to the skull are rare (Ortner and Putschar 1984:341). Where cranial involvement is observed, the diploë and cranial sinuses can become obliterated. In contrast, the densely formed bone characteristic of osteopetrosis is irregular in nature and cement lines are frequently observed in the cross-section (Aufderheide and Rodríguez-Martín, 1998:363). This compares to the medial sections of the bone, which exhibit well organized, if exceedingly thick, cortical bone. Osteopetrosis cannot be conclusively ruled out based on the above description, conversely, it cannot be confirmed either. The opaque radiographic appearance of osteopetrosis is considered diagnostic evidence.

Adeloye and colleagues (1975:27) measured the skulls of black and white males and females under the age of 10 to over 60 years. In individuals over the age of 20, they reported mean range of thickness between 6.2 to 8.7 mm anterior of the coronal suture, 5.2 to 7.8 mm posterior of the coronal suture, 7.4 to 10.5 mm superior of lambda and 5.4 to 8.2 mm inferior of lambda. Case #3 definitely exhibits greater cranial thickness than those recorded in the study (1975); however, it must be noted that Adeloye et. al. (1975) measured cranial thickness at specific locations that are not necessarily analogous to the case under consideration. Similarly, it would be remiss to suggest that the results of a study derived from modern black and white Americans are directly applicable to a

sample from the late Portuguese Neolithic. Merbs (1980:122) describes the average thickness of a normal American male vault at the mid-parietal adjacent to the sagittal to be 4-5 mm.

Hyperostotic changes to the cranium occur in many disorders. On the basis of the differential diagnosis it can be assumed that Case #3 did not exhibit anemia related marrow hyperplasia, pyncodyostosis, hyperostosis frontalis interna, and Paget's disease. Due to the fragmentary nature of the cranium, the missing mandible and cranio-facial area and the absence of post-cranial indicators, osteopetrosis and acromegaly cannot be definitively ruled out. The thicknesses of the various cross-sections exceed those reported by Adeloye and colleagues (1975), tentatively supporting the abnormal nature of the bone.

As stated, care must be taken in interpreting these results. The cranium is too fragmentary to state reliably the origin and etiology of the disorder that caused hypertrophic bone formation. The differential diagnosis does not suggest changes related to infection/inflammation, osteoarthritis or neoplasia. The abnormal expression of the osteoblastic formation suggests that the disorder is related to a metabolic or endocrine disorder (Ortner and Putschar 1984; Roberts and Manchester 1995).

Case #4 Description of Cranium

Age: Young adult

Sex: Male

The partial cranium consists of most of the superior cranial vault including the complete frontal and nasal bones. The right temporal bone and most of the inferior skull base and basi-occiput are missing. There are small fragments of the left maxilla, sphenoid, ethmoid and vomer articulated in the area around nasion. Below this point, the rest of the face is missing including the mandible. Postmortem erosion has pitted the cortical bone of the outer table along the lateral margins of the parietal bones and resulted in flaking of the cortical surface of the frontal bone. The endocranial surface appears to be in good condition with some calcite deposits on the left temporal bone and sphenoid bone.

Case #4 Pathology

A. Cranial porosity

Description of Abnormal Bone

The glabella, supraorbital tori, parietals and occipital, particularly around lambda, present fine, barely discernable porosity (Buikstra and Ubelaker 1994; Nathan and Haas: 1966; Stuart-Macdam 1985) with no evidence of coalescing foramina and thickening of the diploë. Evidence for porotic hyperostosis was recorded as ectocranial porosis (Williams, 1994). The orbits do not exhibit osseous evidence of change. This individual shows no further evidence of abnormal bone change.

Case #5 Description of Cranium

Age: Middle adult

Sex: Female.

The skull is >90% complete, missing only portions of the left face, including the lateral maxilla and zygomatic bones, both palatine bones and the mandible. The right M^1

and M^2 maxillary molars are still *in situ* and the skull base is complete. The cortical surfaces are well preserved, with slight flaking along the left coronal and lambdoidal sutures. There are calcite deposits on the left skull base and left temporal bone. While the base of the skull is intact, significant postmortem damage in the form of large, angular fractures with multiple perpendicular fracture lines are evident. Case # 5 has one of the few intact skull bases in the sample (Plate 8).

Case #5 Pathology

A. Perforated Mandibular Fossa

Description of Abnormal Bone

There is a small lytic defect that perforates the articular fossa of the right temporomandibular joint on the temporal bone. The defect is a singular circular lesion with an inside diameter of 2.3 mm. A slight inward bevel can be detected using a 10x hand lens. If the beveled boundary is included, the lesion's diameter measures 5.1 mm. The margins are slightly scalloped and sharp and exhibit no evidence of a sclerotic reaction or healing. The left articular fossa is unobservable due to the obstruction of a calcite deposit. A similar deposit also obscures a small portion of the lesion on the medial portion of the right articular fossa. There is no evidence of porosity and bony sclerosis indicative of periostitis. Further, there is no evidence for coalescing porosity, eburnation or osteophytic lipping of the articular fossa. The mastoid process, zygomatic process, and external auditory meatus do not exhibit evidence of a bony reaction. The mandible was not available for analysis.

Differential Diagnosis

The differential diagnosis of a small lytic lesion of the mandibular fossa should include, ear infections related to otitis media or mastoiditis postmortem damage, and osteoarthritis. The inability to observe the left temporo-mandibular fossa and the associated mandibular condyles is a major limiting factor in this diagnosis.

Otitis media is an infection of the middle ear that may produce inflammatory and destructive changes to the endocranial and ectocranial surface of the bone and its internal osseous structures (Aufderheide and Rodríguez-Martín 1998: 253; Mann 1991:165). Otitis media and its sequela mastoiditis may present perforations of the bone surrounding the mastoid and external auditory meatus; however, middle ear and mastoid infections are essentially osteomyelitic. Therefore, if the perforation observed on the mandibular fossa were the result of a draining sinus, associated porosity and bony sclerosis would be expected (Mann et al. 1994). The normal appearance of the mastoid and surrounding area and the lack of an inflammatory reaction suggest that the perforation is not a result of otitis media or mastoiditis.

Osteoarthritis is among the most frequently observed disease changes in the archaeological record (Alpagut 1979). The early stage of joint degeneration is characterized by fine porosity that gradually coalesces to form larger pores. Osteophyte formation and continued degeneration leading to eburnation and changes to the joint structure characterize the later stages of osteoarthritis. In the absence of diagnostic eburnation, Rogers and Waldron (1995:44) recommend that at least two other criteria should be present. Other than the perforation, the remaining surface of the fossa exhibits no evidence of porosity, osteophyte formation or changes to the joint. Evidence for

osteoarthritis is often dependent on bilateral observations of both joint surfaces. It must be noted that only one of the four TMJ surfaces was available for observation. On this limited basis, osteoarthritis is excluded from the differential diagnosis.

Rheumatoid arthritis is rarely diagnosed in the archaeological record due to misidentification and inability to assess the distribution of lesions (Roberts and Manchester 1995:116). Rheumatoid arthritis is more common in females, and the involvement of the TMJ occurs relatively late in the disorder. Smooth, well-rounded, lytic lesions, with little or no sclerotic bone characterize lesions of the TMJ (Aufderheide and Rodríguez-Martín 1998: 101). While this is consistent with the description of Case #5, recognition of rheumatoid arthritis is dependent upon the pattern of distribution throughout the skeleton. Given that only one joint is observable, the disorder can be neither confirmed nor excluded from the differential diagnosis.

Damage to the mandibular fossa in the postmortem interval is another option strongly suggested by the presence of calcite on the fossa. The perforation is slightly obscured by a calcareous deposit that appears to have covered the entire perforation at one point. There are several examples from ABS where the post-excavation removal of calcite led to the exfoliation of the periosteal surface of the bone. It is possible that calcite was either purposely or accidentally removed during the curation phase resulting in the perforation, and it is also feasible that the perforation formed as a result of the repeated dripping of calcite on the thin bony structure. However, the presence of a beveled, slightly scalloped margin suggests that the perforation is of biological origin.

It is not possible to identify the origin of the perforation on the left mandibular fossa of Case #5. Otitis media and osteoarthritis are excluded because they lack evidence of the typical inflammatory reaction. Rheumatoid arthritis cannot be confirmed nor excluded on the basis of the observable bone changes. Finally, while scalloped margins are often associated with abnormal bone loss due to disease processes, postmortem damage cannot be ruled out. Calcite deposits on the TM joints, seems to suggest a direct link with the abnormal perforation and damage.

B. Porotic Hyperostosis

Description of Abnormal Bone

There is diffuse, generalized porosity located along the sagittal and lambdoid sutures as well as the parietal bosses medial to the squamousal suture. The frontal bone exhibits minimal involvement, with discrete porosity on glabella and along the supraorbital margins. The right side of the frontal bone appears to be slightly more affected than the left. The vault exhibits bilateral evidence of small, uniform sized pores. Under a 10x hand lens, slight coalescence and thickening of the underlying structure can be observed. The lesions were recorded as ectocranial porosity and present a mixture of sharp and rounded margins indicating that the lesions were in the process of healing. On the supraorbital margins of the frontal bone, the majority of the pores show rounded margins indicating that the lesions were healed and inactive. The orbits do not present evidence of cribra orbitalia (Buikstra and Ubelaker 1994; Nathan and Haas: 1966; Stuart-Macdam 1985; Williams 1994).

Case #6 Description of Cranium

Age: Adult

Sex: Probable female.

The partially complete cranium is composed of three main fragments. The left side of the skull is almost complete, including the parietal bone, temporal bone, and the left frontal body and orbits. The right side of the skull is incomplete with the right portion of the frontal bone broken at glabella, and continuing back to the coronal suture. As a result of the damage, the frontal sinus is exposed. A partial right parietal bone is articulated along the anterior sagittal suture. While the lambdoidal area of the occipital bone is present, there is no skull base. The cranium is also missing the face and mandible. While the observable exterior cortical surface is reasonably well preserved, the rest of the cranium is in relatively poor condition. There is a large conglomeration of calcite bone, rock and sediment adhering to the endocranial surface. The conglomerated mass also obscures much of the right portion of the frontal bone. The deposit is very heavy and makes assessment very difficult.

Case #6 Pathology

A. Mixed reaction on the temporomandibular fossa

Description of Abnormal Bone

The inferior surface of the left zygomatic process, anterior to the temporo-mandibular fossa, exhibits a singular circular lesion with deep perforations circumscribed by a slightly sclerotic margin. The outer circumference of the lesion measures 5.5 mm in diameter; the largest of the four perforations measures 1.8 mm in diameter and is composed of three coalesced holes. The outer border of the circular lesion is raised with a sharp, slightly scalloped margin. Posterior to the circular lesion, the surface of the mandibular fossa exhibits slight pinpoint porosity and barely discernable lipping circumscribing 1/3-2/3 of the fossa margin. Eburnation is observable along the superior, lateral and medial margins of the fossa. Unfortunately, the right temporal and mandibular condyles are not available to inspect for corresponding lesions. The above description suggests that the lesion was active at the time of death. The lesion was recorded as 'severe' osteoarthritis of the left TM joint.

B. Abnormal Bone Loss on the Petrous Portion

Description of Abnormal Bone

On the superior-lateral surface (tegmen tympani) of the left petrous portion, a sharp lytic lesion perforates the ceiling of the auditory canal. The lesion's outer margin exposes the diploë and measures 6.7 mm in diameter (Plate 10). It appears that the outer margin was enhanced by postmortem damage. The inner perforation, through which the external auditory meatus can be observed, measures 4.0 mm in diameter. Posterior to the larger lytic lesion, there is another area of lytic destruction, consisting of irregular sized pores with very sharp margins that expose the underlying cancellous tissue. While the diploë do not appear greatly altered, it is difficult to determine if the erosion of the cortical surface is proceeding from the outer surface to the inner surface, or vice versa. The margins of the petrosal perforation and the area of lytic focus are sharp, and there is no evidence of bony sclerosis. This suggests that the lesions were active at the time of death; however, it is possible that some or all the damage observed is actually the result of postmortem

erosion. Examination of the photographs of the ectocranial aspect of the mastoid and external auditory meatus also suggests additional features not originally observed during data collection. First, there are small fissures on the upper aspect of the mastoid and temporal bone; however, postmortem damage to the squamosal suture obscures the extent and the upper limitation of the fissures. Second, the external auditory meatus is relatively large in comparison to those of other crania in this sample and those pictured in various texts (McMinn and Hutchings 1977). This observation is purely qualitative; there are no measurements to support this statement.

Differential Diagnosis

The differential diagnosis of erosive lesions on the temporal bone must include otitis media and postmortem destruction. Progressive and reoccurring otitis media may lead to changes in the mastoid and a possible cholesteaoma in advanced cases. On Case #6, the area of sharp erosive activity on the tegmen tympani accompanied with a perforated superior surface of the auditory canal is consistent with the cases of otitis media and secondary cholesteaoma described by Schultz (1979), Gregg and Gregg (1987) and Mann (1991). A discernible porosis circumscribes an apparently enlarged external auditory meatus; while neither feature is indicative of the disorder, they are suggestive that the area was a site of abnormal activity. Morphological changes on the surfaces of the temporal bone do not provide adequate evidence for otitis media. Most palaeopathologist's rely upon direct observation of the auditory ossicles with an otoscope, or use x – ray imaging to confirm changes to the middle ear. Postmortem damage to the thin cortical surface of the inner temporal is not uncommon, and the sharp margins of the erosive region are consistent with possible postmortem destruction. It is possible that both biological and taphonomic processes are responsible for the lesions observed on the interior surface of the temporal bone. On this basis, otitis media can be neither confirmed nor excluded.

C. Abnormal Porosity on the Frontal

Description of Abnormal Bone

The left orbital part of the frontal exhibits a very small area of porosity with coalescing foramina and a discrete capillary pattern. There is no involvement of the orbital margin, and only barely discernable porosity on the frontal glabella and supraorbital ridge. The right orbit was not observable due to postmortem damage. Both sharp and well-rounded margins characterize the orbital porosity, suggesting a mixed reaction of activity and healing at the time of death. Cribra orbitalia was recorded as 'medium' and ectocranial porosis was noted as present under porotic hyperostosis (Nathan and Haas 1966; Stuart-Macdam 1989b).

Case #7 Description of Cranium

Age: Adult

Sex: Female

The partial calvarium is comprised of a complete right parietal bone; a partial left parietal bone, a partial frontal bone including the superior orbital roofs and an incomplete right temporal bone. The occipital bone includes the inferior lambdoid and right nuchal areas. The base of the cranium is completely missing, along with all the bones of the face

and mandible. Postmortem damage is likely responsible for the numerous fragments that required refitting during the curation process. While there is recent evidence of cracking and flaking, the cortical surfaces are in relatively good condition. There is fine deposition of calcite and fine white patches of newly exposed bone on the endocranial surface. The occipital exhibits unusual postmortem damage. A large rounded portion of the right cerebral fossa is missing. Along the inferior margin is a 'finger' of bone, also with a somewhat rounded edge, which is broken inferiorly along the cerebellar fossa. The bone was partially refitted during the curation process; however, the medial aspect of the occipital is still missing, hiding the full extent of the damage. While the lesion has curvilinear margins, the newly exposed diploic bone and light coloured fracture lines suggest the damage is postmortem in origin (Hurlbut 2000; Lovell 1997a; Rothschild and Martin 1993). Case #7 also exhibits several observable cross sections of the diploë.

Case #7 Pathology

A. Circular hole on cranium

Description of Abnormal Bone

There is a singular circular area of abnormal bone loss located on the sagittal suture, 52.5 mm inferior to bregma, and approximately 42.2 mm superior to lambdoid. The lesion is 3.8 mm deep and perforates to the internal table of the cranium (Plate 11). The lesion is symmetrical and divided into left and right halves by the sagittal suture. The inferior left margin of the lesion is missing along with the entire inferior part of the parietal. While the full extent of the aperture cannot be determined, the medio-lateral diameter is 23.6 mm and the diagonal diameter is 23.1 mm. On the endocranial aspect of the vault, the sagittal sulcus begins just posterior to the coronal suture and runs along the sagittal suture to the lesion (Plate 12). It then diverts laterally around the anterior margin of the aperture, running along the inferior, right margin of the lesion and continuing along the sagittal suture once again. Along the anterior margin of the aperture, the sulcus is shallow, but discernible to both observation and touch. On the inferior, right margin of the aperture (the inferior left margin is missing) the sulcus is much deeper and is bisected by the sagittal suture, which resumes slightly lateral to its original orientation. The margins have a slight internal bevel on the endocranial surface. Evidence of well-remodeled margins with minimal exposure of the diploë and no evidence of reactive inflammatory bone suggests that the lesion is well healed and of long standing. Additionally, the bones of the calvarium are very thin and light. The average thickness of the parietals is less than 4 mm, and exposed cross sections of the cranium exhibit reduced thickness of the cortical layers and a reduced number of diploic spaces. As will be discussed below, the superior margins of the orbits exhibit evidence of cribra orbitalia.

Differential Diagnosis

The differential diagnosis of a singular circular defect of the cranial vault located on the midline should include: postmortem damage, osteoporosis, osteolytic neoplasms, trephination, depressed fracture, and congenital/developmental disorders (Kaufman et al. 1997).

Given the extent of postmortem damage to the inferior nuchal area, postmortem damage is a possible explanation for the aperture on the mid-sagittal; however, unlike the damage described above, several features distinguish it immediately. The lesion is an

almost perfect circle with smooth, well-remodeled margins indicating that it formed and healed well before death. Further, the sagittal sulcus runs around the aperture suggesting that significant osseous activity followed the formation of the lesion. Based on this evidence, postmortem damage can be excluded from the differential diagnosis.

According to Lodge (1967), osteoporotic thinning of the cortical surface of the vault may eventually lead to the perforation of the cranial tables. Marked thinning of the cortices and reduction in the number and size of the diploë are also features of the disorder (Zimmerman and Kelley 1982); however, thinning of the cortical surfaces frequently occurs bilaterally (biparietal atrophy) and irregularly shaped perforations tend to form on either side of the sagittal. This contrasts with the single circular defect located directly on the sagittal suture. Further, increased porosity leading to a thinned outer table in the zone surrounding the perforation common to osteoporosis is not a feature of Case #7. Osteoporosis leading to biparietal atrophy is common in older individuals, which is not consistent with the osteobiographical information for this individual (Ortner and Putschar 1984:292).

Both multiple myeloma and osteolytic metastatic carcinoma present well-defined circular defects on the cranium (Aufderheide and Rodríguez-Martín 1998; Strouhal 1991); however, they can be distinguished from Case #7 on a number of criteria. First, both diseases predilect older individuals (Aufderheide and Rodríguez-Martín 1998:351; Roberts and Manchester 1995:190) and while Case #7 is classified as 'adult', the minimal closure exhibited by the observable cranial sutures suggests that the individual was likely a young adult at death. Osteolytic metastatic carcinoma most frequently forms multiple diffuse lesions, which is in apparent contrast with the single aperture; however, this cannot be definitively stated because a large portion of the left cranial vault is missing. While multiple myeloma may present a single defect, numerous lesions commonly characterize it. Multiple myeloma also tends to exhibit a "punched-out" appearance with very sharp margins; this contrasts with the smooth, remodeled border of Case #7. While osteolytic metastatic carcinomas exhibit slightly more sclerotic margins, this form of neoplasia also presents multiple resorptive holes and a "moth-eaten" appearance in the periphery of the circular defects (Webb 1995). As far as is macroscopically visible, the bone, while thin, does not appear to exhibit generalized osteopenia in the area surrounding the aperture. Neither form of neoplasia is consistent with the description of Case #7.

Most trephinations described in the literature are typified by a singular oval or circular perforation of the cranial tables (Chege et. al. 1996; Germanà and Fornaciari 1992; Lillie 1998; Mallegni and Valassina 1996; Mallin & Rathburn 1976; McKinley 1992a,b; Persson 1979; Robb and Mallegni 1994; Smith 1990; Zimmerman et al. 1981). According to many researchers, trephination is rare along the sagittal suture; however, a review of the literature suggests that although uncommon in comparison to trephinations on the left parietal, there are a number of examples of the procedure occurring at bregma on the frontal and parietal bones and on the sutures (Jennebert 1991; Mogle and Zias; Parker and Miles 1990). Several features of the lesion in question are inconsistent with a diagnosis of trephination. First, there is no evidence of cut marks associated with scalp reflection or scars/cuts/scrapes that are frequently associated with the operative method. While these features are considered diagnostic, their absence may only reflect a significant degree of healing. Most importantly, Case #7 exhibits an internal bevel rather

than the external bevel characteristic of most trephinations (Campillo 1984; Lisowski 1967; Mallegni and Bertodi 1997; Novak and Knüsel 1997; Robb 1997). The lack of either a sclerotic margin and/or evidence of inflammation in the presence of a healed trephination are uncommon (Brothwell 1994; Campillo 1984). Most importantly, Case #7 exhibits a sagittal sinus that is redirected around the aperture. This unusual feature is not described in the literature on trephination. According to Dean O'Laughlin (1996), the development of unusual endocranial vasculature may reflect the altered growth trajectories of cranial deformation and craniosynostosis. A study by Dean (1995:1), exploring the changes in sinus and meningeal vessel patterns as a result of artificial deformation found that these traits were "developmentally plastic" and influenced by environmental factors. While the role that acute diseases and other forms of cultural modification play on endocranial features is not yet understood, changes to the vasculature suggest that the defect is longstanding. While it is reasonable to expect that a trephination occurring along the sagittal suture during the developmental stages of the cranium (i.e. growth) may result in a reorientation of the sagittal sulcus, trephination of young individuals is very rare in Neolithic Europe and other places (Piroreschi 1991).

The circular defect of Case #7 presents several characteristics that are similar to an antemortem depressed fracture. First, the size, shape and singular nature of the perforation are features that are frequently shared with traumatic lesions of the cranium (Walker 1989). The presence of an internal bevel is consistent with blunt force trauma to the cranial vault and the smooth, well-rounded margins could suggest a healed antemortem injury (Berryman and Haun 1996; Hurlbut 2000; Lambert 1997). While there are no observable fracture lines, the inferior left portion of the lesion is missing due to postmortem damage, therefore, the presence of an associated fracture cannot be ruled out. Conversely, several characteristics suggest that healed blunt force trauma is not the most parsimonious diagnosis for the data presented. First, depressed fractures usually exhibit endocranial spalling and present a larger lesion on the inside of the cranium than on the outside (Lambert 1997; Frayer 1997), which is also in apparent contrast with the above description. It is also reasonable to speculate that a traumatic event sufficient to completely displace the cranial bone of a thin-tabled skull would have resulted in a much more complex fracture pattern, possibly causing death, thereby preventing evidence of healing. Finally, the location of the aperture on the sagittal suture directly above the venous sinus is also inconsistent with the observation that the lesion is healed. Trauma sustained in such a location is very serious, and likely fatal (Webb 1995:242). Evidence to distinguish antemortem trauma from the aperture observed on Case #7 is essentially circumstantial.

A number of congenital/developmental disorders resulting from the failure of the cranium to properly ossify should also be considered. Parietal fenestrae (Catlin Marks) present circular defects with smooth, beveled edges (Kaufman et al. 1997); however, they are frequently observed biparietally and occur on either side of the sagittal suture adjacent to the parietal foramina. This contrasts with the singular nature of the defect located on the sagittal suture. Singular lesions of the cranial vault characterized by a congenital failure of ossification include meningocele/encephalocele and partial dysostosis. According to Kaufman et al. (1997; 193), the location of the aperture on the sagittal suture and the presence of smooth, well-remodeled margins is suggestive of a congenital defect. Meningoceles and encephaloceles are the developmental reflection on

the cranium of the herniation of cerebrospinal fluid or neural tissue (Barnes 1994; Kaufman et al. 1997). They present as a midline defect, usually on the sagittal or occipital. The perforations are often of an irregular shape and are situated within a larger saucer-shaped depression that is usually circular (Aufderheide and Rodríguez-Martín 1998; Stewart 1975; Webb 1995; Webb and Thorne 1985). Case #7 does not exhibit a saucer shaped depression; the contour of the skull is normal in the zone surrounding the defect. Kaufman and colleagues, (1997:196) present an image of a skull with partial dysostosis likely caused by a meningocele that is very similar in appearance to Case # 7; however, according to the description it has a slightly beveled margin. While the above congenital/ developmental disorders are inconsistent to some degree with the description of the circular defect presented in Case #7, the redirected sagittal sinus and suture is suggestive of a condition that developed during the formation of the aperture. As stated above, intentional cranial modification can result in premature synostosis of the sutures and unusual vasculature (Dean-O'Laughlin 1996; White 1996). If this is the case, then it not unreasonable to suppose that other forms of cranial alteration, given time, can effect the orientation and location of cranial features. The smoothly rounded margins that are not especially sclerotic, the normal contour and slight internal bevel suggest a longstanding lesion. The presence of the aperture along the midline, its circular and symmetrical nature also imply that it is a congenital failure of ossification, or a developmental defect relating to tissue herniation.

The lack of a saucer-shaped depression in association with a mid-sagittal aperture casts significant doubt on a diagnosis of meningocele or encephalocele; however, other evidence, particularly the redirected sagittal sulcus, midline location and symmetrical shape suggest that the lesion is developmental in origin. The lesion on Case #7 is tentatively classified as a congenital/developmental disorder.

B. Cribra orbitalia

Cribra orbitalia characterized by coalescing foramina with increased thickening was observed on both the left and right orbits (Plate 13). Due to postmortem damage, the inferior portion of the orbital roofs is missing; therefore, the extent of the lesions cannot be fully determined. The orbits exhibit both sharp and thickened margins, suggesting a mixture of active and healed lesions. The cribra orbitalia is recorded as medium.

Case #8 Description of Cranium

Age: Old adult

Sex: Probable female

The skull is complete, missing only the mandible. Most of the cranial vault was covered in a crystalline matrix that was removed during the curation process. The removal of the calcite matrix damaged the underlying cortical surface. Some of the matrix still adheres to the surface.

Case #8 Pathology

Case #8 does not exhibit observable evidence of pathology.

Case #9 Description of Cranium

Age: Old adult

Sex: Male

The cranium is complete including the cranial base and face, except for a small central portion of the left zygomatic bone and damaged nasal bones. All of the maxillary dentition is present except for the four upper incisors lost in the postmortem interval. There is no mandible associated with the cranium. The skull is in exceptional condition in terms of general continuity and completeness.

Unfortunately, there is a layer of calcite covering most of the cranial vault obscuring the outer cortical surface. While certain abnormal bone changes could be partially observed where the deposits have flaked away, it was determined that the removal of the calcite would result in the significant damage to the outer table (see Case #8). The inner surface of the vault also exhibits significant calcite deposits. As will be discussed below, the skull base exhibits significant trauma that may or may not represent postmortem damage.

Case #9 Pathology

A. Cranial Fractures

Description of Abnormal Bone

The right parietal exhibits two small concentric depressed fractures. The first is located just above the temporal line, on what is likely the coronal suture, although extensive calcite deposits and an obliterated suture obscure the precise relationship. The lesion is elliptical, measuring 11.8 mm medial-lateral and 6.0mm superior-posterior. The anterior margin is deeper and exhibits a slightly steeper edge. The fracture is a singular entity with no radiating fracture lines. While it is difficult to assess the endocranial involvement, inspection via the foramen magnum suggests that both the diploë and the inner table are affected. There is no evidence for healing or associated infection.

A second small concentric fracture is located on the posterior right parietal, 15.5 mm above the lambdoidal suture and 37.2 mm lateral to the sagittal. The lesion is elliptical, measuring 5.3 mm medial-lateral and 7.3 mm superior-posterior. The endocranial surface of the lesion is larger than the ectocranial surface and exhibits a slightly lighter colour. A small fragment of the depressed inner table was found inside the vault. Like the above fracture, there is no evidence for healing or associated infection. A linear fracture, measuring 53.9 mm, radiates towards/from a large fracture on the right lateral aspect of the occipital bone, approximately 10.0 mm superior to the foramen magnum (Plate 14). The fracture is characterized by the complete perforation of the occipital with no depressed occipital bone adhering to the margins. The lesion is somewhat "keyhole" shaped with a circular anterior margin and a triangular/pointed posterior margin. The fracture is 45.2 mm at its longest length (anterior-posterior) and 22.6 mm along across its widest margin (medial-lateral). There is no evidence of healing or associated infection. The linear fracture passes through the large area of missing bone to a small circular lesion, 10.3 mm in diameter between the right occipital condyle and the right mastoid groove, posterior to the jugular foramen. The smooth-walled anterior margin of the lesion is formed by the jugular foramen, while the posterior margin

presents a sharp edge suggestive of recent damage. There is no evidence of healing or associated infection.

Differential Diagnosis

Case 9 exhibits three, possibly four, unhealed lesions of various sizes that are consistent with features of a depressed cranial fracture (Lovell 1997a; Gurdjian et al. 1950:336). While depressed cranial lesions can form under penetrating and sharp force trauma, the force required to inwardly displace the cranial table is usually associated with blunt force trauma (Maples 1985). Small depressed fractures that primarily exhibit external deformation of the cranial vault, minimal involvement of the inner table and the presence or absence of associated fractures suggest impact with a slow moving blunt object (Gurdijan 1950:336). The complete perforation and the obvious detachment of the occipital bone in the keyhole fracture indicate that it formed as a result of a high velocity impact with a blunt object (Gurdijan 1950). The nature of the smaller lesion adjacent to the occipital condyle and the mastoid groove is unclear. Although it appears to be associated with the traumatic event(s) on the superior occipital and parietals, postmortem damage has obscured much of the posterior margin. The relationship of the smaller depressed fractures to the larger keyhole fracture is also unclear. Once the antemortem/perimortem or postmortem origin of the trauma is clarified, it may be possible to establish the sequence of traumatic events.

The margins of the two small, unhealed parietal lesions exhibit concentric fracture lines and depressed cranial bone that is contiguous with the rest of the vault. These features are consistent with perimortem fracture (Berryman and Haun 1996; Hurlbut 2000; Lovell 1997a; Maples 1985; Ubelaker and Adams 1995). However, the margins of both lesions are slightly lighter colour than the rest of the cranium suggesting that the fractures were formed in the postmortem period (Buikstra and Ubelaker 1994; Hurlbut 2000). The perimortem/ postmortem nature of the keyhole fracture is no clearer. The absence of healing and infection, lack of colour contrast, as well as the curvilinear nature of the anterior margin suggests that this lesion formed in the perimortem period (Buikstra and Ubelaker 1994; Maples 1985). While the keyhole fracture's angular and jagged posterior margin, and the recent nature of the damage to the associated jugular foramen is somewhat suggestive of postmortem damage, neither of these are sufficient to clearly distinguish the source of the trauma. It is similarly difficult to establish the origin and timing of the linear fractures. Linear fractures propagate to those areas that are weak and unsupported such as the foramen magnum and jugular fossa (Lovell 1997a: 155; Gurdjian et al. 1950) or similarly, to those areas already weakened by a previous disease or traumatic episode. It is possible that the linear fracture initiated at either the parietal bone or occipital fractures.

While it is clear that the lesions observed on Case #9 are the result of blunt force trauma, neither the perimortem/postmortem origin nor the timing of the lesions can be firmly established. Case #9 is classified as undetermined (undifferentiated) perimortem-postmortem trauma.

B. Porotic Hyperostosis

The frontal glabella and the supraorbital processes exhibit porosity with coalescing foramina and thickening. While the bone has a somewhat spongy appearance, the lesions

do not appear active. There is no observable porosity on the orbital roofs and cranial vault, although much of the vault is obscured with calcite. The porotic hyperostosis was recorded as medium and healed (Buikstra and Ubelaker 1994; Nathan and Haas: 1966; Stuart-Macdam 1985).

C. Periapical lesions of the Right Maxilla

The alveolar margin of the right maxilla exhibits two small circular lytic defects above M^2 and M^3. The defect above M^2 measures 6.0 mm in diameter and the defect above M^3 is 5.9 mm in diameter. The margins are well rounded and there is slight porosity in the zone surrounding the lesions and along the alveolar border suggestive of an actively healing lesion. Despite postmortem damage, the occlusal-mesial surface of the right M^2 exhibits evidence of a large carious lesion (Ortner and Putschar 1984). M^3 is missing postmortem. All the teeth exhibit significant wear. The degree of dentine or pulp exposure was not noted.

While it is not the focus of this thesis to identify and interpret dental disease, the above description is consistent with a diagnosis of periapical abscess secondary to dental caries (Ortner and Putschar 1984:422).

Case #10 Description of Cranium

Age: Adult

Sex: Unknown sex

The partial cranium consists of many refitted and fragmentary pieces. Most of the superior cranial vault, right temporal bone and basi-cranium are preserved. The left temporal bone, face and mandible are missing. The left and right parietal bones and the frontal were refitted from fragments. The anterior parietals and frontal bone were refitted during the curation process, however, portions of both parietal bones are missing the area around bregma and the squamosal suture area of the left parietal is missing as well. The fragmentary frontal bone includes an almost complete coronal suture (>90%) when pieced together. The anterior frontal, including the glabella and orbits are missing and broken across the frontal eminence. Large fractures and small fissures due to postmortem damage occur throughout the superior cranial vault.

Despite postmortem breakage, the cortical surface is in good condition with minimal flaking and spalling. While irremovable sediment stills adheres to some of the surfaces, there are relatively few calcite deposits. Damage due to carnivore/rodent activity is indicated by parallel and approximately equal sized punctures on two small vault fragments. Other eroded patches that may or may not be postmortem in origin will be discussed in the differential diagnosis.

Case #10 Pathology

A. Lytic foci on the mastoid

The mastoid process of the right temporal bone exhibits an area of lytic activity on the outer surface (Plate 15). Six irregularly shaped resorptive foci appear to be comprised of coalescing and irregularly shaped defects. The lesions perforate into the diploë of the mastoid. The smaller lesions, which are not as extensively remodeled as the larger foci, reveal a sub-cortical expansion of the diploic structure. The two larger resorptive foci present smoothly excavated lesions, through which the diploë cannot be observed; the

walls, floor and border are smooth sided. The resorptive defects present a mixture of sharp and well-rounded margins. There is no further evidence of porosity or sclerotic bone formation in the area surrounding the lesions on the mastoid or the rest of the temporal bone. The left temporal bone is missing, therefore no corresponding lesion could be observed.

Differential Diagnosis

Resorptive lesions on the mastoid may be the result of several processes. The differential diagnosis should include normal variation, postmortem damage, and infection, particularly mastoiditis.

According to Mann and Murphy (1990:27), care should be taken to distinguish evidence of mastoid disease from natural morphological variation. Natural fissures on the surface of the mastoid are usually much smaller than those exhibited on the mastoid of Case #10. Further, the sharp-margined lesions that expose the sub-cortical surface may simply reveal the normal air cells of the mastoid; however, the smoothly sided and floored nature of several of the resorptive foci is suggestive of a disease process. Wells (1967) cites the posterior-lateral destruction of the cortical surface of the mastoid process as a common postmortem alteration. In the ABS sample, damage to the mastoid is frequently observed. In Case #10, several of the sharp margined resorptive foci exhibit slight damage incurred in the postmortem interval, however, the well-rounded, smooth sided and coalescing nature of the lesions is indicative of an antemortem disease process.

Mastoiditis is an uncommon infection normally associated with otitis media. While there is no observable evidence (other than the mastoid) of inflammation of the external auditory canal and its surrounding structures, this is not unusual. Evidence of otitis media is often observable only via radiograph or otoscope/endoscope. The resorption and perforation of the mastoid to form pockets within the air cells of the bone are features of mastoiditis (Gregg and Gregg 1987; Mann et al. 1994; Schultz, 1979). These features are consistent with the description of lesions on the mastoid of Case #10. The sharp and well-rounded margins also suggest that the disease may have been chronic and/or was undergoing healing at the time of death.

It is difficult to establish the cause or etiology of lytic lesions on the cranium. On the basis of the above description, mastoiditis is a reasonable diagnosis. Further research, including radiographic evidence of mastoiditis or otitis media, would provide the necessary confirmation. Case #10 is classified as possible mastoiditis dependant on future confirmation from radiographic and otoscope analysis.

Case #11 Description of Cranium

Age: Adult

Sex: Undetermined sex

The partial calvarium consists of portions of the vault excluding the cranial base as well as the face, and mandible. The partial frontal bone is composed of several refitted fragments that include most of the body and the coronal suture; however, it is broken below the crest and excludes frontal glabella and the orbits. A complete left parietal composed of at least five refitted pieces is missing several small fragments along the coronal suture. Most of the anterior 2/3 of the sagittal suture and a small segment of the anterior squamous suture are observable. The cortical surface of the outer table is in poor

condition, and unlike the majority of the cranial elements in this sample, this calvarium has an extremely weathered appearance. Cortical bone on the frontal bone and to a lesser extent on the left parietal bone is marked by a cracked and flaking surface. Many small fracture lines and fissures are particularly noticeable along the temporal line and parietal boss of the parietal bone and frontal body. The endocranial surface is in relatively good condition in comparison with the outer table. There are several decent cross sections of the cranial tables and diploë available for observation.

Case #11 Pathology

A. Lytic lesions of the vault; abnormal bone formation of the diploic table

Description of Abnormal Bone

This cranium exhibits erosive lesions on the endocranial surface of the left parietal bone and on the ectocranial surface of the frontal bone. While both exhibit unusual and destructive bone loss, the lesions present morphological differences that may possibly relate to different etiologies or to different formative/healing stages. Each will be considered separately under this heading.

The endocranial surface of the left parietal exhibits a single destructive focus approximately 25.0 mm adjacent of the sagittal suture and approximately 50.0 mm posterior of bregma (Plate 16). The lesion has an irregular ovoid shape measuring 22.8 mm by 16.5 mm and exhibits extensive involvement of the inner table and diploë. The margins are ill defined and irregular with no apparent sclerotic bone. The cortical surface is completely eroded, revealing grainy diploic bone. The center of the erosive lesion is permeated with deep holes that invade the entire diploic structure. The diploë present a mixture of sharp and indistinct cancellous spaces that appear almost dissolved. There is no observable evidence of new bone formation thickening. The lesion has a distinct brownish colour that is darker and a slightly different shade than other exposed cross-sections of the cranium. Postmortem damage to the erosive lesion has resulted in a white-margin of newly exposed bone circumscribing the lesion.

A second lytic defect can be observed on the left aspect of the frontal body superior to the temporal line on the frontal bone. The lesion is circular, measuring 5.9mm wide and extending 4.6 mm into the diploic layer. Postmortem damage to the anterior border of the frontal obscures the complete extent of the lesion. Unlike the destructive foci described above, this lytic lesion appears smoothly excavated with fine diploic spaces and limited expansion of the cancellous spaces. Significantly, this lesion lacks the "dissolving" appearance characteristic of the above lesion. Despite the postmortem damage on the surface, a slight zone of fine porosity surrounds the defect. The smooth diploë and well-defined margin possibly indicate that the process of abnormal bone loss was slow and chronic; alternatively, these features may also imply that the lesion was in the process of healing.

Cross-sections of the frontal and parietal bones reveal a hypertrophic cranial table. The cortical surfaces of the inner and outer tables are thin, while the diploë appear expanded. Some individual diploic spaces are slightly larger than usual, although most appear normal. The maximum thickness of the cranium measured along the medial exposure of the left parietal bone is 8.4 mm. The outer cortical surface exhibits postmortem damage that is unusual in comparison with the rest of the ABS sample. Small

cracks that give the vault a mosaic-like pattern cover most of the outer surface, and resemble the affects of weathering.

Differential Diagnosis

The following differential diagnosis of Case #11 will consider disease conditions that may explain the relationship of the three abnormal features with the cognizance that the three may be entirely unrelated. The following conditions under consideration are: fibrous dysplasia, infection: non-specific and specific (tuberculosis; mycotic), Paget's disease, eosinophilic granuloma, and hyperparathyroidism.

A mixture of osteoclastic and osteoblastic activity that affects only one bone (monostotic) or many bones (polystotic) of the skeleton characterizes fibrous dysplasia (Aufderheide and Rodríguez-Martín 1998; Olmsted 1981; Ortner and Putschar 1984). While cyst-like destruction, and proliferation of new woven bone characterize this disorder, lesions are usually localized and solitary in expression. Fibrous osseous tissue tends to form in singular round masses, which if unmineralized at death may present in dry bone as a smooth sided bony cyst. If the ossification process is complete at death, the diploë may appear coarse and irregular (Aufderheide and Rodríguez-Martín 1998; Ortner and Putschar 1984:317). This differs from the generalized hyperostotic appearance of the vault and the irregularly trabeculated lytic lesion of Case #11. Fibrous dysplasia usually presents a thin smooth shell of new bone on the periosteal surface and ridged woven bone on the endosteal surface, further distinguishing the normal if somewhat thinned cortex presented in Case #11.

Infectious conditions such as osteomyelitis, mycotic infection and tuberculosis can leave osteolytic lesions on the cranium. Osteomyelitis usually presents as a process of simultaneous bone destruction and bone repair (Ortner and Putschar 1984). The lesion on the ectocranial surface of the frontal bone penetrates deep into the diploic structure, leaving no evidence of endocranial involvement. The lesion vaguely resembles the cloaca or fistula that are often a feature of suppurative osteomyelitic infections; however, the ectocranial frontal lesion does not exhibit the smoothly sided walls or complete perforation characterized by osteomyelitic lesions (Resnick and Niwayama 1988:2565; Roberts and Manchester 1995:127; Rogers and Waldron 1989:612). While the lesion on the frontal may be the early expression of an osteomyelitic infection, the combination of the two lesions in association with the hyperostotic cranial bone suggests that this is not the best explanation. The erosive defect on the endocranial surface of the parietal bone is purely lytic and does not exhibit evidence of bony repair, while the lesion on the ectocranial surface of the frontal bone does exhibit evidence of healing and inflammation.

Fungal or mycotic infections exhibit lytic lesions that are generally of a non-specific nature and are difficult to distinguish in dry bone (Aufderheide and Rodríguez-Martín 1998:213; Ortner and Putschar 1984:224; Roberts and Manchester 1995:73). Both blastomycosis and coccidioidmycosis can be excluded because they are geographically discrete diseases, not known for the Mediterranean (Rothschild and Martin 1993:73, 76). Cryptococcosis presents multiple, discrete lytic lesions occasionally in association with sclerosis and sub-periosteal bone deposition (Aufderheide and Rodríguez-Martín 1998:218), however the lesions are non-specific in both distribution and morphology. It is most frequently observed in older individuals, which may contrast with Case #11. Cryptococcosis does not provide an adequate explanation for the hyperostotic cranial

bone. If only the lytic lesions are considered, then the non-specific nature of the disease means it cannot be entirely excluded.

Tuberculosis is an infectious condition that may leave osteolytic lesions on the cranial vault, although this is uncommon in adults (Ortner and Putschar 1984). When it does express itself on the skull, the large, solitary lesions are primarily lytic with irregular margins that exhibit little or no osteoblastic activity (Aufderheide and Rodríguez-Martín 1998:140; Ortner and Putschar 1984; 162). Osteoclastic resorption tends to begin on the inner surface of the cranial vault and extend towards the outer table. This is similar to the description of the endocranial lesion, but different from the ectocranial lesion. According to Hackett (1975:235), a small sequestrum on the outer surface and extensive superficial erosion on the inner surface is a diagnostic feature of tuberculosis. There is no evidence of a sequestrum associated with either of the lytic lesions. There are similarities between the morphological features of cranial tuberculosis and the description presented for Case #11; however, cranial changes in tuberculosis are generally non-specific and essentially osteomyelitic. Diagnostic evidence for tuberculosis is found on the vertebrae and in the overall patterning of lesions (Manchester 1984; Ortner and Putschar 1984:170; Powell 1991; Roberts and Manchester 1995, Steinbock 1976). The fragmentary cranial vault simply cannot provide the necessary information to accept tuberculosis as an explanation for the lesions observed. Finally, tuberculosis does not adequately account for the well-remodeled lesion on the ectocranial surface, or the hyperostotic cranial vault.

Paget's disease may present a possible explanation for the abnormal changes exhibited on the fragmentary cranium of Case #11. Paget's disease is common disorder that predilects older adults. While the open sutures suggest that the individual was a young adult at death, the exact age cannot be determined, and therefore Paget's disease cannot be ruled out on this basis. In its early stages, Paget's is distinguished by osteoclastic activity on the periosteal and endosteal surfaces leading to osteoporosis circumscripta (Lodge 1967; Rothschild and Martin 1993; Striland, 1991b). Osteoporosis circumscripta is characterized by resorptive loss of diploë. The ectocranial surface of Case #11 exhibits clear evidence of resorbed diploic tissue; however, according to Aufderheide and Rodríguez-Martín, (1998:414), Pagetic trabeculae usually exhibit thickening. This is in contrast with the defect in Case #11, which presents a lytic lesion characterized by greatly diminished trabeculae that appear to dissolve. In Paget's disease the proliferation of the cancellous bone onto the ectocranial surface of the skull results in a patchy "pumice' or "cotton-wool' appearance that is considered diagnostic of the condition (Aufderheide and Rodríguez-Martín 1998:415; Barnes and Peel 1990:132; Olmsted 1981:707). While Case #11 exhibits expansive diploë, the change in trabecular structure is minimal and confined to the sub-cortical region. On the above inconsistencies, Paget's can be ruled out as the cause of the abnormal morphology of Case #11.

Eosinophilic granuloma is one of the three disorders under the umbrella of Histiocytosis X (Aufderheide and Rodríguez-Martín 1998:354; Olmsted 1981:708; Ortner and Putschar 1984:249; Resnick and Niwayama, 1988:2429). Lesions are common to the skull (particularly the frontal bone), where they present as small, localized osteolytic defects with irregular and undulating borders (Olmsted 1981: 709; Ortner and Putschar 1984:250; Resnick and Niwayama 1988:2431). The lytic lesions on the endocranial surface of Case #11 are consistent with the above description. Erosion to the

cortical surface of the vault may also be associated with reactive periostitis and may occasionally form a sequestrum. Lesions may or may not perforate the cranium, and frequently multiple lesions will coalesce and present beveled margins (Aufderheide and Rodríguez-Martín 1998; Barnes and Ortner, 1997: Conway and Hayes 1993; Ortner and Putschar 1984). The lytic defect on the endocranial surface of Case #11 does not exhibit evidence of periosteal reaction, formation of a sequestrum, or beveled margins. It is possible, however, that the white margin that rings the lesion is evidence of a postmortem process that has obscured a circumferential zone of reactive bone. Schüller-Christian's disease, also a disorder of Histiocytosis X, is a chronic disorder that presents multiple lytic lesions throughout the vault and displays varying degrees of healing that may explain the relationship between the two lytic defects. The unusual colour, fuzzy/dissolving trabeculation and hyperostotic diploë are not accounted for in the description of eosinophilic granuloma and histiocytosis X. Based on the above evidence, eosinophilic granuloma cannot be accepted nor entirely dismissed.

The mixture of osteoclastic activity (lytic defects) and osteoblastic activity (expanded diploë), as well as the age and sex criteria observed in Case #11 are descriptively reminiscent of hyperparathyroidism (PTH disorder) (Tam 1989; Zimmerman and Kelley 1982). Hyperparathyroidism is characterized by excessive osteoblastic activity leading to an increase in the deposition of the fibrous matrix (Cook et al. 1998), while unchecked osteoclastic activity results in osteitis fibrosa cystica. Occasionally these cysts (or brown tumors) may present a brownish discolouration (Aufderheide and Rodríguez-Martín 1998:332; Revell 1986:118; Woods 1994:260). While the colour of the lytic lesion on the endocranial surface of Case #11 is brown, the fuzzy trabecular bone does not appear to be consistent. Usually brown tumors present benign non-neoplastic lesions that are smoothly excavated (Aufderheide and Rodríguez-Martín 1998; Ortner and Putschar 1984). Similarly, while there is some excessive deposition of the fibrous matrix, it is usually under mineralized (Aufderheide and Rodríguez-Martín, 1998:331). Unchecked osteoclastic activity leads to diffuse osteopenia, the predominant feature of hyperparathyroidism (Aufderheide and Rodríguez-Martín 1998; Cook et al. 1988). In Case #11, the inner and outer cortical surfaces appear considerably reduced, while the expanded, but otherwise normal looking diploë contrasts with the osteopenia expected in PTH disorder. Hyperparathyroidism cannot be completely dismissed from the differential diagnosis. The unusual colour of the endocranial lesion is suggestive of a brown tumor, however the morphology of the lesion presents a considerable contrast. It may be possible that Case #11 represents the early stages of the formation of a brown tumor. Examination of the post-cranial material for further examples of osteitis fibrosa cystica, as well as histological examination may provide the definitive answer (Aufderheide and Rodríguez-Martín 1998).

Lytic lesions of the cranium present considerable diagnostic problems (Waldron, 1987b). The above discussion considered infectious, metabolic, endocrine and miscellaneous disorders. None could provide a definitive explanation regarding the relationship between the two lytic lesions and hyperstotic vault. It is possible that each aspect of osseous change presented in Case #11 represents a different disease process. Equally, it is possible that the fragmentary and isolated nature of the vault fragments do not present evidence diagnostic of a specific condition. Case #11 is classified as undetermined erosive lesions associated with hyperostotic changes.

Case #12 Description of Cranium
Age: Adult
Sex: Female

Case # 12 is a partially complete cranium.

Case #12 Pathology

Case # 12 does not exhibit evidence of pathology.

Case #13 Description of Cranium
Age: Adult
Sex: Undetermined sex.

This partial cranium consists of the superior cranial vault and excludes the face, cranial base and mandible. A small portion of the frontal bone adheres to the anterior margin, although it is difficult to determine its exact extent because both postmortem damage and pathological changes have obscured the anterior portion of the cranium. Traces of the sagittal and lambdoidal sutures are observable, however, again due to postmortem and pathological changes, most of the sutures are obscured. Partial left and right parietal bones are articulated at the sagittal suture. The right parietal bone is articulated to a partial temporal bone that includes a broken petrous portion and mastoid. The occipital bone includes the superior nuchal area and left and right segments of the medial lambdoidal suture. The occipital is broken below the nuchal area and does not include the base of the cranium. Preservation of the cortical surface is poor. The outer surface appears eroded due to both antemortem disease changes and postmortem processes. Calcite deposition on the left parietal bone, occipital bone, frontal bone and endocranial surface partially obscures the bony changes. While several cross-sections of the cranium are evident along the broken parietal margins, postmortem erosion has obscured their potential usefulness.

Case #13 Pathology
A. Diffuse mixed reaction on cranial vault
Description of Abnormal Bone

This case exhibits diffuse abnormal bone loss and abnormal bone formation on the cranial vault (Plate 17). The cortical bone of the outer table is almost entirely obscured by diffuse abnormal porosity, osteolytic resorption interspersed with new woven and densely sclerotic bone formation. The entire abnormal region of the vault is ill defined and poorly organized. Resorption of the cortical surface has exposed portions of the diploë, and in some cases the periosteal surface of the inner table. In the areas where the cortical bone is intact, discernible porosity with pores >1 mm in diameter proliferates. The porosity exhibits foramina with a mixture of sharp and rounded margins, with little to no coalescing and thickening of the pores. In the areas adjacent to the sagittal suture the lateral mid-right parietal and the occipital lambda the integrity of the outer table is compromised by the resorption of cortical bone and the exposure of diploë. This eroded area is characterized by a slight, extremely irregular looking "worm-eaten" appearance (Hackett 1975; 1976). The resorption of the cortical bone in the occipital lambdoid region has exposed the sutural impressions and sclerotic formation has thickened the serrated margin. Along the mid-sagittal suture, pores with well-rounded margins dot the surface

and abnormal bone with a fine grainy texture has filled in the serrated spaces. Towards the anterior sagittal suture, cortical resorption has exposed portions of the diploic layer, while proliferative bone formation obscures the sutural impressions and covers the anterior portion of the left parietal. The origin of the proliferative bone is difficult to establish. It does not appear to be the result of periosteal apposition; in some places the diploë seems to expand from beneath the thinned cortical bone, and in others, dense sclerotic bone appears remodeled into the cortex. Unfortunately, the cross-sections exhibit considerable postmortem damage and therefore cannot clarify the nature of the osseous formation. The mixture of cortical resorption, porosity and abnormal deposition of bone with a grainy texture give the cranium a "woolly" texture. The overall appearance is a slightly hyperostotic skull with osteitis and eroded cortical surfaces. The endocranial surface and the right temporal appear relatively normal without porosity or new bone formation. The combination of sharp and well-rounded margins as well as both woven and sclerotic bone suggests that the cranium may exhibit mixed healed and active lesions or similarly present a disease process in variable stages of expression.

Differential Diagnosis

Case #13 presents a very unusual looking mixture of erosion and proliferation over most of the observable calvarium. The differential diagnosis will consider the following: neoplasia, fibrous dysplasia, rickets, treponemal disease, osteomyelitis and Paget's disease.

The mixture of osteoclastic erosion and irregular osteoblastic proliferation on the vault of Case #13 is reminiscent of the description of hemangioma and osteosarcoma. Hemangioma usually exhibits cortical resorption thickened with bony spicules that result in a "bubbly" or "honey-combed" appearance. Lesions are usually singular (Anderson 1992; Rothschild and Martin 1993:183; Zimmerman and Kelley 1982:120). This contrasts with Case #13, which has a fuzzy rather than a bubbly appearance. Involvement of the calvarium is very diffuse in Case #13 and not restricted to a single locus (Zimmerman and Kelley 1982:120). Osteosarcoma may result in exuberant bony proliferation and destruction that begins in the diploë and eventually breaks through the thinned cortical surface (Aufderheide and Rodríguez-Martín 1998; Roberts and Manchester 1995; Strouhal et al. 1997; Suzuki 1987). Bone may be characterized by radially arranged spicules that create a sunburst effect or by multilaminate deposition that results in an onionskin appearance (Aufderheide and Rodríguez-Martín 1998:378; Rothschild and Martin 1993:178). While the rough, thickened surface of Case #13 is similar to osteosarcoma in its osseous process, the abnormal appearance of the cranium in question is not as dramatic as most neoplastic conditions (i.e. does not exhibit sun-burst or multilaminated bone). Finally, while osteosarcoma affects a larger area than most neoplasms, nothing in the literature suggests that it affects the entire cranial vault. Pictures of osteosarcoma depict a disease process that is usually restricted to a localized (if large) area (Aufderheide and Rodríguez-Martín 1998; Strouhal et al. 1997).

The mixed osteoclastic/osteoblastic reaction of fibrous dysplasia requires consideration in Case #13. Intense osteoclastic resorption followed by the proliferation of woven fibrous bone is reminiscent of the changes observed on the abnormal vault; however, there are two features that readily differentiate the observed changes from fibrous dysplasia. First, monostotic and polystotic types of fibrous dysplasia are observed

to present single or multiple loci, with well-demarcated margins (Olmsted 1981). This is in clear contrast with Case #13, which is characterized by a diffuse reaction on the observable portions of the vault. Second, while fibrous dysplasia may thin the periosteal surface, the cortex remains smooth and largely intact (Wood 1994:300). Deposition of rough, fibrous bone occurs along the endosteal surface. The surface of the vault in Case #13 is not smooth, and both the periosteal and endosteal surfaces appear to be subject to intense erosion and proliferation. On this basis, fibrous dysplasia is excluded from the diagnosis.

Rickets should also be considered in the differential diagnosis of this cranium. Rachitic changes are essentially characterized by thinned and porotic cortices as well as generalized osteopenia due to the failure of bone to properly mineralize osteoid (Mankin 1974; Resnick and Niwayama 1988: 2096). During the healing stage, the accumulated organic matrix mineralizes resulting in a fine pumice-like appearance (Ortner and Putschar, 1984:274). While diffuse porosity and the fuzzy or woolly appearance of the cranium in Case #13 are reminiscent of rachitic changes, a vitamin D deficiency does not explain the more sclerotic areas of osseous change. Other features noted in rachitic skulls such as craniotabes, and marked frontal/parietal bossing are not observed on the cranium of Case #13; however given the adult age and diffuse abnormal changes it is possible that such features may be obscured. Vitamin D deficiency seems an unlikely possibility in a coastal Mediterranean climate; however, it must be emphasized that nutritional deficiencies may be the result of a host of factors including physiological or malabsorptive disorders, generalized malnutrition and cultural practices (Resnick and Niwayama 1988; Zimmerman and Kelley 1982). Rickets cannot necessarily be discarded from the differential diagnosis, nor can it be accepted based on the observed features. Post-cranial evidence, namely deformities of the weight-bearing skeleton is an essential aspect of any rickets diagnosis (Ortner and Putschar 1984).

The origins of treponemal disease is one of the most emotive and controversial debates in palaeopathology; however, this is more appropriately discussed elsewhere[1]. In short, treponemal disease is not definitively established (i.e. accepted by everyone) for the Old World prior to 1493, although there are an increasing number of putative (and hotly debated) cases (Blondiaux and Alduc-Le Bagousse 1994; Henneberg and Henneberg 1994; Pálfi et al. 1992; Roberts 1994; Striland 1991a; 1994). Despite this, treponemal disease in considered in this differential diagnosis because the osseous features of Case #13 display certain similarities. According to Ortner and Putschar (1984:29), it is generally unwise to eliminate from consideration a condition that is thought not to occur in a given time or region. Our understanding of disease and its prevalence and distribution in the past is continuously evolving, and what we "know" today may be contradicted tomorrow.

Erosive lesions accompanied by extensive, irregular bony regeneration characterize treponemal disease (Roberts and Manchester 1995:152). This is reminiscent of the cranium in question, which is characterized by diffuse abnormal bone loss and bone formation. Erosive lesions on the vault of Case #13 have a somewhat "worm-eaten" appearance that is similar of serpiginous cavitation in the caries sicca sequence (Hackett 1975:233). Dense, extensively remodeled sclerotic bone is characteristic of bony proliferation in treponemal disease. This is in an apparent contrast with Case #13,

[1] See Baker and Armelagos (1988), Dutour et al., (1994), Striland (1991a).

because while there is evidence of bony sclerosis on the cranium, the vault is characterized primarily by the appearance of "fuzzy" or "woolly" cancellous bone and diffuse porosity. Case #13 exhibits extensive involvement of the cranial sutures, including bony sclerosis of the sutural serrations that is not observed in syphilis or other treponemal diseases. Finally, Case #13 does not exhibit evidence of circumvallate depressions, radial scars or the caries sicca sequence that are considered pathognomonic of the treponemal process (Aufderheide and Rodríguez-Martín 1998; Hackett 1975). It is possible that a nongummatous reaction characterized by rough, patchy bone may explain the nature of osseous change, however, this process is not diagnostic of syphilis. The presence of treponemal disease in the absence of orofacial and post-cranial indicators cannot be established (Aufderheide and Rodríguez-Martín 1998).

Paget's disease also presents features that are consistent with the observed osseous changes in Case #13. In its early stages, the disease is characterized by bony resorption leading to *osteoporosis circumscripta* and an overall porous appearance of the skull. Case #13 exhibits diffuse porosity over most of the vault and the outer cortical table is virtually obscured due to mixed osseous activity. According to Aufderheide and Rodríguez-Martín (1998:414), erosion of the diplöe during the initial lytic phase may eventually lead to individual trabeculae thickening during the sclerotic phase. Thickened diplöe seem to expand through the compromised cortical surface. Over time, the proliferation of fine porous bone gives the skull a "cotton-wool" or pumice-like appearance (Olmsted 1981:707). The vault in Case #13, exhibits a mixture of resorption, diffuse porosity and proliferative changes (osteitis) that result in a fuzzy and grainy appearance. While deformation is common in the later stages of the disease, the incomplete nature of the vault makes this difficult to assess (Mirra 1987: 162). Because of the non-specific and extremely variable changes to the vault in question, supporting evidence is required before Paget's could be reliably diagnosed. Images of Paget's disease are highly variable according to each example's unique progression and the stage of the disease. The normally useful cross-section cannot be assessed in Case #13 for the presence of bony nodules, or alternating layers of dense sclerotic and porous pumice-like bone (Olmsted 1981:707; Ortner and Putschar 1984:311). Resnick and Niwayama (1988) consider the ground-glass radiographic appearance, mosaic-patterned microstructure and post-cranial changes to be diagnostic evidence of Paget's disease. Paget's disease is a possible diagnosis for the changes observed in Case #13; however, further research is required.

The diffuse osteitis accompanied with marked sclerosis supports osteomyelitis as a diagnostic possibility. Given the incomplete nature of the calvarium, it is not possible to determine if trauma or a sinus/mastoid/ear infection served as a primary site of direct infection. The lack of bony inflammation on the inner table, (Resnick and Niwayama 1988: 2542) suggests that the putative infection is more likely the result of a primary contiguous scalp infection. While erosion to the outer table has resulted in exposure of the diploë and inner table, the lack of cloacae and sequestrum suggest that the possible infection is non-pyogenic in origin (Rothschild and Martin 1993:63). Non-specific infection or osteomyelitis is difficult to establish beyond a reasonable doubt. The diffuse nature of the lesion in addition to the mixture of sharp pores with densely remodeled bone suggest that the inflammatory changes to the vault are longstanding and at various stages of healing.

While neoplasia and fibrous dysplasia can be excluded from the diagnosis on the basis of their morphological manifestations, treponemal disease and rickets present features that are not readily dismissible. Both Paget's disease and osteomyelitis are potential causes of the changes observed in Case #13. Non-specific inflammatory changes to the vault, resulting in diffuse porosity and bony sclerosis are characteristic of both conditions. Neither Paget's nor osteomyelitis can be ruled out on the basis of the vault evidence presented, and both conditions present non-specific macroscopic changes. In the absence of diagnostic evidence for Paget's disease, it is extremely difficult to determine if the abnormal bone is the result of an infection. Case #13 is classified as an inflammatory response of undetermined origin.

Case #14 Description of Cranium

Age: Young adult

Sex: Probable female.

The partially complete skull is missing most of the face, the anterior cranial base and the mandible. The parietal bones, left temporal bone, superior occipital and frontal body are complete and in articulation. The anterior petrous portion is broken along with the zygomatic process of the left temporal bone. The superior 2/3 of the occipital bone is present but broken posterior to the foramen magnum. Postmortem damage has broken the anterior frontal bone at the left supraorbital foramen and along the frontal glabella exposing the frontal sinus. The intact right supraorbital process is broken towards the frontal temporal line. The left aspect of the greater wing of the sphenoid bone is still in articulation. The cortical bone of the inner and outer tables is in good condition with minimal amounts of sediment adhering to the surface. There is a small section of diploë exposed on the right aspect of the frontal bone that may prove useful in the differential diagnosis.

Case #14 Pathology

A. Bone mass on the frontal

Description of Pathological Bone

On the anterior right frontal boss, medial to the temporal line, there is a singular focus of abnormal bone formation. The formation is characterized by a dense sclerotic bone with an "ivory-like" texture that is restricted to the periosteal surface of the outer table. The margins are indistinct from the contour of the bone. The sclerotic bone appears to have formed in layers, giving the formation the onionskin appearance described by Buikstra and Ubelaker (1994:138) after Ragsdale (1993:467) and typically associated with a number of periosteal reactions including osteosarcoma, eosinophilic granuloma, treponemal disease and normal growth. The lesion measures 9.7mm across the anterior-posterior diameter. Small fissures radiate from the raised area of bone formation. The left frontal boss also exhibits similar fissures, however the area is only slightly raised above the contour of the bone. Postmortem damage has resulted in the cortical surface flaking away. It is possible that the onionskin layers are a product of postmortem damage.

Differential Diagnosis

Ragsdale (1993:467) alternatively describes two periosteal responses that are consistent with the osseous lesion described in Case #14. The first is a solid continuous

periosteal reaction and the second is the "onion-skin" lamellated, continuous, periosteal mass; both apply, because slight postmortem damage to the lesion may have enhanced the layered appearance of the lesion. Conditions presented by Ragsdale (1993) relevant to this differential diagnosis include: eosinophilic granuloma, osteomyelitis, osteosarcoma, osteoid osteoma. Ewing's sarcoma, stress fractures and enchondromas are not typically observed on the frontal bone, and therefore not considered here (Aufderheide and Rodríguez-Martín 1998; Ortner and Putschar 1984). Eosinophilic granuloma was likely included in Ragsdale's (1993) differential diagnosis of dense bony lesions because the osteoclastic activity that characterizes the disorder may occasionally stimulate periostitis on the overlying cortex and sclerotic margins along the lytic lesion (Aufderheide and Rodríguez-Martín 1998: 354; Ortner and Putschar 1984:250). This contrasts strongly with the lesion observed on the frontal bone of Case #14, where there is no evidence of osteolytic activity or diffuse periostitis. Similarly, active osteomyelitis can be rejected as responsible for the lesion because it does not exhibit the increase in porosity or vascularity commonly associated with bony inflammation (Aufderheide and Rodríguez-Martín 1998; Steinbock 1976). Healed osteomyelitis or idiopathic osteitis are possible explanations, however remodeling usually leads to irregular, bulky surfaces (Zimmerman and Kelley 1982) with evidence of the healed porosity. This contrasts with the discrete and smoothly sided lesion observed in Case #14.

Cranial osteosarcoma is also frequently responsible for the formation of dense bony deposits (Suzuki 1987). It can be excluded from this differential diagnosis on the basis that is most commonly observed in older individuals with Paget's disease (Aufderheide and Rodríguez-Martín 1998; Waldron 1996) and Case # 14 was a young adult at death However, while it is extremely rare, it is important to note that Strouhal et al. (1997) identified a cranial osteosarcoma in a young adult in a late medieval, Eastern European context. The fact remains that osteosarcoma is rare in young adults, and is therefore not the most parsimonious explanation.

Osteoma is a benign primary neoplasm that predilects the frontal bone and parietal bones. It is characterized by the formation of dense, "rock-hard" (Capasso 1997:618) tissue (Ortner and Putschar 1984:368). Usually this forms as a 'button osteoma', which is a dense, ivory-like formation usually smaller than 2cm in diameter. A circular constricting margin is common (Ortner and Putschar, 1984:368). The lack of a constricting margin contrasts with the mass on the frontal bone Case #14; however, Ortner and Putschar (1984:368) state that the margin is "usually" observed, and therefore not without exception. Also, Capasso (1997) notes that osteoma (he does not refer specifically to 'button osteoma') is most commonly found on the frontal bone. His descriptions of osteomas as hyperostotic, protruding masses do not refer to constricting circular margins. The differential diagnosis of Case #15 suggests that osteoma is a tentative explanation for the abnormal changes observed on the frontal. Further examination of the cross-section and microstructure of the lesion may confirm this diagnosis. Increasing micro-structural density, a reduction of trabecular spaces and a widening of the individual trabeculae are indicative of osteoma (Capasso 1997).

Case #15 Description of Cranium

Age: Old adult

Sex: Probable female.

The partial cranium includes articulated parietal bones, frontal bone, superior occipital bone including a partial basi-cranium and foramen magnum, a complete left temporal bone and a partially complete right temporal bone broken to the anterior of the squamous suture. The face and mandible are missing. While the cortical surface is in relatively good condition, there is a large depressed fracture on the left parietal boss. The fresh, white margin suggests that the fracture is of recent postmortem origin.

Case #15 Pathology

A. Mixed reaction on cranial vault

Description of Abnormal Bone

There is a large, discretely circumscribed area of mixed abnormal bone loss and abnormal formation located anterior to bregma on the frontal bone and extending posteriorly along the sagittal suture on the right parietal bone (Plate 18). The lesion covers between 1/3 and 2/3 of the superior vault, measuring 81.0mm x 40.5mm. A number of (>10) circular depressions with smooth surfaces and densely sclerotic borders are located within the confines of the circumscribed area. The depressions vary in size with the average is measuring 13.0mm in diameter, and the largest 16.2mm in diameter. The centers of several of the depressions exhibit small circular pits (<2 mm) with smooth floors and walls with well-defined sclerotic margins. Four circular depressions with slightly raised outer-margins delineate the left-anterior portion of the circumscribed area. In between the depressions, raised areas or 'hills' of dense sclerotic bone can be noted. The right-anterior portion of the border of the circumscribed area is partially defined by a large mound of bone that protrudes considerably above the contour of the vault. The posterior margin of the lesion is defined by five depressions that also exhibit densely sclerotic margins. The five depressions located anteriorly on the frontal bone do not cross the coronal suture; similarly, the five located at the posterior boundary of the lesion do not cross the sagittal suture of the right parietal bone. Between the two major concentrations of depressions, there is an area of bone formation, raised above the contour of the vault in a plateau of bone. This plateau exhibits a well-remodeled mixture of lamellar bone and very slight perpendicular spicules. Along the plateau and within the posterior depressions there are small well-remodeled areas of diploic exposure, but no penetration of the inner table. Anteriorly exposed diploë are remodeled and discrete, while the posteriorly exposed diploë (within the depression) present sharper margins. The entire surface of the lesion is covered with fine porosity and a slightly striated/crenellated appearance. In addition, small pores with well-rounded margins, particularly on the anterior lesion suggest that the anterior portion of the lesion demonstrates longstanding activity in comparison to the posterior aspect lesion. While most of the abnormal bone loss and bone formation is restricted to the outer table and diploë, the endocranial surface of the cranium exhibits slightly convex areas that reflect the ectocranial depressions. As well, the interior of cranium has a slightly feathery, vascular appearance in the area of the lesion and along the sagittal and lambdoid sutures. Transillumination of the vault does not reveal significant osteopenia.

Differential Diagnosis

The mixed resorptive and proliferative lesions on the cranial vault of Case #15 may be the result of a number of different processes. This differential diagnosis will consider trephination, Paget's disease, scalping, treponemal disease, and non-specific infection.

The circumscribed ovid and sclerotic nature of the lesion is suggestive on first observation of a large, well healed trephination. Upon further analysis, there are a number of features that distinguish Case # 15 from trephination. First, there is no evidence of cut marks, scraping or grooving, serrated edges or externally beveled margins that are considered indicative of the operative procedure (Campillo 1984:277; Lisowski 1967:662-664). Second, the transition of abnormal bone to normal bone is circumscribed on the cranium of Case #15 and is very discrete. In comparison, trephinated margins even those that are well healed, are usually extremely well defined (Campillo 1991; Germanà and Fornaciari 1992; Lisowki 1967). While the individual depressions on the cranium in question lie below the contour of the skull, the overall circumscribed area lies above the contour of the skull. This is not an observation that is consistent with a procedure that removes significant amounts of bone. While well-remodeled surfaces are common on trephinated skulls (Bennike 1985; Campillo 1991; Germanà and Fornaciari 1992; Jennbert 1991), the series of small depressions, each with their own sclerotic border, small circular pits, and finely crenellated bone are suggestive of another process.

While Case #15 does not present bone that is typical of full-blown Paget's, the observable features on the calvarium are reminiscent of the description of the disorder presented in Case #13[2]. The advanced age of the individual in question is consistent with a diagnosis of Paget's because it is broadly considered a disease of old age (Aufderheide and Rodríguez-Martín 1998; Resnick and Niwayama 1988; Striland 1991b; Wood, 1994). The circular depressions on the cranium are comparable to those found in early Paget's (Lodge 1967:407; Ortner and Putschar 1984: 309; Wood 1994: 266), although they do not exhibit cortical porosity or thinning (Ortner and Putschar 1984). The endocranial surface of the cranium is feathery and exhibits numerous small vascular markings that are consistent with the hypervascularity of Pagetic bone (Bell and Jones 1991; Roberts and Manchester 1995; Zimmerman and Kelley 1982). As well, the circumscribed margin of the lesion on the calvarium is analogous to the sharply marked and advancing border of resorption that is observed in Paget's disease (Ortner and Putschar 1984:309; Wood 1994:266). However, neither of these characteristics is diagnostic of the disease process. Finally, the dense lamellar bone formations are not consistent with the fine, pumice-like diploic bone that proliferates over most of the cranium and presents Paget's most notable macroscopic feature (Aufderheide and Rodríguez-Martín 1998:415; Barnes and Peel 1990:132; Ortner and Putschar 1984:310). The mosaic-patterned microstructure and the radiographic "ground-glass" appearance provide diagnostic evidence of the disease (Resnick and Niwayama 1988; Olmsted 1981). While, further analysis is required to definitively rule out Paget's disease, other diagnoses are more consistent with the osseous changes of Case #15.

The circumscribed lesion with diffuse osteitis exhibited on the superior cranium of Case #15 should be distinguished from scalping. Cutmarks, circumventing the skull or lesion, are generally considered diagnostic evidence of scalping. There is no evidence for

[2] Dry bone pictures of early Paget's disease or osteoporosis circumscripta are rare in the palaeopathological and clinical literature.

cutmarks on the calvarium in question; however, given the degree of dense bony remodeling it is conceivable that such evidence would be obscured. Removal of bony tissue is rare in archaeological examples of scalping (Steinbock 1976:25), and in any event, it does not adequately explain the geographic surface of the lesion. A more likely possibility would be osteomyelitic infection due to a contiguous infection via the traumatized periosteum and scalp. Florid infection of the vault due to scalping frequently exhibits an osteoclastic ring analogous to the sequestrum, bony necrosis and granular formation of new bone (Hamperl 1967:632; Hollimon and Owsely 1994). Less severe reactions may exhibit diffuse periostitis/osteitis. Both reactions eventually resorb into the cortex to form a smooth uneven surface (Hamperl 1967:632; Hollimon and Owsely 1994:350-351; Steinbock 1976:27). While Case #15 does not exhibit the osteoclastic ring or extensive bony necrosis, the densely remodeled surface is consistent with the well-healed osteitis observed on the calvarium in question. Based on the above description, scalping cannot be ruled out, however, it must be noted that there is a lack of precedents in Europe at this time. In the absence of other supporting evidence of scalping, it is more reasonable to suggest that the skull exhibits a possible inflammation that may be the result of a number of routes, including scalping.

The justification for considering treponemal disease has been previously addressed (see Case #13). The multiple circular depressions surrounded by sclerotic bony nodules and diffuse periostitis is very similar to the appearance of the circumvallate cavitations of discrete series gummatous syphilis (Aufderheide and Rodríguez-Martín 1998; Hackett 1975). The small circular pits and slightly striated surface of the densely remodeled depressed foci are also reminiscent of the radial scar that forms from the healed circumvallate cavitation (Hackett 1976). Steinbock (1976:150, Figure 62) presents a photograph of an Australian Aborigine with cranial yaws exhibiting two circular depressions with similar morphology to those exhibited on the calvarium of Case #15. While not considered diagnostic of syphilis, plaques of dense bone may be observed on the outer surface of the cranial vault in the early stages of syphilis (Hackett 1976:67). In time, this may be remodeled into the cortical surface leading to a greatly thickened cortex. This is in keeping with the observation of a plateau of bone between the areas of the depressed lesions (Aufderheide and Rodríguez-Martín 1998:162). While the inner table is infrequently involved in treponemal disease (Aufderheide and Rodríguez-Martín 1998:163; Ortner and Putschar 1984) the mild endocranial features observed on the cranium are not necessarily inconsistent with a diagnosis of syphilis (see for example Hackett 1976:37).

The features that were observed on the calvarium of Case #15 are consistent in many respects with the description of treponemal disease, and in particular acquired syphilis. Aufderheide and Rodríguez-Martín's (1998: 162) characterization of a "geographic" skull with smooth valleys and depressions intersected with plateaus of bone is certainly reminiscent of the abnormal bone in question. However, neither the "rolled over" circumvallate cavitation nor the classic caries sicca scars that are considered diagnostic criteria are observed on the calvarium in question. While there are slight similarities in morphology, the depressions cannot be confidently described as a radial scars, nor is diffuse periostitis/osteitis inconsistent with other disease conditions (particularly non-specific infection). Finally, there is no evidence of treponemal disease

in Europe on the order of 4500 bp[3]. While the disease was considered because it provides a reasonable explanation for the morphology of the lesions, the temporal and areal context of ABS in addition to the accepted disease history recommends a conservative judgment. Without the supporting rhinomaxillary and in particular post-cranial evidence, the ascription of the calvarium to treponemal disease would be extremely premature and misguided. Other avenues of investigation such as microscopy, DNA and immunological studies may provide evidence that is more conclusive[4].

As mentioned above, the mixed bony resorption and proliferation observed above is consistent with bony inflammation due to non-specific infection; however, it must be noted that Case #15 does not exhibit the characteristic hallmarks of pyogenic infection, namely the sequestrum and cloacae. Infections that result from hematogenous spread (secondary source) or via a compound fracture tends to exhibit erosive effects to the inner table that eventually work their way to the outer table (Resnick and Niwayama 1988:2542). This is not consistent with the above description of Case #15. According to Ortner and Putschar (1984:119), contiguous scalp infections often lead to circumscribed areas of "perifocal sclerosis" that are limited to the outer table and diploë. This is more consistent with the observed features of the calvarium in question. Chronic nonsuppurative osteomyelitis (osteomyelitis of Garré), characterized by dense "fusiform thickening of the cortex" and no evidence of a cloaca or sequestrum (Aufderheide and Rodríguez-Martín 1998:178) is also consistent with the description of the abnormal bone in question. By its very nature, non-specific infection is generally indistinguishable from other lesions exhibiting mixed reactions. The smooth-walled depressions, rounded margins, slight porosity and advanced osteitis in conjunction with areas of differential activity/healing, suggest that the most parsimonious explanation for this lesion may be a long standing infectious inflammatory condition.

Case #16 Description of Cranium

Age: Adult

Age: Probable female

The partial cranium includes parietal bones articulated to the superior occipital bone that is broken below the inferior nuchal line, and a partial frontal bone. Both the complete left temporal bone and partial right temporal bone were refitted during the curation process. The partial frontal bone is broken just above the frontal glabella and the supraorbital arches. The face, cranial base and mandible are missing. The cortical surface is in good condition except for the adherence of sediments to the endocranial surface. The ectocranial surface exhibits some sediment and small black patches or stains of unknown origin[5]. The anterior right frontal exhibits evidence of postmortem crushing. While cross sections of the diploë are exposed, they are not directly associated with the areas of diseased bone.

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[3] Steinbock (1976:97) reports that several long bones dated 100-800 B.C, two tibiae dated 500-200 B.C and three skulls dated 100-700 A.D. from Siberia are the oldest examples in the Old World. The oldest purported examples of acquired syphilis in Western Europe date to 580-250 AD in southern Italy (Henneberg and Henneberg, 1994) and 3rd-5th AD in France (Palfi et al., 1994).

[4] See Dutour et. al. (1994): L'Origine de la Syphilis en Europe, Avant ou Après 1493.

[5] Some of the fragmentary pieces from both the adult and sub adult sample exhibit similar small black patches that appear to be post-mortem in origin. The cause of the staining is unknown.

Case #16 Pathology

A. Diffuse abnormal formation on the cranial vault.

Description of the Abnormal Bone

The cranial vault exhibits diffuse abnormal bone formation with two main foci. The larger of the two foci is located on the posterior frontal bone and the superior regions of the left and right parietal bones (Plate 20). The vault exhibits differentially defined margins, which present marked differences in transition from normal to abnormal bone. The amorphous margins make it difficult to acknowledge and measure the extent of the affected area. The anterior of the lesion is comparatively well defined, with an irregular margin of densely formed sclerotic bone that protrudes 9.0 mm above the contour of the vault. This thick bony prominence continues around marking the lateral-right margin of the lesion. The lateral-left and posterior aspects present an amorphous boundary (as opposed to margin) that does not exhibit a marked transition with the normal bone. The maximum outside anterior-posterior diameter of the lesion is approximately 125.0mm and the maximum outside left-right diameter is approximately 101.0 mm. This large focus has a "geographical surface" characterized by slight depressions circumscribed by well-rounded sclerotic prominences and plateaus of bone. The area within the boundary of the lesion presents an irregular mixture of features. Shallow circular depressions with gradual and slightly palpable margins are interspersed with plateaus and patches of rough, finely porous bone, spicules of newly formed periosteal bone and densely remodeled spicules that have a shiny, ivory-like appearance. Several of the dense ivory-like spicules exhibit a striated appearance that resembles the "combed" bone described by Hackett (1976).

Diffuse reactive bone, characterized by porosity extends beyond the central focus onto the posterior region of the parietal bones and occipital. Three small areas of coalescing lytic activity occur outside the main lesion. On the anterior right parietal bone, adjacent the coronal suture there is an area of coalescing porosity that has permeated the cortex into the diploë. It is <10.0 mm in diameter and appears slightly damaged due to postmortem processes. The second lytic lesion is located on the left parietal bone adjacent to the sagittal suture inside the main lesion. The third lesion is located outside the main lesion on the posterior portion of the right parietal bone, just below the parietal foramen. Both lesions present sharply margined pores that are beginning to coalesce. A fine hairline crack extends from the anterior region of the frontal bone to the area surrounding the parietal foramen that appears to be postmortem in origin. Other than the small lytic focus on the right parietal, the skull does not exhibit involvement of the diploë or inner table. Given the diffuse, remodeled nature of the periosteal bone, the large bony prominences and small, shallow valleys, Case #16 can be described as exhibiting diffuse osteitis.

The second of the two major foci is located on the lateral, posterior right parietal and appears to be an isolated extension of the larger focus. The lesion is a discrete oval (35.3mm maximum diameter) that lacks a definitive border of bone. It is most noticeable when the skull is palpated. The lesion is characterized as a flat tableau of bone that does not appear to have formed through periosteal apposition or osteoclastic resorption. Rather, it appears to be a well-remodeled and restricted portion of the cortex. The bone on the surface exhibits a slight grainy porosity coupled with sclerotic bone formation that is similar to the rest of the skull. The endocranial surface appears normal; however, there

is a generally fuzzy or grainy texture to the bone that is similar to the endocranial surfaces of Case #15.

Differential Diagnosis

The diffuse osteitis of the cranial vault accompanied with minimal osteoclastic resorption does not suggest many diagnostic possibilities. The reaction exhibited by the cranial vault is too diffuse and poorly marginated to be considered neoplastic. Trauma, congenital disease and trephination do not present lesions similar in morphological expression, size and extent. Porosity, bony prominences, and spicules of variably remodeled new bone do not suggest anemia or other hematological or endocrine disorders. The osseous reactions of the two metabolic disorders, Paget's disease and flourosis, should be briefly considered, followed by infectious inflammation typical to treponemal and non-specific infection.

Thinned cortices and trabecular thinning with sharply marked borders characterize osteoporosis circumscripta previously described in Case #13 (Lodge 1967; Ortner and Putschar 1984). However, Case #16 does not exhibit a significant amount of bone loss, nor is the newly formed bone reminiscent of the fine "pumice-like" cancellous bone commonly associated with Paget's disease. Therefore, Paget's disease can be quickly excluded from the differential diagnosis.

Fluorosis is very rare in archaeological contexts (Aufderheide and Rodríguez-Martín 1998:318) and is usually recognized due to dental alterations and dramatic formation of thick sheets of periosteal bone and large excrescences (Littleton 1999: 466; Zimmerman and Kelley 1982:69). While involvement of the skull is very rare (Resnick and Niwayama 1988:3072), the presence of osseous spicules and considerable thickening of the cancellous spaces of fluorosis are reminiscent of Case #16. In contrast with fluorosis is the diffuse porosity observed over much of the vault. Malformation and discoloration of the teeth as well as post-cranial evidence of florid new bone formation would provide the diagnostic evidence needed to accurately recognize this disorder. Based on the observed information it is not possible to discount fluorosis; nonetheless, given the rarity of the disorder in the past and the non-dramatic nature of the lesion, a more suitable explanation may be found.

Inflammatory processes due to specific or non-specific infection also need to be considered in the differential diagnosis. Similar to Case #15, the cranium in question exhibits morphological characteristics that are reminiscent of treponemal disease. The diffuse cranial reaction featuring periosteal inflammation, porosity, rough patches, remodeled spicules and irregular cortical thickening are also typical non-gummatous syphilis (Steinbock 1976:116; Zimmerman and Kelley 1982:99). According to Resnick and Niwayama (1988: 2700), nongummatous syphilis is usually limited in its extent and may lead to the occlusion of the marrow and diploic cavities; however, there are no cross-sections on the cranium available to test this observation.

While non-gummatous syphilis can occur in conjunction with gummatous syphilis, erosion and subsequent regeneration characteristic of the caries sicca sequence do not coincide with the bony prominences on the frontal bone of Case #16. The area adjacent to the sclerotic bone formations exhibits insufficient erosion to suggest that either of the discrete or contiguous caries sicca sequence is present. The morphology of this aspect of the lesion is puzzling. The superficial, coalescing cavitations in the area peripheral to the

larger lesion are suggestive of the focal bone destruction and clustered pits of early syphilis (Hackett 1976:34). Nevertheless, none of the observed features of Case #16 are considered to be diagnostic of treponemal disease (Hackett 1976). While syphilis/ treponemal disease cannot be ruled out of the differential diagnosis, Steinbock (1976:64) warns that the diagnosis of treponemal disease based upon the expression of non-specific periostitis or osteitis on a single bone is difficult and prone to error. In this regard, the analysis of the post-cranial material for evidence from the individual associated with the cranium or from the population (which would likely be the case in a collective burial) should be the next avenue of research.

The differential diagnosis of non-specific infection in Case #16 is again similar to Case #15. The osteitis does not involve the inner table, which suggests that the infection is not the result of hematogenous spread or the direct introduction of a pathogen via a depressed fracture (Resnick and Niwayama 1988:2542). Second, the superficial nature of the inflammation and the lack of a sequestrum or cloaca suggest that pyogenic infection is not responsible for the morphological features observed on the cranium. Inflammation due to a contiguous scalp infection provides the most conservative explanation. The circumscribed nature of the anterior lesion, in addition to the periosteal formation of bony spicules, diffuse porosity and irregularly thickened cortex are also consistent with Ortner's and Putschar's (1984:119) description of "perifocal sclerosis". Similar to Case #15, and despite its rarity, the possibility of chronic nonsuppurative osteomyelitis should also not be discounted (Zimmerman and Kelley 1982). Inflammation of the vault due to a scalping incident cannot be discounted either. While there are no cut marks to suggest the traumatic avulsion of the scalp, the degree of remodeling exhibited by the vault would likely obscure such evidence (Hollimon and Owsley 1994; Steinbock 1976). In addition, it is not infrequent to observe a well-circumscribed area of osteitis with diffuse periostitis in cases of scalping (Hamprel 1967; Hollimon and Owsley 1994).

The infectious inflammation of the cranial vault in Case #16 appears to be of longstanding origin, however, small areas of active porosity suggest that the process is still active. The origin of infection is unknown; however, this is not surprising given the non-specific nature of the inflammation. Case #16 is classified as nonspecific infection of the vault resulting from an unknown secondary origin.

Case #17 Description of the Cranium

Age: Old adult

Sex: Male

The partial cranium is comprised of a partial vault with articulated parietal bones, a partial superior occipital bone along the lambdoidal suture and the complete left face. Postmortem damage has resulted in a missing right face, superior right parietal bone and the basi-occiput. No mandible was found in association with the cranium. The face is comprised of a partial frontal bone including the orbital margins, the left and right nasal bones and both maxillae, and *in situ* teeth. The supraorbital margins of the frontal bone are present but the right is broken medially of the zygomatic process. Postmortem damage to the frontal bone around glabella has exposed the frontal sinus. Damage to the right area of the frontal bone and parietal bones commence above the right orbit and curves posteriorly to the midsagittal area of the parietals. The damaged margin in this region is missing large scallops of bone. Most of the anterior and mid-portions of the

right parietal bone are missing, along with the anterior portion of the left parietal bone. There is no frontal or parietal bregma, and most of the coronal suture and the anterior part of the sagittal suture are missing. The left PM^1, PM^2, M^1 and M^2 and the right PM^2, M^1, and M^2 remain *in situ*, while the left M^3 was lost in the antemortem period and the socket resorbed. Postmortem damage to the left C, I^2 and the right PM^1 and M^3 has left only the roots. The anterior alveolar margin also exhibits postmortem damage.

Along the broken margins, the exposed diploë exhibit a similar colour to the rest of the vault suggesting that the postmortem damage is old. The rounded margins suggest that the lesion formed when the bone was still fresh/green; however, the extensive nature of the damage, the sharp nature of the diploë and lack of reactive bone suggests either a perimortem lesion or postmortem damage. Animal activity may provide a possible explanation for the damage observed on the superior cranial vault. Two small parallel punctures on the frontal, small fissures likely due to compressive forces and a raggedly scalloped border are suggestive of carnivore activity. According to Binford cranial attrition by carnivores may remove a "skull disc" (Binford 1981:62), which, early researchers have misinterpreted as evidence of human modification (see Brueil 1939; Dart 1962; Kitching 1963). The curved or scalloped margins of the remaining vault may reflect such a process. Further, the externally beveled margin suggests that damage was not a result of blunt-force trauma (Berryman and Haun 1996:4; Lambert 1997:84). While externally beveled margins are commonly associated with trephination, the lack diagnostic evidence such as cutting or scraping marks (which would be expected if the damage was of perimortem origin) suggests that surgical intervention was not responsible (Campillo 1984). While, the full extent of the damage cannot be appreciated, the breakage to the superior vault in Case # 17 appears to be postmortem in origin. The preserved surfaces of the cranium are in good condition with no evidence of calcite deposits.

Case #17 Pathology

A. Perforation of the left parietal

Description of Abnormal Bone

The left temporal bone and left parietal bone share a circular perforation immediately adjacent the parietal-mastoid and occipital-mastoid articular region and less than 5.0 mm superior of the lambdoidal suture (Plate 21). The ectocranial aspect of the lesion is well defined and regular, measuring 19.1 mm in maximum diameter, with the lesion on the endocranial surface measuring 18.85 mm in maximum diameter. A small densely remodeled osteophyte projects into the perforation from the posterior margin. The margins exhibit a steep, smoothly remodeled external bevel, and the zone around the lesion is palpably depressed. The lesion has a "punched in" appearance, despite exhibiting significant remodeling. Part of the region adjacent the lesion exhibits slight porosity and a plaque of new bone with thickening lamina/striations - evidence of an initial woven response. The margins and area surrounding the perforation are characterized by smooth, densely remodeled sclerotic bone. In places, this sclerotic bone is slightly raised above the depressed contour of the vault. The area of remodeled sclerotic bone is thickest nearest the lesion and thins as it proceeds superiorly onto the left parietal bone.

Approximately 25.0 mm above the perforation is a small lytic focus of exposed diploë. The lytic lesion is well defined with a scalloped margin and measures 5.0 mm in diameter. The exposed trabeculae are rough and irregular, while the margins surrounding the lesion exhibit slight evidence of remodeling. Above this small lytic lesion are two slight depressions that are notable when palpated and represent the superior extent of the entire affected area. The endocranial aspect of the perforation provides no evidence of a reaction or a reflection of the above sclerotic activity. The region adjacent to the lambdoidal and sagittal sutures exhibit an area of diffuse finely remodeled porosity indicative of periostitis.

Differential Diagnosis

The differential diagnosis of a singular circular defect of the cranial vault with an associated osseous response should include: osteolytic neoplasms, eosinophilic granuloma, treponemal disease and tuberculosis infection, depressed fracture, trephination and osteomyelitis (Kaufman et al. 1997). Congenital abnormalities can be excluded due the location of the lesion and the intense bony reaction associated with the perforation. Postmortem damage can also be excluded given the obvious osseous reaction.

Both multiple myeloma and metastatic carcinoma are osteolytic neoplasms that present well-defined circular defects on the cranium (Aufderheide and Rodríguez-Martín 1998; Strouhal 1991); nonetheless, they can be distinguished from Case #17 on a number of criteria. While multiple myeloma may present as a singular lesion, they almost never exhibit sclerotic margins (Strouhal 1991), which contrasts with the densely sclerotic bone surrounding the lytic lesion in Case #17. Osteolytic metastatic carcinoma frequently forms multiple diffuse lytic lesions associated with a "motheaten" periphery and slightly sclerotic margins (Ragsdale 1993; Strouhal 1991; Webb 1995). While the possibility of further lytic lesions cannot be conclusively discarded given the incomplete nature of the cranium, the lesion is singular in its expression on the remaining material. The periphery of the lesion exhibits a thickened cortex due to an apparent osseous inflammation. Further, densely remodeled bone in the region of the lesion and along the margin of the lytic defect is remarkable in comparison to the slightly sclerotic nature of the margins in metastatic carcinoma. Neither forms of neoplasia appear to be consistent with the description of Case #17.

Another osteolytic neoplasm that frequently presents as a singular lytic defect is the epidermoid bone cyst. The cysts are characterized by circular perforations that measure between 1-5cm in diameter. These can have densely sclerotic margins that may exhibit a raised bony contour and a "highly vascular appearance" (Brothwell 1967:320). While these features are consistent with the above description, according to Resnick and Niwayama, (1988: 3831) epidermoid bone cysts can be differentiated from eosinophilic granuloma because they lack a beveled margin. The steep, external bevel is a notable feature of the perforation in Case #17.

Eosinophilic granuloma presents lytic defects with similar features to those described in Case #17. Single or multiple resorptive defects with irregular, beveled and slightly sclerotic margins as well as a button sequestrum are features of the eosinophilic granulomatous lesion (Barnes and Ortner 1997; Resnick and Niwayama 1988). Again, while eosinophilic granuloma may exhibit sclerotic margins, there is no indication in the

literature that the lytic defects are associated with a broad zone of remodeled inflammatory bone such as observed in the specimen in question. Further, eosinophilic granuloma is a disorder that predilects juveniles and young adults. The individual in Case #17 is clearly an older adult, and while the lesion is significantly healed, there also appears to be a differential degree of healing in the areas surrounding the lesion (porosity and striated bone). In addition, a small resorptive defect in conjunction with the advanced age of the remains suggests that these lesions occurred later in life.

Treponemal infections may result in osteoclastic resorption leading to perforation of the cortex (Aufderheide and Rodríguez-Martín 1998; Steinbock, 1976). Syphilitic lesions predilect the frontal and parietal regions of the vault (Hackett 1975, 1976; Herskovitz et al. 1994, 86; Rogers and Waldron 1989:620), therefore a singular perforation low in the parietal-temporal area and occipital region would be very unusual. While portions of the right vault are missing, the presence of an unaffected frontal and left parietal bone suggests that the remaining vault did not exhibit corresponding lesions. Tuberculosis lesions may also result in a perforated cortex. The lesion usually forms on the inner table and proceeds outwards. The margins of the perforations are usually sharp and do not exhibit evidence of proliferative bone (Ortner and Putschar 1984). Frequently a sequestrum with an associated zone of superficial erosion is found on the endocranial surface (Hackett 1976:73; Murray 1990). According to Hackett (1975:235), this constellation of features is considered diagnostic of cranial tuberculosis. These features of tuberculosis are inconsistent with the abnormal changes observed on the cranium of Case #17. Therefore, a *Mycobacterium tuberculosis* infection can be excluded from the differential diagnosis.

A well-healed depressed fracture may also provide a reasonable explanation for the circular defect and associated bony inflammation observed in Case #17. The size, shape and singular nature of the lesion are features shared with traumatic lesions of the cranium (Walker 1989). While its location low on the left side of the vault is unusual, cranial fractures have been noted on most regions of the vault. The small osteophyte that projects into the circular perforation may be a well-remodeled portion of the original bone that possibly relates to the fragments of bone that are often found on the margins of depressed fractures. The remodeled sclerotic bone surrounding the lytic lesion in Case #17 corresponds with inflammatory bone noted to form as a response to traumatic infection. The palpable region of depressed bone surrounding the lesion may also reflect the broad extent of a larger depressed fracture or a remolded linear fracture. In contrast, several characteristics suggest that healed blunt force trauma is not the best diagnosis for the data presented. Usually, depressed fractures exhibit endocranial spalling and present a larger lesion on the inside of the cranium than on the outside (Frayer 1997; Hurlbut 2000; Lambert 1997). The smaller diameter of the inner aspect of the lesion and the lack of endocranial involvement of any type, including spalling is inconsistent with most descriptions of antemortem healed trauma. According to Berryman and Haun (1996:4), blunt force trauma usually results in an internally beveled margin. This contrasts notably with the external bevel on the temporal bone lesion of Case #17. While certain features of the perforation in question correspond to an antemortem depressed fracture, the absence of an internal bevel is significant in distinguishing trauma from other disease processes. It is not, however considered pathognomonic of trauma, therefore antemortem trauma cannot be *definitively* ruled out.

As noted previously, trephinated skulls also exhibit singular circular defects. The identifying criteria for trephination include cutting or scraping marks that relate to scalp reflection and operative procedure. Case #17 does not exhibit evidence of such marks, however, given the degree of healing that the lesion demonstrates it is possible that they were obscured in the remodeling process. The lesion exhibits a steep external bevel, typical of the grooving method of trephination (Campillo 1984; Lisowski 1967); however, while the bevel is an important feature of trephination, it is not considered diagnostic of the surgical procedure. While most trephinations are commonly located on the upper parietals, review of the literature reveals evidence for trephination on almost any surface of the outer vault (Campillo 1984; Kaufman 1997). Nevertheless, it should be acknowledged that the location of the lesion low on the vault is a *very* uncommon site for trephination. In order to identify the lesion in question as an example of trephination, further osseous evidence or precedents within the current sample or archaeological context are necessary. As will be discussed below, trephination is identified in this study, however the lesion in Case #17 is not similar to the other putatively identified trephinations. While the degree of osseous reaction is certainly consistent with bony inflammation as a response to infection, other disease processes may be equally responsible. Given the nature of the evidence, trephination cannot be excluded nor accepted.

Osteomyelitic infection is another possible explanation for the lesion observed on the cranium of Case #17. The smooth sided hole and a surrounding region of proliferative new bone that exhibits extensive remodeling are consistent with the features of osteomylelitis (Aufderheide and Rodríguez-Martín 1998; Resnick and Niwayama 1988; Revell 1986; Rogers and Waldron 1989); however, it is not clear whether cloacae present externally beveled margins. It is reasonable to assume that if the infection arose in the marrow cavity via hematogenous dissemination, the elimination of the exudate would have resulted in an internal bevel. The lack of reactive bone on the endocranial surface, and the direction of the bevel suggest that the infection did not arise on this surface. A primary infection resulting from an overlying scalp inflammation may provide an explanation for the external bevel. According to Ortner and Putschar, (1984:119), these infections lead to localized reactions restricted to the outer surface and may lead to "perifocal sclerosis" associated with a central lytic lesion and possible sequestrum. While there is no evidence for a sequestrum, the rest of the description is consistent with the features observed on the cranium in question. The small lytic lesion and its associated reactive bone superior to the large perforation suggest that the inflamed area is differentially healed. The abnormal features observed on the vault of Case #17 are consistent with an osteomyelitic infection possibly derived via direct dissemination from the scalp.

It is clear from the above description and diagnostic considerations that the lesion in Case #17 is non-specific in nature. While tuberculosis and neoplasia could be definitively excluded from the differential diagnosis, eosinophilic granuloma, trephination, and osteomyelitis cannot be excluded at this time. Case #17 is classified as an undetermined perforation of the left parietal.

B. Dental and Maxillary Lesions

There is a large dental abscess on the periapical margin of the left maxilla. The perforation measures, 4.6 mm and is an associated with slight zonal porosity. The left M^3 was lost in the antemortem period and the alveolar surface demonstrates significant remodeling. While M^1 and M^2 exhibit significant wear, the degree of dentine exposure was not recorded.

Case #18 Description of the Cranium

Age: Adult

Sex: Undetermined sex

Case #18 represents an incomplete individual. The fragmentary cranial vault is comprised of portions of the frontal, parietal and occipital bones refitted during the curation process. There is no cranial base, face or mandible associated with these fragments. The almost complete right parietal bone is articulated with an incomplete portion of the left parietal bone along the sagittal suture. A partial occipital bone, including most of the left lateral region, the superior sagittal sinus and the internal occipital protuberance, are articulated to the left parietal bone. Most of the right frontal body, was refitted in the area of the coronal suture. The suture is not visible due remodeling and postmortem damage. The anterior frontal bone including the orbital margins, frontal glabella and frontal sinus were lost postmortem.

The remains are poorly preserved relative to the rest of the ABS sample. The outer cortical surfaces of the parietal bones and occipital are eroded, with a "worn" appearance that may be the result of disease and/or postmortem changes. Along the sagittal suture and superior occipital bone, a slight colour change and patches of white bone (newly damaged) together with small shallow pits similar to pock marks give the appearance of weathering. The cortical surface of the frontal bone is in relatively good condition in comparison to the rest of the cranial vault. Because the frontal bone was refitted during the curation process, it is reasonable to suppose that the sections were dispersed within the burial environment.

Case #18 Pathology

A. Abnormal bone loss and appearance on the endocranial cranial surface.

Description of Abnormal Bone

The endocranial surface of the frontal, right and left parietal bones exhibit an area of abnormal bone loss and slight bone formation. Most (>2/3) of the observable frontal bone and right parietal bone exhibit a series of changes most active directly on the endocranial surface below the cranial vertex and extending along the groove for the middle meningeal artery. This groove is unusually deep and numerous fissures, averaging 10-15 mm extend from it in a labyrinth-like pattern (Plate 22). Further from the groove, on the right lateral parietal bone and anterior frontal bone, an elaborate network of fissures exhibits significant remodeling and smoothly dense bone. Thickening of the table and slight ridges of bone interspersed among the fissures are palpable. Along the cranial vertex on the frontal bone, directly adjacent to the groove for the middle meningeal artery, numerous (~30) deep, coalescing cavities and pits extend into the diploë. The pits correspond to the general location of the pacchionian bodies. The lytic activity is focused in area about 40.8 mm by 18.6 mm. The larger holes measure from a maximum of 5.6

mm to 1.0 mm in diameter while smaller pits and generalized porosity are adjacent to both the holes and fissures. There is a slight serpigenous track that runs among the pits slightly to the right of the probable location of the sagittal suture. It is reminiscent of a sequestrum. On the ectocranial surface there is extensive porosity resulting in a slight depression directly above the endocranial foci of lytic activity. The margins of the endocranial cavities exhibit remodeling, while the margins of the ectocranial porosity are sharp. The exposed cranial tables along the damaged margins of the cranium do not exhibit significant morphological changes.

Differential Diagnosis

Increased vascular markings, a distended groove for the middle meningeal artery and smooth-walled lytic lesions on the endocranial surface are an unusual cluster of features. Included in the differential diagnosis are eosinophilic granuloma, meningioma and increased vascular marking due to increased age and infection.

Eosinophilic granuloma is a disorder common to children and young adults characterized by solitary or multiple-coalescing frontal bone lesions with irregular and undulating borders (Ortner and Putschar 1984:250; Resnick and Niwayama 1988:2431). The later stages of the disease demonstrate slight osteoblastic activity indicative of healing. Although the age of Case #18 is unknown, the observable evidence suggests that the individual is an adult, possibly of advanced age. This is contrary to the known predilection of eosinophilic granuloma. Further, while lytic lesions of the frontal bone characterize the disorder, it is not usually associated with increased vascular markings, the most distinctive feature of Case #18. On this basis, eosinophilic granuloma can be excluded from the diagnosis.

Osteolytic meningioma presents endocranial depressions and erosive lesions in association with marked vascular grooving and distended meningeal vessels (Campillo 1994; Crawford et al. 1995; Waldron 1998) in response to abnormal tissue growth in the arachnoid granulations of the vault. The deep, smooth-walled pits and exaggerated arterial groove of Case #18 are consistent with clinical and palaeopathological descriptions of meningioma. Further, the lesions are similar to those described and pictured by both Waldron (1998) and Campillo (1991). Missing, is the sizable shallow depression usually described in the osteolytic forms of the disorder (Waldron 1998). Given the incomplete nature of the vault, it cannot be conclusively stated that this feature is absent from this case. As well, it should be noted that Campillo (1991:227) describes distended vascular grooves as the "least specific characteristic" of meningioma. On the basis of the above information, osteolytic meningioma cannot be disregarded as a possible explanation for the abnormal mixed reaction of Case #18. Other conditions and/or further research (osteological analysis of the remaining material, sex determination, radiograph) may rule out competing explanations.

According to Mann and Murphy (1990:34), ossification of the arachnoid granulations lead to the expansion of the pacchionian pits and is a common consequence of increasing age. Initially the pits are small with sharply defined margins. With increasing age, the lesions become "cauliflower shaped" pits of varying size. Disease and increasing age may also result in pits along the groove for the middle meningeal artery; however, the thickening vault, with slight ridges and furrows, porosity and possible sequestrum are suggestive of a more active process than ageing. Further, given the

incomplete nature of the remains, there is no direct information of the actual age of the individual. Evidence from microscopic age-estimation or associated osteological material may help to resolve if advanced age is responsible for the abnormal changes observed on the endocranial surface of Case #18, however, another explanation may clarify the cause.

The deep, smoothly remodeled pits, distended arterial groove, thickened cranial tables and slight sequestrum suggest that an epidural osteomyelitic infection is the cause of the usual cluster of features (Aufderheide and Rodríguez-Martín 1998:175; Steinbock 1976:68). The deep pits extending anteriorly from the arterial groove are surrounded with zonal porosity and well-remodeled bone. The pits are larger towards the centre of the frontal bone and become smaller and sharper towards the outer margin of the bone. The possible sequestrum appears to navigate across the groove for the middle meningeal artery and encompasses four of the small pits. Small fissures and furrows also extend from the groove and sequestrum (Hackett 1976). The above features are consistent with non-specific osteomyelitic infection (Aufderheide and Rodríguez-Martín 1998:175; Steinbock 1976:68). From the incomplete cranium, it is difficult to determine if trauma leading to primary infection is the cause of the endocranial lesion; however, the involvement of the arterial groove on the inner surface of the vault suggests that the infection was secondary to hematogenous spread (Resnick and Niwayama 1988; Rothschild and Martin 1993).

Case #18 represents an incomplete adult of unknown age and sex. While meningioma predilects female's 2:1 (Campillo 1991;Waldron 1998), and advanced age is associated with increased vascular markings and expansion of the pacchionian pits, neither possibility provides an adequate explanation for the constellation of features observed on the fragmentary cranium. Osteomyelitic infection, possibly spread via a hematogenous route is more consistent with the unusual lesions and a likely explanation.

B. Parietal porosity

Description of Abnormal Bone

The preserved parietal region exhibits discernible pinprick sized pores identified as ectocranial porosis. The pores exhibit a mixture of sharp, healed margins, and are located directly above the infectious process observed on the endocranial surface. Neither of the orbits are available for consultation. Porotic hyperostosis of the cranial vault was recorded as ectocranial porosis (Williams 1994).

Case #19 Description of Cranium

Age: Adult

Sex: Undetermined sex

Case #19 represents an incomplete individual. The cranium includes the articulated anterior portions of the parietal bones. The right parietal terminates above the squamous suture, while the medial two thirds of the coronal suture, and less than half of the anterior sagittal suture is still observable. This specimen is associated with a small-unsided fragment of a temporal. There is no cranial base, face or mandible associated with the incomplete cranial fragments.

The incomplete vault is in poor condition with an eroded cortical surface and evidence of pockmarks. The right parietal bone exhibits a large, circular area of postmortem erosion with exposed diplöe and a small perforation. The margins of the

exposed diploë are sharp and slightly whitish. The outer margin of the eroded area is smooth and blunted with sediments and calcite deposits. The endocranial surface exhibits the unusual black and brown staining that is found on several other specimens (Case #3, #16). There are no cross sections available for observation.

Case #19 Pathology

Case #19 does not exhibit evidence of pathology.

Case A Description of Cranium

Age: Adult
Sex: Female

The partially complete cranium is comprised of the articulated parietal bones, frontal bone, right temporal bone and superior occipital bone. A small incomplete fragment of the right maxilla is articulated along the frontal maxillary suture and small portions of the left and right nasal bones are also present. An unarticulated left temporal bone was found in association. The basi-cranium, remaining face and mandible are missing. The cortical surfaces of the bone are in good condition; however, the bone is soft and susceptible to breakage. Sediment also remains adhered to the lateral ectocranial and anterior endocranial surfaces.

Case A Pathology

A. Large Depression at Bregma

Description of Abnormal Bone

There is a singular ovoid-shaped depression with associated reactive bone located at bregma on the frontal and parietal bones (Plate 23). Two-thirds of the lesion is located on the right parietal bone, orientated 45° to the right of bregma. The remaining lesion is located on the posterior frontal and left parietal bones. The principal disturbance is to the outer layer, except for a small perforation (5.2 mm) of the inner table in the right corner of the lesion on the right parietal. The lesion is large with a thick sclerotic margin circumscribing the depression. The inside and outside diameters of the lesion are demonstrated below (Table 4.1 and Figure 4.1).

The ectocranial surface of the depression exhibits a wide external bevel. Along the anterior margin, the bevel is the steepest, measuring 5.2 mm below the contour of the vault, while the posterior bevel is much shallower. In the center of the depression, there is a table or platform of dense sclerotic and reactive woven bone. The adjacent perforation is surrounded by a concentration of woven bone. The endocranial surface does not reflect the extent of the ectocranial lesion; it does however, display a localized area of porosity on the perforated surface

The mixture of sclerotic and woven bone in the center of the lesion along with the sclerotic margins and reactive bone on the adjacent cortical surfaces suggests that the lesion was in the process of healing. The coronal and sagittal sutures are completely remodeled and obliterated along their entire length. While this may suggest that the closure was due to advanced age as opposed to accelerated remodeling due to abnormal bone changes, Campillo (1994:148) asserts that trauma and trephination can result in synostosis of the suture. There is no macroscopic evidence of cut marks or scratch marks surrounding the lesion or on the surface of the vault.

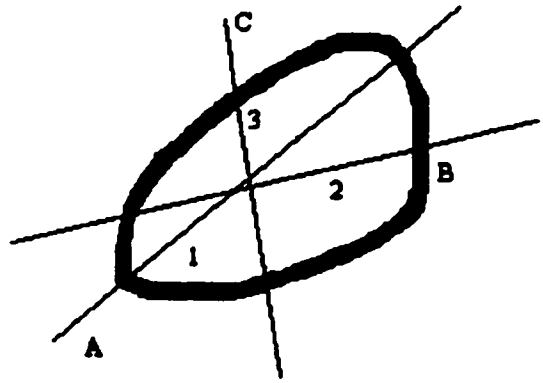


Figure 4.1 Schematic drawing of the lesion on Case A. Not to scale

Table 4.1 Diameters of the lesion on Case A

Inside Diameters	Outside Diameters
1 = 47.8 mm	A = 54.75 mm
2 = 30.0 mm	B = 34.1mm
3 = 39.3 mm	C = 48.8 mm

The endocranial surface suggests the possibility of an infection related to the ectocranial lesion and perforation of the inner table. The perforated portion of the endocranium truncates the groove for the middle meningeal artery, along which there are small patches of reactive bone. On either terminus of the groove, there is a small perforation with sharp well-defined margins. The perforations appear to have occurred sometime in the perimortem/postmortem interval. On the lateral aspect of the right parietal bone, there is an 8 mm perforation and another directly below it on the right temporal measuring 33.4 mm in diameter. On the left parietal bone, a perforation measuring 15.4 mm in diameter is directly parallel to the perforation on the right parietal. Postmortem breakage may have enhanced the size of the lesions on the right temporal bone and parietal bones; however, the possibility of an infection associated with the formation of the lesion on the superior vault is strong.

Differential Diagnosis

Case A presents a well-defined depression associated with reactive bone (osteitis) of the vault. The differential diagnosis should include: infection, trauma, scalping and trephination. Walker (1989:313) recommends that the differential diagnosis of abnormal bone loss accompanied by proliferative bone should include infection. Primary infection, spread from an overlying inflammation of a contiguous surface is a possible explanation for the well-circumscribed depression and diffuse reactive bone on the calvarium. Although rare, infections of the cutaneous surface associated with bacterial (e.g. *stapholoccocus*) or parasitic infections (e.g. *Dermatobia hominis)* may result in superficial inflammation of the outer table of cortical bone (Resnick and Niwayama 1988; Roberts and Manchester 1995). While the endocranial surface of the vault does not demonstrate similar inflammatory involvement, the ectocranial aspect of the depression cannot be described as "superficial". The depth of the depression, perforated cranial

tables and distended arterial groove indicate that the diploë and the outer and inner tables were involved. The reactive bone on the depression and the immediately adjacent surfaces of the vault, exhibit sclerotic margins suggestive of healing. The proliferative bone and perforations on the lateral portions of the cranium appear to have formed after the depression formed. Further, the existence of an external bevel with well-defined margins and the significant table of bone argue for another explanation.

The perforations at either side of the cranium, and along the distended arterial groove are suggestive of an infection secondary to the large depression and perforation of the cranial vertex. The lesions have very sharp margins (and may exhibit postmortem damage) with slight porosity in their peripheries. The sclerotic margins of the large superior vault lesion and its associated reactive bone indicate that they occurred prior to the lateral vault perforations.

Due to the location, shape, size and single nature of the cranial lesion, blunt force trauma is considered in the differential diagnosis (Walker 1989). While singular lesions with well-delineated margins in circular or ellipsoid shapes are consistently associated with blunt force trauma (Walker 1989:313), several features are incompatible with a diagnosis of trauma. First, while fractures to the vault are the most common form of cranial trauma in the osteological record (Lovell 1997a; Merbs 1989; Roberts and Manchester 1995), *healed* depressed fractures on the cranial vertex are rare (Webb 1995) because they usually result in death. Further, while the endocranium exhibits a small perforation, there is no evidence of endocranial spalling that usually accompanies severe blunt force trauma. Most importantly, the internal bevel is a diagnostic feature of blunt trauma (Frayer 1997:189; Hurlbut 2000:7; Lambert; 1997:84). The margins of Case A clearly exhibit an external bevel. On this basis alone, blunt force trauma can be excluded from the diagnosis.

The criteria for scalping were presented in Case #15. While Case A does not exhibit the diagnostic cutmarks on the vault, it is possible to scalp without leaving marks (Bueschgen and Case 1996:233). Further, given the degree sutural remodeling and evidence of osteitis and healing it is conceivable that the marks were remodeled (Miller, 1994; Willey, 1990). The location, general morphology and osteitis adjacent the well-circumscribed lesions are at least superficially similar to descriptions of a healed scalping (Hamperl 1967:632; Hollimon and Owsely 1994; Miller 1994). Bony necrosis leading to an osteoclastic (resorptive) ring and exfoliation of the outer table is frequently characteristic of healed scalpings (Bueschgen and Case 1996; Hollimon and Owsely 1994; Miller 1994; Snow 1942). The outer table of Case A demonstrates a broad area of eroded bone associated with mild bony inflammation and while the entire depression provides evidence of erosion, the margin of Case A is sclerotic and well rounded. This contrasts with the diagnostic osteoclastic ring that surrounds the area of necrosis or inflammation in scalping cases (Snow 1942:401). The excision of the bone on scalping victims is exceedingly rare (Steinbock 1976) and the morphological surface of the depression, small perforation and dense table of bone find no parallel in the descriptions of scalping. Scalping is therefore excluded from the differential diagnosis.

Lesions resulting from trephination are usually singular, circular, oval, or ellipsoid in shape and are frequently located on the frontal bone or parietal bones (Brothwell 1994; Campillo 1984; Kaufmann 1997; Lisowski 1967; Zias and Pomeranz 1992). Case A presents a single, ovoid-shaped depression at bregma on the frontal bone and parietal

bones. Novak and Knüsel (1997:556) debate the origin of a putative trephination at bregma, claiming that it would "in all likelihood be lethal in that the patient would quickly succumb to bleeding from the sagittal sinus". However, a review of the literature provides numerous examples of healed trephinations at bregma and along the sagittal sinus (Bennike 1985; Germanà and Fornaciari 1992; Jennbert 1991; McKinley 1992b; Parker et al. 1986; Persson 1977; Smith 1990).

Additional criteria for trephination relates to the operative procedure. Case A does not present diagnostic evidence of the procedure in the form of cutmarks, scraping, bore holes and abrasion. Not only are these marks considered diagnostic of trephination, they are also representative of the surgical process (Lisowski 1967). While many trephined crania exhibit clear indications of the technique involved, (McKinley 1992; Parker 1986; Richards 1995; Stevens and Wakely 1994), advanced healing and/or infection may obscure the original process (Germanà and Fornaciari 1992; McKinley 1992b). McKinley (1992b), identifies a possible trephination from Baldock, England (1992:339), however, she notes that the unusual shape and absence of operative features renders the diagnosis "debatable". Other recognized trephinations lack scratch marks and other features indicative of the operation (Barnes and Ortner 1997; Campillo 1991; Mckinley 1992b; Robb and Mallegni 1994; Smith 1990).

While there are no surgical marks available to identify the operative procedure of the putative trephination, the dearth of these marks may be an important clue. According to Campillo (1991), the scraping method of trephination results in the most dramatic evidence of healing of all the procedures. The table of bone in the centre of the depression and mild reactive bone are a feature of remodeling and evidence of healing (Campillo 1991). The wide external bevel and associated with a thick sloping and sclerotic margin further support the hypothesis that scraping was the method involved (Brothwell 1994; Campillo 1984; Lisowski 1967). Finally, the ovoid shape and similarity to other such trephinations suggests that the depression was formed by scraping a stone (or shell) implement back and forth along the vault (Lisowski 1967; Stevens and Wakely 1994). While the small aperture in the centre of the depression suggests that the putative trephination may have perforated both the inner and outer table of the cranium, it may also be related to the subsequent infectious process previously identified. The timing of the small perforation is undetermined.

Most palaeopathologists and archaeologists are familiar with the spectacular trephinations identified from Peru and elsewhere. Large apertures, and clear evidence of the operative procedure in the form of cross-natch marks, bore holes and cutmarks characterize these trephinations, (Hrdlička 1939; Koca and Schultz 1994; Lisowski 1967; Richards 1995; Rifkinson-Mann 1988). While these "classic examples"[6] of trephination dominate the literature on the subject, it is important for researchers to remember that just as most disease conditions vary in expression from classic to non-specific, so too can trephination. Germanà and Fornaciari (1992) Manolis (1994), Parker et al. (1986), provide examples of partial or incomplete trephinations. It is important to recognize that while we assume that the goal of the prehistoric trephiner was to remove of a rondel of bone from the cranium, it is just as likely that surgeon only intended to remove an insignificant portion of the cranial vault. Further, Barnes and Ortner (1997), (Campillo, 1991) McKinley (1992b), Robb and Mallegni (1994) and Smith, (1990) present

[6] See Chapter 6 for more discussion on the role of the "classic example" in palaeopathology.

trephinations that provide only speculative evidence of the operative procedures involved (scraping). The presence of a wide external bevel (increasing from endocranium to ectocranium), a pronounced sclerotic margin, a table of woven bone in the centre and mild evidence of osteitis are all consistent with a diagnosis of trephination (Berrymann and Haus 1996; Campillo 1991; Germanà and Fornaciari 1992; Lisowski 1967; Mallegni and Bertodi 1997; Novak and Knüsel 1997; Rifikison-Mann 1988).

According to Steinbock (1976:35), precedents or collaborating evidence from the population under examination are necessary to establish the authenticity of the trephination. As this is the fist examination of the ABS material, there are no previous examples from the site; however, the following case (Case B) exhibits a remarkably similar lesion. Further, as will be discussed in Chapter Six, while trephination is rare in the Portuguese Neolithic, it is not completely unknown (Delgado 1880; Lietão et al. 1987; Piggot 1940).

On the basis of the above information, Case A is classified as a probable incomplete healed trephination. The trephination provides a possible explanation for the origin of the infection/inflammation on the endocranial surface of the frontal and ectocranial surface of the temporal.

B. Osteoarthritis of the TM Joint

Description and Diagnosis of the Abnormal Bone

The right mandibular fossa exhibits coalescing pinpoint porosity and presents evidence of grooving, slight eburnation and pinpoint porosity along the articular tubercle (Plate 24). The above observations are considered diagnostic of degenerate joint disease of the mandibular joint (Rogers and Waldron 1995:13). The fossa and articular tubercle were recorded as 'severe' osteoarthritis after Richards (1988:1529-1531). The left fossa does not exhibit similar lesions. The mandible was not recovered.

Case B Description of Cranium

Age: Adult

Sex: Male

The almost complete cranium is comprised of an articulated vault and face including the supraorbital margins, nasal bones, left sphenoid bone, and left alveolar and palatal regions of the maxilla. While most of the skull base is complete, a small anterior portion is missing. Also missing is the right temporal bone, and portions of the right anterior face including most of the right maxilla, sphenoid bone and all of the right zygomatic bone. The anterior alveolar margins of the left and right maxillae show considerable postmortem destruction. Several teeth remain *in situ*, including the left and right M^1, PM^2 and the left PM^1. Much of the cranium is obscured with very sticky sediments. The preserved cortical surface is in relatively good condition. Six horizontal scratches score the frontal above the right orbits while two deep vertical gashes mark the anterior frontal body. These marks will be discussed below in the abnormal description.

Case B Pathology

A. Large Depression at Bregma

Description of Abnormal bone

There is a large ovoid- shaped depression situated on the posterior margin of the frontal bone at bregma (Plate 25). The lesion is similar in size, shape, location and general morphology to the depression on the cranial vault of Case A. More than 2/3rds of the lesion is located on the right parietal along the first third of the sagittal suture. The anterior margin of the lesion is 17.3 mm from the coronal suture and the posterior margin is located 38.4 mm from bregma. While, the lesion does not perforate the inner table of the vault, and there is no exposed diploë, the surface of the depression and the degree of healing suggest that the diploë was initially exposed, and eventually obscured due to extensive remodeling. The depression is shallow and lacks the platform of bone characteristic of the lesion on Case A. The lesion on Case B is uniformly flat with a defined margin of dense sclerotic bone. The inside and outside diameters of the lesion are as follows (Figure 4.2 and Table 4.2):

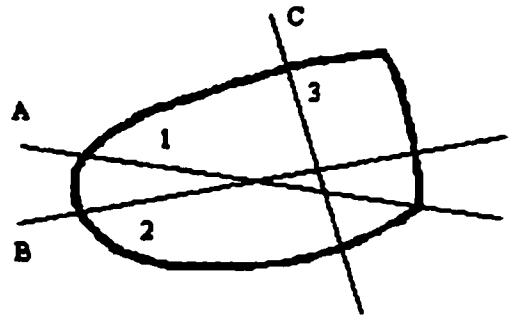


Figure 4.2. Schematic drawing of the lesion on Case B. Not to scale.

Table.4.2 Diameters of the lesion on Case B

Inside Diameters	Outside Diameters*
1 = 46.4 mm	A = 52.95 mm
2 = 29.3 mm	B = 35.7 mm
3 = 12.3 mm	C = 33.8 mm

* *Measurements are approximate*

In contrast to Case A, the outside margin of the depression is not as well defined as the inner margin. Despite its shallow nature, the depression exhibits a palpable external bevel on the ectocranial surface.

The anterior margin of the depression and surrounding area exhibits extensive porosity and coalescing porosity indicative of an active inflammation of the cortical surface. The margins of the porotic bone on the anterior of the depression are sharper than the pores in the surrounding areas. Palpable ridges of dense sclerotic bone extend across the depression and on to the surrounding vault surface. The billowy surface with thickened porosity indicates that the inflammation extended on to the posterior parietals and occipital. The extensive remodeling has obscured much of the sagittal and lambdoidal sutures.

Six parallel horizontal scratches score the surface of the anterior frontal bone, above the right orbit (Plate 26). The scratches vary in length from approximately 60 mm to 90 mm in length. The left orbit does not exhibit similar scratches. Two 'nicks' or gashes with irregular margins are located one above the other, orientated 135° to the left on the anterior/superior aspect of the frontal body above glabella. The lesions measure 12.3mm and 7.2 mm in length. Close observations revel that the nicks are lined with parallel striations. The margins of the lesions and the striations are slightly blunted but sharper than the margins of the depression on the frontal bone and parietal bones. Sediment obscures much of the surrounding area, however initial observations indicate no evidence of inflammation or healing.

Differential Diagnosis

The depression at bregma on the frontal and parietal bones of Case B is very similar to the trephination identified in Case A. The lesion presents a well-defined depression associated with extensive osteitis of the cranial vault. Similar to Case A, the differential diagnosis includes: trauma, infection, scalping and trephination.

Again, the location, shape, size and single nature of the lesion require that blunt force trauma be considered in the differential diagnosis (Walker 1989). Similar to the above case, trauma can be dismissed on the basis of the external bevel, healed location at bregma, and lack of endocranial spalling (Lambert 1997; Lovell 1997a; Merbs 1989; Roberts and Manchester 1995; Walker 1989).

Primary infection, spread from the cutaneous surface of the scalp to the underlying tissue presents a reasonable explanation for the shallow depression on the skull (Resnick and Niwayama 1988; Roberts and Manchester 1995). The lesion at bregma on the frontal bone and parietal bones is shallower and more superficial than the depression observed on Case A. Further, it lacks the table of bone, perforation and endocranial reaction evident in the above case. The reactive bone is much more active and extensive, covering most of superior vault on the posterior parietals and occipital. It is clear that an inflammatory reaction is associated with the depression; however, the distinct shape, margin, bevel and similarity to the previous case recommend that scalping and trephination be considered.

Scalping was ruled out for Case A on the basis of morphology, and the absence of cutmarks. In contrast, Case B presents clear evidence of parallel horizontal cutmarks across the frontal bone (Bridges 1996; Bueschgen and Case 1996; During and Nilsson 1991; Roberts and Manchester 1995). According to Hurlbut (2000), slicing and scraping marks are generally longer than chopping marks, suggesting that the horizontally orientated marks may be associated with scalp reflection The cutmarks demonstrate sharper margins than the depression and its surrounding tissue suggesting disparate timing of the two lesions; however, it is possible that this reflects a differential rate of inflammation and healing instead. Indeed, the superficial inflammation of the depression and vault display varying degrees of healing. The presence of osteitis on the cranial vault also supports the possibility of scalping (Hollimon and Owsely 1994:350; Steinbock 1976:26; Hamperl 1967:632). However, as is evident from the material presented in Appendix two, neither diffuse inflammation nor parallel cutmarks are exclusive to scalping. The lack of an osteoclastic ring, the well–defined and sclerotic margin of the

depression and, most importantly its similarity to the putative trephination in Case A recommend further investigation.

The similarity of the lesion in Case B to the possible trephination in Case A requires that the operative procedure be considered in this diagnosis. The depression is virtually identical in size, shape and location to the identified trephination, differing only in orientation, depth of lesion and degree of beveling. The depression is broad and shallow with a thin, well-defined, sclerotic margin and a palpable external bevel. Like the previous case, these features are consistent with trephination (Berrymann and Haus 1996; Germanà and Fornaciari 1992; Lisowski 1967; Mallegni and Bertodi 1997; Novak and Knüsel 1997; Rifikison-Mann 1988). There is no perforation or steep platform of bone in the centre of the lesion. Such evidence was likely remodeled given the presence of extensive osteitis and inflammation across the depression and adjacent surfaces.

The depression and surrounding areas do not exhibit cutmarks or evidence of the possible operative procedure. Again, given the above, any such evidence was likely remodeled in the post- surgical period. As noted above, the series of parallel scratches and horizontal cutmarks on the frontal bone do not demonstrate the same degree of activity. It is possible they represent differential healing of the same event, equally however, they may be the result of another antemortem event or postmortem process (Hurlbut 2000). Hurlbut (2000) suggests that randomly placed cutmarks maybe indicative of bone defleshing in the postmortem period (Hurlbut 2000). The cutmarks on the frontal, while intriguing, do not constitute diagnostic evidence of trephination.

Following the operative criteria identified in Appendix two, scraping is the most probable method used to remove bone from the calvarium (Brothwell 1994: 133; Campillo 1984:277; Lisowski 1967:662; Smith 1990:90). While the lesion demonstrates significant remodeling, the lack of endocranial reaction suggests that the initial operation was "unsuccessful" in penetrating both cranial tables. As previously stated there are numerous examples of trephination identified in the literature that lack the classic features popularly associated with the procedure. Due to features consistent with trephination and its similarity to Case A, Case B is classified as a possible incomplete trephination associated with extensive healing and a probable superficial infection of the cranial vault. The cutmarks remain ambiguous.

CHAPTER 5

DISSCUSSION

5.1 Introduction

This chapter evaluates the osteological sample from Algar do Bom Santo and considers the possible ramifications for the data. The results are then presented with the prevalence of lesions according to disease and trauma categories. Prevalence of the total sample as well as its age and sex distribution is provided. Finally, the results are discussed in the context of ABS.

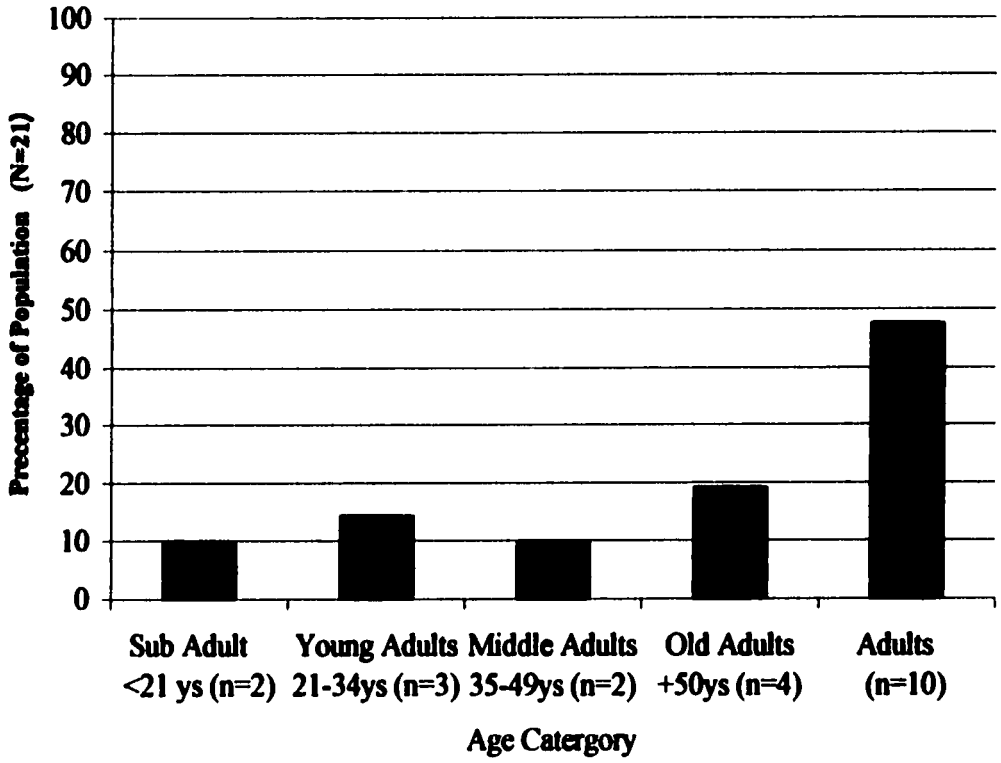
5.2 The Sample

Prevalence statistics are provided to facilitate future comparisons of the ABS sample to newly excavated data from the site and other archaeological samples. While comparisons frequently appear in the palaeopathology literature, it is not the intention of this thesis to compare the disease rates of the Late Neolithic site with living populations (Cohen 1989; Wood et. al. 1992). In most studies, there are too many inherent inadequacies and inconsistencies with archaeological samples to draw reliable comparisons to living populations (Waldron 1994:10; Wood et al. 1992). The fundamental difference between clinical and palaeopathological research is the fact that the studied population is comprised of *dead* individuals (Wood et al. 1992). Further differences relate to how the sample is derived. A skeletal sample is obtained by taking all of the dead from a living population and burying a proportion of it the mortuary site. A proportion of those buried at the site will be preserved at the time of discovery, of which the archaeologist will recover only a segment. Finally, a study sample is taken from a further proportion of the segment recovered from the site (Waldron 1994:12; Wood et. al. 1992:344).

Another factor limiting comparability is the time scale from which the sample is derived. Skeletal or palaeopathological samples may be derived from a "population" that lived over several hundred years, while most modern demographic and epidemiological studies consider 30 years to be a long term (Waldron 1994). Archaeological evidence and radiocarbon dates suggest that ABS may have been used as a mortuary site for upwards of 500 years (Duarte 1999 personal communication). Another important distinction between living and archaeological populations is the ability of the researcher to estimate accurately age and sex. The reliability of the age and sex distribution depends upon the relative ambiguity of the age and sex characteristics of the individual, the preservation of the materials and the ability of the researcher to provide an accurate estimation. The problems specific to ABS will be discussed below.

According to Waldron (1994:11), a skeletal/palaeopathological sample almost never meets the random criteria of the medical epidemiologist. While a random sample from a biased parent sample can be obtained, this does not in any way ameliorate the inherent bias of the parent. The sample from ABS was taken from the partially excavated rooms immediate to the entrance and does not represent the entirety of the cave. Further, there was no intention of creating a strictly random sample from the excavated material. The unit of analysis for this study is the cranium. Crania obtained in the1994, and 1995

Table 5.1 Proportion of population by age for the Algar do Bom Santo Sample. Includes subadults.



excavations were examined and only those that were deemed representative of "individuals" (complete and partially complete) were included. Further, under the justification of establishing the nature of the abnormal bone, two of the *in situ* crania noted to have trephination-like lesions were removed from unexcavated portions of the cave for analysis. The two crania were considered in this project for evidence of pathology; however, they *were not* included as part of the main study sample. Although their relevance to the ABS population will be considered, the descriptive statistics do not include either of the crania. One skull was taken from the unexcavated Sala C, (cranium will be referred to as: Case A) and another from the partially excavated Sala A, (cranium will be referred to as: Case B).

As reviewed in Chapter four, the sample is comprised primarily of partially complete crania. This will have implications on the calculations of the prevalence of disease. First, missing material means that the presence or absence of a particular disorder on the missing elements will not be represented in the sample. In this situation, it is difficult to determine if the calculated prevalence under or over estimates the rate of the disorder in the population (Waldron 1994: 54). Further, the inability to recognize a disorder where the patterning of lesions is essential to recognition will hinder the process of diagnosis (Roberts and Manchester 1995; Rogers and Waldron 1989; Rothschild and Rothschild 1995b) and as a result, the prevalence of certain diseases will be underestimated. There are two other problems with the demographic profile of ABS. The first is the age distribution and the second is the ratio of females to males.

With the two sub adults added to the profile, the distribution does not conform to the expected U-shape of a pre-industrial population (Waldron 1994: 89). This is demonstrated in Table 5.1. With the juvenile individuals included, the chart indicates that fewer individuals belong in the sub-adult category than the young adult and old adult categories. There are several possible explanations for this. First, the age distribution was derived from the sample of complete and partially complete individual crania and not from the MNI based on all the material including isolated remains. While a minimum number of seven juveniles was determined from the excavated skeletal remains, the adult MNI was not broken down into Young Adult, Middle Adult and Old Adult categories; as a result, it was not possible to construct a profile that properly represents the juvenile portion. Of note, juveniles represent 22.5% of the population when the adult and sub adult MNIs are combined. Second, the sample is comprised primarily of partially complete crania for which a significant proportion are missing one or more of the suture sites necessary for age estimation. As a result, 52.6 % of the 19 adult individuals could not be assigned a cranial age estimation more reliable than adult. Similarly, Sub Adult #1 demonstrates completely open sutures, while Sub Adult #2 presents an erupted M^2 (no wear) and an M^3 as yet encrypted. Neither juvenile vault could be assigned a precise age and both were classified as 'sub adult'. Thirdly, cranial age estimation provides broad age-ranges and large standard deviations; as a result, researchers are usually encouraged to use it as a supplementary technique (Meindl and Lovejoy 1985:5). Unfortunately, given the nature of the data and the poor preservation of other sites of cranial age assessment (e.g. teeth), ectocranial age estimation is all that was available. Finally, studies show that the juvenile and elderly material is subject to poor preservation and under representation in skeletal samples (Walker et al. 1988).

The ratio of identified females to males is 8:5 (9:5 including juveniles), suggesting that the sample does not conform to the expectation of unity (Waldron 1994:23) (Table 5.2). However, for 31.6% (33.3 % including juveniles) of the sample, sex could not be determined, and was labeled "unknown". While sex can usually be determined relatively reliably based on cranial material (Bass 1987), the incomplete nature of much of the sample is likely responsible for the skewed ratio. Given the above limitations, prevalence data for different disease categories at the age and sex sub-samples must be carefully considered before the data are compared to other populations.

5.3. Results & Discussion

The preceding chapter provided the descriptions and differential diagnosis of the 19 individuals from the ABS cranial sample. Also included were the two individuals from Sala A and Sala C. The process of differential diagnosis was moderately successful in determining the specific etiology of the pathological crania. The following section provides the prevalence of lesions in each of the following categories: 1) Trauma, 2) Metabolic, Endocrine and Hematological Disorders; 3) Infection; 4) Neoplasia, 5) Congenital and Developmental Disorders, 6) Joint Disease, 7) Human Modification and 8) Undetermined. Table 5.3 provides the prevalence of each of the disease categories according to the total sample. It further calculates the age and sex prevalence of each of the disease sub samples. Table 5.4 provides the prevalence of each disorder in the age and sex subsamples.

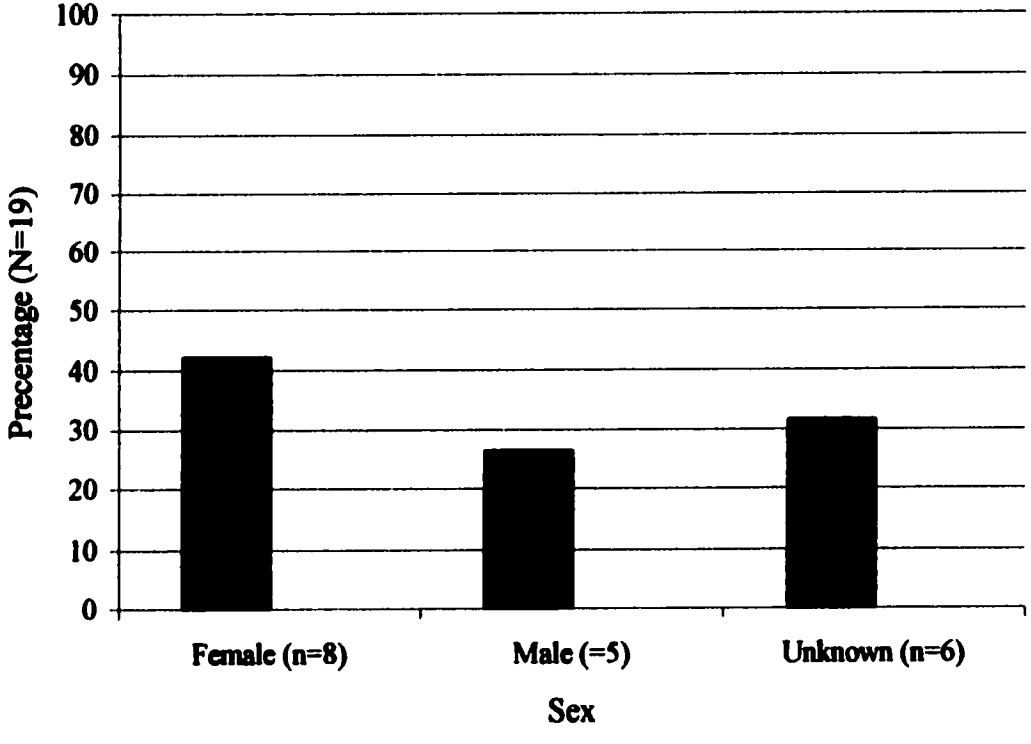
5.3.1 Trauma

A total of two depressed fractures (Case #1 and #2) were identified from the 19 crania (10.5%) of the ABS sample. Both lesions occur on the left parietal and were likely caused by blunt force impact. Case # 1 exhibits no evidence of healing or infection, while Case #2 demonstrates significant remodeling indicating that it was likely a longstanding lesion. Both fractures were identified on male crania (40 % of the male crania), and both were from Sala B. One other skull (Case #9 a male from Sala A), exhibits possible evidence of perimortem/ postmortem blunt force trauma to the right parietal. While the lesions are clearly identified as depressed factures, they are ambiguous in their timing.

Partial or incomplete crania represent 84 % of the sample, and as a result, the percentage of cranial trauma in the 19 individuals from ABS is likely underestimated. There were no examples of trauma to the facial region. In the ABS sample, only seven of 38 expected maxillae (18.4%) and six of the 38 possible zygomatics (16.8%) were identified. According to Waldron (1987), the poor preservation of the cranio-facial area is frequently associated with the under representation of trauma and other disorders in this region.

Two important observations arise from the examination of the fracture on Case #1. First, while it is generally very difficult for palaeopathologist to comment decisively on the cause of death (Roberts 1996:129), severe cranial trauma is frequently fatal (Knight 1991). Evidence for unhealed trauma, as in Case #1 is one of the few scenarios where a palaeopathologist may confidently speculate on the cause of death (Roberts 1996:129). It is very unlikely given the degree of comminution and the lack of healing or infection that the individual survived very long after impact. Second, depressed fractures that form as a result

Table 5.2. Proportions of the Algar do Bom Santo population by sex (juvenilles not included).



of blunt force trauma may provide potential evidence for the implement involved (stone tool, rock etc.) (Lambert 1997; Merbs 1989; Webb 1995). The shape of the depressed fracture for Case #1 is distinct, and while no implements can be attributed to the lesion at this time, it bears further consideration. Case #2 presents a well-healed non-lethal depressed fracture (possibly pond) with no evidence of infection. Due to the significant degree of remodeling, it was not possible to fully evaluate the kind of impact, and type of implement involved or to compare the shape and size of the depression to Case #1. The degree of remodeling and lack of reactive bone suggests that Case #2 survived for a very long time after the injury.

Trauma is frequently used to interpret the socio-cultural and environmental context of human behaviour in prehistory. Evidence for injury may provide clues as to the individual life history, socio-economic status, occupational hazards, and interpersonal violence (Gauer and Roberts 1996; Hulburt 2000; Hutchinson 1996; Jurmain and Bellifemine 1997; Lovell 1997; Papathanasiou et al. 2000; Smith 1996). Cranial fractures, in particular are considered an indication of aggression and interpersonal violence (Jurmain and Bellifemine 1997; Lovell 1997; Martin 1997; Martin et al. 1993; Papathanasiou et al. 2000; Walker 1989; Webb 1995). Cranial injuries located on the frontal and parietals may be the result of hand –to-hand fighting (Roberts and Manchester 1995:79) and depressed fractures found on the left side of the vault, particularly the parietal, are often interpreted as representing a "frontal assault by a right-handed person" (Webb 1995:205). Although highly geographic and temporally variable, male crania tend to exhibit trauma more frequently than females (Jurmain and Bellifemine 1997; Walker 1989). It is interesting then, that both depressed fractures from ABS are located on the left parietals of adult males.

While it is tempting to draw an immediate conclusion regarding the origin of the injuries, more data on the patterning of fractures throughout the skeleton and population are needed before an explanation of interpersonal violence can be accepted (Lovell 1997a). Further information regarding the social/cultural context of the individual crania and the site is required. While archaeological evidence (mostly indirect) suggests that Late Neolithic people were agro-pastoralists (Whittle 1996; Zilhão 1993), little is known about the specifics of their life ways. The post-cranial material and a re-examination of the isolated facial and mandibular elements, both typically the most common site of interpersonal injury (Lovell 1997a) may facilitate interpretation of the above data.

5.3.2 Metabolic, Endocrine and Hematological Disorders

One skull (5.3%), Case #3, demonstrates ambiguous evidence of a metabolic or endocrine disorder. Hyperostotic changes usually reflect a disruption to the normal osteoblastic activity of bone and are frequently found in association with metabolic and endocrine disorders (Revell 1986). Given the incomplete nature of the remains, there was insufficient evidence to refine further the pathological possibilities. The skull is of unknown age and sex representing 10.0% and 16.7% of those samples respectively. One other crania (Case #13) presents inflammation that may represent the kind of disruption in cellular activity that is frequently associated with metabolic disorders; unfortunately, it could not be distinguished from competing diagnoses. There is no other conclusive evidence of endocrine or specific nutritional deficiencies from the ABS sample.

Porotic hyperostosis and cribra orbitalia are frequently associated with anemia (Lovell 1997b; Stuart-Macdam 1992). The prevalence of cranial porosity in the form of ectocranial porosis/porotic hyperostosis was calculated for the entire sample. Seven out of 19 individuals (36.8%) presented some degree of porosity. Of those individuals, 57.1% were male, 28.6 % were female and 14.3 % were unknown. Young adults accounted for 28.6 % of the crania with porotic hyperostosis, middle adult's 14.3 %, old adult's 14.3% and unknown's accounted for 42.9 %. More interesting is the fact that 80 % (4/5) of the total male sample exhibits some form of porotic hyperostosis and 66.7 % (2/3) of the young adult sample presents evidence of the disorder. Six of the seven cases of porotic hyperostosis (85.7%) are represented by ectocranial porosis, and only one-presented lesions scored as medium.

The prevalence of cribra orbitalia was calculated using the number of identified orbits in the sample. Of the 18 orbits counted[1], four (22.0%) had evidence of cribra orbitalia. Of these, two of the examples were from the same individual (Case #7). Seventy-five percent (including the two orbits from the same individual) were identified from adult female crania. One (25%) was identified from a young adult male. In other words, three individuals from the ABS sample exhibited cribra orbitalia: two females (Case #6 and #7) and one male (Case #1). Twenty-three percent (23.7%) of the female orbital sample from ABS exhibited cribra orbitalia. Two individuals (Case # 1 and # 6) exhibited orbital and vault lesions, three (Case #4, #5 & #9) presented only vault lesions, and one only orbital lesions. For two crania (Case #2 & #18), the relationship between orbital lesions and vault lesions could not be evaluated because of postmortem damage. The above samples are very small, particularly for cribra orbitalia, and care must be taken when interpreting the above data.

Evidence for the probable cause(s) of anemia at ABS is limited. Genetic and congenital anemias can be ruled out as explanations for the cause of the porotic hyperostosis and cribra orbitalia on the basis that they typically exhibit severe changes to the vault characterized by a "honeycombed" and "hair-on-end" appearance (Aufderheide and Rodríguez-Martín 1998: 347; Resnick and Niwayama 1988: 2351). While the face and mandible are frequently involved in genetic anemia, the poor preservation of the cranio-facial area in the ABS sample makes this difficult to assess, although Case #6, represented by a relatively complete skull exhibits light cribra orbitalia and does not demonstrate any changes to the face. Also, according to Stuart-Macadam (1992:40), the present day distribution of congenital anemia suggests they were rare in the past. Iron-deficiency anemia is the most common form of the disorder and widely recognized in the palaeopathological literature (Roberts and Manchester 1995; Stuart-Macdam 1989b). For the most part, the ABS crania exhibit ectocranial porosis on the vault and mild lesions on the orbits suggesting that iron deficiency related anemia is the probable cause (Lovell 1997b).

Increased rates of anemia in antiquity have been previously interpreted as

[1] A MNI of 10 was counted for the 18 orbits.

Table 5.3. Prevalence of Disease from the Total Algar do Bom Santo Sample. Prevalence is also calculated by sex and age for each disease category.

Disease Categories	Disease Prevalence		Sex Distribution						Age Distribution							
			Male		Female		Unknown		Young		Middle		Old		Adult	
	#/N	%	#/n	%	#/n	%	#/n	%	#/n	%	#/n	%	#/n	%	#/n	#
Trauma	2/19	10.5	2/2	100.	0/2	0.0	0/2	0.0	1/2	50.0	1/2	50.0	0/2	0.0	0/2	0.0
Metabolic, Endocrine & Hematological Disorders	1/19	5.3	0/1	0.0	0/1	0.0	1/1	100	0/1	0.0	0/1	0.0	0/1	0.0	1/1	100
Porotic Hyperostosis	7/19	36.8	4/7	57.1	2/7	28.6	1/7	14.3	2/7	28.6	1/7	14.3	1/7	14.3	3/7	42.9
Cribra Orbitalia	4/18[1]	22.0	1/4	25.0	3/4	75.0	0/4	0.0	1/4	25.0	0/4	0.0	0/4	0.0	3/4	75.0
Infection	3/19	15.8	0/3	0.0	2/3	66.7	1/3	33.3	0/3	0.0	0/3	0.0	1/3	33.3	2/3	66.6
Otitis Media & Mastoiditis	2/18[2]	11.1	0/2	0.0	1/2	50.0	1/2	50.0	0/2	0.0	0/2	0.0	0/2	0.0	2/2	100
Periapical Abscess	2/7[3]	28.6	2/2	100	0/2	0.0	0/2	0.0	0/2	0.0	0/2	0.0	2/2	100	0/2	0.0
Neoplasia	1/19	5.3	0/1	0.0	1/1	100	0/1	0.0	1/1	100	0/1	0.0	0/1	0.0	0/1	0.0
Congenital Disorders	1/19	5.3	0/1	0.0	1/1	100	0/1	0.0	0/1	0.0	0/1	0.0	0/1	0.0	1/1	100
Joint Disease																
TMJ	1/14[4]	7.1	0/1	0.0	1/1	100	0/1	0.0	0/1	0.0	0/1	0.0	0/1	0.0	1/1	100
Human Modification	0/19	0.0	0/0	0.0	0/0	0.0	0/0	0.0	0/0	0.0	0/0	0.0	0/0	0.0	0/0	0.0..
Undetermined	4/19	21.1	1/5	20.0	1/5	20.0	2/5	40.0	0/5	0.0	1/5	20.0	1/5	20.0	2/5	40.0

Note: '#' = number of individuals; n= sub sample; N= total sample. Total sample is 19 unless otherwise noted. % = percentage of individuals within the sample or sub sample.

[1] N=18 (9 left and 9 right) orbits were identified from the sample. Prevalence was calculated based on the number of individual bones recovered.
[2] N=16 (11 left and 7 right) temporal bones were recovered from the sample. Prevalence was calculated based on the number of individual bones recovered.
[3] N=7 (4 left and 3 right) maxillae were identified from the sample.
[4] N=14 (8 left and 6 right) TM joints were identified from the sample.

representing a decline in dietary iron related to nutritional inadequacy at the transition to agriculture (Carlson et al. 1974; Cohen and Armelagos 1984; El-Najjar 1977; Palkovich, 1987; 1987; Stuart-Macadam 1989b; 1991,1992a). According to Lovell (1997b) however, iron deficiency anemia should be recognized as a symptom of disease (infection) or an indicator of non-specific stress, rather than a representation of a specific deficiency disease. Recent research suggests that acquired iron deficiencies due to chronic infection or excessive blood loss due to gastrointestinal parasites are also important etiological considerations (Lovell 1997; Mensforth et al. 1978). Anemia due to the lack of iron may have less to do with a poor diet, than increased population densities and domestication of animals leading to poor sanitation, increased pathogen loads and endemic infection. These features of incipient agriculture may result in generalized stress leading ultimately to marrow hyperplasia and the morphological expression of porotic hyperostosis and cribra orbitalia (Hengen 1971; Kent 1990; Roberts and Manchester 1995; Stuart-Macadam 1989b; 1992a; 1992b).

Stable isotope analysis from Gruta do Caldeirão, suggest that the (Early) Neolithic diet was liberally based on terrestrial resources (Jackes and Lubell 1992:271). Archaeological evidence from several Early and Middle Neolithic sites suggests that in addition to terrestrial fauna, marine resources continued to play an important dietary role (see Lubell et. al. 1994:207 for the specific sites and assemblages). In his analysis of coastal Scandinavia populations, Walker (1986), found that diets high in iron-rich shellfish demonstrated increased rates of iron-deficiency anemia. He concluded that contaminated water and fish bound parasites were the most likely cause of the disorder. Archaeological evidence from the Portuguese Mesolithic demonstrates that these populations relied heavily on shellfish and marine resources (Lubell et al. 1994; Zilhão 1993). To date, while little data have been presented on the rates of porotic hyperostosis and cribra orbitalia for both Mesolithic and Neolithic, it would not be unreasonable to expect that if fish-born parasites were responsible for the iron-deficiency anemia, then the Mesolithic rates should be higher than the Neolithic rates. While this is not the case, it is likely that the data from both periods are too limited to evaluate effectively. Indirect dietary evidence from the south of Portugal (Goldar) suggests that (Middle) Neolithic populations consumed cereals, olives, pistachios and acorns. According to Lovell (1997:119) a limited diet or specific deficiency (e.g. ascorbic acid, beta-carotene) may predispose the individual to iron deficiency anemia, however, these insufficiencies are usually rare in light of a well rounded diet as suggested above.

Anemia is frequently associated with individuals comprimised because of infection (Roberts and Manchester 1995; Stuart-Macdam 1992b). Some parasitic, viral, and fungal infections reduce bio-available iron leading to iron-deficiency anemia, while others such as tuberculosis and osteomyelitis may result in anemia related to chronic infection (Stuart-Macadam 1992:159). Meiklejohn and colleagues (1984:81) report a general increase in the prevalence of cranial infection in their review of western Mediterranean palaeopathology. A number of individuals in the ABS sample (n~7) display evidence of otitis media, periapical abcess, mastoiditis and non-specific osteomyelitic infection. It is possible that the mild porotic hyperostosis and cribra orbitalia is a reflection of the level of infectious disease in this population.

Anemia related porotic hyperostosis and cribra orbitalia are also interpreted as evidence of generalized stress and an overall decline in health. Meiklejohn and

colleagues (1984:81), reported limited evidence of cribra orbitaia in their review of pathological crania from the western Mediterranean. They report no cases of cribra orbitalia from the Mesolithic sites and only four examples from the Neolithic sites. Jackes, Lubell and Meiklejohn's 1997(b) paper reports that there is limited evidence for an increase in cribra orbitalia in the PortugueseNeolithic; however, because of diffuculties with the sample (Casa da Moura) they decline to interpret this as representing a deterioration of nutrional status. Further, the skeletal, dental and palaeodemographic evidence from the Early Neolithic of Portugal does not support a substantive decline in health at the transition to agriculture (Jackes et. al. 1997b: 65). On the other hand, they do point out "future studies may discern changes in skeletal biology towards the end of the Neolithic period which might in part be responses to agricultural intensification". As yet, little is understood about the palaeoeconomy and agricultural life styles of the populations associated with ABS and the rest of the Portuguese Late Neolithic. It is hoped that this thesis may provide a foundation for examining the health status of Portuguese Late Neolithic populations, and the implications of agricultural intensification.

5.3.3 Infection

A total of three (15.8%) crania from the ABS sample exhibit evidence of non-specific infection. Both Case #15 and #16 are female, while #18 is undetermined. All three exhibit evidence for advanced age, although only Case #15 could be reliably aged. Cases #15 and #16 represent 25% of the female sample. Case #18 represents 16.7 % of the unknown sample, while Case #15 represents 25 % of the old adult sample and Cases # 16 and #18 represent 20% of the adult sample. All three crania were from Sala A. Two other crania (Case #13 and #17) from ABS exhibit possible evidence for inflammation due to non-specific infection; unfortunately, neither could be distinguished from competing conditions.

Case #15 and #16 exhibit primary infections that were longstanding and possibly chronic. Although there are differences in the morphological expressions of the infection on each cranium, the differential diagnoses were markedly similar. Treponemal infection was considered for each case and while each exhibited similarities to gummatous and non-gummatous syphilis (non-diagnostic), the disorder was excluded because there is no universally accepted evidence of the disease in Europe before 1492 (Baker and Armelagos 1988). Post-cranial or immunological evidence from ABS or another temporally and geographically similar site would provide a justification for a re-evaluation of the data. Case # 18 presents evidence for a longstanding pyogenic infection on the endocranial surface. The location and association with the vascular vessels suggest that it was spread hematogenously from another area of the skull or post-cranium (Resnick and Niwayama 1988; Rothschild and Martin 1993).

Evidence for otitis media and mastoiditis was calculated from the 18 temporal bones[2] recovered from the site. One example of otitis media and one example of mastoiditis were recorded for a combined percentage of 11.1 % of the temporal bones. The otitis media was identified from Case #6, an adult female, while the mastoiditis was identified from Case #10 an adult of undetermined sex. Care must be taken when interpreting these results for two reasons. First, both diagnoses are tentative. The

[2] A MNI of 13 was counted for the 18 temporal bones.

disorders are difficult to diagnose on the basis of macroscopic description; supporting evidence (x-ray and otoscope) is required to confirm the initial diagnosis. Second, because the fragmentary nature of the temporal sample may not accurately reflect the actual occurrence of the disease, the recorded prevalence may either underestimate or over estimate the frequency of the disorders.

As stated previously, it is not the intent of this thesis to evaluate dental disease; however, two examples (28.6%) of periapical abscesses were recognized from the seven maxillary[3] bones recovered. Both abscesses were recorded on old adult male crania representing 50 % of the male maxillae identified in the sample and 33.3 % of the old adult maxillary sample. The abscess on Case #9 is associated with a carious lesion of the right M^2 and exhibits evidence of healing. The abscess on Case #17 is associated with caries of the left M^2 and AMTL of the left M^3.

The two crania from outside the sample (Case A and B) both exhibit osteitis of the cranial vault. In both cases, the inflammatory bone is in apparent response to a previous probable trephination. Case A also exhibits evidence of possible osteomyelitis that is on the endocranial surface directly underneath the lesion. Given the lack of healing it likely formed after the probable trephination. The prevalence of infectious disease in the ABS sample may be underrepresented for several reasons. First, the incomplete and fragmentary nature of much of the sample contributes to an under representation of the actual number of affected individuals and elements. Further, the identified infectious lesions of the vault are characterized by abnormal bone formation in association with small areas of erosion (see Cases #15, #16, A and B). Lytic lesions are commonly associated with infection and inflammation. Those elements with predominantly lytic lesions are more likely to be damaged in the postmortem process than are those whose osteoblastic activities make them resistant to damage (Powell 1991:175). Further, lytic lesions are common in many disorders and may frequently be confused with postmortem damage. Finally, osteitis/periostitis are inflammatory bone responses to many different diseases (Ortner and Putschar 1984) and may or may not be confused with other disorders.

Osteomyelitis of the skull is rare (Hackett 1976:72; Ortner and Putschar 1984:117; Steinbock 1976:81). Cranial infections are usually primary, caused by the introduction of a pathogen to the vault via trauma or a contiguous skin lesion. Case A and Case B are good examples of primary infections related to trauma and disturbances of the scalp. For the most part, the infection is limited to the outer periosteal surface of the vault, progressing only to the endocranial surface of Case A via a perforation in the vault. Cases #15, #16 and #18 represent nonspecific infection with evidence of involvement in the two tables and diploë. The severe outer table and inner table reactions do not indicate the cause or origin of the infections; although, certainly, the crania appear to have undergone long-term involvement.

The three crania in this category represent the most common disorder recognized in the ABS sample other than porotic hyperostosis/ectocranial porosis. In his review of 46 sites from the western European, including Portugal, Meiklejohn and colleagues (1984:81) reported that there was an overall increase in the rate of cranial infection from the Mesolithic to the Neolithic. Although the methods and level of analysis were limited, they interpreted the increased level of infection in the larger populations typical of the

[3] A MNI of 5 was recognized for the 7 maxillae.

more sedentary Neolithic period (Larsen 1985: 199; Meiklejohn et al. 1984: 8; Ortner and Putschar 1984:105; Roberts and Manchester 1995:131). Larger populations provide a reservoir for endemic infections and facilitate their transmission through the community. A general decrease in the health status of the population and/or individuals may also provide an explanation for the causes of infection. Individuals compromised as a result of dietary insufficiency, malnutrition and anemia may be susceptible to infection. However, as stated previously, Jackes, Lubell and Meiklejohn (1997b) do not feel that there was a decline in the overall health of the populations associated with the Early Neolithic. The reasons for the infections remain elusive.

Infection, in the context of this thesis, also sheds some important insights into the nature of the sample and the method of differential diagnosis. The category of infection and inflammation underlines why the commingling of the material is so restrictive to understanding disease. Cases #15, #16, #18 and #13 present interesting lesions, possibly reminiscent of specific infection; however, unlike trauma and trephination, evidence from other areas of the skeleton can be critical to resolving the etiology of infectious lesions and other disorders. While it is very likely that the abnormal bone will remain non-specific upon review of the other skeletal material from ABS, the dearth of unassociated and the unanalyzed post-cranial material prohibits a holistic approach to the diagnosis (Rogers and Waldron 1989), and limits the ability of the researcher to interpret the material.

In contrast, this disease category also underlines why the rather lengthy descriptive process and differential diagnosis are so important. At first, the cranial lesions of Cases #15, #16 and #18 are very unusual and difficult to qualify. The description process, pinpointed and immediately eliminated postmortem destruction from consideration (e.g. weathering on the outer table of Case #18) clarified the osteoblastic/ osteoclastic relationships (mixed reaction on Case #15 and abnormal formation on Case #16) and emphasized important features (bony spicules and geographic surface on Case #16; pits and possible sequestrum on Case #18). This facilitated comparison of the abnormal crania to the literature and created a limited list of competing possibilities. The process of differential diagnosis eliminated conditions that upon superficial examination appeared consistent, but with further research were revealed incompatible (e.g. trephination, scalping, Paget's disease). Because non-specific infection by its very nature presents ambiguous lesions, it is important to evaluate the constellation of features with reference to each other (Miller et. al. 1996). To approach cases such as these without rigorously describing and comparing competing disorders may have resulted in premature conclusions. On the other hand, differential diagnosis may have helped to establish the etiology or pathogensis of a specific disease process (Palkovich 1987). Finally, the descriptions and differential diagnoses provided in this thesis have laid the foundation for future research and interpretation (Ortner 1991).

5.3.4 Neoplasia

In stark distinction to the important role that cancer plays in the modern world, evidence of neoplasia is rare in the archaeological record. According to Brothwell (1967: 320), the incidence of neoplasia is likely underrepresented in the palaeopathological literature. Waldron (1996) feels that the relative infrequency of neoplastic conditions may

Table 5.4. Prevalence of Each Disease Category from the Algar do Bom Santo Sample According to Age and Sex.

Disease Categories	Sex Distribution						Age Distribution							
	Male		Female		Unknown		Young		Middle		Old		Adult	
	#/n	%	#/n	%	#/n	%	#/n	%	#/n	%	#/n	%	#/n	#
Trauma	2/5	40.0	0/8	0.0	0/6	0.0	1/3	33.3	1/2	50.0	0/4	0.0	0/10	0.0
Metabolic, Endocrine & Hematological Disorders	0/5	0.0	0/8	0.0	1/6	16.7	0/3	0.0	0/2	0.0	0/4	0.0	1/10	10.0
Porotic Hyperostosis	4/5	80.0	2/8	25.0	1/6	16.7	2/3	66.7	1/2	50.0	1/4	25.0	3/10	30.0
Cribra Orbitalia[1]	1/7	14.3	3/11	27.3	0/0	0.0	1/4	25.0	0/2	0.0	0/8	0.0	3/4	75.0
Infection	0/5	00.0	2/8	25.0	1/6	16.7	0/3	0.0	0/2	0.0	1/4	25.0	2/10	20.0
Otitis Media & Mastoiditis[2]	0/6	0.0	1/9	11.1	1/3	33.3	0/4	0.0	0/2	0.0	0/7	0.0	2/5	40.0
Periapical Abscess[3]	2/4	50.0	0/3	0.0	0/0	0.0	0/0	0.0	0/1	0.0	2/6	33.3	0/0	0.0
Neoplasia	0/5	0.0	1/8	12.5	0/6	0.0	1/3	33.3	0/2	0.0	0/4	0.0	0/10	0.0
Congenital Disorders	0/5	0.0	1/8	12.5	0/6	0.0	0/3	0.0	0/2	0.0	0/4	0.0	1/10	10.0
Joint Disease														
TMJ[4]	0/4	0.0	1/7	14.3	0/3	0.0	0/2	0.0	0/1	0.0	0/7	0.0	1/5	20.0
Human Modification	0/5	0.0	0/8	0.0	0/6	0.0	0/3	0.0	0/2	0.0	0/4	0.0	0/10	0.0..
Undetermined	1/5	20.0	1/8	12.5	2/6	33.3	0/3	0.0	1/2	50.0	1/4	25.5	2/10	2.0

Note: '#" = number of individuals; n= sub sample; % = percentage of individuals within the sample or sub sample

[1] N=18 (9 left and 9 right) orbits were identified from the sample. Prevalence was calculated based on the number of individual bones recovered.

[2] N=16 (11 left and 7 right) temporal bones were recovered from the sample. Prevalence was calculated based on the number of individual bones recovered.

[3] N=7 (4 left and 3 right) maxillae were identified from the sample.

[4] N=14 (8 left and 6 right) TM joints were identified from the sample.

be more an artifact of the archaeological record, poor preservation of pathological specimens and small sample sizes. The failure to identify neoplastic growths in antiquity may also be the result of 1) the nature of the osseous lesions; 2) the limitations of macroscopic evaluation; 3) a poor understanding of the etiological factors involved; and 4) lower life expectancies in the past and the correlation of increasing age and neoplasia (Ortner 1981; Resnick and Niwayama 1988; Roberts and Manchester 1995; Rothschild and Rothschild 1995; Rúa et al. 1995; Strouhal 1991).

The ABS sample contains one example (5.3%) of a neoplastic disorder. The lesion on Case #14 was identified as an osteoma of the frontal vault. The skull is a young adult female. There is no other evidence of neoplasia from the ABS sample. Osteomas are generally considered to have no symptoms or clinical significance (Capasso 1997). The consequences for the individual from ABS would have been limited to non-existent (Roberts and Manchester 1995:189).

5.3.5 Congenital and Developmental Disorders

Severe congenital defects are unusual findings in the archaeological record. Most affected individuals die in childbirth, shortly after or during infancy (Roberts and Manchester 1995:). The fragile nature of immature skeletons, particularly those with congenital defects would have facilitated their early elimination from the archaeological record (Aufderheide and Rodríguez-Martín 1998; Barnes 1994). In addition to the fragmentary nature of immature remains, multiple etiologies, complex presentations and the frequent association of many congenital abnormalities, make it difficult to differentiate between disorders (Pedersen and Anton 1998; Richards and Anton 1991).

One cranium (5.3%) from ABS was tentatively classified as exhibiting a congenital or developmental disorder. While the exact disorder cannot be established, the specific features recognized through the process of description and differential diagnosis indicate that the lesion is developmental in origin. Case #7 is a female of undetermined age, although active cribra orbitalia and open sutures suggest that the individual was young.

5.3.6 Joint Disease

The prevalence of joint disease was calculated for the atlanto-occipital joint and the TM joint. According to Waldron (1994:54-55), prevalence should be counted using each joint as the unit of analysis. Since only cranial material was evaluated in this thesis, the atlanto-occipital joint was represented by the occipital condyles. Of the four-recorded basi-occiputs, none presented evidence of osteoarthritis. No mandibles were associated with the 19 individuals in this sample; as a result, the temporomandibular joint was represented solely by the temporal fossa. Of the 14 recorded TM joints[4], only one (7.1%) exhibited evidence of osteoarthritis. Case #6, an adult female, exhibited severe degeneration of the left joint. Unfortunately, the right temporal was not recovered. This female represents 14.3% of the female TM joint sample.

Case A, from outside the main sample, exhibits severe unilateral osteoarthritis of the right TM joint. As will be discussed below, it is possible that the severe nature of the arthritis precipitated the possible trephination located at bregma.

[4] A MNI of 11 was recorded for the 14 recorded TM joints.

It is obvious that care must be taken in interpreting the above data. Given that joint disease is the most commonly observed skeletal disorder in prehistory (Alpagut 1979: 571; Aufderheide and Rodríguez-Martín 1998:93; Roberts and Manchester 1995:100; Waldron 1994:57) the prevalence of joint disease is in all likelihood underestimated for the ABS sample. According to Waldron (1994:57), osteoarthritis should be the most common affliction of the skeleton, no matter the provenance. If this is not the case, as with ABS, the researcher can be reasonably certain that there is something wrong with the sample. Given that not only is the sample small and likely biased, the fact that the cranium contains only two joints susceptible to arthritis, it is not surprising that the data do not conform to expectations. Examination of isolated cranial elements should provide further evidence for osteoarthritis.

Dental trauma, ATML, malocclusion and biomechanical stress are the most common etiological factors for osteoarthritis. However, due to postmortem damage, none of these predisposing factors were observable on either cranium. As will be discussed below, the severe osteoarthritis on the right articular fossa of Case A may present a relationship with the skull's possible trephination.

5.3.7 Human Modification

There is no evidence of human or cultural modification in the 19 crania. One undetermined crania, (Case #17), includes a healed complete trephination as one of the competing explanations for the perforation located on its inferior left parietal. There is not enough evidence to support a diagnosis of trephination, and another explanation, osteomyelitis, is more compelling.

Two crania from outside the main sample exhibit evidence of trephination. Case A is an adult female with a probable complete trephination located at bregma. Scraping or abrading was the likely method of operation (Campillo 1984; Lisowski 1967; Parker et al. 1986; Stevens and Wakely 1994). The trephination is partially healed and exhibits associated osteitis of the vault, with further evidence of a more severe infection of the endocranium below the trephination. Given the location of infection directly below and lateral the perforated portion of the vault, it is likely that the two are related. Most intriguing is the evidence for osteoarthritis on the right TMJ. The lesion was recorded as 'severe', which suggests that it is longstanding and predates the possible trephination.

Case B is an adult male with a probable complete or incomplete trephination located at bregma. The lesion is very similar in morphology to Case A. Again, scraping or abrading was the most likely method of operation (Campillo 1984; Lisowski 1967; Parker et al. 1986; Stevens and Wakely 1994). The probable trephination exhibits significant healing and evidence of diffuse periostitis, with no evidence of a presupposing condition. The diffuse porosity and extensive thickening over much of the parietal bones and occipital bone in association with healing suggest that the individual survived an extended period of time after the probable trephination (Chege et al. 1996). Of note are the two sets of cutmarks across the frontal bone. The first set are shallow and run in a horizontal direction in parallel bunches, while the second set are much deeper and run in a vertical direction. They do not exhibit the same degree of bony inflammation associated with the probable trephination and superior vault. While it is possible for the vault to exhibit differential healing, another explanation may be that the scalp was reflected at a later date

Figure 5.1 Putative trephination from the Eneolithic Site Grotte de Furninha. Fig. 68 is the cranial fragment with round fossa possibly suggestive of a "turning" trephination. From Delgado (1880:219).

to examine the original trephination. Alternatively, they may be related to a postmortem ritual as yet unidentified.

At this time, there is limited evidence for trephination in Portugal. Piggott (1940: 127) in his extensive review of European trephination refers to two trephinations from Grotte de Furninha and Casa da Moura [5]. Piggott summarizes the evidence from Casa da Moura as a "trepanned skull in eneolithic burial cave". Delgado (1880: 219) describes a cranial fragment from Grotte de Furninha, as exhibiting a round ectocranial fossa, 20 mm wide and 5 mm deep with little evidence of healing. He suggests that the lesion was possibly related to superstition or early surgical procedure, similar to trephination Further, he feels that the round shape of the lesion indicates the use of a "turning instrument " (Delgado 1880: 219). The putative trephination is pictured in Figure 6.1. Another cranium (adult male) from the Late Neolithic site Gruta do Lugar do Canto is reported to exhibit four scraping trephinations and various other "pathologies" including fractures, periostitis, arthritis, and a collapsed vertebrae (Leitão et. al. 1987).

While neither Case A and B exhibit direct evidence in the form of diagnostic trephinning marks, the shape, relatively shallow nature and small perforation (of Case A) suggests that scraping was the operative method (Brothwell 1994; Chege et al., 1996: Lisowski 1967; Smith 1990). After reflection of the scalp, a stone or shell implement was likely abraded gently across the cranium, eventually exposing the dura. Unlike other methods where a roundel of trephined bone is eventually acquired, the scraping method removes bone in a powder form (Piggot 1940). The gradual erosion of cranial bone results in a wide bevel around a shallow lesion, at the centre of which a small perforation may form. It is reasonable to hypothesize that for trephinations along the sagittal, the scraping method was preferred. Scraping allows for greater control than other methods and demonstrates a higher post-operative survival rate (Parker et al. 1986; Zias and Pomeranz 1992). In Europe, and elsewhere the scraping method of trephination is the most common, (Brothwell 1994; Jennbert 1991; Mallin & Rathburn 1976; Parker et. al. 1986; Perrson 1977; Piggott. 1940), therefore, it is not surprising then that it is the identified method for the probable ABS trephinations.

Although the first trephination reported in the literature met with certain skepticism, it was not long before the medical and anthropological community was fascinated by this evidence of "a peculiar type of prehistoric surgery" (MacCurdy 1905). From the first publication on trephination by Paul Broca in 1867[6] to today, debate over the prehistoric motives of the procedure has dominated almost every treatise on the subject (Campillo 1984; Jennbert 1991; Hrdlička 1939; Lisowski 1967; Margetts 1967; Rifkinson-Mann 1988). The possible explanations for trephination include a) treatment of trauma or therapeutic relief of headaches and illness, b) for religious and ritual purposes, and c) to obtain amulets from the living or dead. In modern medicine, trephination is preformed in the treatment of cranial fractures and to relieve intracranial blood clots (Aufderheide and Rodríguez-Martín, 1998; Bennike, 1985: 65); a number of apparent prehistoric examples exist in the literature (Capasso and Di Tota, 1996; Chege et al. 1996; Mallegni and Valassima 1996; McKinley 1992b). In the absence of traumatic lesions or evidence of what Brothwell, (1994:134) terms "rational surgical treatment" many researchers look towards ethnographic sources for an explanation. Margetts

[5] Unfortunately, an extensive search for Cartailhac (1886) was unsuccessful.
[6] Discovered by E.G. Squire in Cuzco Peru in the mid-1800s

(1967:682) records that the Lugbara of Uganda perform trephinations to "let out the evil spirit which was causing an intractable headache".

A review of the literature suggests that a majority of reported European and Middle Eastern trephinations were preformed on males (Alt et. al. 1997; Germanà and Fornaciari 1992; Koca and Schultz 1994; Lillie 1998; Mallegni and Valassima 1996; Manolis et. al. 1994; McKinley 1992b; Jennbert 1991; Parker et al. 1986; Persson 1977) as opposed to females (Capasso and Di Tota 1996; McKinley 1992a; Parker et al. 1986; Robb and Mallegni 1994; Zias and Pomeranz 1992). Parker and colleagues (1986) review 28 trephined crania from English sites of which 16 (57 %) were identified as male, two (7%) were female and the rest were unidentified. In Sweden, Jennbert (1991) comments that trephination presents a strong male dominance from the Stone Age to the Bronze Age and Prioreschi (1991), notes that adult males represent the majority of trephinations on Neolithic skulls. It is interesting to note that while predominance of male trephination is very obvious, few researchers comment on the tendency.

Cranial trauma is evoked as being especially indicative of interpersonal violence, usually common in males. It is not difficult to reach the conclusion that the high rate of trephination in males may be related to their high rate of trauma (Walker et al., 1989). That being said, most trephinations cannot be directly attributed to a prior cause, traumatic or non-traumatic. As Campillo rightly points out, the preponderance of "nuerosurgical affections do not predominate in the left side of the skull, neither the parietal nor in adult males" (*sic* 1984:279). The fact that a high proportion of trephinations from Neolithic Europe are male suggests that there may be a ritual dimension to the procedure. According to Parker et al. (1986:153), evidence of ritual trephination requires supporting evidence from differential treatment of the archaeological or skeletal remains.

According to Jennbert (1991), there is a preoccupation among palaeopathologists, archaeologists and medical historians for discovering the medical explanation for trephination. She argues that the close link between medicine and religion is overlooked in the stridently rational approach that dominates 20th-century research. Indeed, as the above quote from the Lugbara implies, it is unreasonable to suggest that medicine, religion and ritual played separate and distinct roles in prehistoric communities. In all likelihood, the practitioners of ritual and religion, and medicine and healing were the same individuals (Jennbert 1991).

An examination of both the possibly trephined crania from ABS is interesting in light of the above discussion. Case A is an adult female, unusual in the context of trephination in Neolithic Europe. It also exhibits evidence of severe, unilateral TMJ osteoarthritis. It is possible, and even tempting to suggest that the trephination was a palliative attempt to cure the pain caused by the arthritis. There are other examples of a trephination associated with non-traumatic disorders. Barnes and Ortner (1997) identified lesions consistent with multifocal eosinophilic granuloma in association with a trephination. While Mann (1991), suggests that a trephination on a Peurvian skull may have been an attempt to relieve the pain and discomfort of a chronic ear infection and cholestoma[7]. In one of the most compelling examples of non-traumatic palliative trephination, Zias and Pomeranz (1992) identify on a skull from Jericho, serial surgical lesions associated with an advancing intracranial infection. They propose that a

[7] In this case, it is important to note that the trephination occurs on the side opposite the ear infection.

craniectomy to relieve a frontal sinus infection lead to the spread of the infectious process to the outer surface of the skull. The degree of healing suggests that the individual survived still another trephination designed to relieve the symptoms. A final trephination exhibits no evidence of healing. While these cases are interesting and even compelling, it very difficult to authoritatively link non-traumatic trephination to a specific cause (Richards 1995).

Case B is an adult male with no evidence for a predisposing lesion, however a portion of the vault, including the right temporal is missing. Like most trephinations, there is no evidence to suggest the cause of the procedure (Campillo 1984:279-280). Case A was found in small cluster of crania (~5) in Sala C (Pedagagos); however, this is not an unusual feature at ABS and there is, at this time, no further evidence to suggest differential treatment of either case. Also of interest are the cutmarks on the frontal of Case B. While they may or may not be related to the possible trephination, they provide further evidence of the kinds of cultural activities that are reflected on the bones of ABS.

5.3.5 Unidentified Material

The differential diagnosis of four crania (21.1%) proved unable to reveal the etiology of the lesion. The undetermined crania represent two of the cases for which age and sex could not be determined (Cases #11, and #13), while a middle adult female (Case # 5) represented one and an old adult male (Case #17) the other. Surprisingly, or perhaps not so surprisingly given the nature of palaeopathology, it was not only the incomplete crania that went undiagnosed, but also both complete and partial crania presented problems for diagnosis (Cases # 5, # 11, #13, and #17).

For two of the cases the differential diagnosis suggested a list of possibilities, Case #13 presents an inflammatory condition of possible infectious (osteomyelitis) or metabolic origin (Paget's disease), while Case #5 presents a small perforation of the temporomandibular articular fossa than may be the result of postmortem damage or rheumatoid arthritis. Two of the crania, Case #11 and Case #17 presented, such ambiguous evidence that none of the possibilities considered in the differential diagnosis were sufficient given the current state of information.

Of the four undiagnosed cases, two were represented by lesions that were primarily lytic in origin (Cases #5, Case #17), while another (Case #11) presented lytic lesions in association with hyperostotic changes. Lytic lesions are difficult to diagnosis for several reasons. First, they are more susceptible to postmortem damage than abnormal hyperostotic bone (Waldron 1987). Second, if the lytic lesion occurred in the perimortem period they may be difficult to distinguish from postmortem damage. Third numerous conditions are characterized by osteoclastic or lytic lesions while osteoblastic or hyperostotic bone represents fewer disorders. Finally, lytic lesions tend to present more non-specific evidence than osteoblastic lesions (Ortner and Putschar 1984).

CHAPTER 6

CONCLUSIONS

6.1 Evaluation of Differential Diagnosis

This thesis presented the descriptions and potential differential diagnoses from a sample of 19 crania from Algar do Bom Santo, Portugal. Two other crania from the site were also analyzed, but not included in the main sample. Because of the commingled and fragmentary nature of much of the sample, it was hypothesized that the most effective means of identifying the origin of abnormal bone change was differential diagnosis. Differential diagnosis is a method of analysis that places emphasis on description and careful consideration of competing conditions (Molto 1990; Roberts and Manchester 1995:6; Waldron 1994:28). While the ultimate goal of most diagnoses is to identify the etiology or origin of bone change, for the purposes of this thesis there were three realistic conclusions: 1) a diagnosis or a 'possible' diagnosis, 2) recognition of disease category (e.g. trauma, congenital disorder) and 3) unknown cause. If the cause of the bony changes remained undetermined, every effort was made to recognize the predominant osseous activity (e.g. abnormal bone loss, abnormal bone formation, postmortem alteration). Several important observations were made regarding the effectiveness of differential diagnosis in evaluating fragmentary and commingled remains.

Differential diagnosis was reasonably effective as a means of identifying the pathological conditions of the crania. It was most successful at recognizing trauma, trephination and osteoarthritis; however, it is not altogether unsurprising that singular and morphologically discrete lesions were the easiest to identify. Trauma, (Cases #1, #2) postmortem damage (#9) trephination (A and B) and osteoarthritis of the TMJ (#6, #20 and A) do not require evidence from diverse parts of the skeleton to confirm their diagnosis. Further, the operational criteria and diagnostic characteristics are reasonably clear for trauma and osteoarthritis because of their prevalence in archaeological remains. In several cases (Case #1 and #6) the differential diagnosis was used more to clarify osseous changes than to identify them per se. For example, in Case #1, a full differential diagnosis was not required to establish the pathogenesis of the lesion, rather the process was used to evaluate the type of fracture and its timing, while in Case #2 the differential diagnosis was used to distinguish a healed depressed fracture from several possibilities.

Differential diagnosis was moderately successful at determining the categories of disease for several of the crania with more ambivalent lesions. In Cases #3, #6, #7, #10, #15, #16 and #18 the potential disease category was recognized through the descriptions of osseous change and the differential diagnosis that eliminated inconsistent explanations. Differential diagnosis was limited in ability to recognize fully the actual disease process for a number of reasons: poor preservation, fragmentary and isolated material, disassociation from the post-cranial skeleton, lack of diagnostic features on bone and the non-specific nature of osseous change reflecting the ambiguity of the condition. In other words, for some of the crania the incomplete nature of the remains was a barrier to effective diagnosis, while for others, associated cranial and post-cranial material may not have provided the answer because the condition is inherently non-specific. Cases #13 #15, and #16 effectively illustrate the last point. While hypothetical evidence from the post-cranial skeleton may provide additional clues as to the etiology of the ambiguous

and/or non-specific vault lesions, it is just as likely that the post-cranial skeleton would not provide an indication as to the cause of the changes. In situations such as these, the differential diagnosis was effective in determining what the lesions *did not* represent.

It is tempting to consider an "undetermined" or "unknown" differential diagnosis a failure; however, a differential diagnosis may still provide interpretable evidence. In this thesis, several of the undetermined crania (Case # 3, #7, #9) were classified as representing one or more of the possible disease categories. For the unknown material, (Case # 5 # 13 # 11 and # 17) the description and differential diagnosis was at least helpful in determining the kinds of osteological processes that may have been involved. It is possible that a more succinct classification of the abnormal changes may not be obtainable because of the significant overlap that several conditions and general disease categories display (Miller et. al. 1996). Finally, as stated previously, differential diagnosis requires that inconsistencies are considered and evaluated against the consistent data. As a result, differential diagnosis, whether successful in identifying the disorder or not, ensures a reasonable degree of reliability in the final analysis (Roberts and Manchester, 1995:199)

In light of the above project, there remain several limitations to differential diagnosis. Some of these are inherent to palaeopathology and the study of archaeological skeletal material. First, as stated above, differential diagnosis could not definitively identify those lesions representative of disorders that affect diverse parts of the skeleton. Second, it can be a long and occasionally cumbersome process that is heavily reliant on the literature (both clinical and palaeopathological) to facilitate diagnosis. While the clinical research is a good source of information, most descriptions are based on soft tissue morphology and radiographic observations and as such frequently require "translation" in relation to dry bone morphology. Further, epidemiological profiles and morphological descriptions presented in the literature are derived from modern cases, which may contrast profoundly from archaeological examples (Miller et. al. 1996; Ortner 1991). The effects of medical intervention, iatrogenic pathology, improved health status and increased longevity need to be filtered from clinical descriptions in order to be applied to past specimens. Conditions that were acute and frequently resulted in death in prehistory may be prolonged, drastically altered, and even cured with modern medical intervention (Molto 1990).

The focus of palaeopathological literature upon the 'classic' morphological expressions of disease can and should be considered a limitation to both differential diagnosis and palaeopathology. According to Miller et al. (1996:225), the classic expression of a given disorder frequently represents the "narrow tails of the normal distribution". In this thesis, very few crania exhibit 'classic' expressions of their identified disorders or disease categories (Case #1 is the possible exception). Cases A and B illustrate this point effectively. While the criteria they present are consistent with trephination, they lack the classic or typical features many researchers have come to expect in trephination: gaping perforations, bore holes, cross-hatch marks and cutting or scraping marks. These features, typical to the earliest examined trephinations from Peru are not always identified on European crania where the scraping method of bone removal was the most common (Brothwell 1994; Jennbert 1991; Mallin & Rathburn 1976; Parker et. al. 1986; Perrson 1977; Piggott 1940).

According to Powell, (1991:179) "differential diagnosis should always be sensitive to the possibility of multiple pathological conditions simultaneously affecting an individual". It can be difficult to mediate the multiple effects of life and death including: growth, disease, healing, degeneration, cultural practices, postmortem alteration and processing, burial, taphonomy, excavation and curation. Like the archaeological palimpsest, all of these processes may be compressed into one layer and require careful consideration and interpretation. This is demonstrated in Case A, where the visible effects of osteoarthritis, trephination, healing, possibly two courses of infection, and postmortem destruction all demonstrate osseous changes identified in the process of differential diagnosis. While care was taken in this thesis to identify the various processes at work, it was not always possible to fully appreciate their relationship to the changes observed on the skull. For example in Case B, the relationship between cutmarks, trephination, infection and healing is ambiguous and remains unclear. In another example, the effect of weathering, multiple healed and unhealed lytic lesions associated with hyperstotic bone formation in conjuction with fragmentary cranial remains preclude the diagnosis of Case #11.

In a clinical setting, the differential diagnosis is contingent upon the collection of reliable and relevant information in the form of histories, physical examination and laboratory analysis. Within a palaeopathological context, emphasis on reliability takes on a new level. Bone changes pathognomonic of specific disease states are rare, and the function of time, taphonomy and burial environment can obscure both diagnostic and non-specific lesions. Differential preservation of bone can obscure the prevalence of abnormal bone on the level of the element, individual and population. For these reasons, the palaeopathologist must reconcile the fact that a definitive diagnosis may not always be the likely outcome of the diagnostic pursuit. By emphasizing descriptive information and ruling out competing etiologies, the palaeopathologist has established a viable framework not only for the classification of abnormal bone but also for further research.

6.2 Evidence for Palaeopathology.

As stated in the introduction, the objectives of this thesis were the description and differential diagnosis of the cranial material, recognition of different disease categories, identification of the presence or absence of trephination and the calculation of disease prevalence. From these objectives, a number of interpretations were offered and several valuable conclusions can be put forth.

First, the sample did not conform to unity or the u-shaped age distribution expected in pre-industrial societies, suggesting that either mortuary practice or a significant bias skewed the sample (Waldron 1994). However, when the ratio of sub adults to adults was considered, juveniles made up 22.5 % of the population. According to Waldron (1994: 23) in a healthy, nonbiased sample, sub adults may represent upwards of 30 % of the population. At this time, the most parsimonious explanation for the skewed profile relates to the materials (e.g. fragmented, commingled), methods (cranial suture closure) and deliberate sampling bias (crania and partially excavated site), rather than the differential burial of select members of the population. On the other hand, it must be remembered that the evaluation of the mortuary space and the relationship of the skeletal materials is ongoing, and as yet we do not understand the funerary behaviours associated with ABS.

Second, depressed fractures of blunt force origin were identified on two crania from ABS. In both cases, the skulls were young to middle aged males with lesions on the left parietal. According to Roberts and Manchester (1995:79), "skull injury usually represents intentional blows". While there is insufficient evidence to interpret the behavioral context of the fractures, it is interesting to note that it conforms to a 'universally' common pattern of trauma that is frequently interpreted as representing interpersonal violence (Walker 1989). Another vault exhibits evidence of blunt force trauma on the right parietals and skull base but the timing of the lesions remain ambiguous.

Third, the causes of iron-deficiency anemia identified in the ABS sample cannot be established at this time because the life style and dietary patterns of the Late Neolithic people are as yet unknown. Nonetheless, extension of the research from earlier periods does not support the usual contention that there was a decrease in dietary quality or health status in the Neolithic following the transition to agriculture (Jackes et. al. 1997b; Meiklejohn et. al. 1984). Stuart-Macadam (1992:161) points out that coastal populations have higher incidences of porotic hyperostosis than highland populations. This may be related to the proximity of marshlands with high pathogen loads, or the ingestion of marine resources with fish and water born parasites (Walker 1986). Certainly, the location of ABS on Montejunto near the Atlantic Ocean suggests that the population had access to the coast. It is also possible that the rate of infection in the ABS sample is responsible for the mild porotic hyperostosis and cribra orbitalia observed in this sample. Of the crania that demonstrate some evidence of infection, two (n~7 = 28.6%) present porotic hyperostosis or cribra orbitalia.

Fourth, after porotic hyperostosis, infection is the most commonly observed pathological condition in the sample. Common infections including mastoiditis, otitis media, periapical abscesses and infection related to traumatic events (trephination) were all identified with a reasonable degree of reliability. The most interesting evidence for infection represents tantalizing and ambiguous results. Three cases of non-specific infection were recognized in the ABS sample. Two of these present lesions that bear similarities to treponemal disease; however the Late Neolithic date of ABS is several thousand years before the most universally accepted dates in Europe (Roberts and Manchester 1995:158).

While no comparisons were made with other samples, the significant degree of infection could be seen as supporting Meiklejohn's et al.'s (1984) observation that there is an increase in cranial infection from the Mesolithic to the Early Neolithic in Western Europe. This may reflect the increasing population densities and sedentism associated with the shift to agriculture (Larsen 1995:199; Roberts and Manchester 1995:129). Further analysis and comparison may establish that the ABS sample represents a continuation of this trend coinciding with the intensification of agriculture.

Finally, ABS provides evidence for cultural activity related to the skeleton and the body. As noted in Chapter three and five the skull base is under represented in the ABS population and sample. Only four skull bases from 19 crania (26.3%) were at identified as partially intact[1]. Of these, three (75 %) exhibited significant perimortem/ postmortem trauma in the region. Case #5 depicted in Plate 8, presents angular fractures perpendicular to the foramen magnum suggestive of postmortem damage to the skull base. These

[1]Partially intact refers to the presence of at least 30% of the foramen magnum.

fractures may represent evidence of mortuary behaviour, and therefore may have particular significance in terms of understanding the burial context of ABS.

Case B exhibits cut marks that may or may not be related to possible trephination. The series of parallel lines across the frontal are similar to those related to scalp reflection (Hurlbut 2000; Owsely et al. 1994), and while the lack of healing suggests they may not be related directly to the procedure, they do represent intentional modification of the skeleton. Another set of cutmarks that are deeper and exhibit a series of grooves are found on the same frontal, above the parallel marks, orientated in an anterior-lateral direction. Again, because of the lack of observable healing, it is difficult to establish the origin of the marks, but their randomly placed nature may suggest defleshing of bone in the postmortem period (Hurlbut 2000).

Final evidence of cultural activity can be found on the two crania from outside the main ABS sample that demonstrated lesions suggestive of trephination. Through the process of description and differential diagnosis, this hypothesis was established as "probable". Observations of the *in situ* material suggest that there may be evidence of at least one more cranium with a similar lesion. Trephination is the direct result of the deliberate human intention to modify bone. As Richards (1995:207) states it can "bear(s) directly on our extremely limited understanding of medico-surgical and medico-religious practices" of past groups. The similarity in form for both possible trephinations from ABS is striking. Although orientated differently at bregma, the lesions are virtually the same size and unusual shape. The fact that the possible trephinations are so similar might suggest that the morphology of the lesion was determined by a strong regional, cultural or individual influence.

It must be clarified that before such conclusions can be reached, the temporal and spatial relationship of the crania must be resolved. The crania come from two different, although connected regions of the cave and are associated with different dates. Case A, was removed from Sala C, with dates ranging between 4630 ± 60 and 4780 ± 50 BP (uncalibrated), while Case B was taken from unexcavated portion of Sala A with dates ranging from 4030 ± 80 to 4860 ± 100 BP (uncalibrated). According to (Monge Soares, cited in Duarte 1998:113) these dates are not significantly different, and as yet we do not have any information that suggests temporal differences in associated artifacts or mortuary practice. Further consideration of the possible trephinations should be made in light of the other noted examples from Casa da Moura, Grotte de Furninha , and Gruta do Lugar do Canto (Delgado 1880; Leitão et. al., 1987; Piggot 1940).

6.3 Conclusion

Despite the limitation of working with fragmentary and commingled remains, evidence for trauma and a variety of disorders were recognizable on the crania from Algar do Bom Santo. While definitive explanations could not be provided for all the pathological or abnormal crania, differential diagnosis was an effective analytical tool in the recognition and classification of the skulls. The emphasis on description and careful consideration infuses a degree of reliability to the diagnoses that offsets the certain limitations of the procedure (Ortner 1991; Roberts and Manchester 1995).

The biocultural approach advocates using the skeleton as a "window to understanding the population from which the individual is drawn" (Meiklejohn and Zvelebil 1991:129). The small sample size and as yet limited understanding of the

cultural and ecological context of ABS makes it difficult to provide definitive interpretations on what certain diseases and their prevalence's "mean" (Ortner 1991). Nonetheless, the recognition of a variety of disease categories form Algar do Bom Santo inspires questions and provides an important template for further research. Further, disease provides insight into the lives of past individuals, and the skull perhaps more than any other element is the most biologically and culturally meaningful element of the body. Evidence for trephination from Algar do Bom Santo is an elusive and intriguing glimpse into this population.

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APPENDIX 1

BURIAL LOCATION AND OSTEOBIOGRAPHICAL SUMMARY

Table Appendix 1.1 Osteobiographical Profile of Algar do Bom Santo Cranial Sample.

Case #	ABS Identification	Room & Unit	Burial Type	Age	Sex	Completeness	Pathology
Case # 1	ABS.3058/2339	B-C2	secondary	YA	?M	50-90%	Perimortem depressed fracture; porotic hyperostosis/cribra orbitalia
Case # 2	ABS.906	B-B3	primary	MA	?M	50-90%	Possible antemortem healed fracture; porotic hyperostosis/cribra orbitalia
Case # 3	ABS.2304/2312/194/A.D2.11/A.E2.87	A-C2; A-D2; A.D3; A.E2	secondary	Adult	U	50-90%	Possible metabolic/endocrine of the skull
Case # 4	ABS.3191	A-E1	secondary	YA=34ys	M	50-90%	Porotic hyperostosis
Case # 5	ABS.3118	A-E0	secondary	MA(L.V.=40)	F	>90%	Undetermined: postmortem damage versus rheumatoid arthritis; porotic hyperostosis
Case # 6	ABS.3252	A-E0	secondary	A	?F	50-90%	Possible otitis media; cribra orbitalia; osteoarthritis
Case # 7	ABS.631	A-C2	secondary	A	F	50-90%	Possible developmental defect; cribra orbitalia
Case # 8	ABS. 461	A- C5	secondary	OA(L.V=5 6.2)	?F	> 90%	No
Case # 9	ABS.459	A-C5	secondary	OA	M	<90%	Perimortem/postmortem trauma; periapical abscess; porotic hyperostosis
Case # 10	ABS.A.D2.3/ ABS.463/462/477 /500	A–D2 & A-E1	secondary	Adult	U	50-90%	Mastoiditis of the right mastoid.

Case #	ABS Identification	Room & Unit	Burial Type	Age	Sex	Completeness	Pathology
Case # 11	ABS.3248/2291/3 334/2947	B-C2	secondary	Adult	U	50-90%	Undetermined erosive lesions accompanying hyperstotic changes to the vault
Case # 12	ABS.1251	A-D2	secondary	Adult	?F	50-90%	No
Case # 13	ABS.A.E2.19; A. E2. 89	A-E2	secondary	Adult	U	50-90%	Undetermined inflammatory (infectious or metabolic) condition of the vault
Case # 14	ABS.B.B4.65	B-B4	secondary	YA	?F	50-90%	Osteoma of the frontal
Case # 15	ABS.A.E2.31	A-E2	secondary	OA	?F	50-90%	Possible non specific infection of the vault
Case # 16	ABS.3135	A-D1	secondary	Adult	?F	50-90%	Possible non specific infection of the vault
Case # 17	ABS.B.B5.329	B-B5	secondary	Adult	M	50-90%	Undetermined perforation of the left parietal; Caries and dental abcess
Case # 18	ABS.A.D2.58/A. D2.6/A.D2.87	A-D2	secondary	Adult	U	<50%	Possible non specific infection of the vault associated with hypervascularity; ectocranial porosis
Case # 19	ABS.A.E2.38	A - E2	secondary	Adult	U	<50%	No
Case A	ABS.C.J.	Sala de Pedagas (C) Nuclio J	secondary	Adult	F	50-90%	Possible trephination and associated infection; osteoarthritis of the right TMJ
Case B	ABS.A.D5.#	A	secondary	Adult	M	50-90%	Possible trephination and associated infection; cutmarks

APPENDIX 2

PALAEOPATHOLOGY OF THE CRANIUM

2.1 Introduction

Bone is a biological entity that is subject to both extrinsic and intrinsic modification. Many different taphonomic, cultural and biological processes affect the cranium. Biological processes, as well as human/cultural modification frequently manifest upon the cranium. While some conditions predilect the skull or show abnormal changes that are pathognomonic evidence of a specific disease process (e.g. syphilis, cribra orbitalia, and antemortem depressed fracture), others provide only passing or non-specific evidence of their involvement. Postmortem processes may or may not be difficult to distinguish on cranial material, but their elimination from pathological consideration must always be attempted. The skull, especially when considered as an isolated element may not only prove ambiguous – it may be misleading (Waldron 1987; Rogers and Waldron 1989). Care must be taken in recognizing what can and cannot be determined from the cranium.

Following a brief discussion regarding the nature of abnormal bone change, lesions of the cranial vault will be considered in the following categories: taphonomy and pseudopathology; trauma; metabolic, endocrine and hematological disorders; infectious conditions; neoplastic conditions; congenital/developmental disorders; osteoarthritis; miscellaneous conditions and; human and cultural modification. These specific classifications are based in part on Miller, Ragsdale and Ortner (1996), Roberts and Manchester (1995), Mann and Murphy (1991), Ortner and Putschar (1984), and Steinbock (1976). The categories should not be considered mutually exclusive, nor should this discussion be considered comprehensive of all conditions affecting the skull. A brief differential diagnosis of the major competing conditions associated with specific pathological conditions found on the skull is also provided.

The following section provides a review of the possible abnormal lesions that can be found on the skull in palaeopathological contexts. Given the vast amount of clinical and palaeopathological literature devoted to the description and identification of disease and trauma it is not remotely possible to provide a definitive review of cranial pathology. While brief details will be provided regarding the etiology, epidemiology, and palaeopathology of the specific disease processes, the focus of this chapter is the predominant osseous response and the defining morphological characteristics that can be observed on the cranium. In most instances however, further details of a particular condition will be provided if necessary in the Differential Diagnosis and Discussion parts of this thesis.

2.2 Taphonomy & Pseudopathology

Care must be taken to distinguish the results of taphonomic change in cave environments and postmortem damage as well as recognize the role of normal human variation when considering abnormal conditions of the skull. Pseudopathology refers to the extrinsic factors (postmortem damage) and intrinsic features (human variation) that act upon the skeleton to affect structural changes which mimic antemortem pathology (Aufderheide and Rodríguez-Martín 1998:11; Roberts and Manchester 1995; Wells 1964, Wells 1967). While a review of all the pseudopathological sources of postmortem and

biological variation of the cranium is beyond the scope of this chapter, several factors should be borne in mind when observing cranial material. Among the postmortem alterations that should be considered are: burial environment, animal and insect activity, breakage and human modification.

Care should be taken to reckon the role of the burial environment and its potential to cause deformation, postmortem fractures and erosive lesions on the skull (Henderson 1987; Wells 1968). Over time, the burial environment may exert pressure on the vault leading to deformation and/or fracture of the skull that may mimic certain congenital disorders or intentional cranial deformation (Aufderheide and Rodríguez-Martín 1998; Henderson 1987). Areas of the skull that have already undergone antemortem disease and traumatic processes as well as the structurally weak areas of the skull, such as the base and face are more susceptible to postmortem damage and loss because of soil pressure. This may result in an under representation of these areas and the diseases that affect them (Waldron 1987). Further, soil pressure as well as acidic burial environments, microbiological, and floral/fungal agents may also undermine the cortical surfaces of the skull giving the appearance of resorptive lesions (Aufderheide and Rodríguez-Martín 1998: 16-17; Fulcheri et al. 1986:73; Schiffer 1987:182-189).

In cave environments, a host of post-depositional changes can alter the burial environment and result in changes the skeletal material. Chemical processes may be responsible for macroscopic changes the bone's surface and microscopic changes to its mineral matrix. Depending on the conditions, water percolating through limestone cave sediments may result in the cementation or dissolution of the cave's constituents (rock, soil, bone, artifacts etc.) (Karkanas et al. 2000:916). Calcite deposits may obscure lesions, damage fragile bones, and destroy the cortical surface upon removal (personal observation).

Animals and insects may also affect pseudopathological changes on the cranium (Aufderheide and Rodríguez-Martín 1998; Micozzi 1991; Roberts and Manchester 1995; Wells 1967). Small animals may leave tooth marks that require differentiation from antemortem stab wounds postmortem cutmarks (Fulcheri et al 1986; Micozzi 1991:64), and bony inflammation (Aufderheide and Rodríguez-Martín 1998:16). Larger animals may leave considerable damage to the large bones. Tooth/bite marks and crushing may mimic perimortem trauma and postmortem human modification. Animals often remove bones or parts of bones resulting in the under representations of those elements (Henderson 1987; Micozzi 1991; Schiffer 1987; Waldron 1987).

As will be discussed below in the section on trauma, postmortem damage and perimortem trauma require differentiation in cranial remains (Lovell 1997a; Ubelaker and Adams 1995). This can be very difficult given that the diagnostic evidence for antemortem trauma is bony reaction in the form of healing or infection that may not be macroscopically observable for upwards of three weeks after the injury (Lovell 1997a: p). Palaeopathologists must therefore rely on other measures to distinguish perimortem and postmortem trauma (Ubelaker and Adams 1995). A final consideration is that pre-existing antemortem trauma or disease may facilitate postmortem destruction, obscuring the abnormal bone (Aufderheide and Rodríguez-Martín 1998; Henderson 1987; Ortner and Putschar 1984) and subsequently resulting in the under representation of disease and trauma. Finally, as with all palaeopathological diagnoses, it must remembered that it may not always be possible to distinguish from postmortem and antemortem events

(Aufderheide and Rodríguez-Martín 1998; Roberts and Manchester 1995).

2.3 Trauma

Trauma is defined as any internal or external force that results in an injury or wound to the body (Lovell 1997a: 139; Roberts and Manchester 1995:65). Trauma occurs as a result of environmental and occupational hazards (accidents, falls), interpersonal/ inter-group violence, suicide, intentional modification and surgery (Lovell 1997a; Merbs, 1989). While most palaeopathologists regard intentional modification and surgery as (inherently) traumatic events, for the purposes of this discussion they will be considered separately (Aufderheide and Rodríguez-Martín 1998; Merbs 1989; Ortner and Putschar 1984; Roberts and Manchester 1995).

Ortner and Putschar (1984:55) have classified osseous trauma into four categories: a partial or complete break of the bone; an artificially induced shape or contour; abnormal displacement of the bone or dislocation of a joint; and a disruption to the nerve or blood supply of the bone. While all categories of trauma are important, the first is the most significant for diagnosing and interpreting non-surgical trauma in cranial remains.

A fracture can be defined as the complete or partial disruption in the continuity of bone because of a traumatic event (Gauer and Roberts 1996; Lovell 1997a; Merbs 1989; Rogers 1992:19; Roberts and Manchester 1995:67). Fractures occur as a result of abnormal stress applied to the bone. Usually this stress is dynamic and results in a sudden, acute discontinuation of the bone's integrity. Occasionally however, fractures can occur due to pre-existing diseases, intrinsic weaknesses and repeated loading of stresses. Pathological and stress fractures are commonly observed in the post-cranial skeleton, but are less common in the cranium due to the lack of biomechanical stress (with the exception of the mandible) Harkess et. al 1991).

Proper description of the fracture is the most important step in the identification of trauma. From this basis, the "mechanism" (Lovell 1997a: 148) or "cause" (Roberts 1995: 129) of the injury (e.g. direct/indirect impact; blunt/sharp/penetrating force trauma etc.) can be determined, followed by the possible behaviour (i.e. manner) or event that ultimately instigated the fracture (e.g. fall, hammer blow) (Roberts 1995:129). Harkess and colleagues (1991:1) provide three important descriptive categories including: 1) anatomical location; 2) direction of the fracture line; and 3) whether the fracture is linear, depressed and/or comminuted. Fractures can be further classified as a result of "direct impact" (force applied directly to bone), or "indirect impact" (force transmitted from impact in another area) (Rogers 1992:23). The most commonly observed forms of trauma are the result of direct impact due to 1) blunt force trauma; 2) sharp force trauma (including penetrating and non-penetrating trauma); and 3) trauma related to less clearly defined mechanisms such as falls and crushing. Of these, blunt force trauma is responsible for most of the cranial fractures observed in archaeological contexts (Roberts 1995:131). Fractures resulting from both blunt and sharp force trauma are usually described as linear, depressed, penetrating (Lovell 1997a: 149) or a combination of the three.

Linear fractures are usually straight or curved and they may begin at the site of impact or arise at a distance in the structurally weak areas of the skull (Gurdjian et al. 1950; Knight 1991:165; Roberts 1995: 131). Singular linear fractures are usually the

result of a low velocity impact with an object or a fall (DiMaio and DiMaio 1989: 142; Tedeschi 1977:40). Linear fractures can be described as concentric, radial, spider or mosaic. *Concentric* fractures form as a series of independent arcs between the fracture lines that radiate from the margin of the impacted region (Smith et al. 1987). *Radial* fractures are usually described as one or more fissures that form out from the point of impact or transmit to the point of impact depending on the force and area involved. Knight (1991:166), defines *spider* or *mosaic* fractures as comminuted fractures with at least one, and usually multiple fissures radiating from the point of impact in a spider web or mosaic-like pattern. These fractures usually form under severe focal impact with or with out associated depression (DiMaio and DiMaio 1987; Knight 1991; Polson 1965).

Depressed fractures are usually the result of (relatively) high velocity blows from a small surface area that causes cranial bone to be driven inwards. The degree of force usually predicates the extent of comminution and inward cranial displacement (DiMaio and DiMaio 1989; Lovell 1997a). Depressed fractures may also present a number of variations including comminuted, stellate, and pond fractures, as well as spider or mosaic and concentric fractures. *Comminuted*, fractures occur when there are at least two linear fractures associated with an area of inbending (Roberts 1995:131). There can be a complicated mixture of linear fractures and depressed fragments of varying lengths and sizes that can be categorized as either expressed (displaced from the cranium) or intact (Polson 1965; Roberts 1995). Comminuted fractures form as a result of very high velocities or the repeated loading of multiple impacts (Gurdjian et al. 1953: 113). *Stellate* fractures form with moderate levels of velocity, causing radial fractures to form in the area of depressed bone (DiMaio and DiMaio 1989:142). *Pond* fractures form as a shallow concaved dent that may present small fissures and sharp edges on the periphery. On the pliable bones of children and infants, they are known as "ping pong" fractures (analogous to greenstick fractures) (Knight 1991:166; Polson 1965:133). *Concentric* fractures, when associated with depressed fractures radiate in a series of arcs from the margin of the inbended region (Smith et al. 1987). The fracture lines usually start on the outer table and propagate towards the inner table resulting in an internal bevel (Berryman and Haun 1996).

Penetrating fractures are usually the result of impact with small, sharp-edged objects (Lovell 1997a: 150) that usually do not generate enough force to completely penetrate the skull. The result is often clean-cut, elongated lesions that are V-shaped in cross-section (Stewart 1979:78). Occasionally linear fractures may result from penetrating trauma, and when the force is great enough depressed fractures may result (Lovell 1997a).

Other noteworthy types of fractures include: basal, ring and diastatic. Fractures to the skull base, or *basal* fractures form as a result of impact to almost any region of the cranium, in particular the vertex. The forces of the impact cause the fracture to transverse the fragile floor of the skull and separate it into two halves (Gurdjian 1953:229). *Ring* fractures form around the foramen magnum as a result of indirect impact from a fall. In falls from a considerable height the energy may not be completely absorbed by the post-cranium, and transmit up the vertebral column, resulting in the penetration of the cervical spine into the base of the skull (Knight 1991:162; Rogers 1992:45). Finally, *diastatic* fractures result from the separation of one or more cranial sutures due to traumatic impact. It is most common in the fusing and pliable skulls of infants and sub-adults. The

fracture may initiate as a linear fracture in one area of the skull and run partially down the length of the suture. The most commonly separated suture is the lambdoid, followed by the sagittal and coronal sutures (Moritz 1954; Grossart and Samuel 1961; Tedeschi, 1977).

In archaeological contexts, fractures to the cranial vault are more common than fractures to the skull base and cranio-facial area (Lovell 1997a; Merbs 1989; Roberts and Manchester 1995). While this is most frequently ascribed to the low preservation rate of these areas of the skull (Waldron 1987), it is also attributable to the fact that the vault provides a larger surface area and is easily accessible (in comparison to the skull base). Also, fractures to the vault and base are more likely cause death due to their proximity to the brain (Knight 1991:156; Roberts 1991:131). As a result, they are more likely to be observed in a perimortem context, while craniofacial fractures are usually non-lethal (Walker 1989), and are therefore remodeled before death.

The type of fracture is determined by a number of both extrinsic and intrinsic factors (Berryman and Haun 1996:3; Harkess et al. 1984; Roberts 1995:131). Extrinsic factors include the velocity, force, and duration with which the cranium is impacted, direction and angle of the impact as well as the size and shape of the implement or surface involved. The region of the skull that is struck is also important in determining the type of fracture and its severity. For example, traumatic impact to the mandible would not necessarily be as serious as an impact to the temporal bone. Intrinsic factors include the physical characteristics of the region of impact including, the scalp, hair, thickness and density of the bone, and elasticity of the skull (Berryman and Haun 1996). Further, as the skull is a unit, impact to one area may result in fracture in another. Fractures can migrate from or to the place of impact from a structurally weak area of the skull (Knight 1991: 165). For example, a blow or fall to the occipital may result in a fracture through the posterior fossa to the foramen magnum. If severe enough, the traumatic force will transmit through the brain and fracture the orbital plates (Knight 1991:165).

The most important distinction to be made in any analysis of cranial trauma is the differentiation of antemortem, perimortem and postmortem events (Lambert 1997; Ortner and Putschar, 1984). While macroscopic evidence of healing is evident within the first few weeks of a traumatic event (Lovell 1997a: 145; Rothschild and Martin 1993:58) the absence of a bony reaction is not in itself indicative of a postmortem event. Trauma occurring at or near the time of death – in the perimortem interval- does not have time to manifest reactive changes on the skeleton, therefore it can be very difficult to distinguish perimortem and postmortem events. The most commonly used criteria to determine the perimortem origin of the event are: 1) uniform colour of the bone surface, fracture margins and sutures; 2) round margins, radiating and depressed fractures; and 3) depressed fragments adhering the margins of the fracture. Also important is the presence of an inward bevel and spalling of the endocranial surface on the perimortem fracture (Hurlbut 2000:7). In contrast, postmortem events are most often characterized by a colour contrast and angular fractures with smaller fragments characteristic of dry, brittle bone. Regardless of these distinctions, it may be very difficult if not impossible to distinguish between trauma –peri or post- that occurs around the period of death (Berrymann and Haun 1996; Campillo 1991; Frayer 1997; Hurlbut 2000; Mann and Murphy 1990; Ortner and Putschar 1984; Robb 1997; Ubelaker and Adams 1995).

Antemortem trauma that displays evidence of healing, most notably depressed

fractures will require distinction from other abnormal processes. Cranial dyasphrism, syphilis, tuberculosis, metastatic cancer and trephination are all examples of disorders that manifest circular lesions to the cranium (Lambert 1997; Ortner and Putschar, 1984). Walker (1989:313), established the following criteria to aid in distinguishing healed trauma from other abnormal physiological processes: 1) absence of proliferative bone that would suggest the lesion is of infectious origin; 2) a tendency towards singular lesions; 3) lesions with well-defined and delineated margins in circular or ellipsoid shapes; and 4) the presence and/or retention of fracture lines associated at the periphery of the point of impact. In addition, Lambert (1997:84) notes that healed depressed fractures can be further distinguished from other forms of cranial pathology because of their smooth rim and internal bevel (widening from the ectocranium to the endocranium) (Berrymann and Haun 1996; Robb 1997) and the possibility that they have retained the shape of the implement involved (Courville 1967; Frayer 1997; Merbs 1989).

2.4 Metabolic, Endocrine and Hematological Disorders

The classification of diseases attributed to metabolic, endocrine and hematological dysfunction is by no means uniform in the clinical and palaeopathological literature (Miller et al. 1996; Ortner and Putschar 1984; Resnick and Niwayama 1988; Revell 1986; Roberts and Manchester 1995; Steinbock 1976; Zimmerman and Kelley 1982). This is likely a reflection of the interconnected relationship between these systems, the multi-etiological nature of the conditions ascribed to them, and the limited number of ways in which bone can change in response to stress. The fundamental basis of all bone disease is a disruption in the balance between the functions of bone resorption, deposition and mineralization (Ortner and Putschar 1984:36). In disorders of a metabolic, endocrine and hematological nature, this osseous disruption is highlighted. In the following section, particular attention will be placed upon the morphological expression of bone cell imbalance.

2.4.1. Metabolic Disease

According to Roberts and Manchester, (1995:163) disorders of the metabolic system can be loosely " considered as abnormalities of deficiency or excess" in both dietary constituents and hormones. While considerable focus has been placed upon metabolic diseases over the last 20 years to provide evidence of dietary stress (or lack of stress) in relation to major technological and social changes (e.g. agricultural revolution) (Cohen and Armelagos 1984; Meiklejohn and Zvelebil 1991), anthropologists have become increasingly aware that most metabolic diseases characterized by dietary deficiencies are actually more indicative of generalized stress (Martin et al. 1985). The term bioavailability refers to whether a specific nutrient or other substance is biologically available to meet physiological needs of the body (Dorlands 1982). The decreased bioavailability of a specific nutrient may be related dietary intake, inability to synthesize or absorb a particular nutrient, malnutrition, gastrointestinal disease, infectious conditions, genetic factors, growth of the skeleton and other constitutional factors (Martin et al. 1985; Palkovich 1987). Metabolic conditions reflect an imbalance at the cellular level of bone; therefore, particular attention should be paid to the primary osseous activity. Finally, it must be remembered that many metabolic conditions overlap and individuals under one form of stress easily become susceptible to other forms of stress

(e.g. anemia and scurvy can often be found in association in clinical studies) (Murray et al. 1990:594).

2.4.1.1. Abnormal Loss

Osteoporosis

Osteoporosis is the most frequently encountered manifestation of metabolic bone disease (Resnick and Niwayama 1988:2022). According to Revell (1986:183), the general definition of osteoporosis is a "diminution of bone mass without detectable differences from normal in the relative proportions of mineralized and non-mineralized matrix". Increased porosity marked thinning of cortices and reduction in the size and number of trabeculae (Zimmerman and Kelley 1982:56) are the characteristic features of the disorder. Osteoporosis reflects a prolonged imbalance between osteoclastic and osteoblastic activity that may be a condition in and of itself, or secondary response to other disease processes, dietary deficiencies, biomechanical needs, and non-specific stressors (Martin et al. 1985; Roberts and Manchester 1995). Primary forms are usually associated with older individuals, particularly postmenopausal women in whom there is a dramatic reduction in the osteoclastic inhibitor estrogen. Secondary forms are related to dietary, environmental and physiological availability of calcium, other vitamins, minerals and hormones essential to the body's ability to synthesize bone (Roberts and Manchester 1995). Other metabolic disorders, neoplasia, localized infection and trauma also exhibit signs of osteoporotic bone loss (Zimmerman and Kelley 1982).

Changes to the skull are usually mild, occurring late in both life and/or the course of the condition (Ortner and Putschar 1984). Occasionally biparietal thinning/atrophy caused by abnormal bone loss to the diploic and cortical tables of the cranial vault leads to smooth, bilateral depressions on either side of the sagittal suture. The depressions are large, ovid/ oblong, and may present perforated bone (Aufderheide and Rodríguez-Martín 1998:316; Lodge 1967:405). According to Ortner and Putschar, (1984:292), biparietal atrophy may be related to senile onset osteoporosis (primary).

Diagnosis is often confined to radiographic analyses and other clinical diagnostic methods (SEM, microradiography etc.), although the comparatively light nature of osteoporotic bone is often indicative of bone loss (Ortner and Putschar 1984). The differential diagnosis should include osteomyelitis (periosteal reaction) and postmortem damage. In the case of a perforated cranium, congenital failure of ossification, trephination and trauma should also be considered (Lodge 1967:408).

Rickets and Osteomalacia

Rickets and osteomalacia are two related metabolic disorders caused by an insufficient bioavailability of vitamin D. Skeletal abnormalities result from a failure of the organic matrix of bone or osteoid to mineralize. Vitamin D stimulates the intestinal resorption of the calcium and phosphate required to properly mineralize bone. Continued osteoclastic resorption of pre-existing bone in addition to its failure to mineralize effectively results in abnormal bone loss and generalized osteopenia (Resnick and Niwayama 1988:2096). While the trabeculae become thin and sparse, the cortical bone exhibits increasing porosity (Mankin 1974) and the continued deposition of osteoid results in softened bone subject to deformities of the weight-bearing skeleton (Murray 1990:561; Ortner and Putschar 1984: 274; Stuart Macadam 1989a). Rickets predilects

infants between four months and two years of age while, osteomalacia is the clinical manifestation of rickets in adults (Ortner and Putschar 1984; Roberts and Manchester 1995). It is most common in young adult females, and associated with a combination of poor diet, pregnancy, and a lack of sunshine (Resnick and Niwayama 1988: 2101; Zimmerman and Kelley 1982:60). Rickets and osteomalacia may be the result of inhospitable environmental contexts, physiological and malabsorptive disorders, cultural practices, and generalized protein-calorie malnutrition (Resnick and Niwayama 1988: 2100-2101).

Rickets and osteomalacia are most marked in those bones that have the highest remodeling rates, and therefore the greatest need for mineralization (Ortner and Putschar 1984:280). In infants, rapid skull growth requires accelerated remodeling resulting in softened calvarium overlain by thinned cortices. This abnormality, common to the parietals and occipital is known as craniotabes (Klein 1993:264; Mankin 1974:117; Ortner and Putschar 1984: 274) and may lead to marked flattening or "squaring" due to pressure against the head (Mann and Murphy: 1990:123). In rachitic infants, the persistence and widening of the cranial sutures in addition to marked bossing of the frontal and parietals creates a characteristic cruciate pattern known as "hot-cross-bun skull" (Mankin 1974:117). Resorption of the cortical surfaces and individual trabeculae continues along with the subperiosteal deposition of osteoid. The accumulated organic matrix is mineralized during the healing process presenting a fine pumice-like appearance that in cross-section resembles diploic expansion (Ortner and Putschar 1984:274). Decrease in skull-base-height and basilar invagination due to the weight bearing pressure of the skull has been observed in both rickets and osteomalacia (Angel 1982; Barnes, 1994; Murray 1990). Additional changes to the skeleton in osteomalacia are restricted to the post-cranium (Ortner and Putschar 1984:281).

Changes to the cranium in rickets (and not necessarily osteomalacia) can be suggestive given the required constellation of features. Differential diagnosis should include the identification of those changes due to rachitic malformation and those due to postmortem deformation, artificial cranial modification, and delayed suture closure. Paget's disease, hyperparathyroidism and changes due to iron deficiency anemia should also be considered. However, it should be noted that osteomalacia is often associated with secondary hyperparathyroidism and rickets commonly occurs in concert with other metabolic and protein-calorie deficiencies disorders such as anemia (Ortner and Putschar 1984:308).

Scurvy

Scurvy is a metabolic bone disease that occurs as a result of an insufficient bioavailability of vitamin C. While the primary etiological factor is presumably dietary, clinical studies suggest that it is unlikely for a broadly based diet to be specifically deficient in vitamin C. Scurvy therefore, is probably associated with general protein-calorie malnutrition (Gómez-Castro et al. 1994). Vitamin C is necessary for the body to successfully synthesize stable collagen the critical component of osteoid, cellular cementum, the periosteum and the periodontal ligaments (Murray et al. 1990:594; Ortner 1984:81). While there is no specific age predilection, individuals under the age of two and men (seafarers) are the most susceptible (Ramar et al. 1993; Stuart-Macadam 1989).

Changes to the skeleton are the result of two processes: those directly related to

the inability to synthesize stable collagen, and secondary lesions caused by the effect of trauma to the unstable skeletal tissues (Ortner and Putschar 1984:270). The inability to synthesize collagen results in reduced osteoblastic activity, while osteoclastic activity remains normal leading to abnormal bone loss, irregularly organized trabeculae and brittle bone (Resnick and Niwayama 1988). The primary affects are most marked in children in whom the demands of growth require large amounts of osteoid. In adults, where remodeling rates and physiological needs are significantly lower, generalized osteopenia is the most significant primary change.

Changes to the cranium are usually the result of secondary phenomenon. Subperiosteal hematoma with associated new bone formation (Ortner and Putschar 1984:270; Roberts and Manchester 1995; Williams 1994) is a common feature found on the frontal and parietal bosses, mandible, maxilla, and orbital roofs (Caffey 1978: 1460). In response to traumatic hemorrhaging, new bone is deposited under the elevated periosteum, eroding the cortical bone from the surface. This results in finely porotic reactive bone adjacent to the area of the hemorrhage (Ortner 1984). Orbital lesions reminiscent of the anemic cribra orbitalia, form as "vertically arranged trabeculae" deposited upon thin, partially absorbed cortical bone (Ortner and Putschar 1984:272-273), however, they do not exhibit the thickened and expansive quality of anemic lesions. Because the periosteum is tightly attached to the bone in adults, subperiosteal hematoma is more likely to occur in juveniles (Ortner and Putschar 1984:271). The failure of viable collagen also leads to hemorrhaging of the gums, weakened periodontal ligaments leading to gingival inflammation and periodontal disease. Alveolar resorption of the mandible and maxilla may eventually result in the exfoliation (ATML) of the teeth, particularly the anterior dentition (Ortner and Putschar 1984: 442).

Diagnostic changes to the skeleton in scurvy are primarily found in the post-cranial skeleton. If scorbutic orbital lesions can be differentiated from those of anemia, the constellation of features including subperiosteal hematoma, alveolar resorption and ATML as well as reactive porosity of the anterior face may provide suggestive evidence. The differential diagnosis should also consider whether the subperiosteal hematoma, alveolar resorption and ATML are secondary to, or independent of a scorbutic instability. It is also important to note that scurvy may be part of generalized protein-calorie malnutrition and that other specific dietary deficiencies and non-specific stressors may be attendant (i.e. iron deficiency anemia) (Ortner and Putschar 1984).

2.4.1.2. Abnormal Gain

Hyperostosis

Hyperostosis refers to the generalized abnormal increase of bone formation and ossification in the skeleton (Capasso 1997). The related term, osteosclerosis refers to an "increase in bone density without alternation to the overall shape" (Revell 1986:169) of the bone. The osteosclerotic apposition of new bone is often found in association with healing of traumatic and disease lesions, osteoblastic neoplasia, localized inflammation, and metabolic diseases (Revell 1986). Palaeopathologists use the term sclerosis or osteosclerosis to refer to dense new bone formation, while a more discrete, localized hyperostosis is commonly referred to as osteophytosis (Capasso 1997). While most metabolic conditions are characterized by bony destruction, there are several endocrine disturbances that exhibit abnormal bone formation.

Hyperostosis Frontalis Interna

Hyperostosis frontalis interna (HFI) is an obscure metabolic disease of possible pituitary origin (Aufderheide and Rodríguez-Martín 1998). Clinical studies demonstrate that it is most common in females who are post-menopausal, obese or pregnant (Barber et al. 1997; Roberts and Manchester 1995) although it can occur in males with hormonal disturbances (Hershkovitz et al. 1999).

Cranial changes are characterized by "mild to massive" new bone formation most common to the frontal (Armelagos and Chrisman 1988:27) although it has been observed on the temporal and parietal bones (Ortner and Putschar 1984:294). Aufderheide and Rodríguez-Martín (1998:419) and Ortner and Putschar (1984) report that the cross section reveals proliferative cancellous bone obscuring the diploic table and retaining only a thin inner table of cortical bone. However, according to an exhaustive study by Hershkovitz et al. (1999:304), there is almost no evidence of diploic or ectocranial involvement. Smooth, thick, tumor-like swellings or bony nodules give the endocranial surface a bumpy or 'rugose' surface (Armelagos and Chrisman 1988; Barber et al. 1997:158; Revell 1986; Roberts and Manchester 1995; 182; Zimmerman and Kelley 1982). HFI changes range from small islands or "endocranial elevations" through to osseous thickening and coalescence of the nodules so that their individual margins are no longer distinguishable. In advanced cases, bone formation continues and spreads to form "pancake-shaped" (Herskovitz et at. 1999:307) lesions with sharp margins and large nodules clustered together. HFI tends to form bilaterally across the frontal and exhibit well defined margins. Hershkovitz and colleagues (1999), also report that the periosteal surface of the outer table did not demonstrate an associated reaction. The differential diagnosis of HFI should include a consideration of Paget's disease, acromegaly, and leontiasis ossea, osteopetrosis, meningioma, osteoid osteoma and fibrous dysplasia (Armelagos and Chrisman 1988; Hershkovitz et al. 1999).

Leontiasis Ossea

Leontiasis ossea is a chronic condition in which the balance in osseous dynamics is grossly biased in favour of bone formation. Excessive bone formation of the cranial vault, mandible and facial bones present a gross distortion of the normal features and a disruption of the mechanical function. Onset is typically prior to adulthood. (Aufderheide and Rodríguez-Martín 1998; Ortner and Putschar 1984).

While leontasis ossea is a poorly understood condition, researchers recognize two different forms of the disease. In the first, proliferative new bone forms in nodules in the paranasal sinuses and orbits eventually occluding them. Thick and bumpy new bone also forms in 'creeping' layers on the periosteal surface of the face and vault. The underlying bone of the diploic table thickens and expands in size. In the second, the pre-existing bone is resorbed internally and replaced by areas of irregular new bone that result in an expansion and thickening of the diploic table (Aufderheide and Rodríguez-Martín 1998; Zimmerman and Kelley 1982). Individual trabeculae exhibit marked bony sclerosis, and the outer surface of the skull is much smoother (Ortner and Putschar 1984:294). The face maybe grossly deformed with a leonine appearance. Leontaisis ossea may be difficult to distinguish from Paget's without the post-cranial material or access to other diagnostic methods (microscopy and radiography).

Fluorosis

Typically caused by the prolonged and excessive ingestion or inhalation of fluorine, fluorosis results in a metabolic disruption of osseous activity (Littleton 1999:465). Fluoride directly stimulates osteoblastic bone formation that may form as thick sheets of new periosteal bone or bony excrescences. Unchecked osteoblastic activity leads to generalized osteosclerosis of the skeleton. Changes to the skull are very rare (Resnick and Niwayama 1988:3072). Trabecular thickening eventually leading to the obliteration of the diploë and thick bony excescresences have been observed (Aufderheide and Roderiguez-Martin 1998:317; Littleton 1999:466). Teeth become chalky white and commonly exhibit a yellow and brown mottling. Fluoride can also cause problems in dentine formation resulting in teeth of abnormal size and shape (Aufderheide and Rodríguez-Martín 1998:317; Zimmerman and Kelley 1982:69).

Paget's Disease (Osteitis deformans)

The specific etiology of Paget's disease is unknown. There are a number of possibilities including, viral infection, hereditary disorder, hormonal dysfunction and neoplasia (Revell 1986:163; Olmsted 1981:706). Consequently, it is often classified as a 'miscellaneous' disorder by palaeopathologists (Aufderheide and Rodríguez-Martín 1998; Ortner and Putschar 1984). Excessive remodeling and an imbalance in osseous activity characterize the condition (Wood 1994:263). For this reason, many clinicians and palaeopathologists classify it as metabolic disease (Resnick and Niwayama 1988; Roberts and Manchester 1995; Zimmerman and Kelley 1982).

Paget's disease is a very common disorder predilecting older individuals. Clinical studies reveal a postmortem prevalence around 3% in those over 40 years (Revell 1986:145). It is slightly more common in males than females (Barnes and Peel 1990:132; Resnick and Niwayama 1988: 2128; Striland 1991b: 173). While both osteoblastic and osteoclastic activity are involved, over time, abnormal bone formation becomes the predominant feature. In the initial stages of the disease, excessive bone resorption to both the periosteal and endosteal surfaces leads to well marked areas of abnormal bone loss, known as osteoporosis circumscripta (Lodge 1967:407; Rothschild and Martin 1993). This may present as single or multiple asymmetrical patches of cortical thinning and trabecular resorption (Mirra 1987: 162; Ortner and Putschar 1984: 309). The patches are sharply marked in comparison to normal, non-pagetic bone (Wood 1994:266). In the following stage, the excessive osteoclastic resorption stimulates localized proliferation on the endocranial and ectocranial surface of new, fibrous bone (Wood 1994; Striland 1991b). The new bone is poorly-organized, abnormal in structure and exhibits a sponge-like appearance. The surface of the skull, covered with rough, patchy Pagetic bone has also been described as having a "cotton-wool" or "pumice" appearance (Aufderheide and Rodríguez-Martín 1998:415; Barnes and Peel 1990:132; Olmsted 1981:707). Mirra (1987:164) describes the process as a "waves of intense osteoclastic resorption... followed by waves of osteoblastic hyperplasia resulting in the formation of irregular bone". In the late phase, the deposition of excessive new woven/fibrous bone may lead to a noticeably enlarged cranium, obfuscation of the sinuses, invagination of the skull base and possible vault deformation (Revell 1986:149 Ortner and Putschar 1984:311). Excessive bone formation may result in an enlarged vault may appear to "spread over the

ears and back of neck" (Wood 1994:264). The cross-section reveals alternating sclerosis and porous tables with the occasional nodular mass. The meningeal vessels on the endosteal surface become prominent due to increased blood flow (Bell and Jones 1991; Roberts and Manchester 1995; Striland 1991b; Zimmerman and Kelley 1982). Cranial osteosarcomas are frequently associated with older Pagetic adults (Aufderheide and Rodríguez-Martín 1998; Steinbock 1976; Wood 1994).

The macroscopic appearance of late-stage Paget's disease from the cranium is reasonably easy to diagnose although leontiasis ossea and hyperostosis frontalis interna should be considered. Care must be taken to differentiate the disease in its osteoclastic or mixed stage form fibrous dysplasia, osteoblastic metastatic carcinoma, hyperparathyroidism, rickets/osteomalacia, the anemias and tertiary syphilis. The ground glass radiographic appearance and the "mosaic-patterned" microstructure are considered pathognomonic of Paget's (Bell and Jones 1991; Olmsted 1981; Resnick and Niwayama 1988).

2.4.2. Endocrine Disorders

Endocrine disorders result from the dysfunction of the ductless glands (pituitary and thyroid are relevant to palaeopathology) leading to a qualitative change in hormone secretion and therefore disrupting the cellular dynamic of bone. For this reason, endocrine disorders are often classified as or with metabolic diseases (Roberts and Manchester, 1995; Wells, 1967). Endocrine disorders are very rare in palaeopathological contexts (Roberts and Manchester, 1995:180).

2.4.2.1. Abnormal Bone Loss

Hypopituitarism (Pituitary Dwarfism)

Hypopituitarism, similar to hyperpituitarism, is also caused by a dysfunctional pituitary due to tumorus interference. Failure of the pituitary during sub-adult growth causes pituitary dwarfism, resulting in a small, gracile but proportionate skeleton (Zimmerman and Kelley 1982:65). The cranial sutures remain patent into adulthood and the eruption of the adult dentition may be delayed. In contrast to hypothyroidism, the mandible is small and underdeveloped in comparison to the maxilla leading to an overbite (Aufderheide and Rodríguez-Martín 1998:329). Similar to hypothyroidism, the sella turcica may exhibit resorptive damage (Aufderheide and Rodríguez-Martín 1998).

Hypothyroidism (Cretinism)

Stunted physical (and mental) growth may be the result of dysfunction or atrophy to the thyroid during fetal growth. Individuals with this disorder are of small stature and disproportionate size. The skull may exhibit thickened cranial bones (Ortner and Putschar 1984; Zimmerman and Kelley 1982).

Hypopituitarism and hypothyroidism may be difficult to differentiate without reference to the extremities, however the comparatively gracile nature of hypopituitarism contrasts with the sclerotic bones of hypothyroidism. If observable, the sella turcica may exhibit damage accountable of hypopituitarism. Achondroplasia, hydrocephalus and microcephalus should also be considered in the differential diagnosis (Aufderheide and Rodríguez-Martín 1998:329; Zimmerman and Kelley 1982:65).

2.4.2.2. Abnormal Bone Gain

Hyperpituitarism (Gigantism and Acromegaly)

Malfunction of the pituitary gland due to tumorus invasion may lead to gigantism in subadults and acromegaly in adults (Zimmerman and Kelley 1982:63). Gigantism is a very rare condition characterized by excessive skeletal growth leading to statures above seven feet. The appendicular skeleton and mandible may become disproportionately large. While acromegaly, a much more common condition may exhibit limited stature increase, the most pronounced feature is the enlarged, prognathic nature of the mandible. Bony buildup along the alveolar margins of the mandible and maxilla may eventually lead to separation of the teeth. Periosteal bone deposition on the face, nasal bones and cranial vault results in their prominence. Occasionally the small tumors of the pituitary may enlarge the sella turcica (Aufderheide and Rodríguez-Martín 1998:327; Ortner and Putschar 1984:300; Zimmerman and Kelley 1982:63). Gigantism is usually apparent given the enlarged size of the bones. Acromegaly should be distinguished from other hyperostotic conditions such as hyperostosis frontalis interna, Paget's disease and the anemias.

2.4.2.3. Mixed Reaction

Hyperparathyroidism

Hyperparathyroidism is precipitated by primary or secondary excess of parathyroid hormone (PTH) (Cook et al. 1988). The primary hyperparathyroidism predilects females of middle age, while the secondary form of the disease affect both children and adults (Zimmerman and Kelley 1982:63). In clinical studies only 5% of those individuals with the disorder actually exhibit skeletal involvement (Resnick and Niwayama 1988; Tam 1989). Excessive PTH lowers calcium levels, which stimulates diffuse osteoclastic resorption of the trabeculae resulting in abnormal bone loss, characterized by generalized osteopenia and a speckled "salt and pepper" appearance (Olmsted 1981:706; Rothschild and Martin 1993:145; Zimmerman and Kelley 1982:63). Osteoblastic activity results in the deposition of abnormal fibrous bone. Osteoclastic activity continues, outpacing bone formation leading to the formation of osteolytic cysts and brown tumors (Revell 1986; Cook et al. 1988). Brown cysts form in the cancellous bone of the mandible, face and skull expanding to thin the overlying cortex. According to Revell (1986:118), the tumor can have a variegated yellow or brow surface. This process is also known as *osteitis fibrosa cystica* (Aufderheide and Rodríguez-Martín 1998:331). Profound demineralization of bone can result in deformities, although these are typically restricted to the weight bearing bones (Aufderheide and Rodríguez-Martín 1998:331). Differential diagnosis should include osteomalacia, early Paget's disease, fibrous dysplasia, and hyperostosis frontalis interna. The cysts and brown tumors may require differentiation from metastatic cancer, multiple myeloma and tuberculosis. (Ortner and Putschar 1984: 308)

2.4.3. Hematological Disorders

Blood is manufactured in the marrow cavities of bone consequently disorders in the manufacture and proliferation of blood may result in abnormalities to the skeleton (Aufderheide and Rodríguez-Martín 1998:345). These disorders are caused by a variety of etiological factors including hereditary and developmental abnormalities, dietary

insufficiencies, malabsorption, infection and generalized stress. Given the multifactoral nature of most blood disorders, skeletal changes usually appear to be of a non-specific nature (Zimmerman and Kelley 1982).

2.4.3.1. Abnormal Bone Loss

Histiocytosis X (Langerhans cell granulomatosis)

Histiocytosis X includes three childhood conditions of unknown etiology, caused by the proliferation of histiocytes (Olmsted 1981: 708; Ortner and Putschar 1984: 250). Each has a different clinical manifestation and age-specific predilection thereby separating them into three separate entities, namely: Eosinophilic granuloma, Hand-Schüller-Christian disease and Letterer-Siwe disease (Aufderheide and Rodríguez-Martín 1998:354; Ortner and Putschar 1984:249; Resnick and Niwayama 1988:2429). In all three conditions, the skull is the most frequently involved. Lesions are characterized by a predominantly osteoclastic activity that typically perforates the cranium and exhibits little or no osteoblastic reaction in the early stages. Later stages may exhibit evidence of bony sclerosis indicative of healing.

Eosinophilic granuloma is the most common of the three disorders predilecting children and young adults. While multiple bone lesions can occur, eosinophilic granuloma is characterized by a "solitary, purely lytic round or oval defect" (Ortner and Putschar 1984:250; Resnick and Niwayama 1988:2431) with irregular, undulating borders. Multiple lytic lesions can coalesce into large scalloped or geographically margined holes with beveled borders (Barnes and Ortner 1997: 545; Olmsted 1981:709). Osteoclastic activity may stimulate a periosteal reaction and a "button" sequestrum may occasionally be observed (Aufderheide and Rodríguez-Martín 1998:354; Ortner and Putschar 1984:350). The lytic lesions of the other two conditions are similar in morphological appearance. Letterer-Siwe is an acute disease that affects infants below the age of two and is usually fatal (Barnes and Ortner 1997). Multiple lesions affect the vault, sphenoid and occasionally the face (Barnes and Ortner 1997). Schüller-Christian's disease is a chronic disorder that predilects immature individuals and young adults, most commonly males (Aufderheide and Rodríguez-Martín 1998:354). This condition is characterized by scattered multiple lytic lesions involving the calvarium, base and sphenoid. Since the lesions occur over a period of years, they will exhibit varying degrees of healing. Otitis media, periodontal inflammation and tooth disruption are occasionally associated with all three (Aufderheide and Rodríguez-Martín 1998; Mann et al. 1994).

Differential diagnosis of solitary lesions should consider localized infections, neoplasia, and fibrous dysplasia. Multiple lesions should be distinguished from multiple myeloma, osteolytic metastatic carcinoma, hyperparathyroidism, leukemia and Gaucher's disease (Aufderheide and Rodríguez-Martín 1998).

Leukemia

Leukemia is a malignant disease of the bone marrow, that is usually acute in children and either chronic or acute in adults (Aufderheide and Rodríguez-Martín 1998:355). According to Rothschild and colleagues, (1997:481) the disease is more common in children and more likely to exhibit osseous changes than in adults. The acute form exhibits a greater severity of osseous reaction in comparison to the chronic form. While skeletal changes predilect the marrow producing areas of the skeleton,

occasionally the cranial vault may exhibit single or multiple osteolytic lesions. Periosteal deposition that may result in marked pitting and a rough, porous surface in association with the osteolytic lesions (Ortner and Putschar 1984:264; Resnick and Niwayama1988: 2462; Rothschild et. al. 1997:482). Diffuse osteopenia resulting from metabolic disruption and marrow irregularities is the most common observation, however it is not likely to be observable without a radiograph (Aufderheide and Rodríguez-Martín 1998: 355; Resnick and Niwayama 1988; Rothschild et. al. 1997:484). According to Resnick and Niwayama, (1988:2464), the differential diagnosis should consider: infectious inflammation, neoplasias, anemias and syphilis.

Gaucher's Disease

Gaucher's Disease is a lipid storage disorder primarily of the post-cranium. The disease is fatal in children, however, in adult's life expectancy is normal (Aufderheide and Rodríguez-Martín 1998:370). While the skeletal changes are primarily due to a mixed osteoclastic and osteoblastic activity, the cranium primarily exhibits trabecular destruction and bilateral thinning of the vault tables (Rothschild and Martin 1993). Destruction of the marrow cavities, bony necrosis, osteomyelitis, pathological fractures and osteoarthritis are several disorders/features are commonly associated with this disease.

2.4.3.2. Mixed Reactions

Anemia

Anemia is a hematological disorder that results in the excessive destruction and the underproduction of the red blood cells. Clinically there are two types of anemias-congenital and acquired. Congenital anemias such as thalasemmia and sickle-cell anemia are hereditary disorders not related to external influences on the bioavailability of specific nutrients. In contrast, iron-deficiency anemia results from the inadequate availability of iron due to dietary insufficiency, impaired absorption, parasitic infection and other external influences. When iron is not present in sufficient amounts, haemoglobin formation is impaired, stimulating accelerated production of bone marrow (Garrow and James 1993: 181-182; Resnick and Niwayama 1988). In cases where the deficiency is long-standing, the activity of hyperplastic marrow will result in significant changes to the skeletal system.

While iron-deficiency anemia can be acquired by any individual with inadequate levels of bioavailable iron, menstruating and pregnant females, children and infants between the ages of nine months and three years are the most susceptible to iron deficiencies (Garn 1990; Kent 1990; Resnick and Niwayama 1988). This supports Stuart-Macadam's, (1985) findings, which suggest that skeletal manifestations of anemia observed in adults, actually represent a childhood affliction. Congenital anemias are usually incipient at birth.

Anemias exhibit mixed osteoclastic and osteoblastic activity. Increased resorption of the cortical and cancellous bone occurs simultaneously with increased deposition along the individual trabeculae in order to provide a rearranged diploic space that can incorporate the body's demand for more marrow. While there is some debate regarding the relationship of cranial changes to anemia, clinical and palaeopathological studies support the observation that porotic hyperostosis and cribra orbitalia are related to

marrow hyperplasia (Lovell 1997b; Stuart-Macadam 1987). Porotic hyperostosis is identified by the marked reduction of the individual trabeculae associated with subsequent thickening (hypertrophy). The cortical surface becomes thinned and demonstrates osteolytic lesions that vary in size from fine, pumic-like foramina to larger pores in excess of two millimeters (Aufderheide and Rodríguez-Martín 1998:349; Ortner and Putschar 1984:43). These larger pores become linked and reveal interconnecting trabeculae that may project above the contour of the outer table. Porotic hyperostosis generally affects the cranial vault bilaterally and symmetrically, predilecting the parietals, occipital, and frontal, particularly adjacent to the sutures; there is usually little involvement of the face and mandible (Resnick and Niwayama 1988; Stuart-Macadam 1987). Cribra orbitalia forms in a similar manner to porotic hyperostosis (Stuart-Macadam 1989b). Trabecular involution and hypertrophy stimulate the formation of thickened trabeculae. As the cortical bone is eroded, the fine foramina begin to coalesce revealing a labyrinthine like pattern on the orbital roof. The lesions are usually, but not always distributed bilaterally on the superior margins in a sickle-like shape (Hengen 1971:60). According to Stuart-Macadam (1989b), vault lesions occur predominantly in concert with orbital lesions, while orbital lesions have been noted to occur with and without vault lesions. Radiographically, diploic thickening of the orbits and vault as well as the "hair-on-end" trabeculation pattern are diagnostic of anemias in general. Reduced or non-existent sinuses, retention of the metopic suture and an increased frequency of caries and enamel defects are also associated with porotic hyperostosis/cribra orbitalia (Caffey 1978; Stuart-Macadam 1985, 1987).

Ectocranial porosis is one of the most frequently observed lesions on the cranial vault. It is characterized by the pitting of the vault surface without subsequent thickening of the cortex. While many researchers associate it with iron-deficiency anemia, its specific etiology is unknown. Many now feel that before ectocranial porosis can be attributed to anemia, thickening of the cortex must be observed (thereby identifying it as porotic hyperostosis) (Williams 1994). Ectocranial porosis may be indicative of generalized stress, rather than a specific disorder.

Iron-deficiency anemia

Iron-deficiency anemia attracts a lot of attention in the palaeopathological literature (Carlson et al. 1974; El-Najjar 1977; Lovell 1997b; Palkovich 1987; Stuart-Macadam 1989a). While it is considered to be the most wide spread type of anemia, one of the most common disorders of humankind, and one of the most universally observed skeletal abnormalities in antiquity (Stuart-Macadam 1989b: 212), there is considerable debate regarding the specific etiology of iron-deficiency anemia. Recent research points to a number of factors, however, iron-deficiency anemia is likely a symptom of generalized stress resulting from one or more etiological factors acting synergistically (Lallo et al. 1977; Lovell 1997b; Mensforth et al., 1978; Walker 1986). The above description of cranial changes associated with anemia is relevant to iron-deficiency anemia.

Congential Anemias: Thalasemmia and Sickle-Cell Anemia

Beta-Thalassemia (thalassemia) is a congenital anemia[1] incipient at or near birth, and common in Eastern Mediterranean populations. According to Resnick and Niwayama (1988), skeletal manifestations of thalassemia are more severe in comparison iron-deficiency anemia. Thalassemia is characterized by the destruction of the paranasal sinuses, lateral displacement of the orbits, and osseous expansion of the diploë into the maxilla, mandible and zygomatics resulting in facial deformity, disorderly eruption of the teeth and associated malocclusion (Ortner and Putschar 1984:253; Zimmerman and Kelley:). Sickle-cell anemia is common in African derived and circum-Mediterranean populations. In sickle-cell anemia, it is common to find only mild manifestations (in comparison to thalassemia) of the characteristic hair-on-end pattern (Resnick and Niwayama 1988:2324). As well, there is little or no involvement of the frontal bone, which may serve to differentiate the disorder from other anemias. Congenital anemias differ from iron-deficiency anemia in that they manifest over the course of the individual's life while iron-deficiency anemia is predominantly expressed in younger individuals.

While differentiation between thalasemmia, sickle-cell anemia, and iron-deficiency anemia is difficult on the clinical level, it is even more difficult at the palaeopathological level. Marrow hyperplasia is responsible for the skeletal manifestations of all anemia and consequently it can be virtually impossible to differentiate between the three (Ortner and Putschar 1984:258); however, slight distinctions can be made. For example, lesions of thalassemia and sickle-cell anemia are usually more severe than those of iron-deficiency anemia, exhibiting a honeycombed and "hair-on-end" appearance (Aufderheide and Rodríguez-Martín 1998: 347; Resnick and Niwayama 1988: 2351). Congenital anemia, particularly thalassemia, exhibit facial porosity, while there is little or no involvement of the face and mandible in iron-deficiency anemia. Finally, it is suggested that palaeopathological examples of porotic hyperostosis/cribra orbitalia represent iron-deficiency anemia rather than congenital anemia because the present-day distribution of congenital anemia suggests that they were not prevalent in the past (Stuart-Macadam 1989a). Conditions that produce hyperostotic changes including fibrous dysplasia, leonstatis ossea, hyperostosis frontalis interna, acromegaly, Paget's disease, hyperparathyroidism and osteolytic conditions such as osteoporosis, scurvy, rickets and periostitis should be considered if the cranial lesions are ambiguous (Ortner and Putschar 1984; Resnick and Niwayama 1988).

2.5 Infection

Changes to bone as the result of an infectious agent are among the most commonly observed lesions in the archaeological record (Kelley 1989; Roberts and Manchester 1995). Infection was likely the most common cause of morbidity and mortality in antiquity and probably figured strongly in its biological, cultural, sociological and psychological impact (Ortner and Putschar 1984). Infection provides the anthropologist an important window into the synergistic and multifaceted relationship of disease to a variety of biological, cultural, environmental, geographical and immunological factors (Kelley 1989). Despite the importance of infectious diseases to

[1] There are 56 recognized anemic disorders under the umbrella of Thalassemia. Their expressions range from very mild too severe (Russell Personal Communication, 2002).

our understanding of prehistory, anthropologists are severely limited with respect to identifying and interpreting most conditions. While numerous infectious agents and their attenuating clinical manifestations affect human health and vitality, only a small number of these produce skeletal lesions. Infections in prehistory were likely acute and primarily affected the soft tissues, resolving either through death or healing long before leaving evidence on bone. Only conditions that were relatively long-standing and chronic will manifest an inflammatory lesion on bone. According to (Roberts and Manchester 1995:125) bacteria were the most likely agents of bony infection in prehistory because viral infections (e.g. small pox) usually resolve before bony lesions can occur. Clinical studies show that the bacteria most commonly involved in bone infection is staphylococci, streptococci and pneumococci - these likely reflect the common agents of prehistoric infection as well (Roberts and Manchester 1995:126). Because bone changes in a limited number of ways, the majority of infectious reactions cannot be ascribed to a specific agent or event. Most osseous infections identified in the archaeological record are considered non-specific inflammations. Only a few infections result in lesions or patterns of lesions that are pathognmonic of the clinical manifestations of the infectious agent. These include tuberculosis, leprosy and treponemal disease (Rogers and Waldron 1989). Occasionally a specific etiology can be ascribed to lesions resulting from fungal or parasitic infections; however, these are the exception and not the rule (Zimmerman and Kelley 1982).

Inflammation refers to the non-specific changes associated with bony reaction to trauma, neoplasia, osteoarthritis, generalized stress, metabolic disruptions and infection (Gregg and Gregg 1987:51). Changes to bone are generally a combination of abnormal bone loss and abnormal bone formation (Roberts and Manchester 1995: 125-126) with one or the other usually predominating. When soft tissue or bone is infected, a cellular reaction is precipitated against the invading organism (virus, bacterium, parasite or fungus), that if chronic and ongoing will demonstrate osseous response. The following discussion considers non-specific infections, otitis media, mastoiditis and sinusitis, the specific infections of leprosy, tuberculosis, treponemal disease as well as mycotic and parasitic infections. Descriptions of typical skull lesions will be provided with particular attention to the type of osseous reaction (osteoblastic/ osteoclastic/mixed) involved.

2.5.1. Non-Specific Infections

Osteomyelitis

Most changes to bone via a variety of agents are non-specific in nature. Changes stemming from one infectious agent are virtually indistinguishable from another (Roberts and Manchester 1995:126). Osteomyelitis, the most common expression of non-specific bone inflammation, may derive from fungal, viral or parasitic agents, however, staphylococcus bacteria is thought to account for upwards of 90% of clinical cases (Aufderheide and Rodríguez-Martín 1998: 172; Ortner and Putschar 1984:106; Resnick and Niwayama, 1988: 2526; Rothschild and Martin 1993: 63). *Osteomyelitis* is commonly defined as an inflammation of the marrow cavity (Revell 1986:235). The term *periostitis* refers to the superficial process of inflammation, characterized by the apposition of a plaque of rough, striated and porotic new bone on the periosteal surface. The term *osteitis* refers to the inflammatory involvement of the cortex that leads to fine porosity, striations and a plaque-like formation that result in notably thickened bone

(Canci et al. 1994; Resnick and Niwayama 1988). Clinical, microscopic and radiographic analyses suggest that inflammation is not as confined to each layer of bone as to require strict definitions (Resnick and Niwayama 1988). Although it may be difficult to determine the involvement of the marrow cavity on the basis of macroscopic evaluation, current practice encourages the use of osteitis and periostitis as descriptive terms within the clinical manifestation of osteomyelitis (Aufderheide and Rodríguez-Martín 1998: 172; Ortner and Putschar 1984:107; Roberts and Manchester 1995:126; Steinbock 1976). In other words, osteomyelitis is the term given to non-specific osseous inflammation with or without direct evidence of marrow cavity involvement. Care should be exercised when using osteitis and periostitis, because as terms of description, they also describe non-infective bony reactions (e.g. psoriasis, neoplasia) (Aufderheide and Rodríguez-Martín 1998; Resnick and Niwayama 1988).

Osteomyelitis can be divided into pyogenic (pus-forming) and nonpyogenic (non pus-forming) forms and clinically manifest at the acute, subacute and chronic level (Rothschild and Martin 1993:63). Clinicians and palaeopathologists recognize several routes of primary and secondary bony contamination: 1) primary infection from direct introduction (via trauma or iatrogenic intervention); 2) primary spread from a contiguous source (skin, sinus); and 3) secondary hematogenous spread from the primary source (Resnick and Niwayama 1988; Rothschild and Martin 1993).

Primary infection as the result of direct infection can occur at any age, however, it is most common in older adults in whom fractures, chronic soft tissue infections, diabetes mellitus, malignant disease and periodontal disease have a higher incidence (Revell 1986:236; Steinbock 1976: 73). Primary osteomyelitis as a result of direct infection also predilects males possibly reflecting a higher prevalence of trauma in males in comparison to females. The shafts of the long bones are the most commonly affected areas, however, the mandible and cranial vault are also frequently affected. In rare cases, an overlying chronic and primary skin or sinus infection can spread to the underlying bone resulting in a superficial inflammation of the bony surface (Resnick and Niwayama 1988; Roberts and Manchester 1995). Acute secondary infection results from the hematogenous dissemination of the infectious agent from a primary inflammatory site in the bone or soft tissue (Steinbock 1976). Secondary hematogenous infections are more common than primary infections of bone, predilecting immature individuals between the ages of 3 and 15 in whom growth is most active (Aufderheide and Rodríguez-Martín 1998; Ortner and Putschar 1984; Rogers and Waldron 1989). In immature individuals, acute secondary infections usually occur on a single bone with the metaphysis of the long bone the most common site infection. In adults, hematogenous spread of infection usually involves multiple secondary sites (Ortner and Putschar 1984; Roberts and Manchester 1995). In the skull, the most commonly affected bone is the mandible (Steinbock 1976:63).

Osteomyelitis presents as a process of simultaneous bone destruction and bone repair. In hematogenous infections, purulent organisms invade the bone via the vascular channels and trabeculae forming pus and increasing the intraosseous pressure (Rothschild and Martin 1993). Within two weeks of the infection's onset, abnormal bone loss is radiographically observable as a reduction in density occurs (Steinbock 1976). Bone destruction occurs first with increasing porosity leading to coalescing pits and cavities (Aufderheide and Rodríguez-Martín, 1998:175; Steinbock 1976:68) spreading through the trabeculae (marrow cavity) to the cortex. Eventually the pus breaks through, spilling

onto the periosteal surface, cutting off the underlying bone and effectively killing it, while stimulating the apposition of new bone along the trabeculae and cortex (Revell 1986; Rogers and Waldron 1989; Zimmerman and Kelley 1982). A hypervascular plaque of thick, short, bony projections comprised of disorganized woven bone characterizes the appearance of the periosteal reaction (periostitis) (Aufderheide and Rodríguez-Martín 1998). Below, the old bone cut off from its nutrients, dies forming the sequestrum. The sequestrum is dead bone often separated from living bone by a line of demarcation or erosion (Hackett 1976). In severe cases continued apposition of new bone may produce an involucrum - a bony shell that surrounds sequestrated bone. Often a fistulae or abscess, known as the cloaca, will perforate the involucrum to drain the pus from the dead bone. Colaca are often smooth sided and deep, extending from the source of infection (medullary cavity) (Resnick and Niwayama 1988: 2526; Roberts and Manchester 1995:127; Rogers and Waldron 1989:612). Direct infection via trauma, surgery or skin condition presents skeletal changes that are similar in process to hematogenous infection, however, they often remain more localized and may or may not involve the marrow cavity/table (Zimmerman and Kelley 1982:94). Continued remodeling of the infected area will produce bony sclerosis with an irregular, bulky surface. The involucrum and surrounding tissue is structurally unsound and pathological fracture in biomechanically active bones is not an uncommon occurrence (Resnick and Niwayama 1988).

Chronic osteomyelitis often develops from acute cases. The failure of the wound to heal may lead to the presence of a low-grade infection that can flare up over a series of months and years. Over time, old sinuses and cloacae are occluded with new bone and tissue stimulating the formation of new cloacae and continued apposition of reparative bone (Steinbock 1976, Zimmerman and Kelley 1982). The bone becomes enlarged presenting an irregular surface riddled with holes. Chronic non-suppurative osteomyelitis (sclerosing osteomyelitis of Garré) may result from a chronic low-grade infection. This rare, non-pus forming, osteoblastic condition most often affects the mandible and occasionally the vault (Resnick and Niwayama 1988:2526). Bone is characterized by dense "fusiform thickening of the cortex" (Aufderheide and Rodríguez-Martín 1998:178) and occlusion of the marrow cavity with no evidence of cloaca.

Osteomyelitis of the skull is rare (Hackett 1976:72; Ortner and Putschar 1984:117; Steinbock 1976:81). The most common method of cranial contamination is via a traumatic opening, blunt force trauma, surgical intervention or scalping. The infection spreads from the scalp through the open wound into the cranium or mandible. According to Ortner and Putschar, (1984:119) these infections usually remain localized, with the lesion more extensive on the outer table than the inner table. Given a prolonged course, the infection will remain localized, circumscribed by an area of "perifocal sclerosis" (1984:119) associated with the central lytic focus and possible sequestrum. Compound fractures may lead to the hematogenous dissemination of the infection through the diploic spaces. Chronic infections of the soft tissue can also spread to the skull and mandible. Infections that spread from a contiguous source tend to have a more erosive effect upon the outer surface of the skull and work inwards towards the diploic and inner tables of the bone. This is generally opposite to the inward-outward effect of infections resulting from hematogenous spread or compound fracture (Resnick and Niwayama 1988:2542). Infections of the frontal sinus may spread through the diploic spaces, venous sinuses and

meningeal vessels throughout the face and cranial vault but do not typically involve the occipital (Ortner and Putschar 1984: 120; Resnick and Niwayama 1988:2542). Middle ear infections can spread to the mastoid, petrous portion and the rest of the temporal (Ortner and Putschar, 1984:120). According to Wood-Jones (1910:283), dental disease accounts for most of the infections observed in the skull and mandible (Ortner and Putschar 1984:123). The mandible and maxilla of children is prone to infection resulting crowded dentition. Orofacial infections commonly manifesting as a periapical abscess resulting from caries or periodontal disease remains common into adulthood (Ortner and Putschar 1984; Resnick and Niwayama 1988).

Bony infection or osteomyelitis, whether early or well healed, can be difficult to recognize. Depending upon the constellation of features, the predominant type of osseous response, the avenue of contamination, the clinical manifestation of severity, the degree of healing and location of the infection a variety of conditions may require differentiation from osteomyelitis of the skull. According to Aufderheide and Rodríguez-Martín (1998:179), Rothschild and Martin (1993:69), and Steinbock (1976:80) these include: osteoarthritis (temporomandibular joint); osteosarcoma; tertiary syphilis; tuberculosis; bone cysts; osteoma; osetoblastoma; and eosinophilic granuloma.

Otitis Media, Mastoiditis and Sinusitis

Otitis media is an infection of the middle ear, most common in immature individuals under one year of age (Mann et al. 1994). Clinical and palaeopathological studies suggest that the disease predilects Native Americans, Inuit and Australian Aborigines (Gregg and Gregg 1987; Mann et al. 1994). Infection is usually via a bacterial contagion (pneumococcus or influenza) of the upper respiratory tract or allergic reaction that causes mucosal secretions to congest the auditory isthmus leading to an inflammatory response in the temporal bone. Erosive changes can be found on the ectocranial and endocranial surfaces, as well as on the internal structure of the temporal. A large erosive space on the endocranial surface of the tengem tympani may be the result of a cholesteaoma, a potentially lethal complication (Gregg et al. 1981:291; Mann 1991:165; Schultz 1979:576) of middle ear disease. Otitis media may be difficult to observe based on surface morphological changes. An otoscope permits evaluation of the tympanic membrane for possible perforation and an endoscope will reveal evidence of erosive destruction of the ear ossicles (Aufderheide and Rodríguez-Martín 1998:253). If ear infections occur during mastoid growth[2] they can result in altered pneumatization and bony sclerosis that is observable via radiographic analysis (Gregg et al. 1981; Gregg and Gregg, 1987; Mann et al. 1994).

Mastoiditis is an uncommon infection, however when it is observed, it is most frequently related to primary otitis media. The radiographic appearance of sclerotic bone and the formation of pockets in the temporal bone and mastoid process are suggestive of the process. According to Mann and Murphy, (1990:27), resorption and possible perforation of the mastoid, the area surrounding the external auditory meatus and other portions of the temporal bone may be observed. Changes are essentially osteomyelitic, often leading to the formation of a fistula/cloaca on the mastoid or squama for the draining of puss (Daniel et al. 1988; Mann et al. 1994; Slater 1994). The fistula may open

[2] Development of the air cell and growth of the mastoid begins before birth and is usually completed between the ages of 4-5. Continued pneumatization occurs into the teens (Gregg and Gregg, 1987:72).

either to the exterior or interior surface of the bone. If the pus drains exteriorly then it will usually exhibit signs of healing or chronic involvement; if the fistula opens to the interior surface, and therefore the brain, it is usually fatal (Roberts and Manchester 1995:132). In extreme cases, the entire mastoid can be completely destroyed (Mann et al. 1994; McKenzie and Brothwell 1967). Care must be taken to distinguish the affects of otitis media and mastoiditis from the erosive effects of postmortem damage to the thin cortical bone overlying the mastoid and petrous portion (Daniel et al. 1988). Further, fissures and small pores on the surface of the mastoid are considered normal variation and should not be mistaken as abnormal bone loss (Mann and Murphy 1990). According to Gregg et al., (1981), radiographic analysis is the most effective means of identifying these disorders.

Chronic sinusitis is likely caused by the hematogenous spread of a primary infection of the ears, throat or respiratory-tract to the sinuses (Roberts and Manchester, 1995:131). Dental abscess and chronic allergies are possible biological causes, while environmental factors such as pollution and poor ventilation may also be responsible (Aufderheide and Rodríguez-Martín 1998: 257; Resnick & Niwayama 1988: 2542; Roberts and Manchester 1995:131). According to Roberts and Manchester (1995:131), sinusitis presents porosity and new bone formation on the interior surface of the sinuses. Infection of the sinus spaces may also spread to the middle ear and the mastoid (Wells 1964). Sinusitis is frequently missed in general palaeopathological surveys. Not only is the fragile area frequently damaged in archaeological contexts, its location in the interior of the skull makes it easy to overlook, especially if the sinus spaces are filled with matrix. (Roberts and Manchester 1995; Wells 1964:80). Care must be taken to recognize and differentiate postmortem breakage. Systematic radiographic analysis is usually the best method of identification.

2.5.2. Specific Infections

Certain infectious diseases produce skeletal lesions that are both diagnostic and nonspecific in nature (Powell 1991). Careful attention to the descriptive process, pattern of abnormal changes and differential diagnosis is essential to their differentiation and identification. The following section provides a brief description of the major morphological expressions and differential diagnosis of three specific infections: tuberculosis, leprosy and treponemal disease.

Tuberculosis

Tuberculosis is a chronic granulomatous infection caused by the bacillus *Mycobacterium tuberculosis* that exists in two forms of significance to palaeopathology (*M. tuberculosis bovis and M. tuberculosis humanis*) (Powell 1991; Rogers and Waldron 1989). Most researchers believe that the bovis form of tuberculosis spread from cattle to humans through the domestication process (Manchester 1984; Roberts and Manchester 1995). The skeleton is affected in only 5-7% of individuals with active tuberculosis (Steinbock 1976:175; Zimmerman and Kelley 1982:103), and clinical studies suggest that it can affect individuals of any age with no significant predilection to either sex (Resnick and Niwayama 1988:2663).

Abnormal changes to the skeleton are derived primarily via hematogenous spread from pulmonary or gastrointestinal foci (Ortner and Putschar 1984:141) to those areas of the skeleton with large amounts of red marrow. Bony changes are primarily destructive

with multiple, sharply defined, lytic lesions and little or no evidence reparative bone or remodeling (Hershkovitz et al. 1998; Pfeiffer, 1984; Roberts and Manchester 1995; Rothschild and Martin 1993; Steinbock 1976; Zimmerman and Kelley 1982:104). Continued osteolytic activity in the area surrounding the lesion may begin to resemble generalized osteoporosis (Ortner and Putschar 1984:144).

Cranial involvement is rare in tuberculosis except in children under 10 in whom there is an abundance of haemopoietic marrow. While children exhibit multiple, small cranial lesions, infected adults' present larger, solitary lesions. The frontal and the parietals are the most commonly affected region of the cranial vault. Active osteoclastic activity with little or no osteoblastic activity leaves resorptive erosions with round irregular margins no more than 2cm in diameter (Aufderheide and Rodríguez-Martín 1998:140; Ortner and Putschar 1984:162). Complete or partial perforation of the inner and outer tables is common (Murray 1990; Ortner and Putschar 1984:162) with changes to the inner surface more marked and larger than the outer surface. Hackett, (1976:73) observes that cranial lesions of tuberculosis exhibit superficial pitting and cavitation beginning on the inner surface and extending outward. A small sequestrum on the outer surface and extensive superficial erosion on the inner surface should be considered diagnostic of tuberculosis (Hackett 1975:235). Osteoclastic destruction of the petrous portion and mastoid process as well as otitis media are occasional observations in immature individuals (Ortner and Putschar 1984:163; Rogers and Waldron 1989: 614). On rare occasions, the cranial base may reflect the destructive lesions of spinal tuberculosis located on C1 and C2 (sub-occipital tuberculosis) (Aufderheide and Rodríguez-Martín 1998:140; Ortner and Putschar 1984:164). Superficial erosive lesions, with little or no proliferative bone, and the occasional abscess along the zygomatic arch characterize cranio-facial involvement (Ortner and Putschar 1984:164). Secondary mucosal infections can also lead to the destruction of the nasal bones similar to leprosy, however this is very rare (Manchester 1994; Ortner and Putschar 1984). Destruction of the nasal area is usually asymmetrical and provides no evidence of repair or smooth remodeling as in leprosy (Manchester 1994:80). The mandible is occasionally involved in infants, while adults may exhibit destruction to the alveolar areas of the maxilla and mandible as extension of oral-mucosal infections (Ortner and Putschar 1984:166).

Because the changes are essentially osteomyelitic and non-specific in nature, there is considerable overlap with other disease lesions. It is the pattern of destructive lesions, in particular those of the vertebrae, that provides the diagnostic evidence for tuberculosis (Manchester 1984; Ortner and Putschar 1984:170; Powell 1991; Roberts and Manchester 1995; Steinbock 1976). Evaluation of the age-at-death, lesion morphology, location and distribution of lesions (Buikstra 1976) is essential to the diagnostic process, however, the non-specific nature of most lesions make it extremely difficult to distinguish skeletal tuberculosis from other etiologies. Differential diagnosis of the cranial lesions of tuberculosis should include: eosinophilic granuloma (histiocytosis-X) and metastatic neuroblastoma, osteomyelitis, bacterial infection, leprosy and tertiary syphilis (Aufderheide and Rodríguez-Martín 1998; Buikstra 1976; Hackett 1976; Pfeiffer 1984; Resnick and Niwayama 1988; Steinbock 1976).

Leprosy

Leprosy is a chronic granulomatous infection precipitated by the bacillus

Mycobacterium leprae. Clinical studies show that there are several forms of the disease that appear to reflect differing levels of immunity (Manchester, 1984:167; Resnick and Niwayama, 1988). The tuberculoid form is milder in comparison to the more severe lepromatous form that is responsible for major destructive abnormalities that characterize leprosy (Zimmerman and Kelley 1982:93). Modern studies show that *M. leprae* infectious are characterized by long incubation periods with a low frequency of skeletal involvement (Zimmerman and Kelley 1982). Leprosy appears to be slightly more common in men than women affecting any age with the average age of manifestation under 20 or over 30 (Resnick and Niwayama 1988:2688; Zimmerman and Kelley 1982: 93). The principal sites of infection are the peripheral nerves and skin (Pálfi 1991). Damage to bone results from trauma sustained to anesthetized digits, necrosis of the skin and bone, and inflammation spread hematogenously and contiguously from infected skin (Ortner and Putschar 1984; Roberts and Manchester 1995; Rogers and Waldron 1989; Steinbock 1976)[3].

Lepromatous changes to the skeleton parallel soft tissue infection, predilecting the face, hands and feet (Resnick and Niwayama 1988: 2688). Extension of the infection from the contiguous skin and mucosal areas results in highly destructive erosive lesions. Abnormal bone loss begins at the periosteum and then spreads slowly to the cortex and marrow leaving little evidence of proliferation or sclerotic new bone (Resnick and Niwayama 1988: 2688). Perhaps the most dramatic changes to the skeleton are the "socially isolating facial deformities" (Aufderheide and Rodríguez-Martín 1998:147) of facies leprosa (Möller-Christensen 1978), characterized by the destructive atrophy of the rhinomaxillary region. The anterior nasal spine exhibits pitting and progressive loss of the cortical bone leading to cancellous exposure and an eventual rounding and widening of the nasal aperture (Aufderheide and Rodríguez-Martín 1998). Smooth resorption with minor surface pitting of the alveolar process is generally confined to the incisal region leading to the eventual ATML of the anterior dentition. While the dominant osseous reaction observed in lepromatous infection is abnormal bone loss, the palate may exhibit superficial evidence of osteoblastic activity. Osteitis of the palate, coalescing pits, and plaques of new bone are occasionally observed (Resnick and Niwayama 1988: 2688; Rogers and Waldron 1989:617; Zimmerman and Kelley 1982:93). Other changes to the skull may include erosive lesions of the cranial vault (Aufderheide and Rodríguez-Martín 1998:151: Ortner and Putschar 1984). A diagnosis of facies leprosa requires the destructive atrophy of the nasal area to be accompanied by at least one of the other characteristic changes to the face (Andersen and Manchester 1992; Manchester 1994; Möller-Christensen 1976).

The pattern of destructive atrophy to the face, hands and feet is usually considered pathognomonic of leprosy. In the absence of the post-cranial material, most researchers consider facies leprosa to be diagnostic (Möller-Christensen 1978), however, care must be taken. Both syphilis and tuberculosis are capable of producing similar manifestations of rhinomaxillary destruction. Tertiary syphilis in particular has been noted to exhibit atrophy of the nasal and alveolar margins without exhibiting the osteosclerotic vault lesions that are pathognomonic of syphilis (Ortner and Putschar 1984:187). The differential diagnosis of cranial leprosy must consider pyogenic osteomyelitis, syphilis, tuberculosis, periodontal disease, frostbite, and fungal infections and postmortem destruction (Aufderheide and Rodríguez-Martín 1998; Hackett 1976; Ortner and Putschar 1984; Rogers and Waldron 1989)..

[3] For an eloquent description and consideration of skeletal changes in leprosy, please see the monograph by Møller-Christensen, 1976.

Treponemal

Treponemal disease includes four chronic granulomatous infections: pinta, yaws, endemic syphilis (bejel) and acquired syphilis (venereal and congenital). Most researchers believe that *Treponema carateum* (pinta), *Treponema pertenue* (yaws), *Treponema pallidum* (endemic syphilis and acquired syphilis) are three related and virtually indistinguishable spirochetes responsible for the treponemal infections (Hackett 1975:229; Striland 1991a: 39). There is considerable debate regarding the origin and relationship of the four spirochetes (Dutour et al. 1994; Hackett 1975; Striland 1991a)[4], however, this is not the format for a detailed discussion. Pinta is primarily an infection of the soft tissue that does not involve the skeleton and therefore it is not of interest in this discussion (Roberts and Manchester 1995).

The basic gross, radiographic and microscopic changes to bone in yaws, endemic and acquired syphilis are virtually indistinguishable and differ mostly in quantity and skeletal distribution and such subjective criteria as severity (Hackett 1975; 1976; Roberts and Manchester 1995; Rogers and Waldron 1989; Rothschild and Rothschild 1995b). Even then, the skeletal presentation of each disease may be difficult to compare and distinguish because different rates of destruction and sclerosis will occur at different stages of each disease (Aufderheide and Rodríguez-Martín 1998:171)[5]. According to Roberts and Manchester (1995: 152), clinical studies reveal a 3-5% skeletal involvement for yaws, 10-12% skeletal involvement for acquired syphilis and somewhere in-between for endemic syphilis. Resnick and Niwayama (1988:2697), report somewhat different rates for modern populations ranging from as low as .15% to as high as 8-20%.

Yaws, endemic syphilis and acquired syphilis are distinguishable primarily through their immunological and epidemiological differences. Clinically speaking, yaws is endemic to tropical and subtropical regions and affects individuals, most commonly males, within the first decade of life. Bejel occurs in rural populations in temperate and nonhumid regions and commonly affects individuals in the first decade of life. Finally, acquired syphilis has a worldwide distribution and predilects urbanized populations. Because venereal syphilis is transmitted through sexual contact, it is most commonly observed in adults between the ages of 15-30 years. Congenital syphilis results from the transplacental spread of venereal syphilis, and is frequently associated with high infant mortality (Aufderheide and Rodríguez-Martín 1998:154-164). For the purposes of this thesis, skeletal changes related to the treponematoses will be discussed generally. For an in depth description and discussion regarding treponemal related changes to the skeleton, Hackett (1976), is the definitive monograph (see also Steinbock, 1976).

The primary stages of infection involve the soft tissues, and it may be as many as 5-10 years before the characteristic osseous changes of tertiary syphilis develop (Aufderheide and Rodríguez-Martín 1998). Multiple osteoclastic lesions accompanied by extensive if irregular osteoblastic regeneration characterize bony change in treponemal infections (Roberts and Manchester 1995:152). Infection moves from the periosteum through the cortex, eventually perforating into the cancellous tissue. On the surface, new periosteal bone is added and eventually remodeled into lamellar bone that is virtually

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[4] Some researchers believe that the spirochete, *Treponema pallidum*. is responsible for all four diseases (Hudson 1958 in Roberts and Manchester, 1995:151).

[5] Recent studies suggest that certain manifestations of destruction and patterning of lesions may be useful in distinguishing various treponemal conditions. These are, however, mostly confined to the post-cranial skeleton (Morse, 1967; Ortner et al., 1992; Rothschild and Rothschild 1995b).

indistinguishable from the cortex (Aufderheide and Rodríguez-Martín 1998:158-159). In yaws, endemic syphilis and acquired syphilis the tibia is the most commonly involved element (Aufderheide and Rodríguez-Martín 1998; Hershkovitz et al. 1994; Ortner and Putschar 1984; Roberts and Manchester 1995). Both yaws and endemic syphilis demonstrate infrequent involvement of the skull (Aufderheide and Rodríguez-Martín 1998; Ortner and Putschar 1984); however, in yaws when the skull *is* affected, it is severe – particularly in the oronasal and palatal regions (Powell 1991:176; Roberts and Manchester 1995:152)[6]. In endemic syphilis, the vault is rarely involved while the face may exhibit oronasal and palatal destruction (Aufderheide and Rodríguez-Martín 1998:157; Roberts and Manchester 1995:152). Cranial vault (frontal and parietals), orofacial and palatal lesions appear to have the highest incidence in venereal or acquired syphilis (Hutchinson and Weaver 1998; Roberts and Manchester 1995:153; Steinbock 1976).

The sequence of destructive and reparative changes to the cranium in late stage acquired syphilis is usually considered diagnostic of the disease (Aufderheide and Rodríguez-Martín 1998:158; Zimmerman and Kelley 1982:102). Syphilis may present as either the common nongummatous form, the highly distinctive gummatous form or a combination of the two (Aufderheide and Rodríguez-Martín 1998:158; Ortner and Putschar 1984:182). The literature regarding nongummatous syphilis is thin considering it is the most common osseous response associated with the disease. This is likely a reflection of the fact that the gummatous "caries sicca" sequence provides the diagnostic criteria for the disease (Aufderheide and Rodríguez-Martín 1998). The nongummatous form exhibits periosteal inflammation, the appearance of hypervascular bone, "roughened external patches" and cortical thickening (Zimmerman and Kelley 1982:99).

In the gummatous sequences, formation of a granulomatous lesion of the soft tissue leads to the spread of the infection to the periosteal surface and the formation of a gumma. The gumma initiates osseous destruction and necrosis that eventually perforates the bones of the skull (Aufderheide and Rodríguez-Martín 1998; Rogers and Waldron 1989; Steinbock 1976). These destructive lesions are usually accompanied by extensive healing and marked periosteal apposition of dense new bone. The new bone resorbs and amalgamates with the cortex so that it is virtually indistinct from the cortical surface (Hershkovitz et al. 1994; Rogers and Waldron 1989). The marrow cavity may become partially obliterated with spongy bone or with sclerotic ivory-like bone. Although the inflammatory reaction is extensive, the formation of a sequestrum is very uncommon (Aufderheide and Rodríguez-Martín 1998; Hackett 1975).

Gummatous changes proceed from the initial stage of the gumma lesion through to either the 1) discrete or 2) contiguous series (Hackett 1976:28). In the discrete series, osteolytic lesions begin on the frontal in the form small pits that eventually become confluent, cavitate and spread to the face and parietals (Hershkovitz et al. 1994:86; Rogers and Waldron 1989:620). As the discrete series progresses, dense new bone forms along the border and "roll(s) over" the margins, smoothly remodeling the lesion and forming a circumvallate cavitation (depression) with a wrinkly radial scar at the center (Aufderheide and Rodríguez-Martín, 1998; Hackett, 1976). The contiguous series leads to the diagnostic caries sicca lesion (Hacket 1975:234: Rogers and Waldron 1989).

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[6] According to Aufderheide and Rodríguez-Martín (1998:156) cranial involvement in yaws is less severe than syphilis. All agree that oronasal destruction is more severe in yaws than in syphilis.

According to Hackett (1975:232), caries sicca refers to the "scar remaining after the healing of superficial gummatous osteitis of the calvaria". The sequence begins similarly as the discrete series with an area of clustered and confluent pits that eventually open to the surface in an irregular "serpiginous" or worm-eaten pattern. As healing proceeds, a mass of dense, smooth, bony nodules form and nestle against each other (Hackett 1975:233; Rogers and Waldron, 1989:621). The inner surface of the cranium is rarely involved, and in the disease's earlier stages, may not cross suture lines (Aufderheide and Rodríguez-Martín 1998; Hackett 1975, 1976; Ortner and Putschar 1984). Aufderheide and Rodríguez-Martín, (1998:162), describe the overall appearance of a syphilitic skull as "geographic" with smooth elevated plateaus of reactive bone, intersected with "valleys", "grooves" and circular depressions. In sum, Hackett (1975:235) recognizes the diagnostic criterion of syphilis to be "crenellated thin-floored depressions and perforations which are surrounded by fine radially striated rims ... relating to circumvallate cavities".

Rhinomaxillary and palatal changes are common in the later stages of the disease. According to Manchester (1994:80), they are usually more massive and "rapidly progressive" than rhinomaxillary changes in leprosy (Manchester, 1994:80). Bilateral loss of the intranasal structures, perforation and cavitation of the palatine process, inflammation and aggressive new bone formation (spiculated) (Hackett 1975:235; Hutchinson and Weaver 1998:447) characterize syphilitic changes in these regions. In congenital syphilis, the notched incisors known as Hutchinson's teeth and the anomalous cusp pattern on the molars known as Mullberry molars are considered diagnostic (Anderson et al. 1986:347; Hackett 1975: 236; Roberts and Manchester 1995:155).

Syphilis is often called the "great imitator" and care should be taken to differentiate it from a number of disorders (Zimmerman and Kelley 1982:101). On the basis of each individual's particular morphological expression and stage of progression at death, the differential diagnosis of treponemal disease of the cranium should include: healed trauma/trephination, osteomyelitis, tuberculosis, leprosy, metastatic carcinoma, multiple myeloma and Paget's disease (Aufderheide and Rodríguez-Martín 1998; Anderson et al. 1986:346).

2.5.3. Other Bacterial, Fungal and Parasitic Infections

Other Bacterial Infections

Actinomycosis

Actinomycosis is a higher bacterial infection caused by the bacillus *Actinomyces israelii* (Ortner and Putschar 1984:218; Resnick and Niwayama 1988:2705; Revell 1986:251). Hematogenous dissemination of the infection results in a mixed osteoclastic and osteoblastic reaction that is frequently found in the mandible, maxilla and ethmoid, and temporal bone (mastoid and TMJ) (Buikstra 1976; Manchester 1994:80; Revell 1986; Rothschild and Martin 1993). Frequently associated with oral trauma, infection or tooth extraction, inflammatory changes are characterized by multiple small resorptive foci with new bone formation limited to the periosteal surface (Ortner and Putschar 1984: 221). The differential diagnosis should include specific infections, periodontal disease and osteoarthritis of the TMJ (Ortner and Putschar 1984).

Fungal Infection

Blastomycosis, Coccidiodomycosis, Cryptococcosis, Mucormycosis Aspergillosis

Fungal infections (mycoses) constitute a very small percentage of infections afflicting the skeleton. Primary infection through trauma or secondary infection through hematogenous dissemination leads to chronic granulomatous lesions that predilect the cancellous spaces and are primarily lytic with little or no reactive bone formation (Aufderheide and Rodríguez-Martín 1998:213; Ortner and Putschar 1984:224; Rothschild and Martin 1993:73). Fungal infections do not present distinctive lesions on dry bone that facilitate their differential diagnosis. Therefore, clinicians and palaeopathologists rely upon the geographical distribution and possible occupational context of the differing mycoses to provide a key to their identification (Aufderheide and Rodríguez-Martín 1998:213). Due to the primarily lytic nature of the lesions, tuberculosis, leprosy and osteoclastic neoplasias should be consulted for the differential diagnosis.

Blastomycosis is an uncommon chronic granulomatous myoctic infection common to North and South America (Aufderheide and Rodríguez-Martín 1998; Resnick and Niwayama 1988), although cases have been reported in Africa (Baily et al. 1991). Cranial inflammation is characterized by small round lytic foci on the ectocranial surface with "fronts of resorption" (viewed only under 10x magnification) (Hershkovitz et al. 1998:49), coalescing cavities and bony remodeling. The endocranial area of the lesion is much larger and exhibits extensive lytic destruction and modest new bone formation (Hershkovitz et al. 1998:57). *Coccidiodomycosis* is fungal infection common today in arid regions of North and South America (Gregg and Gregg 1987:64; Ortner and Putschar 1984; 224; Rothschild and Martin 1993:76). Multiple destructive lesions of the skull with minimal periosteal reaction restricted to the outer table characterize cranial inflammation.

Cryptococcosis is a serious granulomatous infection with worldwide distribution. Clinically seen in individuals with compromised immune systems (Resnick and Niwayama, 1988:2706), the disorder is common in older individuals (Aufderheide and Rodríguez-Martín 1998; 218). The skull is a common site of infection (Gregg and Gregg 1987:64). Multiple non-specific infectious lesions characterized by osteolytic foci with well-defined discrete margins affecting both tables of the skull and "mild sclerosis" limited to the periosteal reaction (Ortner and Putschar 1984:224). *Mucormycosis* is a rare, commonly fatal fungal infection, clinically common in individuals with severely compromised immune systems (Resnick and Niwayama 1988:2723). Changes are usually confined to the face, particularly the sinus regions of the maxilla and ethmoid, however, the frontal and sphenoid can be involved as well (Manchester 1994:80). Profuse osteoclastic activity results in the "extensive dissolution of some of the facial structures" (Resnick and Niwayama 1988:2724). In chronic cases, abnormal bone formation is observed. Finally, *aspergillosis* is a systemic mycotic infection with worldwide distribution and no age or sex predilection. Primary infection of the ear, nasal sinuses, orbits and anterior cranial fossa leads to typical osteomyelitic inflammation and destruction of the surrounding skeletal tissue (Aufderheide and Rodríguez-Martín 1998:220; Ortner and Putschar 1984:226; Resnick and Niwayama 1988:2723).

Parasitic

Parasites have a long history of human involvement. During the agricultural revolution, animals were introduced into the domestic sphere, bringing with them

parasites. As common as the infections probably would have been, the palaeopathological evidence is rare and usually secondary in nature. Very few parasites have a direct impact upon the skeleton, and of the small numbers that exhibit abnormal bone change (e.g. *toxoplasmosis*), an even smaller number of these leave evidence on the skull. Of the diseases that affect the skull, none provide diagnostic evidence. For example, changes to the skeleton in *Echinococciasis* (Hydatid disease) occur in less than two percent of all cases (Zimmerman and Kelley 1982). Changes to the skull are extremely rare and consist of "intraosseous cysts" that occasionally progresses to multiloculated lesions. Zimmerman and Kelley (1982:109) state that it is unlikely that the disease can be differentiated from other osteolytic conditions such as fibrous dysplasia, metastatic carcinoma, and multiple myeloma.

Parasitic infection may leave secondary or indirect evidence of their involvement. Some parasitic infections such as *Filariasis*, *Dermatobia hominis* (bot fly), and *Cochlimyia hominivorax* (screw worm) involve serious skin lesions (Aufderheide and Rodríguez-Martín 1998; Sancho et al. 1996) that may result in a superficial inflammation of the outer table, and possibly the introduction of bacteria to the underlying bone This may culminate in a non-specific primary bone infection. Skeletal changes as a result parasitic infections are often sited as possible etiological factors in metabolic and haemopoetic disorders relating to malapsorption, decreased bioavailability of specific nutrients and generalized stress. For example, cribra orbitalia and porotic hyperostosis, generally considered indicative of iron deficiency anemia, which may be caused by a parasitic infection (Stuart-Macadam 1990).

Viral

According to Aufderheide and Rodríguez-Martín, (1998:207) skeletal involvement in viral infection is particularly rare. Viruses tend to be acute – often causing death before the skeleton has a chance to be involved and respond. Viral infections were rarely chronic and do not usually produce pus and abscesses; therefore, bony reaction is limited and non-descript. Finally, viruses often require a large/ and or specific population bases to take hold and as such, were likely rare in prehistory (e.g. smallpox) (Aufderheide and Rodríguez-Martín 1998: 201)

Smallpox is one of the most common and destructive viral infections in the world; however, it exhibits only minimal, non-specific evidence on the skeleton of its involvement. Changes are similar to bacterial osteomyelitis and do not usually affect the cranium (Ortner and Putschar 1984:228). *Rubella*, while relatively harmless to children and adults, can be very serious to developing fetus (Ortner and Putschar 1984:229). Poor mineralization of the growing bones is most marked in the metaphyseal regions, but can affect the skull (Aufderheide and Rodríguez-Martín 1998:209). Hindered growth, enlargement of the anterior fontanel and poor mineralization are characteristic of changes to the cranium. It is difficult to recognize rubella in the palaeopathological record because of the fragile nature of fetal bones. Further, if the infant survives past three months post-natal, all traces of the disease will remodel, leaving no trace (Ortner and Putschar 1984:229).

2.6. Neoplastic Disease

A neoplasm is typically defined as an abnormal mass of tissue, or tumor, whose

uncoordinated growth exceeds that of normal tissues (Roberts and Manchester 1995:186). Neoplasms vary from relatively harmless, slow forming growths to aggressive, destructive lesions that have serious implications for the quality of life and vitality of the individual. Neoplasms may arise at any place in the body and in their broadest sense can affect any individual regardless of age, sex, biological affinity, health and nutritional status or social group. However, it is important to note that the prevalence of specific neoplasms can vary within these groups thereby providing a possible key to the etiology of the lesions (e.g. in females metastatic cancer is frequently associated with breast cancer while in males it is associated with prostrate cancer) (Roberts and Manchester, 1995:186). While the specific cause of most neoplasms is poorly understood, it is thought that genetic propensity, cultural practices, and in particular environmental disturbances are among the most important etiological factors (Ortner 1981; Rúa et al. 1995).

While the term neoplasia refers to abnormal tissue growth, neoplastic changes observed in dry bone can be characterized as abnormal bone loss, abnormal bone growth or a combination of both. Osteolytic neoplasms usually result from the elimination of osseous tissue due to the impingement of an abnormal growth on an adjacent tissue that produces a "negative" or a "mould" of the abnormality (Brothwell 1967:320). Osteoblastic growths can result in differing qualities of bone, a variety of shapes and contours depending upon the type of growth and the primary or secondary nature of the growth. Neoplastic growths are typically divided into two categories: benign or malignant (Zimmerman and Kelley 1982). Benign growths tend to remain at site of origin without a systemically affecting the body, while malignant growths tend to develop aggressively at or on the site of bone or spread (metastasize) to bone from another location (Roberts and Manchester 1995:186). In the palaeopathological record, benign growths are common in older individuals, while malignancies are more common in the young. Both types of growths can be classified as primary or secondary. Primary neoplasms arise on the bone, are often singular rather than multiple, and tend to predilect the appendicular skeleton as opposed to the axial skeleton (Baraybar and Shimada 1993). Secondary neoplasms arise at another location in the body, and spread to bone. According to Waldron (1994:464), secondary tumors commonly exhibit a mixture of bone resorption and formation and are the most common tumors of the skeleton.

Benign neoplasms are not as significant to the vitality and morbidity of the individual because the negative affects of benign lesions are generally restricted to the impingement of the growth on the surrounding tissues. Osteoblastic growths are usually characterized by dense smooth projections, while well-demarcated borders, short zones of transition and sclerotic margins, often characterize osteolytic reactions. These features are indicative of the slow, eventual growth of most benign neoplasms (Roberts and Manchester 1995; Rothschild and Martin 1993).

Conversely, malignant growths are characterized by their propensity to spread to different areas of the body and destroy tissue, and if left unchecked eventually lead to the death of the individual. Malignant lesions display significant "growth autonomy" (Aufderheide and Rodríguez-Martín 1998:372; Roberts and Manchester 1995:188), meaning they can disseminate through the blood and lymphatic system into any zone of the body. In bone, osteoblastic growths are characterized by expansive, multi-layered, sunburst and spiculated osseous reactions that are indicative of rapid expansion. Variably sized, multi-focal resorptive lesions, with ill-defined margins and broad zones of

transition inferring a destructive and aggressive growth characterize osteolytic reactions (Rothschild and Rothschild 1993). The following discussion considers some of the more common neoplastic conditions of the skull based upon the predominantly observed osseous reaction (osteoblastic, osteoclastic/osteolytic or mixed) and the benign or malignant nature of the neoplasm.

2.6.1. Abnormal Bone Formation

2.6.1.1. Benign Neoplasms

Osteoma, Hereditary Multiple Exostoses and Osteochondroma

Spjut et al. (1971) defines osteoma as a "protruding tumor mass, composed of abnormally dense, but otherwise normal bone, formed in the periosteum". While osteoma is generally classified as pathological condition, Capasso views the hyperostotic lesion to be of a non-pathological, idiopathic nature (1997:619). Human osteoma predilects the skull, particularly the frontal and parietals (Capasso 1997:615). Ortner and Putschar, (1984:368) recognize three types of benign osteoma useful to this discussion. The most common is the button or ivory osteoma. A small (<2cm), slow- growing tumor, comprised of dense, ivory-like bone, often circular and presenting frequently as solitary or multiple lesions on the cranial vault, face and mandible (Ortner and Putschar 1984; Zimmerman and Kelley 1982). Usually the mass presents a "peripheral circular constriction" (Ortner and Putschar, 1984:368). Another type of osteoma forms in the external auditory meatus and is characterized by a small dense, bony projection that can grow to occlude the canal (Ortner 1981; Ortner and Putschar 1984; Zimmerman and Kelley 1982). Auditory exostoses are clinically similar to osteomas and consist of dense lamellar bone. They appear to form as a reaction to thermic stresses, such as prolonged exposure to cold water (Barnes and Peel 1990; Capasso 1997). The third type of osteoma predilects the frontal and paranasal sinuses with a large mass of fibrous woven bone that eventually remodels into dense lamellar bone. This lesion often leads to severe facial disfigurement (Ortner and Putschar 1984). Other benign neoplasms characterized by an osteoblastic reaction are: hereditary multiple exostoses and osteochondromas (singular exostoses). Both tumors occasionally exhibit dense bony projections on the endocranial surface of the skull base (sphenoid and parasellar); however, both are most commonly expressed on the post-cranial skeleton as either a single (osteochondroma) or a multi-focal lesion (hereditary multiple exostoses) (Ortner and Putschar 1984: 373; Resnick and Niwayama 1988:3703).

2.6.1.2. Malignant Neoplasms

Osteoblastic Metastatic Carcinoma

According to Ortner and Putschar, (1984:392) the most common carcinomas that metastasize to the skeleton are breast and prostrate. Carcinomas metastasizing from the breast are predominantly osteolytic and predilect the skull and thoracic region, while carcinomas of the prostrate are characterized by abnormal bone formation and predilect the pelvis and spine, only occasionally spreading to the skull (Baraybar and Shimada 1993:135). Osteoblastic lesions on the skull are characterized by unorganized fibrous and fine bony spicules projecting from the surface of the pre-existing bone (Aufderheide and Rodríguez-Martín 1998; Rúa et al. 1995). Osteoblastic metastatic carcinoma is usually associated with individuals of advanced age as opposed to osteosarcoma, a condition that

primarily affects the young (Aufderheide and Rodríguez-Martín 1998; Suzuki 1987). Care should also be taken to distinguish this condition from Paget's disease (Rúa et al. 1995; Tkocz and Bierring 1984).

Other benign and malignant neoplasms that exhibit abnormal bone formation also exhibit considerable osseous destruction, and for therefore will be considered under 'Mixed Reactions'.

2.6.2. Abnormal Bone Loss

2.6.2.1.Benign Neoplasms

Osteoblastoma

Osteoblastoma is a benign lesion characterized by a predominantly lytic reaction, although both abnormal bone formation and mixed reactions are observed as well (Resnick and Niwayama 1988:3636). While predominantly a condition that predilects the vertebrae and lower extremities, it is occasionally found in the skull with the mandible and the maxilla being the most frequently affected (Ortner and Putschar 1984; Resnick and Niwayama 1988). The lesion is characterized radiographically as a lucent oval of small size with well-circumscribed margins (Resnick and Niwayama 1988) and is most common in individuals under 30 years of age.

Giant Cell Tumor

According to Zimmerman and Kelley (1982:118), giant cell tumors can be either benign or malignant and are characterized by their large resorptive lesions, frequently associated with a "thin cortical shell" expanding from the cortex and "eccentric" locations (Ortner and Putschar 1984:375). Thin bony shells are considered a diagnostic feature of the growth, however, these are easily damaged in archaeological contexts. Giant cell tumors predominantly occur in bones that develop from endochondral ossification, therefore they are not common in the skull because most cranial bones are derived from membranous ossification; however, the sphenoid, temporal, facial bones and temporomandibular joint may occasionally exhibit the tumors (Resnick and Niwayama 1988:3759; Rothschild and Martin. 1993:183). Giant cell tumors need to be differentiated from aneurysmal bone cysts and the 'brown tumors' of hyperparathyroidism (Aufderheide and Rodríguez-Martín 1998:387).

Epidermoid Bone Cysts

Epidermoid bone cysts predilect the hands and skull and are commonly associated with a history of trauma to those regions (Aufderheide and Rodríguez-Martín, 1998). Any bone of the skull can exhibit an epidermoid bone cyst, however the frontal and parietal are the most frequent locations (Resnick and Niwayama 1988:3830). The lesion is characterized by well-defined lytic area with a sclerotic margin. The differential diagnosis should include infections due to middle ear disease, metastatic carcinoma and osteomyelitis (Aufderheide and Rodríguez-Martín 1998:391; Resnick and Niwayama 1988:3831).

Ossifying Fibroma

Ossifying fibroma is a solitary, slow growing benign tumor that is exclusively observed in the vault, face and mandible (Barnes and Peel 1990:134). It is most common

in young and middle-aged adults, however, there is a discrepancy regarding the sex predilection (none vs. women). The lytic lesion is usually oval or spherical with a sharply marked border. Lesions are expansive and associated with cortical thinning (Rothschild and Martin 1993). It should be differentiated from other osteolytic neoplasms, fibrous dysplasia and eosinophilic granuloma.

Desmoplastic Fibroma and Neurilemmoma

Other benign neoplasms exhibiting abnormal bone loss to the skull are desmoplastic fibroma and neurilemmoma. Lucent resorptive lesion generating a 'soap-bubble' pattern on the mandible are characteristic of desmoplastic fibromas (Aufderheide and Rodríguez-Martín 1998:382). It may be associated with reactive periostitis and a sclerotic halo indicative of slow growth. It is usually associated with adults under the age of 30. Neurilemmomas exhibit single, ovoid, resorptive lesions with reactive periostitis on the mandible as well (Aufderheide and Rodríguez-Martín 1998:385). Neurilemmomas predilect adults between the ages of 30 and 50.

2.6.2.2. Malignant Neoplasms

Multiple Myeloma

Multiple myeloma is the most common primary malignancy, affecting almost exclusively older individuals (Aufderheide and Rodríguez-Martín 1998:351; Roberts and Manchester 1995:190). According to Ortner and Putschar, (1984) it is much more common in males than females. Multiple myeloma arises in the red marrow of the bone, and has been classified as a haemopoietic disorder (Strouhal 1991). Characterized by multi-focal circular lytic lesions with a "punched out" appearance (Rothschild and Martin 1993; Strouhal 1991), multiple myeloma can affect the entire skull, particularly the mandible and cranial vault. This condition begins in the diploic table and presents sharply-marginated, with almost no associated sclerotic bone and the occasional pathological fracture (Strouhal 1991). The lesions are often scalloped and of relatively uniform size (3 to 10 mm) (Ortner and Putschar 1984:264). While single lesions are sporadically observable on dry bone, radiographic images demonstrate that there tends to be multiple lytic lesions arising in the diploic table of the skull, progressively penetrating the inner and outer cortexes giving the skull a 'motheaten' appearance (Aufderheide and Rodríguez-Martín 1998). Multiple myeloma is most commonly confused with metastatic carcinoma (see below). Other disorders that should be considered in the differential diagnosis include: histocytosis-X, and tuberculosis. If the skull presents singular lesions, care must be taken to distinguish them from trauma, trephination, bi-parietal thinning and osteoporosis.

Osteolytic Metastatic Carcinoma

According to Roberts and Manchester, (1995: 191) while the aggressive and destructive nature of the lesions of multiple myeloma distinguishes them from syphilis or trauma, the principle condition multiple myeloma must be differentiated from is metastatic carcinoma. Metastatic lesions arise secondarily from primary growths of the soft tissue that then spread hematogenously or lymphatically to distant areas of the body. Metastatic carcinomas, like multiple myeloma predilect older adults, but may differ according to sex based on the primary cancer (e.g. breast cancer in females, prostrate

cancer in males). As discussed above, most forms of metastatic cancer (breast, lung, kidney) produce characteristically lytic lesions, however some forms of cancer (e.g. prostate) will produce blastic metastases (Baraybar and Shimada 1993). In the cranial vault, osteolytic metastatic carcinoma commences in the diplöe table spreading to the inner table first. The disorder usually presents multiple resorptive holes of various sizes that may present a macroscopically observable moth-eaten appearance (Manchester 1983; Ragsdale 1993; Strouhal 1991). Radiographically, the metastatic lesions tend to blend with the surrounding bone in some places exhibiting a shadow of increased density (Rothschild and Rothschild 1995a; Strouhal 1991). This increased density is occasionally observable as circumferential sclerotic bone. Pathological fractures and infections are common sequelae of metastatic cancer (Aufderheide and Rodríguez-Martín 1998; Roberts and Manchester 1995). Rothschild and Rothschild (1995:363), observe that it is difficult to differentiate the multiple myeloma and osteolytic metastatic carcinoma. This a result of the fact that while multiple myeloma is characterized by lesions of uniform size, it may manifest lesions of various sizes, and while metastatic carcinoma is characterized by well-rounded lesions with sclerotic margins, it may occasionally look punched-out (Resnick and Niwayama 1988; Steinbock 1976:423). Other disorders that should be considered in the differential diagnosis include: trauma, histocytosis-X, tuberculosis, trephination, bi-parietal thinning and various congenital disorders.

2.6.3. Mixed Reaction Neoplasms

2.6.3.1.Benign Neoplasms

Hemangioma

Hemangioma is a primary benign vascular neoplasm than predilects the calvarium and vertebrae. The disorder is common in females over 50, hemangiomas are characteristically proliferative lesions associated with areas of peripheral resorption due to increased vascularity. These areas of vascularity are usually single and are thickened due to the formation of "bony spicules perpendicular to the skull tables" (Anderson 1992; Zimmerman and Kelley 1982:120). This gives the skull a "coarse honeycomb, web-like, bubbly or sunburst trabecular patterns" (Rothschild and Martin 1993:183). Care must be taken in distinguishing the spiculated pattern in hemangiomas from the 'sunburst' effect and expansive growth of an osteosarcoma (Zimmerman and Kelley 1982).

Meningioma

Intraosseous meningioma is a benign type of neoplasm that arises in the arachnoid granulations of the endocranial and intracranial regions and may progressively manifest itself onto the ectocranial surface (Anderson 1992; Waldron 1998). Clinical studies divide meningioma into hyperostotic, osteolytic and mixed types (Crawford et. al. 1995). While abnormal bone formation is the commonly observed reaction, osteolytic and mixed reactions are common as well (Campillo 1991; Ortner 1981). Campillo offers a classification scheme with eight types of meningioma, none of which he considers pathognomonic (1991:226). Small endocranial exostoses (< 20mm), thickened cranial tables, and the formation of occasionally dense 'spongy-like' outgrowths characterize osteoblastic reactions (Anderson 1992; Campillo 1991; Waldron 1998). In rare cases, radiant spicules or dense osteosclerotic bone may form destroying the outer table of the vault; these may be virtually indistinguishable from osteosarcoma in dry bone (Ortner

and Putschar 1984: 378). Osteolytic reactions are characterized by small endocranial depressions, small resorptive lesions, increased vascular grooving, expanded diploë, and enlarged meningeal arteries (Waldron 1998). According to Waldron (1998: 212), they are "the commonest cause of pathological vascular markings on the skull". Mixed reactions often present a combination of osteoblastic and osteolytic formations (Campillo 1991:226). Meningioma predilects females and is rarely observed in younger adults and adolescents (Campillo 1991; Resnick and Niwayama 1988). It is important to distinguish hyperostotic meningioma from other neoplasms such as osteoma, osteosarcoma (Roberts and Manchester 1995:189), Paget's disease (Waldron 1998:212) and hyperparathyroidism (Crawford et. al. 1995:914). Osteolytic meningiomas need to be differentiated from hemangioma, epidermoid cyst, myeloma, eosinophilic granuloma, metastatic cancer and fibrous dysplasia (Crawford et al. 1995).

2.6.3.2. Malignant Neoplasms

Osteosarcoma

While clinical studies suggest that osteosarcomas are rare neoplasms (Spjut et al., 1983), according to Waldron (1996:464), it is the most common primary malignancy in the archaeological record. Osteosarcoma predilects the long bones of adolescents and young adults. Lesions on the cranium are frequently associated with Paget's disease, a condition common in older individuals (Aufderheide and Rodríguez-Martín 1998). Cranial osteosarcoma manifests itself primarily in the bones of the vault and mandible and while the lesion is most strikingly in its osteoblastic presentation, the over all pathogenesis is frequently of a mixed osteoblastic/osteoclastic nature (Aufderheide and Rodríguez-Martín 1998: 377; Zimmerman and Kelley 1982). Osteosarcoma is characterized by "frenzied and uncontrolled" (Roberts and Manchester 1995:190) fibrous or woven bone growth that begins in the diploic table breaking through the cortex and occasionally causing a pathological fracture (Stouhal et al.1997; Suzuki 1987). On the cortex a series of radially distributed bone spicules known as the "sunburst effect" provide the diagnostic feature of this condition (Aufderheide and Rodríguez-Martín 1998:378; Ragsdale et al. 1981:751). Osteosarcoma requires differentiation from hemangiomas, meningiomas, osteoblastic metastatic carcinoma. Care must also be taken to differentiate osteosarcoma from callus formation associated with trauma (particularly in the mandible) and osteomylelitis (Suzuki 1987).

2.7 Congenital and Developmental Disorders

Congenital disorders are commonly defined as developmental abnormalities of the soft tissues or skeleton that begin during fetal development and are present at or around birth (Aufderheide and Rodríguez-Martín 1998:51; Roberts and Manchester 1995:31). Congenital disorders may range from minor disruptions in morphology and form, to major abnormalities (Barnes 1994:8) impingent on vitality. Barnes (1994:2) prefers the term 'developmental defects', because while the underlying etiological or genetic factor may be present at birth, many congenital disorders remain undetected until developmental (childhood), functional (adulthood) or environmental stresses precipitate their presentation. Congenital and developmental disorders are also known as defects, malformations, abnormalities, and diseases (Aufderheide and Rodríguez-Martín 1998; Brothwell 1968; Turkel 1989). This myriad of terms, in addition to their poorly

understood etiologies, underlies the major problem of classification that congenital and development disorders present (Resnick and Niwayama 1988; Turkel 1989). It is not the purpose of this discussion to review all the defects or present the possible classifications of congenital disorders; rather the following is abbreviated presentation of some of the possible disorders that can be observed on the skull. Skeletal malformations will be classified by the following categories: abnormality of shape, abnormal bone loss, abnormal bone gain.

2.7.1. Abnormal Contour or Shape

Craniosynostosis

Closure of the cranial sutures is a normal part of the aging process in adults. When this process occurs at an abnormally early age, malformation of the cranium can result. Craniosynostosis (premature closure of the cranial sutures) is one the most common congenital/developmental disorders of the skull (Aufderheide and Rodríguez-Martín 1998:52; Cohen 1986:4). Craniosynostosis may arise from primary genetic causes or from secondary associations with various disorders such as anemia, or rickets or as a result of environmental influences (Cohen 1986:13; Pedersen and Anton 1998: 370; Turkel 1989:111). Aufderheide and Rodríguez-Martín (1998:52) note that less than one-third can be assigned a specific etiology. While the prevalence of craniosynostosis is unknown, however it appears to predilect males over females. Premature synostosis presents completely obliterated sutures, occasionally associated with ridges of bone, and variations in length and form as a function of the particular suture (Aufderheide and Rodríguez-Martín 1998; Dean O'Loughlin 1996; Ortner and Putschar 1984). Deep cerebral impressions from increased intracranial pressure can also be noted. The degree and nature of the malformation depends upon the individual suture(s), the number of sutures involved, the degree of closure, the order of closure, the age of synostosis and the underlying etiology. The several types of craniosynostoses recognized in the palaeopathological and clinical literature are summarized in the table below.

There are a number of syndromes that are associated with premature closure of the cranial sutures including, Apert's syndrome, Crouzon's syndrome and Carpenter's syndrome. Apert's syndrome is often accompanied with hydrocephaly and presents a brachyocephalic/ scapheocephalic cranium with a flattened occipital, possible palatal division, hypoplasia of the maxilla and delayed dental eruption (Aufderheide and Rodríguez-Martín 1998:55; Zimmerman and Kelly 1982: 21). Crouzon's syndrome is characterized by oxycephaly and brachycephly, intercranial pressure causing bossing at bregma, a thinned frontal bone, shallow orbits and wide-set eyes, a long foramen magnum, hypoplasia of the maxilla and paranasal sinuses, prognathism and dental anomalies (Aufderheide and Rodríguez-Martín 1998:54). Carpenter's syndrome is similar to Apert's syndrome and is characterized by early closure of the coronal, sagittal and lambdoidal sutures (Aufderheide and Rodríguez-Martín 1998:55).

The differential diagnosis of craniosynostosis must include other cranial deformities including microcephaly, macrocephaly and hydrocephaly (Zimmerman and Kelley 1982: 23). While care is required in differentiating craniosynostosis and artificial cranial deformation, it must be remembered that most researchers recognize a causal relationship between the artificial deformation and premature suture closure (Dean O'Loughlin 1996; White 1996).

Anencephaly

Anencephaly is a fatal congenital defect of the neural canal resulting in the malformation of the brain and surrounding cranium (Aufderheide and Rodríguez-Martín 1998:55; Ortner and Putschar 1984: 346; Zimmerman and Kelley 1982:23). The cranial vault is absent, leaving the underdeveloped brain exposed. The skull base is deformed and the orbits are usually small and partially formed. In clinical studies, anencephaly appears with higher frequency in populations of lower socio-economic status. There is no sex predilection. While, anencephaly is surprisingly common in modern populations, with an incidence rate of 1 in 1000 births, it is rare in the archaeological record. This is likely a result of prenatal death, the fragile nature of fetal bones of the skull and poor recognition of the remains.

Table Appendix 2.1. Craniosynostoses

Disorder	Suture	Sex	Shape of Vault
Brachycephaly	Coronal	-	short-headed [a]
Turricephaly	Coronal & sagittal,/ sphenofrontal	F	broad skull with a tower-like forehead, wide-set eyes [a, b, e]
Scaphocephaly/ Dolichocephaly	Sagittal	M	elongated & narrow skull with a widened forehead with prominent frontal bosses, narrowed skull base & maxilla with a bony ridge from bregma to lambda [a, b, d, e]
Synostosis of the Lambdoidal suture	Lambdodial	-	Flat occipital [b]
Plagiocephaly	Frontal & Sphenofrontal	Uknown	lopsided skull with a deformed base & orbits [1]
	Occipital	M	flattened of the occipital & prominent forehead [b]
	Coronal, Squamous, Lambdoid	-	multiple fusion on one side of skull [b]
Oxycephaly	Lambdoidal & Coronal	-	high-conical forehead with increased height at bregma [b, c]
Trigonocephaly	Metopic suture	M	Triangle-shaped skull, close set eyes narrow forehead, and bony ridge from glabella to bregma [a, b, e]

a Cohen (1986:4-5,12)
b Aufderheide and Rodríguez-Martín (1998:52-54)
c Barnes (1994)
d Ortner and Putschar (1984:352)
e Zimmerman and Kelley 1982:21

Hydrocephalus

Hydrocephalus is another malformation of the skull occasionally observed in the archaeological record (Kreutz and Schultz 1994; Richards and Anton 1991). This disorder is characterized by an enlargement of the cranial vault due to an accumulation of cerebral spinal fluid in the intracranial spaces (Richards and Anton 1991; Roberts and Manchester 1995). A combination of a normal face with a large globular vault, prominent frontal bossing, thin cranial bones, widely separated sutures, atrophied supraorbital margins, and cranial capacities as large as 3300 and 3900 have been noted (Aufderheide and Rodríguez-Martín 1998:57; Richards and Anton 1991: 191). Hydrocephalus is not strictly a congenital abnormality, often occurring in later childhood or adulthood as

sequelae of other disorders including tumor, infection, or trauma (Aufderheide and Rodríguez-Martín 1998; Ortner and Putschar 1984). Hydrocephalus should be distinguished from macrocephaly and achondroplasia (Zimmerman and Kelley 1982:23).

Macrocephaly

An abnormal enlargement of the brain results in a corresponding enlargement of the cranial vault (Brothwell 1968; Zimmerman and Kelley 1982). A macrocephalic brain weighs between 1600 and 2800 grams (1200 to 1400 g is normal). According to Zimmerman and Kelley (1982:23) it is "difficult or impossible" to distinguish macrocephaly from hydrocephalus on the basis of limited skeletal remains.

Microcephaly

A statistically smaller skull circumference, usually resulting from the failure of the brain to develop characterizes microcephaly (Brothwell 1968; Zimmerman and Kelley 1982). While genetic defects are largely responsible, microcephaly may be associated with congenital infections, toxins, irradiation, Tay-Sachs disease, or phenylketonuria (Zimmerman and Kelley 1982:22). The brain typically weighs between 500 and 600 grams; cranial capacity is always less than 1000 cc and the circumference less than 46 cm (Aufderheide and Rodríguez-Martín 1998: 56). The skull can exhibit irregularities in thickness, producing enlarged lacunae, flattened occipital bones, prominent nasal bones, a receding mental eminence, craniosynostosis, conodially shaped skull and a large face in relation to head (Aufderheide and Rodríguez-Martín 1998: 56).

Achondroplasia

Achondroplasia is a congenital disorder resulting in disproportionate dwarfism and characterized by a strong familial predilection. While 50% of achondroplastic dwarfs die in *utero* and infancy, the rest grow to maturity (Zimmerman and Kelley 1982:40). The limbs are disproportionately small in comparison to the thorax, while the head is disproportionately large. Cranial manifestations include an enlargement of the vault and a shortening of the base, bulbous forehead, wide face, depressed midfacial/nasal region and a deformed foramen magnum. Hydrocephalus may be present to a mild degree (Ortner and Putschar 1984:329; Zimmerman and Kelley 1982:40). Cranial remains require differentiation form hydrocephalus, hypopituitarism and hypothyroidism.

2.7.2. Abnormal Bone Loss

Parietal Fenestrae (Catlin Marks), Meningocoele/Encephalocoele, Partial Dysostosis, Cranial Dysostosis

Another type of congenital disorder results from the failure of the vault to completely ossify, thereby leaving circular defects along the sutures or bones of the skull (Kaufman et al. 1997: 198). These circular defects may take on a number of forms as detailed below.

Parietal fenestrae usually present as bilateral holes along side the parietal foramina (thereby distinguishing them from enlarged parietal foramina). They are oval in shape, equal in size and have smooth edges and beveled margins. According to Kaufman et al. (1997:197), parietal fenestrae or Catlin marks are among the most common of congenital defects of skull.

A meningocoele or an encephalocele is defined as a congenital herniation and perforate the cranial vault (dysraphism) by the meninges and brain. These defects are usually associated with circular, saucer- shaped depressions (Barnes 1994; Stewart 1975; Webb 1995; Webb and Thorne 1985) on the occipital, parietal and nasal regions of the skull (Aufderheide and Rodríguez-Martín 1998). Partial dysostosis, frequently associated with meningoceles are circular lesions commonly found in the lower occipital, sagittal or frontal region. Complete dysostosis presents elongated defects along the sutures, particularly the sagittal and maybe associated with other congenital and developmental disorders (Kaufman et al. 1997).

Cranial holes that result from a congenital failure of the skull to ossify are commonly mistaken for trephination and healed trauma (Stewart 1975). Webb and Anton (1985), include treponemal infection, tuberculosis and multiple myeloma in their differential diagnosis of a parietal meningocoele. According to Kaufman et al. (1997), they can often be distinguished because of their characteristic location along the midline or suture, often-bilateral, symmetrical presentation, and smooth well-remodeled margins.

Cleidocranial dysostosis

Cleidocranial dysostosis is a developmental disorder affecting intramembranous ossification and characterized by agenesis of the clavicles and dramatic changes to the skull (Ortner and Putschar 1984:338). The cranial vault may exhibit incomplete ossification, widening of the sutures and persistence of the fontanels (Aufderheide and Rodríguez-Martín 1998:72; Kaufman et al. 1997). The frontal and parietals are widely splayed, occasionally forming a "metopic fontanel" (Kaufman et al. 1997: 195). The brachycephalic cranial vault is often disproportionately large in comparison with the face. Anomalous ossification may result in numerous small bones that give the skull a mosaic appearance (Aufderheide and Rodríguez-Martín 1998:72; Revell 1986:62). The orbits, mastoid and foramen magnum may be malformed and the skull base shortened. The mandible may not completely fuse at the mental eminence and dental anomalies are often associated (Aufderheide and Rodríguez-Martín 1998:72; Ortner and Putschar 1984: 340). The differential diagnosis should include osteogenisis imperfecta, hydrocephalus, brachycephaly, disorders characterized by the failure to ossify and fibrous dysplasia (Ortner and Putschar 1984: 338).

Cleft Palate and Cleft Lip

Craniofacial deformities are relatively infrequent in the archaeological record due to the fragile nature and poor preservation of the facial bones. Cleft palate and lip are occasionally recognizable because of their diagnostic features (Gregg and Gregg 1987:136; Ortner and Putschar 1984:346-351). Roberts and Manchester (1995:39) point out that cleft defects to the face inhibit breast-feeding; therefore, infants with these disorders in antiquity may have died in early infancy. While the two disorders are often associated, demonstrating a strong familial predilection, cleft palate occurring on its own is thought to be the result of environmental factors (Roberts and Manchester 1995:40). Females exhibit the malformations more frequently than males (Zimmerman and Kelley 1982: 21). Cleft palate is characterized by the arrested development of the palate. It can present as a minor cleft or a large U-shaped defect along the midline leading to an opening between the oral and nasal cavities (Aufderheide and Rodríguez-Martín 1998:

58; Webb 1995:244; Zimmerman and Kelley 1982:21). Cleft lip exhibits a defect on the anterior portion of the maxillae, which may or may not be contiguous with a cleft palate. The anterior dentition (incisors, canines) may be atavistic. According to Ortner and Putschar, (1984:350), infection, trauma, AMTL due to dental disease should be considered in the differential diagnosis.

Osteogenesis Imperfecta

Osteogenisis Imperfecta is a hereditary disorder that shows a strong familial connection. There is no sex predilection. Defective collagen formation and osteoblastic inhibition resulting in generalized osteopenia and brittle bones that fracture easily (Aufderheide and Rodríguez-Martín 1998:365; Ortner and Putschar 1984:337) characterize the disorder. While the disorder predominantly affects the weight bearing bones of the post-cranial skeleton, the skull may exhibit an enlarged vault, patent sutures, thinned cortices, sparse trabeculae and prominent bossing (Zimmerman and Kelley 1982:41-42). Multiple centres of ossification may lead to numerous wormian bones, and a mosaic-like pattern. Deciduous and adult dentitions are usually deformed and the poorly mineralized dentine leads the enamel to chip easily (Aufderheide and Rodríguez-Martín 1998: 365). The fragile nature of the skeletal material make it difficult to identify in archaeological contexts, however, cliedocranial dysostosis and fibrous dysplasia should be considered in the differential diagnosis.

2.7.3. Abnormal Bone Formation

Osteopetrosis

According to Rothschild and Martin (1993:158), osteopetrosis is a rare metabolic/ developmental disorder characterized by the failure of normal osteoclastic action. This disorder shows a strong familial tendency with no sex predilection (Ortner and Putschar 1984: 340). Infantile forms usually lead to death, while adolescent (osteopetrosis tarda) forms survive into adulthood. Unchecked osteoblastic activity leads to heavy, dense bones that are very brittle. Often the presence of cement lines and unorganized bone can be observed in the cross-section. The face and skull base become thick and dense and the passages for the cranial nerves are narrowed. In the tarda form there is little involvement of the skull (Ortner and Putschar 1984:341), however, Aufderheide and Rodríguez-Martín, (1998:363) note that the diploic table and cranial sinuses can become obliterated. Evidence for anemia is occasionally observed and osteomyelitic infection of the mandible is a common sequelae (Revell 1986:56-57).

Pycnodysostosis

Pycnodysostosis is another developmental abnormality observed in early childhood that is characterized by abnormal bone formation that presents dense, thickened bones. According to Ortner and Putschar, (1984:342), the cranial sutures remain patent and the fontanels open. Eventually the intervening areas will fill with wormian bones. Affected individuals frequently exhibit relatively small faces and receding chins with misplaced and frequently carious teeth. This condition should be differentiated from osteopetrosis (Revell 1986:61).

2.8. Joint Disease

Joint disease is among the most recognizable and widely studied group of diseases in palaeopathology. It is generally divided into four categories including: neuromechanical (osteoarthritis), inflammatory (septic or infections arthritis), immune (rheumatoid, ankylosing spondylitis, psoriatic arthritis) and metabolic (gout) (Rogers and Manchester 1995:101). Although there are a number of joint disorders arising from a variety of etiological factors, the structures involved have a limited capacity for change, and as a result, a number of different diseases may produce similar lesions (Rogers et al. 1987:179). Changes to the joint are characterized by predominantly erosive activity with new bone formation along the margins of the articular surface (Rogers and Manchester 1995:101). According to Rodgers and colleagues (1987:179), the identification of joint disease requires a consideration of the skeleton in its entirety. On the cranium, there are only two joints available for consideration: the temporomandibular joint (TMJ) and the basi-occipital.

Osteoarthritis

Osteoarthritic or degenerative changes to the joints of the skeleton are the most frequently observed disease changes in the archaeological record (Alpagut 1979: 571; Aufderheide and Rodríguez-Martín 1998:93; Roberts and Manchester 1995:100). While frequencies of osteoarthritis differ between populations, the universal nature of the disease is directly related to its most significant etiological factor: increasing age (Hodges 1991; Richards 1988). As individuals age, increased biomechanical stress at the joint precipitates a breakdown of the articular cartilage and the surface of the joint. Degenerative joint disease can be classified as either idiopathic or secondary in which the joint is affected by a primary disruption of normal biomechanical activity (e.g. trauma, occupational stress, congenital disorders, infection), increased loading of stress, and cellular activity (e.g. metabolic, vascular and neuotrophic conditions). In clinical studies, idiopathic arthritis is by far the more common of the two (Aufderheide and Rodríguez-Martín 1998:93).

Temporomadibular Joint Arthritis

While osteoarthritis of the TMJ is most clearly linked to increased age, its presence in younger individuals suggests that other eitological factors such as trauma, malocculsion, "dental status" relating to AMTL and dental attrition (Hodges 1991; Richards 1988; Sheridan et al. 1991) have placed significant biomechanical stress on the joint. For example, Hodges, (1991) found a significant correlation between dental attrition[7] and osteoarthritis of the TMJ. In modern populations there is a predilection towards females, however, in archaeological populations a variety of factors related to gender-based activities may favour one sex over another (Merbs 1983; Rogers and Waldron 1995:45). Cultural and dietary practices may lead to stress on the joint, dental attrition, or ATML, resulting in degradation of the joint.

Three processes characterize osseous changes to the temporomandibular joint: erosion, proliferation, and eburnation. Degeneration begins on the anterior aspect of the articular surface of the joint, splitting the cartilage and exposing the underlying bone.

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[7] Attrition and AMTL are both associated with increased age. When age was controlled for, only attrition demonstrated a significant association (Hodges, 1991:375).

This precipitates the resorption of the osseous surface, which is characterized by pitting porosity and occasionally subchondral cysts. Some areas of erosion are directly associated with areas of newly formed sclerotic bone. Proliferation of new bone known as an osteophyte commonly forms around the margin and surfaces of the joint. The new bone is an attempt by the joint to stabilize the structure by diffusing the increased biomechanical load (Roberts and Manchester 1995:101;Rogers and Waldron 1995:35). Ligaments, tendons and fibrocartilage may ossify and new bone may also form in the periosteum (Rogers et al. 1987:180). Extensive formation of new bone may lead to eventual fusion of the joint. The destruction of the cartilage may lead to bone-on-bone contact between the articular surfaces resulting in eburnation. Eburnation is characterized by hard, shiny surfaces, with parallel grooves corresponding to movement of the joint (Rogers and Waldron 1995:36). Eburnation is considered pathognomonic of osteoarthritis (Rogers and Waldron 1995:13). The above changes may lead to overall deformation of the condylar and temporal joint surfaces. Other changes include "roughness" on the surfaces of the joint (Aufderheide and Rodríguez-Martín 1998:400), flattening of the condylar heads and an enlargement of the mandibular fossa. Studies indicate that the mandibular condyle demonstrates fewer changes than the temporal surface of the joint (Hodges 1991:368). ATML, dental attrition, malocclusion, 'abnormal' craniofacial morphology and mandibular or maxillary trauma may predispose the individual to osteoarthritis of the temporomandibular joint and should be considered supporting evidence.

In this study, the presence or absence of a joint was noted and the degree of osteoarthritic change was scored following Richards, (1988:1529-1531). The elements in question were scored as; 'blank' – unobservable; 'normal'; 'moderate' – localized areas of erosion or proliferation; and 'severe' – generalized proliferation and eburnation. Rogers and Waldron (1995) recommend that in the absence of diagnostic eburnation, at lease two of the following are present: presence of new bone on the surface of the joint and/or in the form of marginal osteophytes; pitting or porosity on the articular surface or; alteration of the joint. According to Aufderheide and Rodríguez-Martín, (1998:400), there are a number of disorders that potentially affect the TM joint. Care should be taken to differentiate from osteoarthritis and middle ear infection, trauma, and rheumatoid arthritis.

Rheumatoid Arthritis

According to Roberts and Manchester (1995:116), rheumatoid arthritis is rarely seen in the archaeological record. The erosive lesions of the disorder are often misdiagnosed as other joint disease, in particular osteoarthritis. Identification of the disorder is often dependent upon recognizing the pattern of distribution throughout the entire skeleton. Rheumatoid arthritis is a chronic, inflammatory disorder of the synovial joints (Aufderheide and Rodríguez-Martín 1998:99). In clinical studies, females are three times more likely to suffer from the disorder. Inflammation of the cartilage destroys the joint - leaving lytic lesions along the margins and eventually on the articular surface. The lesions are smooth, with rounded edges and trabeculae (Aufderheide and Rodríguez-Martín 1998: 101) and usually associated with osteoporosis. Lesions form bilaterally and the involvement of the TMJ occurs late in the disorder (Aufderheide and Rodríguez-Martín 1998:99). There is little or no evidence of new bone formation in response to

inflammation. Diagnosis of rheumatoid arthritis on the TMJ alone is very difficult. Osteoarthritis and middle ear disease both produce lytic lesions. However, it should be noted that while both disorders are associated with sclerotic bone formation, only osteoarthritis presents diagnostic eburnation.

2.9. Miscellaneous Conditions

Fibrous Dysplasia

Fibrous dysplasia is a disorder of unknown etiology that predilects individuals under 30 years (Aufderheide and Rodríguez-Martín 1998:420; Barnes and Peel 1990:132; Olmsted 1981:703). There are two forms, monostotic (single bone) that involves the skull in 10% of cases and has an equal sex predilection, and polystotic (multiple bones), which involves the skull upwards of 40% and is most common in females (Barnes and Peel 1990:132). Fibrous dysplasia characterized by a mixed osteoclastic and osteoblastic activity most commonly involving the craniofacial bones and mandible; it is usually localized/solitary and unilateral in expression. The periosteal surface of the cortex is usually thinned and smooth, while the endosteal surface is often rugged with fibro-osseous tissue that expands into the cancellous bone with varying degrees of ossification (Olmsted 1981:704; Wood 1994:300). The lesion may be lytic with a cyst-like appearance, or dense with round masses of irregular, proliferative woven bone and sharply demarcated, sclerotic margins (Aufderheide and Rodríguez-Martín 1998: 420; Barnes and Peel 1990:134; Ortner and Putschar 1984:317; Rothschild and Martin 1993:170; Zimmerman and Kelley 1982:132). The radiographic image of fibrous dysplasia projects a "ground glass quality" representative of the fine trabecular pattern (Gregg and Reed 1980:593; Wood 1994: 300). Hyperparathyroidism, Paget's disease, eosinophilic granuloma and ossifying fibroma should be differentiated from fibrous dysplasia (Ortner and Putschar 1984).

2.10. Human and Cultural modification

Trephination

One of the earliest forms of medical intervention in living patients, trephination is defined as the surgical removal of a portion of the cranium without damaging the underlying meninges and brain (Lisowski 1967:651). The term trepanantion derives from the Greek *trypanon*, which means "borer" (Bennike 1985:65). While written documentation for early surgical techniques extends back to the ancient Egyptian and Classical Greek literature (Jennbert 1991; Bakay 1985; Horrax 1952), the archaeological record provides evidence that trephination was performed at least 5000 years ago (Lisowski 1967), and possibly as early as 7000 or 8000 thousand years ago (Alt et al. 1997; Lillie 1998). In Europe, the surgery appears to reach its zenith in the Neolithic period, becoming more widespread but less frequent after that (Brothwell 1994; Jennbert 1991). Trephination is found throughout much of the ancient world including, Africa, Australia, the Americas, the Middle East and Europe (Campillo 1984; Lisowski 1967). There is considerable debate surrounding the origins and reasons for trephination (Browthwell 1994; Campillo 1984; Jennbert 1991; Lisowski 1967; Margetts 1967; Rifkinson-Mann 1988). While most researchers support a therapeutic explanation (Allison and Pezzia 1976; Barnes and Ortner 1997; Mann 1991; Parker et al. 1986), others feel that it was preformed for a combination of social, medical and ritual purposes

(Campillo 1984; Jennbert 1991). This will be considered further in Chapter 6.

The most common site is the left parietal, although trephinations are also common to the right parietal and frontal. Trephinations are also found on the temporal and the occipital although there are no examples of the procedure on the skull base (Kaufmann 1997; Campillo 1984; Lisowski 1967). While the literature suggests that trephination's are rarely located on the sutures or above the venous sinuses (Novak and Knüsel 1997), ample evidence exists for the procedure at bregma and along the sagittal suture (Jennbert 1991; Mogle and Zias 1995; Parker et al. 1986; Stone and Miles 1990; Bennike 1985; Germanà and Fornaciari 1992).

The lesions are usually singular, although there are many examples of individuals with multiple trephinations (Zias and Pomeranz 1992; Oakely 1959). Cut marks, suggestive of scalp reflection, and evidence of the method used – abrasive scratches, grooves, boreholes and incisive marks are usually considered diagnostic (Aufderheide and Rodríguez-Martín 1998; Ortner and Putschar 1984; Lisowski 1967). Further criteria used to identify trephination, include pronounced margins that usually feature an external bevel that differs from the internally beveled margin of blunt force trauma (Berrymann and Haus 1996; Frayer 1997; Lisowki 1967; Mallegni and Bertodi 1997; Novak and Knüsel 1997). Perimortem trephination (unhealed) will not have time to modify the evidence of the procedure or bevel. In contrast, healed[8] trephinations exhibit closed diploë, smooth borders and the formation of bony clusters or exostoses around the margins (Rifikison-Mann 1988:411) The formation of a platform or step of woven bone at the center of a partial trephination are occasionally observed (Campillo 1991; Germanà and Fornaciari 1992). Often there is a well-defined zone of inflammatory/ reactive bone (osteitis) that frequently advances beyond the original margin (Brothwell 1994; Stewart 1956). Infection is very common corollary of trephination.

Further criteria for trephination relates primarily to the operative procedure. It is generally perceived that there are four methods used to remove cranial bone and each presents characteristics that are indicative of the technique employed. The first method involves the scraping or abrading of the skull surface with a stone implement resulting in lesions that are usually ellipsoid with wide, slightly raised and externally beveled margins (Brothwell 1994: 133; Campillo 1984:277; Lisowski 1967:662; Smith 1990:90). The second method involves grooving the skull with a sharp implement and leaving a circular or polygonal lesion. The grooves are drawn and redrawn until the bone can be pried away. A relatively steep externally beveled margin is common (Lisowski 1967: 663). A third method involves drilling a series of small holes into the skull and using a sharp implement to adjoin the perforations and extract the roundel of bone. These lesions are usually conical dimension and circular in shape with serrated edges. Finally, bone can be removed by using a trepan instrument to remove a circular disc of the cranium (Brothwell 1994:134; Campillo 1984: 277; Lisowski 1967:662-664). While scraping appears to be the most commonly used method, many palaeopathological crania exhibit a combination of these methods (Brothwell 1994; Lisowski 1967; McKinley 1992; Piggot 1940; Zias and Pomeranz 1992). Perimortem or unhealed trephinations should exhibit evidence of the procedure, while healed lesions may be ambiguous.

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[8] The post-operative healing rate is very high as evidenced by the volumes of reports and articles that depict healed trephinations. In one study of 214 Preuvian skulls that showed evidence of trephination, more than 70% have evidence of healing (Rifkinson-Mann 1988: 411; Malins and Rathburn 1985).

Depending on the various morphological features of the lesion, the differential diagnosis should consider: parietal fenestrae; cranial dysraphism, blunt force trauma, neoplasia (osteolytic carcinoma and multiple myeloma), specific infections (syphilis and tuberculosis) and non-specific infections, parietal osteoporosis/osteopenia, postmortem damage (Aufderheide and Rodríguez-Martín 1998; Ortner and Putschar 1984). Palaeopathological precedents within the population or region provide supporting evidence, and it is suggested by Steinbock, (1976:35) that if only one cranium in a large population exhibits evidence of trephination, the diagnosis should be considered tentative. Identified trephinations should be classified as either: complete or partial; antemortem or postmortem; and healed and unhealed (Campillo 1984; Lisowski 1967; Rifkinson-Mann 1988). The cranium should also be carefully examined for pre-existing lesions (e.g. cranial trauma) that may relate to the cause of the trephination (Richards 1995).

Scalping

Scalping has a long history, reaching going back to the works of Herodotus in the fifth century B.C (Merbs 1989:177). While evidence for scalping exists universally, most palaeopathological examples are derived from post-contact North America (Bridges 1996; Bueschgen and Case 1996; Allen et. al. 1985; Ortner and Putschar 1984; Gregg et. al. 1981;Snow 1942). While scalping can be accidental, evidence from prehistory suggests that it is strongly associated with interpersonal violence (Bueschgen and Case 1996; During and Nilsson 1991; Hollimon and Owsely 1994; Miller 1994).

In scalping, a sharp instrument is used to remove a portion of the periosteum and covering skin (scalp) from the underlying bone usually in the area of the forehead (Roberts and Manchester 1995). The sharp instrument leaves short, straight or curved cut marks that may either encircle the skull or cluster on the frontal and parietals. The marks are thin and usually parallel (Bridges 1996; Bueschgen and Case 1996; During and Nilsson 1991). If the scalping occurred at, or near death, or in the postmortem interval, evidence would be restricted to cutmarks alone. It must be noted that it is possible to scalp an individual and leave no marks whatsoever (Miller 1994; Willey 1990)

Depending on the severity, individuals who survived the scalping would exhibit a range of osseous reactions from bony inflammation due to primary infection to complete bony necrosis and sloughing of the outer table. While bone is infrequently removed along with the excised scalp, the deprivation of blood via the periosteum results in extensive osteoclastic resorption and osseous death (Steinbock 1976:26). While the borders of the scalped area are usually irregular and exhibit diffuse bony reaction, occasionally a well-marked osteoclastic resorptive ring will form on the periphery of the scalped region (Hamperl 1967; Hollimon and Owsely 1994). Granular new bone forms on the cortex (osteitis) in response to the necrosis and infection. Increased vascularity may be visible on either the outer or inner tables (Miller, 1994). If the individual survives, the area will become remodeled and characterized by a smooth if uneven depression (Hamperl 1967: 632; Hollimon and Owsely 1994:350-351; Steinbock 1976:27; Roberts and Manchester 1995:85).

According to Bueschgen and Case (1996:232), there is a range of variation in the size of the scalp taken and method used by Native American groups that likely reflects group preferences and cultural differences. An undisputed archaeological context and

regional palaeopathological precedents provide essential supporting evidence (Buseschgen and Case 1996). Cutmarks require distinction from those derived via sharp force trauma, postmortem defleshing, animal tooth marks and excavation and curation damage (Hurlbut 2000; Micozzi 1991; Owsley et al. 1994). Scalping should be differentiated from other non-specific infections of the periosteum, tertiary syphilis, trephination (Miller 1994).

APPENDIX 3

OSTEOBIOGRAPHY

Case #1

While the left mastoid process and the glabella present ambiguous sex characteristics, the nuchal crest on the occipital bone and right supraorbital process of the frontal bone suggest that this individual is a probable male (Buikstra and Ubelaker 1994:20). The broad cranial vault distinguished temporal line, and rugged occipital also supports this assessment (Bass 1987;Ubelaker 1989). Ectocranial suture closure of the external vault sites provides an interdecile age range of 19-44 years of age. The mean age according to Buikstra and Ubelaker (1994: 38) is 31.5 placing this individual in the 'Young Adult' age category. Age estimation from the more accurate Lateral-Anterior sites could not be established because the inferior and superior sphenotemporal and the sphenofrontal suture sites were not available for observation. Minimal closure on the endocranial surface, unfused/disarticulated frontal bone and prominent parietal bosses also suggest a youthful age (Ubelaker 1989).

Case #2

The rugged nuchal area of the occipital bone and a thick supraorbital margin on the frontal bone suggests that this case is a male; however, other than a well-defined temporal line on the parietal bones there are no other features diagnostic of sex available for determination. With only two reliable features available, sex cannot be positively determined therefore the individual is classified as a probable male (Buikstra and Ubelaker 1994:20). Case #2 exhibits exceptional sutural complexity (particularly the lambdoidal) and varying degrees of closure that may have an impact on the age assessment (White1996). Ectocranial suture closure of the external vault sites provides an interdecile age range of 28-44 years of age. The mean age according to Buikstra and Ubelaker (1994:38) is 36, placing this individual in the 'Middle Adult' age category. Age estimation from the more accurate Lateral-Anterior sites could not be established because the inferior and superior sphenotemporal and the sphenofrontal suture sites were not available for observation. The endocranial aspect of the sagittal suture demonstrates significant closure, while the left lambdoid and left coronal demonstrate minimal closure. According to Krogman and Iscan, (1986) and Buikstra and Ubelaker (1994:36), "advanced but incomplete" suture closure is usually indicative of middle adults.

Case # 3

Due to the fragmentary nature of the remains, there are no identifiable criteria for sex determination. Only three ectocranial suture sites are observable for age estimation: the midlambdoid and lambda demonstrated significant closure while obelion was completely closed. On the endocranial surface, the left lambdoid suture was completely closed. Given the morphology and the evidence of extensive suture closure at the observable sites, it is likely that the remains belonged to an adult at death. A more precise estimation is not permitted due to the lack of suitable aging sites (Buikstra and Ubelaker 1994).

Case #4

While the left mastoid process presented ambiguous characteristics and the nuchal crest was scored as probable male, the thick supraorbital processes and the prominent glabella on the frontal bone were identified as male (Buikstra and Ubelaker 1994:20). Ectocranial suture closure of the external vault sites provides an interdecile age range of 23-45 years of age. The mean age according to Buikstra and Ubelaker (1994:38) is 34 years, placing this individual in the 'Young Adult' category. A missing pterion did not effect the estimation of age. The exterior midcoronal and interior coronal sutures were observed as open, the inferior sphenotemporal presented minimal closure, while the interior sagittal demonstrated significant closure. All other sutures were recorded as unobservable.

Case #5

While the nuchal crest was scored as ambiguous, the glabella, left and right mastoid processes and the left and right supraorbital processes were scored as female (Buikstra and Ubelaker 1994:20). Assessment of the cranium as female is also supported by the small, very gracile, and feminine looking nature of the skull (Bass 1987:81). Both the vault and the lateral anterior sites were available for ectocranial age estimation. All of the cranial sutures of the external cranial vault were recorded as open with the exception of the midcoronal, sphenofrontal and inferior sphenotemporal sutures, which demonstrated minimal closure. The external vault sites provided an interdecile age range of 19-44 years with an average of 31.5 years. The lateral-anterior suture sites provided an interdecile age range of 27-51 years with an average of 39 years (Buikstra and Ubelaker 1994: 38). Because the lateral-anterior sites are considered to be more reliable indicator of chronological age than the external vault sites, Case #5 is classified as a "Middle Adult" (Buikstra and Ubelaker 1994: 36; Meindl and Lovejoy 1985:62). The sutures of the internal cranial vault demonstrated minimal closure. The palatine sutures were unobservable.

Case # 6

The nuchal crest, left supraorbital process and the glabella were scored as probable female while the left mastoid process was scored as female (Buikstra and Ubelaker 1994: 20). The lack of other features infuses a degree of uncertainty regarding the sex of this cranium; therefore, probable female is the most reasonable assessment. Age estimation could not be derived from ectocranial suture closure because bregma, pterion, sphenofrontal and sphenotemporal suture sites were unobservable. The observable sites display varying degrees of closure, from minimal at the midlambdoid, to significant at lambda and the anterior sagittal, and complete at obelion and midcoronal. The left temporal is disarticulated along the squamosal suture. The sutures of the interior cranium are not observable. The cranium was placed in the 'Adult' age category.

Case # 7

The nuchal crest, left and right supraorbital processes and the glabella were scored as female according to the standards derived from Buikstra and Ubelaker (1994:20). While the vault appears to exhibit minimal to no closure, the absence of obelion, and the sphenotemporal and sphenofrontal sutures and the presence of a lesion at midsagittal do

not facilitate a reliable age estimation (Buikstra and Ubelaker 1994:36). Therefore, Case #7 is classified as an 'Adult'.

Case # 8

The left mastoid process and right supraorbital process presented as ambiguous sex while the right mastoid process and the left supraorbital process presented as probable female. The nuchal crest and glabella were scored as female (Buikstra and Ubelaker 1994:20). While the gracile nature of the vault was suggestive of the female sex, the ambiguous and probable nature of most of the characteristics requires that the vault be designated 'probable female'. Ectocranial sutures exhibit advanced closure along all observable sutures. Lateral-anterior ectocranial age estimation provides an interdecile range of 49-65 (Meindl and Lovejoy 1985:63). The mean interdecile age is 55 years, however, crania with completely closed sutures are best considered "old adults" as age estimates are not entirely reliable (Buikstra and Ubelaker 1994:36).

Case # 9

While the supraorbital margins are sharp, the rest of the skull presents male characteristics. The mastoid processes, glabella and the nuchal crest were scored as male or probable male. Square orbits, a large foramen magnum and a well-marked temporal also support the observation that the cranium is male (Buikstra and Ubelaker 1994: 20; Ubelaker 1989). Lateral-anterior ectocranial age estimation provides an interdecile range of 49-65 (Meindl and Lovejoy 1985:63). Complete ectocranial suture closure along of the lateral anterior sites suggests that this individual belongs in the "Old Adult' category Buikstra and Ubelaker (1994:36). Given the advanced age of this individual, a more concise age estimate cannot be determined. Obliterated palatal sutures and significant tooth wear also support the observation that this individual is of advanced age.

Case # 10

Due to postmortem damage, only the nuchal crest could be scored as probable female, and the right mastoid process was scored as ambiguous, were available for sex determination. As a result, the sex of Case # 10 remains undetermined (Buikstra and Ubelaker 1994:20). Only four of the ectocranial vault sites were available for age estimation. On the endocranial surface, the sagittal and left lambdoid sites displayed minimal closure. While the partial calvarium exhibits only minimal closure at all available sites, there is not enough information to recommend more precise age estimation.

Case # 11

Sex cannot be determined for Case # 11. There are no available sites for sex determination and the vault is too fragmentary and weathered (Buikstra and Ubelaker 1994:20). Similarly, age could not be estimated using ectocranial suture closure due to the fragmentary and poorly preserved nature of the remains. The small portion of the sagittal that is observable suggests that the sites of bregma and midsagittal were open at death; however, no other sutural sites were scorable and given the large size of the vault, it is reasonable to assume that the individual was an adult at death.

Case # 12

There is no descriptive data for Case # 12 other than an 'adult' designation.

Case # 13

The only available site for sex determination is the nuchal crest, unfortunately it is unscorable due to postmortem and disease changes; therefore, sex is 'undetermined'. Ectocranial age estimation could not be utilized because most sutural sites were either damaged or not present. While midlambdoid, lambda, obelion and the anterior sagittal suture sites were available, they were scored as unobservable due to postmortem and pathological changes. On the internal sagittal and lambdoid sutures, complete closure was observed. No composite age estimates could be derived from the given data, therefore the individual represented in Case # 13 is classified as an 'adult'.

Case #14

While Case # 14 exhibits large mastoid processes that were scored as probable male, the nuchal crest, the right supraorbital process and the glabella were scored as probable female. While the skull is not as gracile or feminine in appearance as Case # 5, the lack of strong muscle markings in addition to the above observations supports the classification of Case # 14 as a probable female (Buikstra and Ubelaker 1994:20). All exterior vault sutures were scored as open except for the inferior sphenotemporal, which was scored as minimal closure. On the endocranial surface, the sagittal and left lambdoid were scored as minimal closure and the left coronal as open. The composite scoring method of vault sites described in Buikstra and Ubelaker (1994) and based upon Meindl and Lovejoy (1985) cannot be used for crania with completely open sutures. The interdecile age range for the lateral-anterior sites is 21 to 42 with an interdecile mean of 31.5 years. This individual falls into the "Young Adult" category.

Case #15

The nuchal crest and glabella present ambiguous sex characteristics, while the left and right mastoid and the left and right supraorbital processes presented features consistent with a designation of probable female (Buikstra and Ubelaker 1994:20). The vault is smooth and slightly gracile and lacks significant muscle markings. The cranium is a probable female. Observations of the ectocranial sutures revealed that the cranium exhibited significant closure at death. An age estimate could not be derived from the external vault sites due to the proximity of the lesion. An ectocranial age estimation derived from the lateral-anterior sites provides an interdecile age range of 40 and 60 + years with a mean of 52 years (Buikstra and Ubelaker 1994:38). Case # 15 is classified as an "Old Adult".

Case #16

The nuchal crest presents ambiguous sex characteristics while the right mastoid was considered a probable female (Buikstra and Ubelaker 1994:20). The forehead and vault have the gracile appearance of a female skull (Bass 1987). There are no other observable features for sex determination, therefore the skull is classified as 'probable female'. The midlambdoid, lambda, obelion, anterior sagittal, bregma, midcoronal and pterion suture sites all exhibit complete closure; however, they are also directly

associated with extensively remodeled bone and therefore cannot be used for age estimation. Age estimation for the lateral-anterior sites cannot be established because the sphenofrontal and sphenotemporal sites are not observable. The interior cranial vault sites were scored as completely closed (Buikstra and Ubelaker 1994:36). While the vault appears to be of an advanced age, it is difficult to provide conclusive age estimation due to the abnormal condition of the bone, therefore the skull is classified as 'adult'.

Case #17

The cranium of Case #17 exhibits definite male characteristics. The nuchal crest was scored as probable male, while the left mastoid, the left and right supraorbital process and the glabella were scored as male. Strongly defined muscle attachments, particularly the temporal line and square orbits support the identification of Case # 17 as a male (Bass 1987). Most observable sites for age estimation were completely closed. The lateral-anterior vault sites provided an interdecile age range of 49-65 years with a mean interdecile age of 57 years (Buikstra and Ubelaker 1994:36; Meindl and Lovejoy 1985:63). All exterior vault sites were scored as completely closed except for bregma, which was scored as unobservable due to postmortem damage. All three interior vault sites were scored as completely closed and the maxillary molars were observed to exhibit significant wear. The above evidence suggests that the Case #17 should be designated as an 'Old Adult'.

Case #18

There are no features available for the determination of sex. Likewise, age is also inestimable given the incomplete nature of the remains and the damaged nature of the vault surface. The midlambdoid, lambda, anterior sagittal and midcoronal suture sites were obliterated and scored as completely closed. The interior cranial vault sutures were scored as unobservable due to abnormal bone loss and formation. While the individual appears to be of advanced age due to the degree of suture closure at the observable sites, it is possible that abnormal bone loss and formation may have enhanced the remodeling process.

Case #19

Sex cannot be determined for Case #19. There are no sites available for sex determination and the vault is too fragmentary to provide a subjective assessment (Buikstra and Ubelaker 1994:20). The anterior sagittal displays significant closure, on both the ectocranial and endocranial surfaces suggesting the individual was of adcanced age: however, given the incomplete nature of the vault, a reliable estimation cannot be provided. Case #19 is classified as an 'adult'.

Case A

The nuchal crest was scored as ambiguous sex, while all the other cranial sites were scored as female. The skull is gracile and does not exhibit extensive muscle attachments (Buikstra and Ubelaker 1994:20). The exterior vault suture sites of midlambdoid, lambda, and obelion demonstrated significant closure, while the anterior sagittal, midcoronal and pterion exhibited complete closure. Bregma was scored as unobservable due to the location of the lesion. The minimum age estimate that can be derived given the missing

site is 31-61 years with a mean age of 45 years. The lateral anterior sites of pterion and midcoronal were scored as completely closed, while the sphenofrontal was scored as significantly closed. The inferior and superior sphenotemporal were unobservable. Again, given the missing suture sites, the minimum age that can be calculated from the lateral-anterior sites is 35-55, with a mean age of 45 years. Interior vault sites were scored as completely closed, confirming that the individual was of advancing age. The significant degree of suture closure exhibited at all the sites suggests an individual of middle to advanced age. As a result, Case A is classified as a 'middle adult'.

Case B

The left and right supraorbital margins of the frontal were scored as ambiguous sex, while the nuchal crest, mastoid process, and glabella were all scored as male. Protruding supraorbital tori, square orbits, strong muscle attachment and a broad nasal aperture also support the observation that the skull is male (Buikstra and Ubelaker 1994:20). While the site at bregma was not observable due to the location of the lesion, ectocranial suture closure of the external vault sites provides a minimum interdecile age range of 31-61 years with an average of 45 years. The more accurate lateral anterior suture sites predominately exhibit significant closure. An interdecile age range of 40-60+ with an average of ~52 years suggests that Case B is an old adult. The complete obliteration of the interior vault sites further supports this estimation.

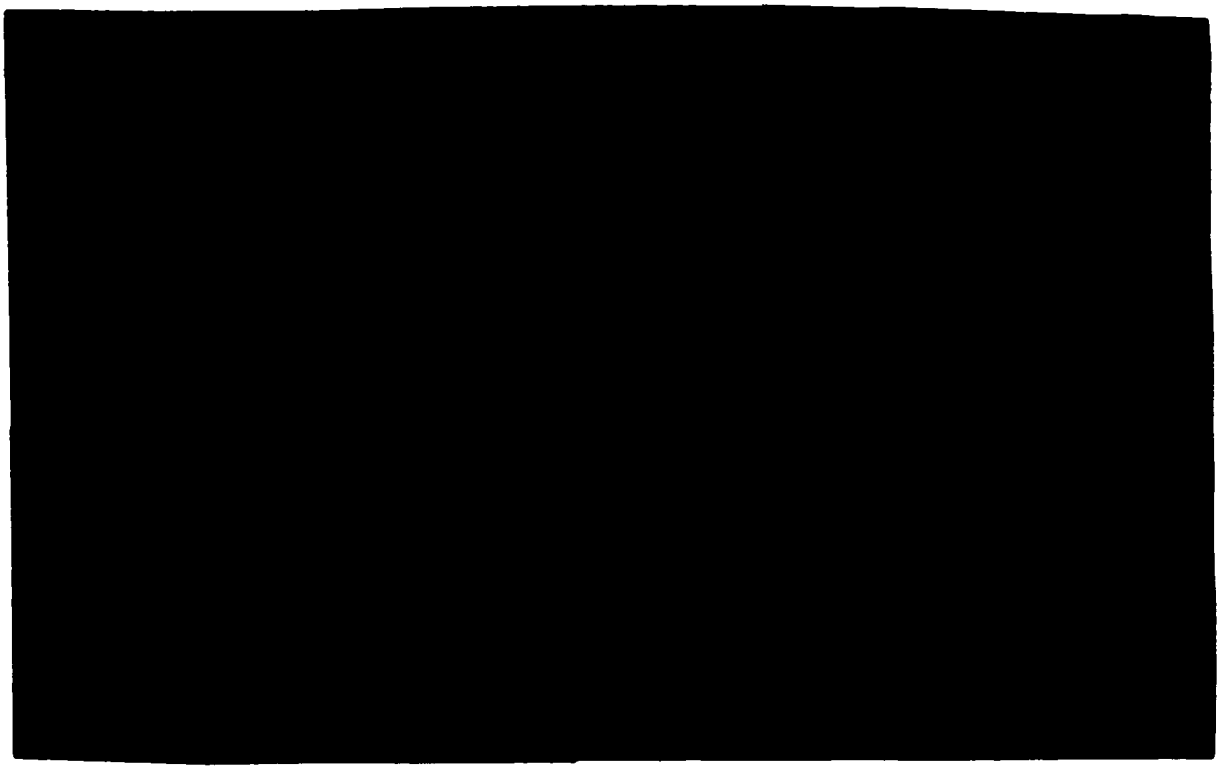

Plate 1. Eastern facing view of Algar do Bom Santo.

Plate 2. Superficial commingled deposits in Sala B. Polished anfibolite axe and a sherd of non-cardial pottery can be seen in the background.

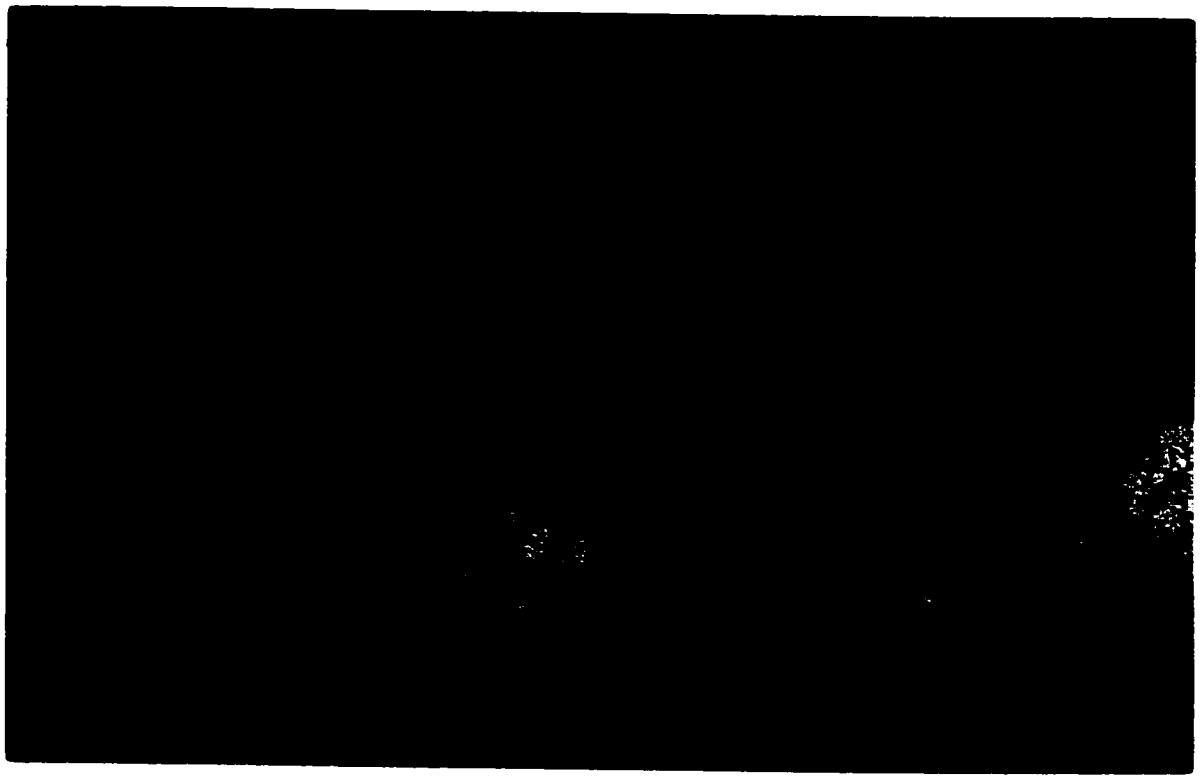

Plate 3. A nucleus of crania in Sala C. Four crania can be distinguished in this picture, including Case A in the foreground.

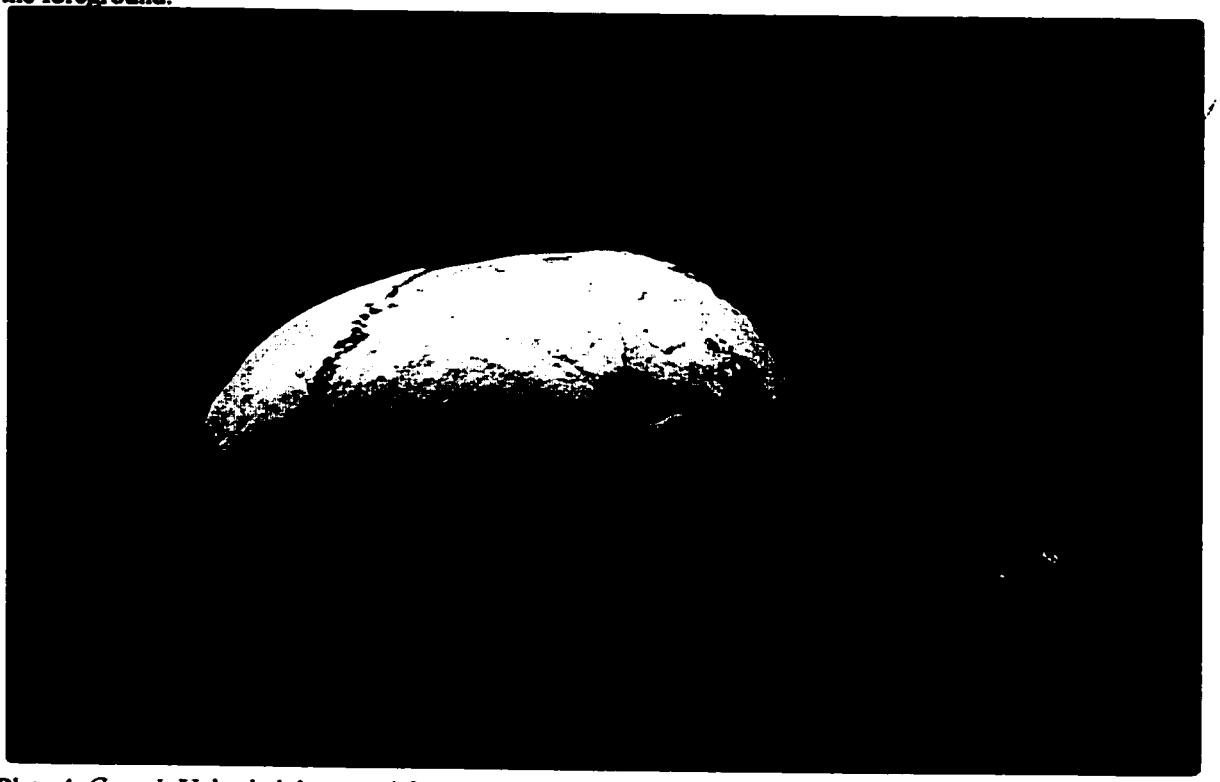

Plate 4. *Case 1*. Unhealed depressed fracture to the left parietal. Note radiating fractures lines and internal bevel and the portions of cranial table displaced into the inner cavity.

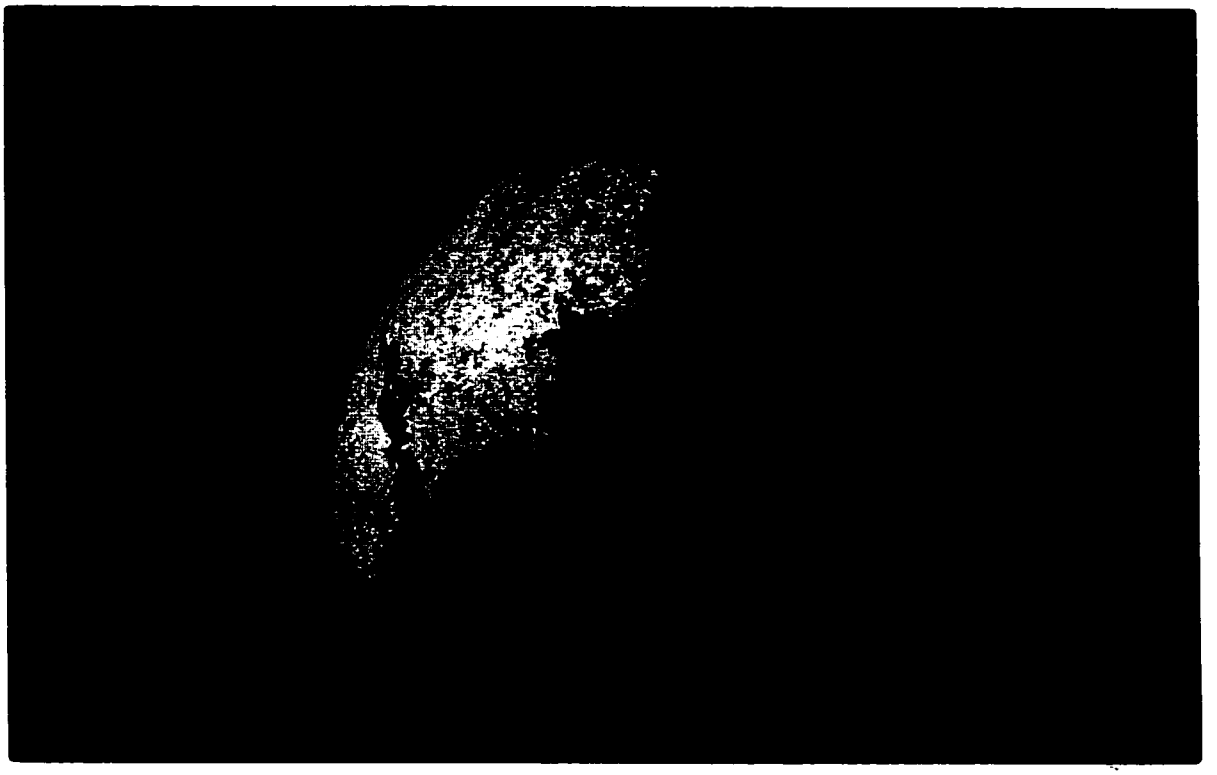

Plate 5. *Case 2*. Healed depressed fracture to the left parietal. Note that the inferior margin of the depressed region is circumscribed by a sharp circular groove.

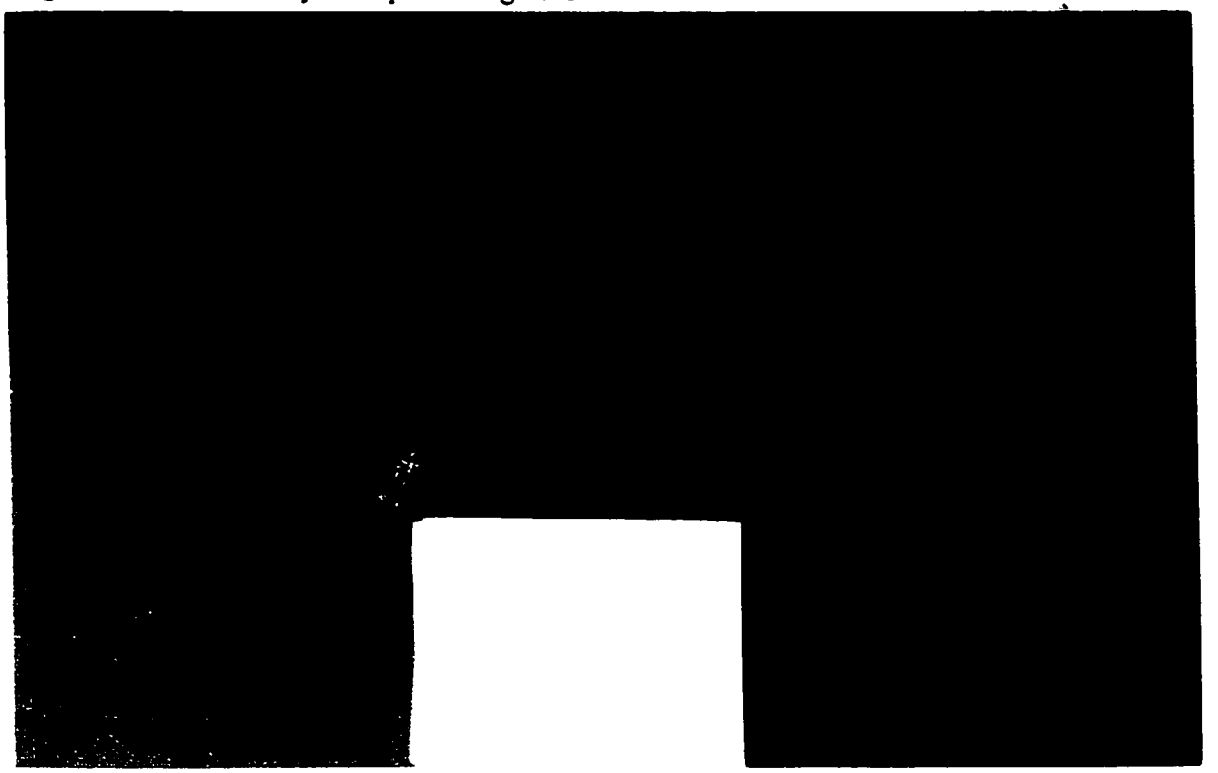

Plate 6. Case 3. Abnormal formation across the cross-section of the frontal bone.

Plate 7. *Case 3*. Close up of the left lateral section of the frontal. The outer and inner tables exhibit dramatic formation of cortical bone, while the inner table is almost obliterated. Abnormal formation may be related to a metabolic or endocrine disturbance

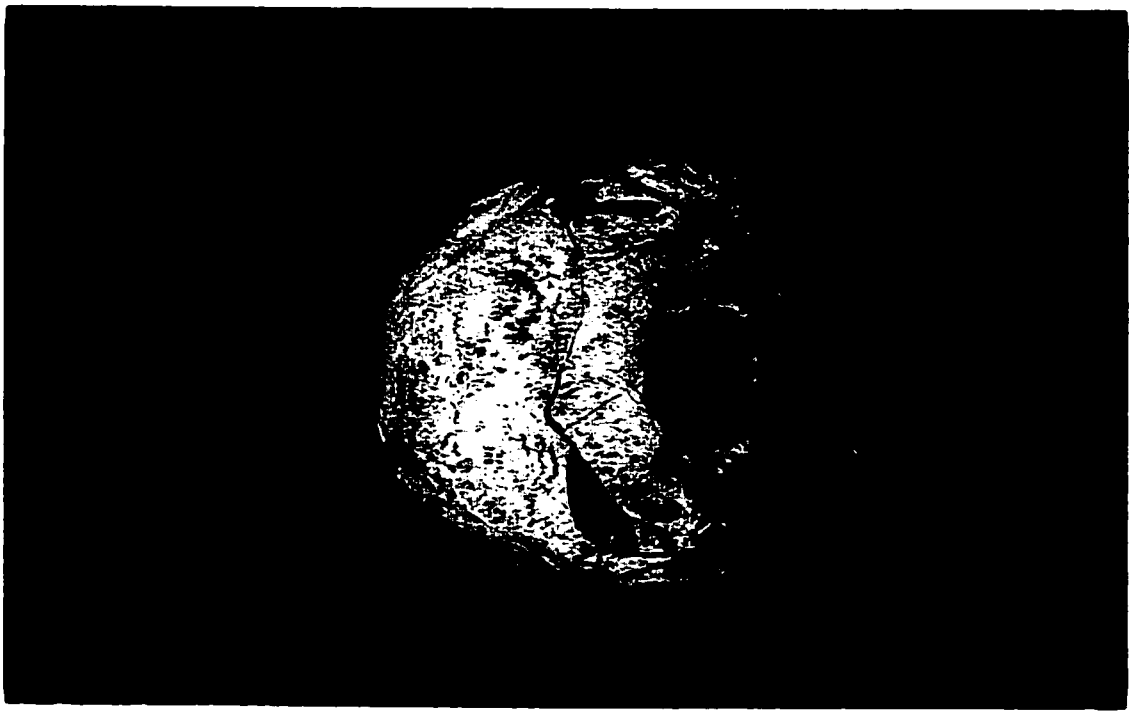

Plate 8. *Case 5*. Postmortem damage to an otherwise intact cranial base. This skull base represents one of the few in the sample.

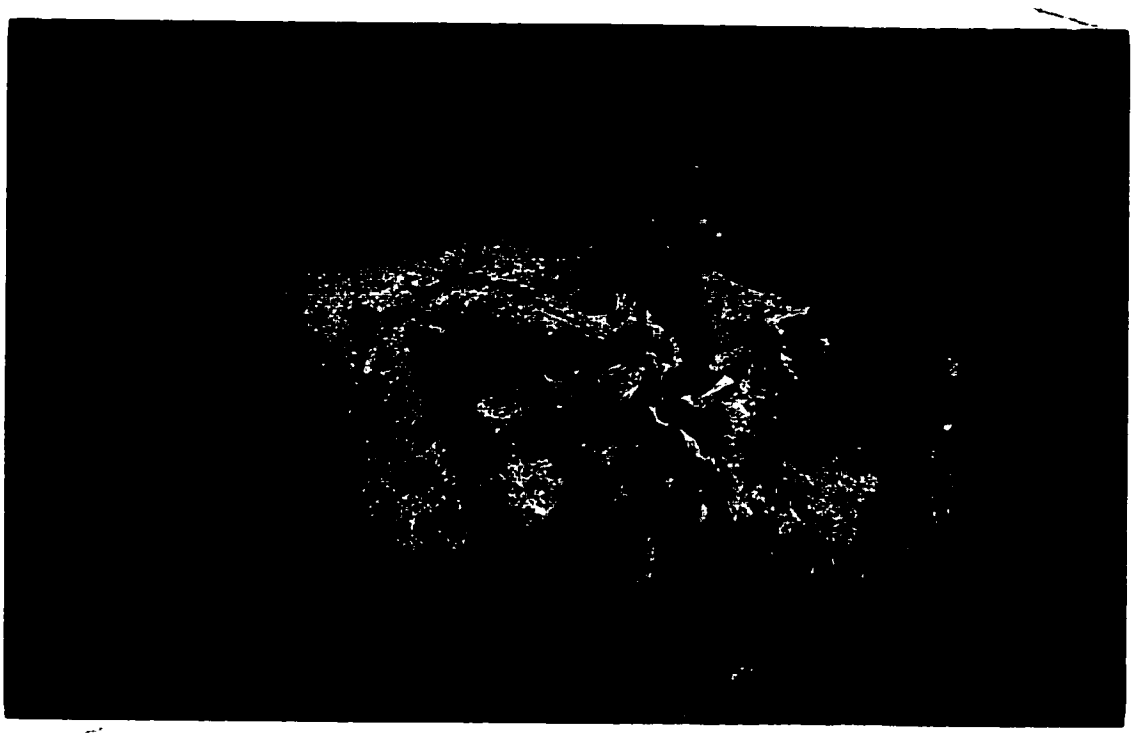

Plate 9. *Case 5*.Small, sharp-margined perforation on the left mandibular fossa. Note the calcite deposit on the anterior margin of the lesion. The origin of the perforation is unidentified.

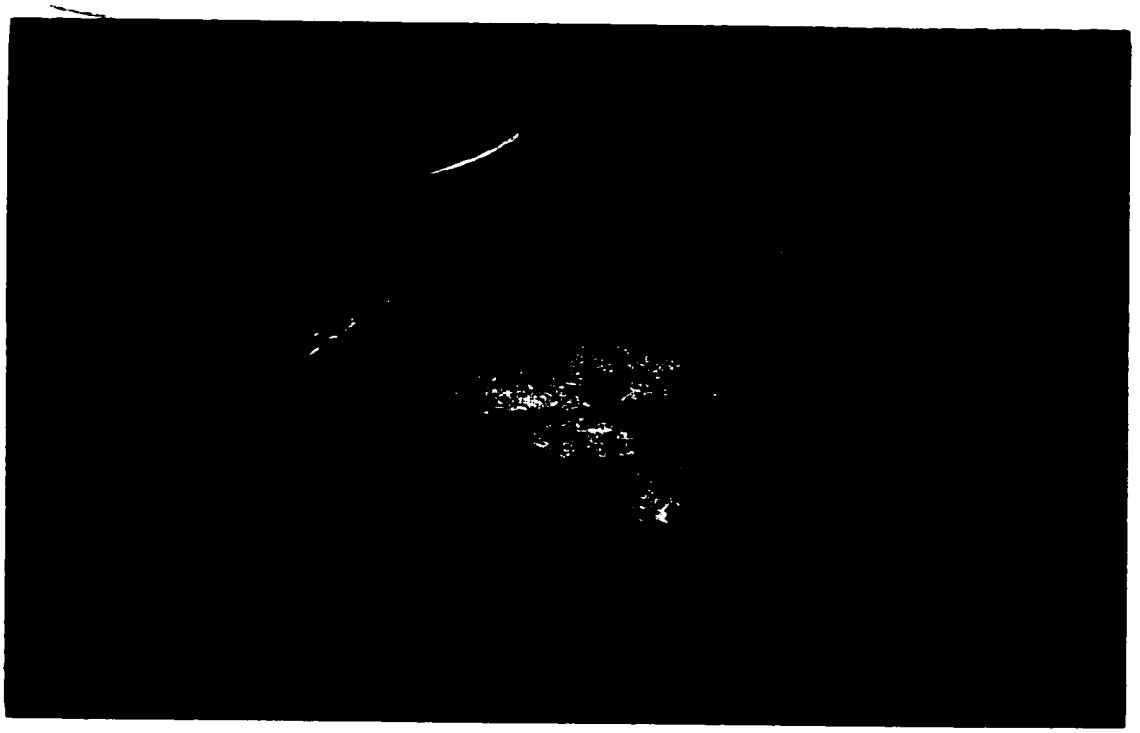

Plate 10. *Case 6*. Erosive lesions on the endocranial surface of the left petrous portion possibly the result of otitis media.

Plate 11. *Case 7.* Perforation of the cranial vault. The perforation is likely the result of a developmental abnormality. Note the rounded margins and the slight deviation of the sagittal suture to the right.

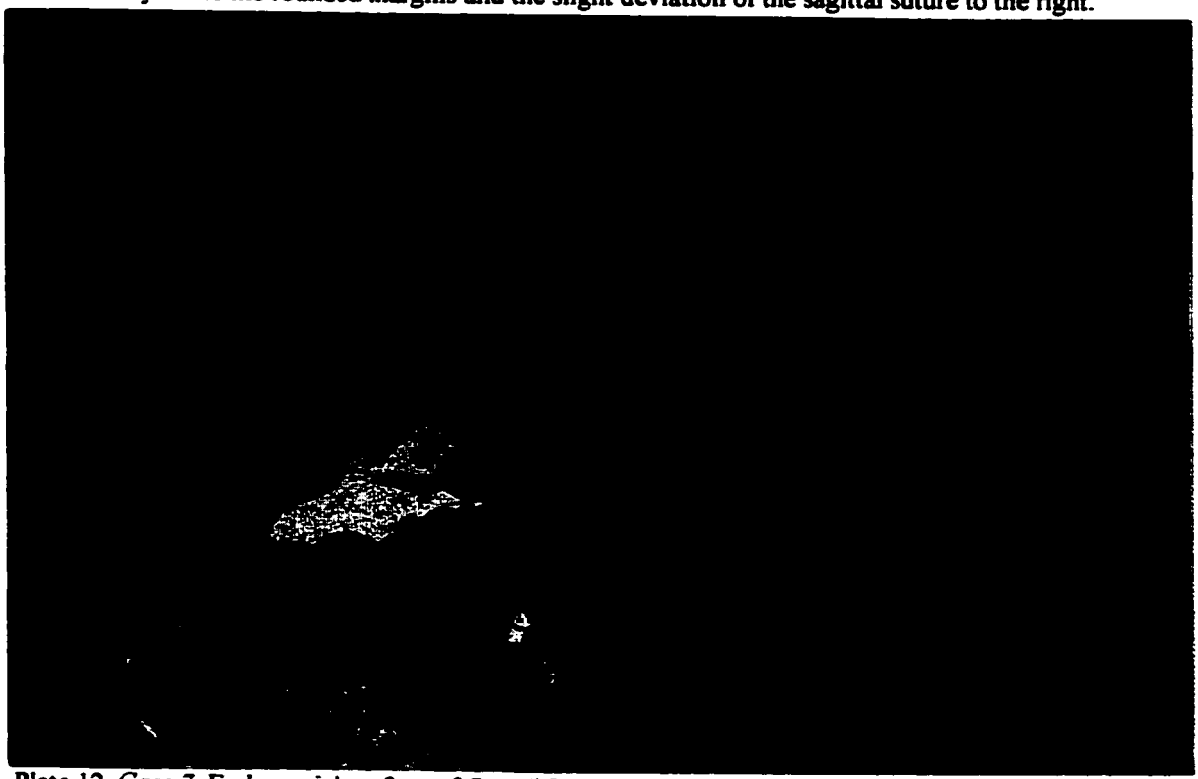

Plate 12. *Case 7.* Endocranial surface of Case # 7. Note the rounded margins and the internal bevel. The sagittal sulcus runs along the margin of the lesion and continues inferiorly, deviating slightly to the left.

Plate 13. *Case 7.* Mixed active and healed cribra orbitalia lesions. Cribra orbitalia was scored as 'medium'.

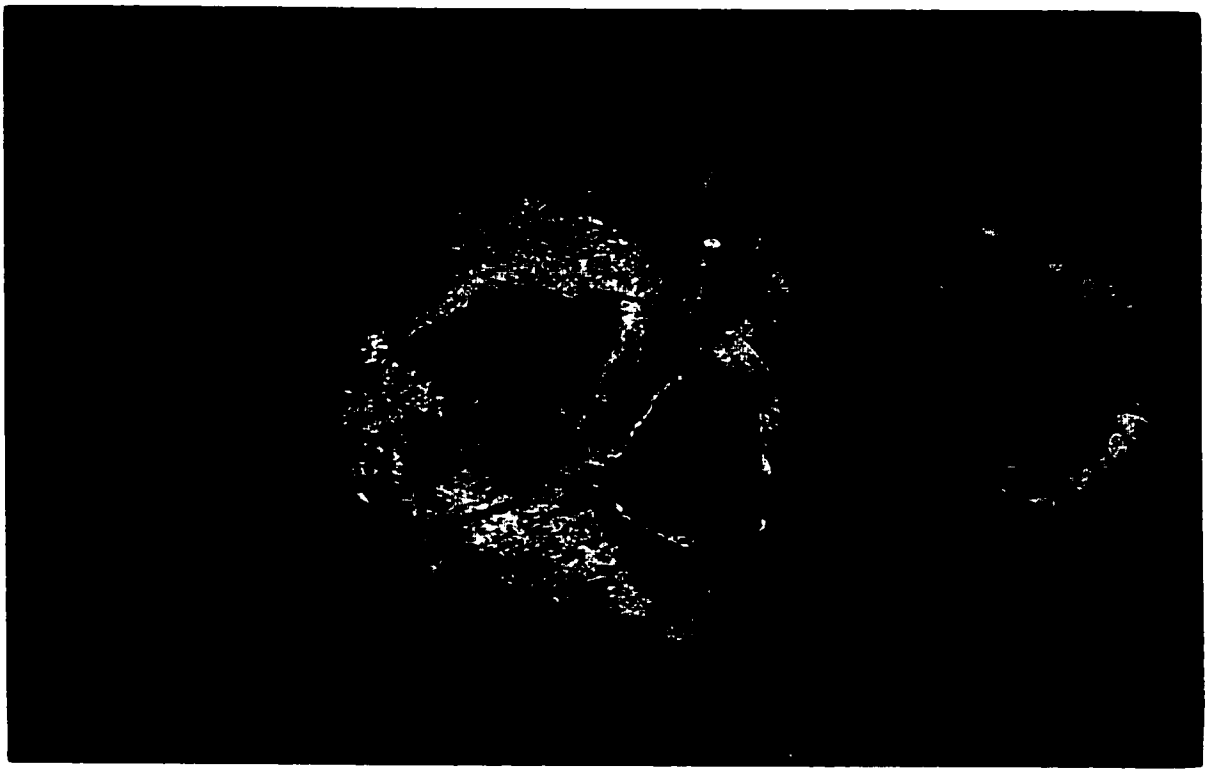

Plate 14.*Case 9.* Large postmortem "keyhole" fracture on the posterior aspect of the occipital. Note the linear fractures radiating from the superior and inferior aspects of the fracture.

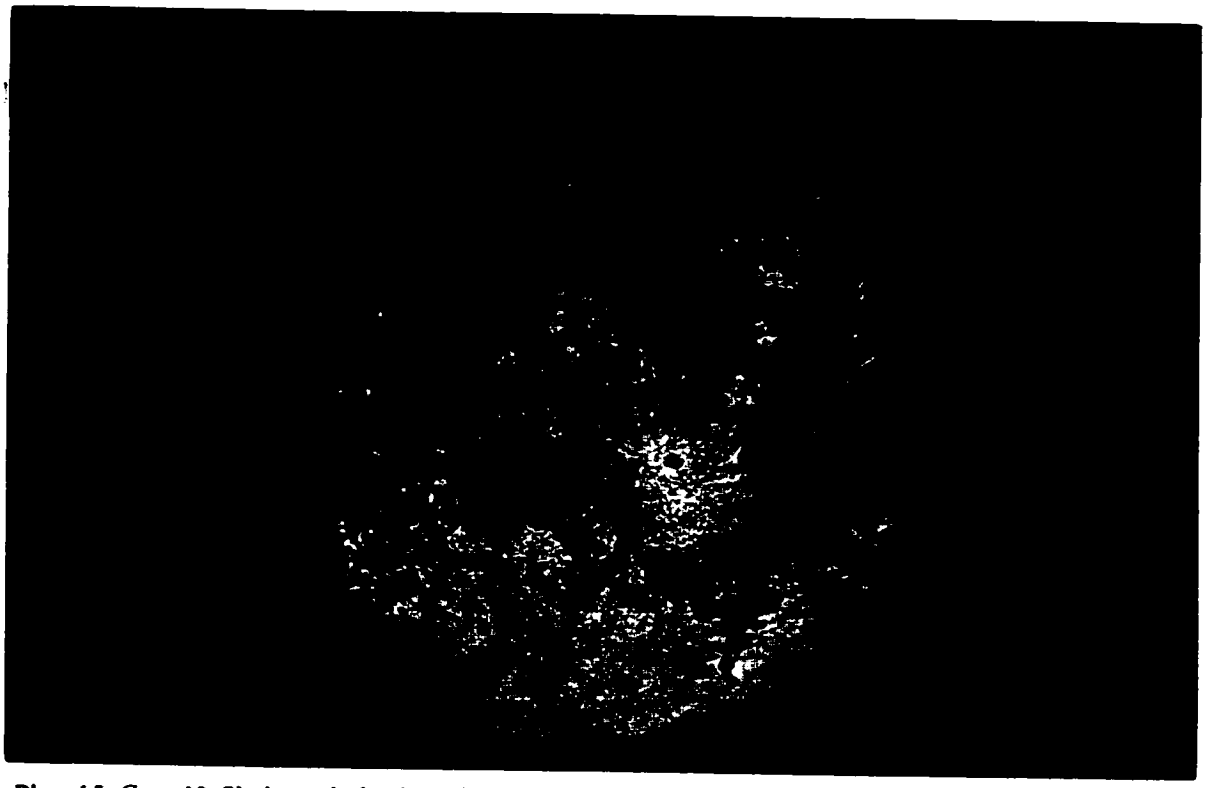

Plate 15. *Case 10.* Six irregularly shaped resorptive foci on the mastoid process of the right temporal bone that are possibly the result of mastoiditis. Note picture is a vertical.

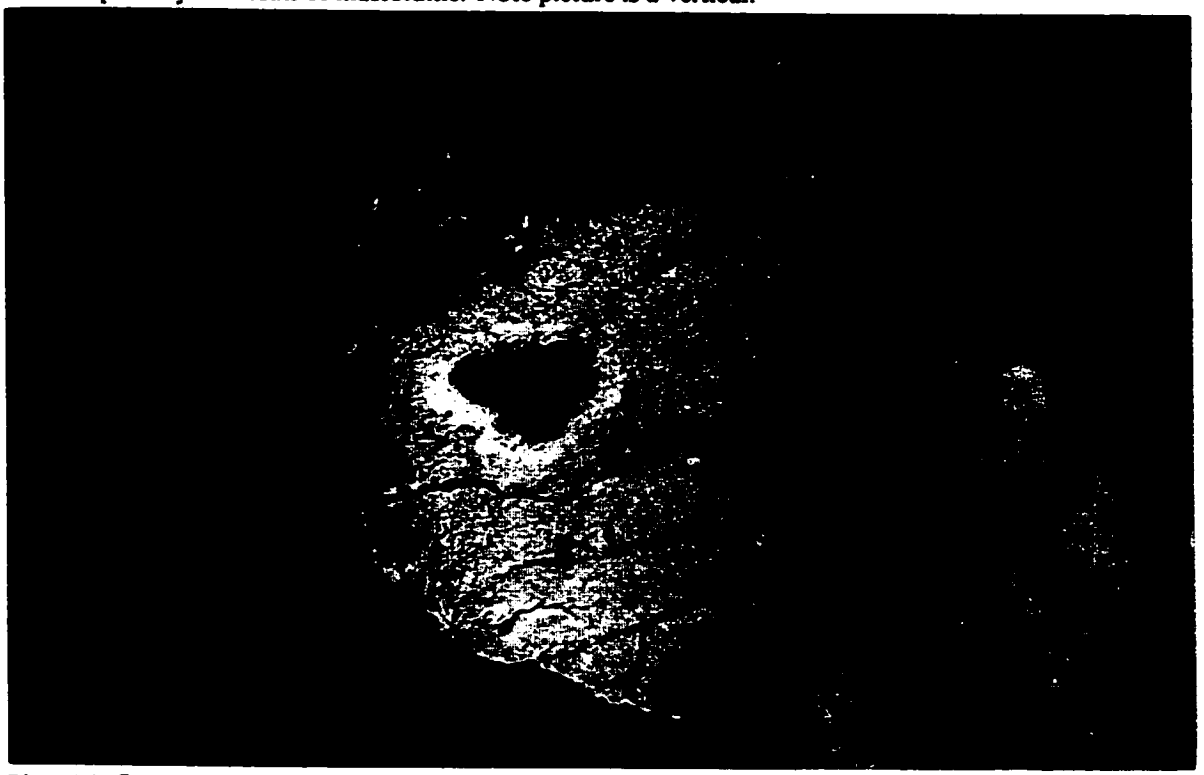

Plate 16. *Case 11.*Erosive lesion on the endocranial surface of the left parietal. The diploë have a "dissolving" appearance and a slightly brownish colour. The margin is white due to postmortem damage. Etiology of lesion is undetermined.

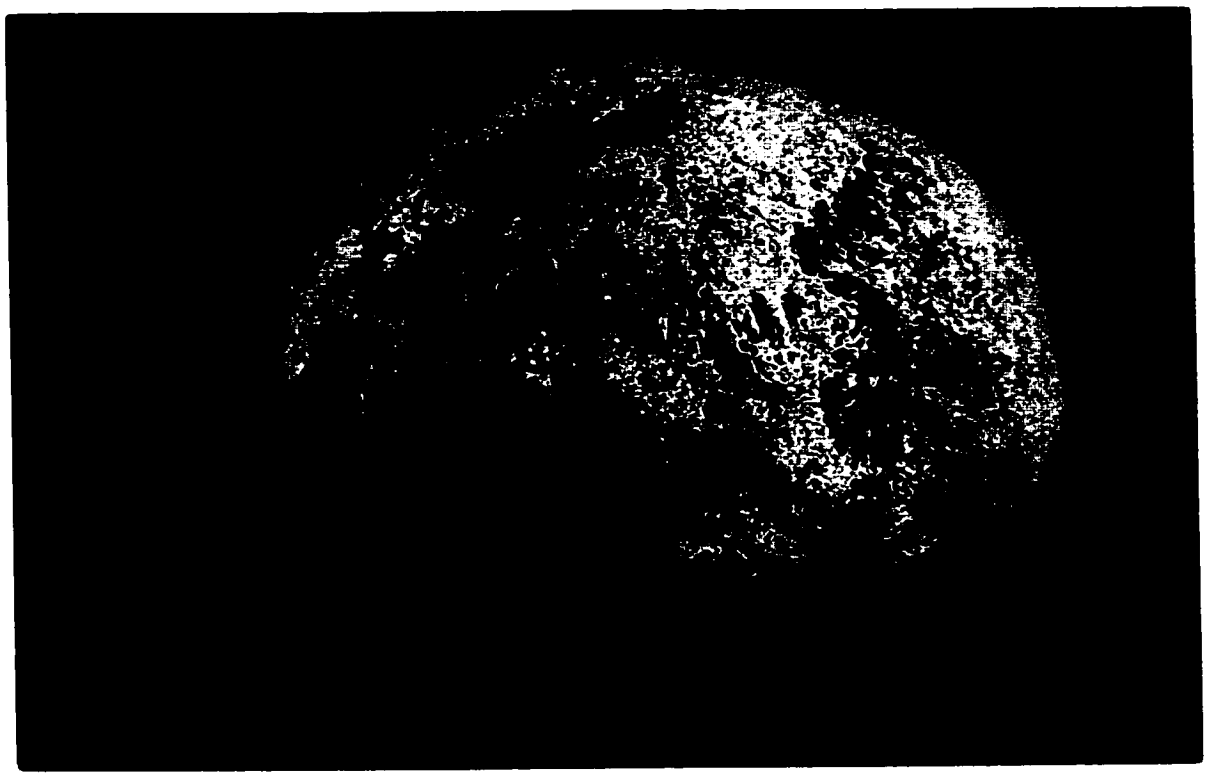

Plate 17. *Case 13.* The cranial vault exhibits diffuse porosity osteolytic resorption in interspersed with new woven and densely sclerotic bone formation.. Note: picture is a vertical. The lesion is classified as an inflammatory condition of undetermined origin.

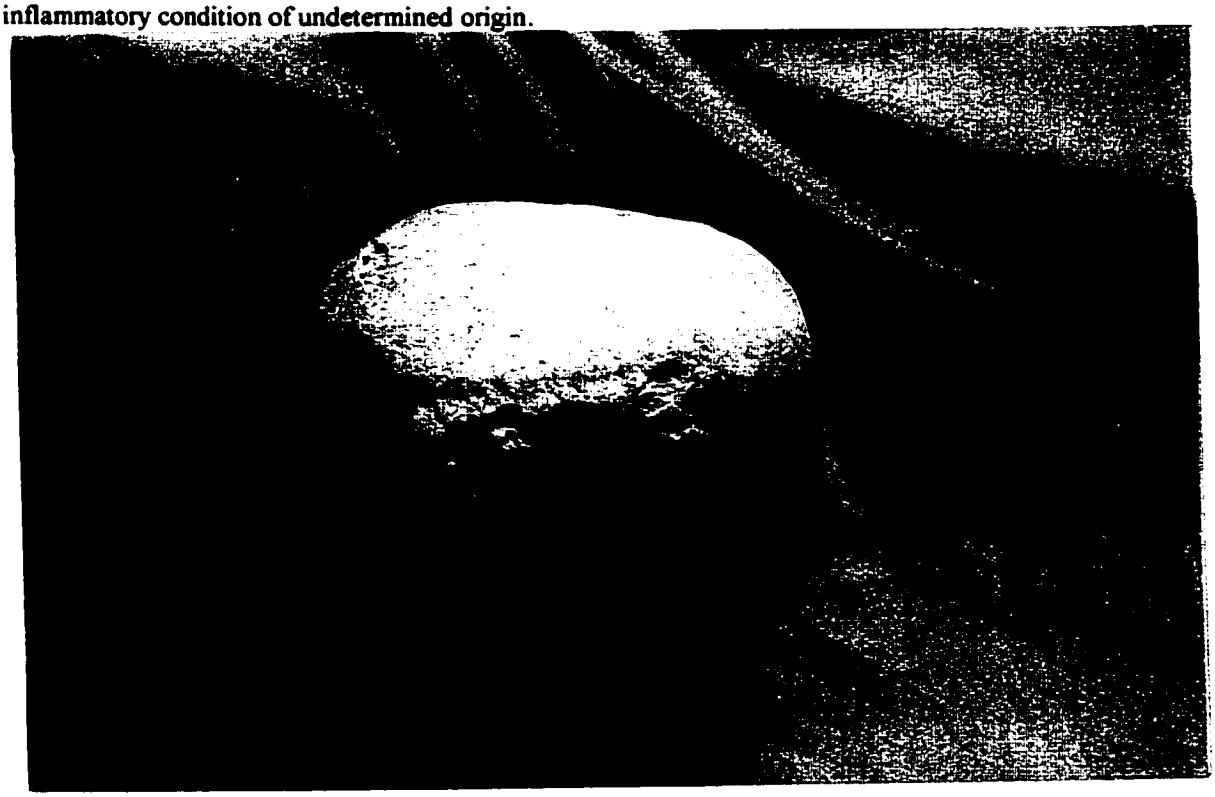

Plate 18. *Case 15.* Discretely circumscribed area of mixed abnormal bone loss and abnormal formation located along the sagittal suture. Note the circular depressions interspersed with plateaus of bone.

Plate 19.*Case 15.* Close up of the lesion. Note the unusual mixture of smoothly remodeled depressions; porosity and sclerotic bone. Also, note the small pits in the depressions along the anterior margin and the small exposure of diploë near the posterior margin. The lesion is classified as a non-specific infection.

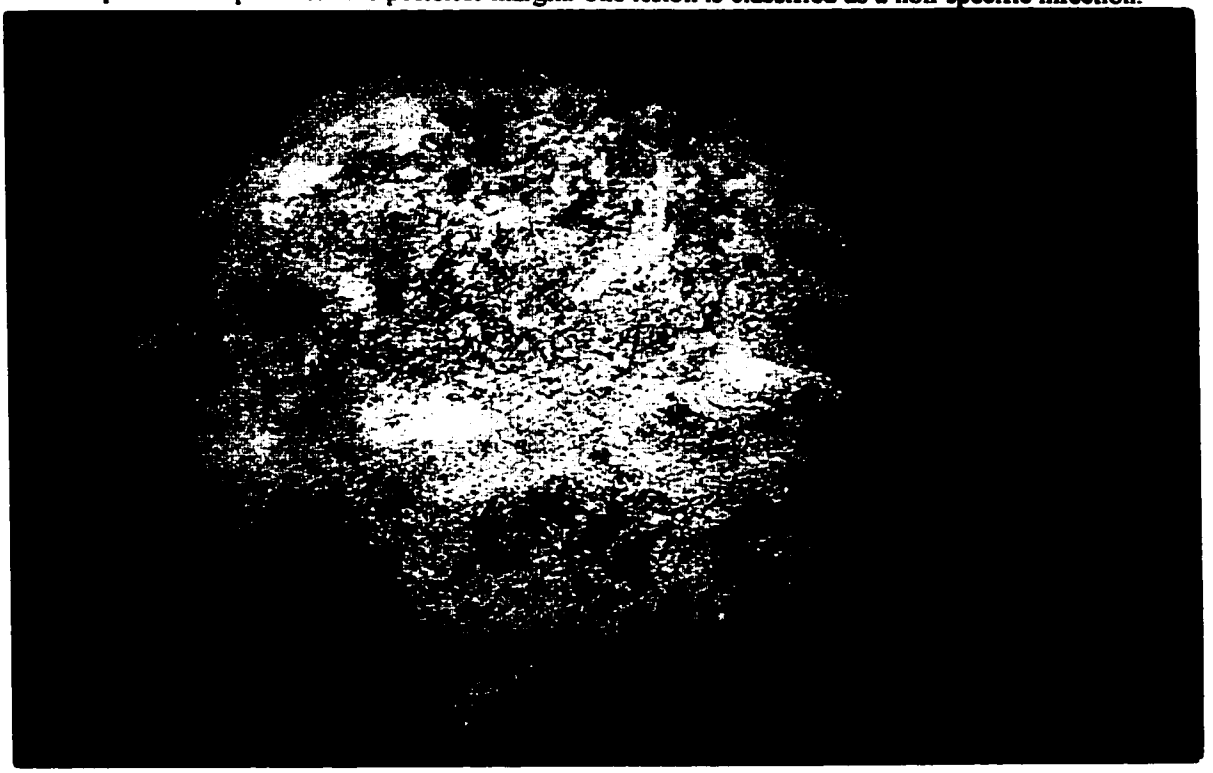

Plate 20. *Case 16.* Superior view of the skull. Note the fine pores interspersed with dense ivory like spicules of periosteal bone. On the anterior aspect of the frontal, a densely remodeled prominence marks the anterior margin of the lesion. Possible non-specific infection.

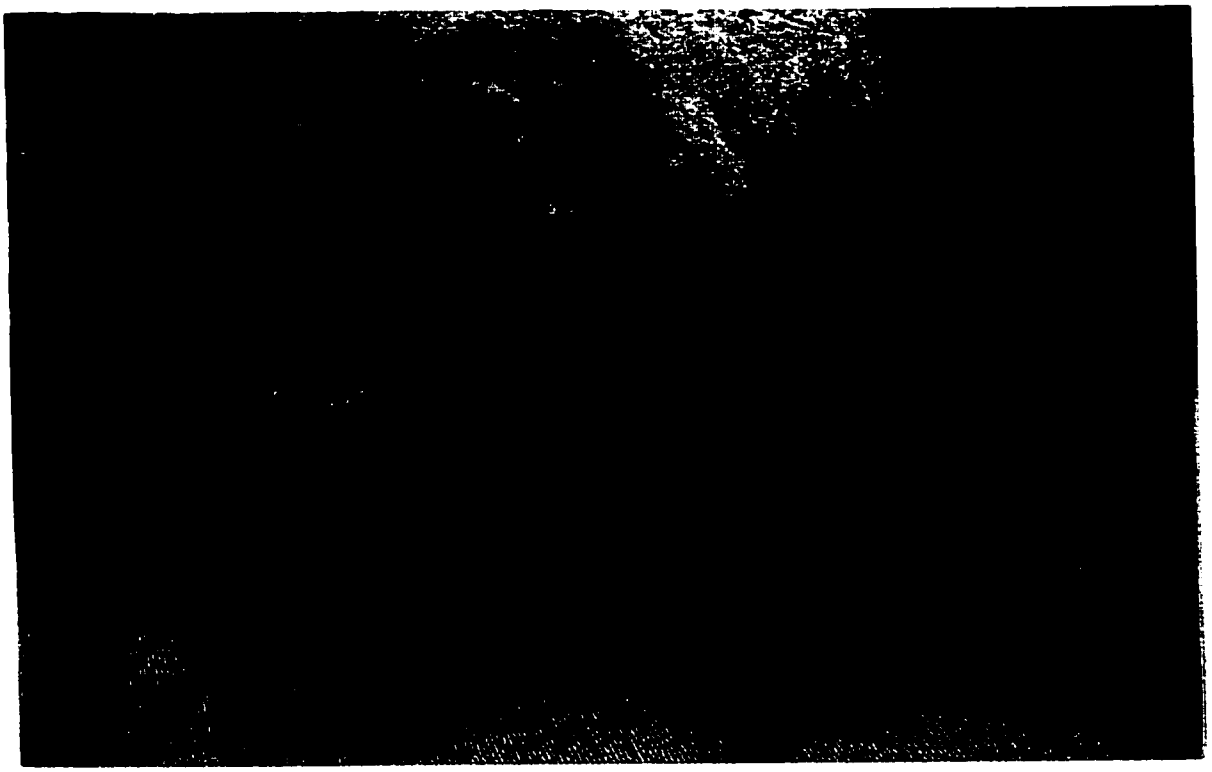

Plate 21. *Case 17.* Perforation in the left temporal- occipital region. The lesion looks "punched in" and exhibits a steep, remodeled external bevel. The zone around the lesion is palpably depressed and surrounded with reactive bone. The cause of the lesion is undetermined.

Plate 22. *Case 18.* Endocranial surface of the frontal and left parietal. The groove for the middle meningeal artery is unusually deep and associated with labyrinthine fissures and serpigenous tracks. Numerous deep, coalescing cavities and pits extend into the diploë. Possible non-specific infection.

Plate 23. *Case A*. Superior view of the cranial vault. The ovoid-shaped depression demonstrates a small perforation in the anterior corner of the lesion. Note the thick sclerotic margin with a wide external bevel. Also, note the platform of sclerotic and reactive woven bone in the centre. Possible trephination.

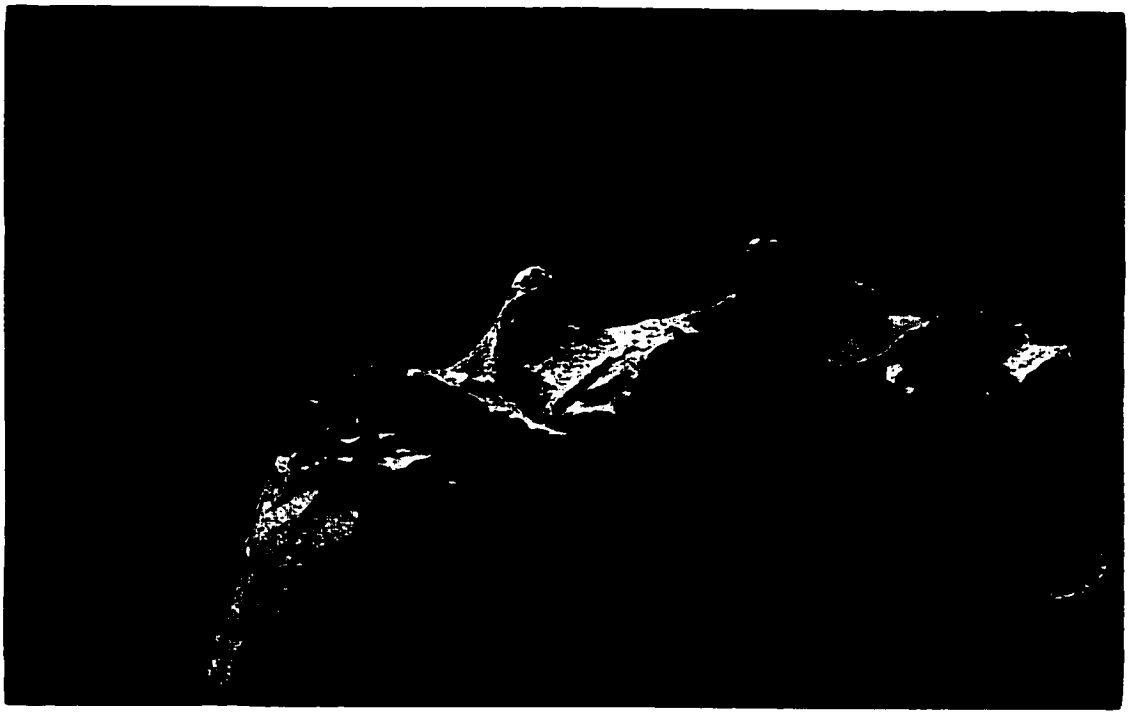

Plate 24. *Case A*. Articular surface of the right mandibular fossa. Errunation, grooving and coalescing porosity provide clear evidence of osteoarthritis.

Plate 25. *Case B*. Superior view of the cranial vault. The ovoid-shaped depression is similar to Case A. The margin is sclerotic with an external bevel. The surface of the vault and lesion is covered with active and remodled pores associated with a bumpy rugose surface. Possible trephination

Plate 26. *Case B*. Anterior view of the frontal. Horizontal cutmarks score the frontal above the left orbit. Deeper, vertically oriented cutmarks are located superior to these. Note the grooving in these marks.