REINTERPRETING CRIBRA ORBITALIA ETIOLOGY IN A COASTAL NORTH CAROLINA ALGONKIAN POPULATION USING COMPUTED TOMOGRAPHY

By

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May 2011

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Cribra orbitalia is visually characterized by porous lesions on the orbital roof and is often attributed to iron deficiency anemia, although other etiologies are possible. The main objective of this study is to reassess the diagnosis of iron-deficiency related cribra orbitalia in a North Carolina coastal Algonkian population (n= 50, AD 295-1460) using non-destructive methods. Microscopic techniques such as thin-ground sectioning have successfully differentiated between diploic expansion attributed to anemia as opposed pathological expressions related to other etiologies. However, such destructive techniques often are not possible with some U.S. samples because of NAGPRA provisions. Thus, we utilize non-invasive computed tomography (CT) scanning an alternative to identifying diploic expansion versus other causes of porosity in the orbital roof.

Out of a total sample size of 183 crania, 45 crania with varied forms of upper orbital lesions and 5 crania without such lesions were selected for CT scanning analysis. The axial anterior-posterior CT images would allow for distinction between diploic expansion and resorption of the corresponding cortical bone and cortical bone porosity. This preliminary study suggests that although in most cases CT scanning observations are the same as simple visual analysis, in some instances CT scanning allows for a more accurate diagnosis of diploic expansion versus porosity. While cribra orbitalia is usually attributed to anemia, orbital lesions can also indicate dietary deficiencies within the population. By more accurately understanding the etiology of cribra orbitalia in archaeological populations, we can better understand their dietary habits, health, quality of life, and overall adaptations to their unique environment. Reconsidering the etiology of cribra orbitalia has important implications for the current interpretations of malnutrition and

infectious disease in earlier human populations. This new non-destructive methodology has implications for paleopathological methodology, archaeology, and Native American history.

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A Thesis

Presented to the Faculty of the Department of Anthropology

East Carolina University

In Partial Fulfillment of the Requirements for the Degree

Master of Arts, Anthropology

By

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ACKNOWLEDGEMENTS

This study was possible because of the generosity of the Department of Cardiovascular Sciences at the Brody School of Medicine, East Carolina University for allowing us the use of their CT scanner free of charge and the enormous help of their scanning technicians, Kelli West and Vickie Davenport. I would also like to acknowledge Dr. West, without whose guidance on the scanner protocol, this project would not have been possible.

I would like to thank Dr. Perry for all of her help and guidance on my thesis. You were able to turn the denial of my initial proposal by the Indian Affairs Council into a very exciting project, and one that I am very proud of.

A huge thank you goes to Mindi Seeman, who has kept me sane and always reminded me that we're still swimming! The biggest thank you of all goes to my parents and to David. Without your constant support, encouragement, and never-ending belief in me I could not have done this. Who else would not think twice about moving across the entire country to chase this crazy dream with me? Thank you.

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CHAPTER 1: INTRODUCTION

Cribra orbitalia was first recognized by Welcker in 1885 and since then researchers have disagreed on its etiology. Rather than a specific disease, cribra orbitalia is a morphological feature of various diseases, and is visually characterized by porous lesions on the superior-lateral margin of the orbital roof. There are many possible causes for these cranial lesions, with iron deficiency anemia, other acquired anemias, and congenital or hereditary anemias most frequently cited as causing orbital roof pathology. Numerous other factors can cause porosity in the orbital roof, including deficiencies in vitamins C, D, and B_{12}, trauma, localized pressure within the eye orbit, and even postmortem erosion. This study aimed to determine if the majority of orbital lesions found in coastal Algonkian populations were due to anemia, or if the lesions should be attributed to a different etiology. By reassessing the causes of cribra orbitalia in a Late Woodland Algonkian sample, researchers can better understand not only their overall health patterns, but also what diseases they were susceptible to and how this population adapted to their unique marine environment.

Traditionally, histological analysis using thin sections of the orbital roof has proven most effective for viewing the internal structures of the cranium. However, destructive techniques using Native American skeletal remains, such as preparing thin sections, has been restricted since the passing of the Native American Graves Protection and Repatriation Act (NAGPRA) in 1990. The purpose of this study was to explore noninvasive techniques for viewing the internal orbital roof structure using computed tomography. This study attempted to determine whether or not computed tomography is a viable alternative to the more destructive traditional methods in determining the etiology of orbital roof pathologies. The current study was unable to conclusively prove that analysis using computed tomography was more accurate or detailed than visual observation, although in 18% of the samples CT scanning did show different results.

This study was completed using five different North Carolina coastal Algonkian ossuaries (31CK9, 31CK22, 31CK24, 31CO5, and 31DR38) radiocarbon dated from AD 360 to 1460. Most of the ossuaries date to the period of the densest occupation, during the Colington phase of the Late Woodland period. This study only included those crania with visible orbital regions, leaving a total sample size of

183 crania (N=232). Half of the 183 crania in this total sample (42% of adults and 80.5% of subadults) exhibited some form of cribra orbitalia. Fifty crania representing the range of severity scores were chosen for further analysis using computed tomography.

Although this Algonkian population has previously been studied (Hutchinson 2002), only preliminary macroscopic analysis was used. By reassessing this population using the more advanced methodology of computed tomography, a more accurate representation of this population was achieved. This more accurate understanding of the various etiologies of cribra orbitalia among this population leads to a better understanding of their diet, how they interacted with their marine environment, their quality of life, what diseases and nutritional deficiencies they were susceptible to, and their overall health status. Due to the various restrictions on scientific research of Native American groups, not as much is known about the overall health and diseases these populations lived with as is known with other populations. This study has implications for not only the current interpretations of malnutrition and infectious disease loads in prehistoric human populations, but also for the fields of archaeology, Native American history, and paleopathological methodology.

CHAPTER 2: BACKGROUND

Researchers have long recognized cribra orbitalia as a pathological condition but still disagree on its etiology. Invasive techniques to identify pathological changes, such as thin-sectioning cross-sections of the orbital roof, have helped to differentiate anemia-related porosity versus other causes. However, the legal implications of the Native American Graves Protection and Repatriation Act hinder destructive analysis such as thin-sectioning on Native American populations. A non-invasive technique, such as computed tomography, could overcome this research hurdle. Using computed tomography, or CT scanning, to analyze cross-sections of the orbital roof allows the internal structures of the orbit to be seen and analyzed without physically taking a sample from the crania.

In order to understand the implications of cribra orbitalia in reconstructing ancient health and disease and difficulties in proper diagnosis of its cause, the etiology and expression of the pathological conditions of cribra orbitalia and porotic hyperostosis, including demographic patterns and histology, will be outlined. This will be followed by a discussion of how the paleopathological study of cribra orbitalia and porotic hyperostosis developed, including the limitations of such studies. In addition, the archaeological and historical implications of cribra orbitalia frequencies in Late Woodland North Carolina Algonkian populations will be addressed. Finally, NAGPRA will be explained within the context of limitations of bioarchaeological techniques that can be used to study ancient Native American populations.

What are Cribra Orbitalia and Porotic Hyperostosis?

Cribra orbitalia, first identified by Welcker (1885, 1888), is visually characterized by porous lesions on the superior-lateral margin of the orbital roof. Cribra orbitalia is not a specific disease; rather, it is a morphological feature of various different diseases. Cribra orbitalia has been found in many other primate species, such as chimpanzees, orangutans, baboons, and macaques (Wells 1973).

Cribra orbitalia is sometimes accompanied by porotic hyperostosis, which causes diploic expansion of the parietal and occipital bones resulting in a porous appearance of the outer table. In radiographs of anemic patients, porotic hyperostosis is characterized by a “hair on end” appearance due to the thinning of the outer table of the skull and the perpendicular orientation of the trabeculae (Roberts

and Manchester 2007:229). The term porotic hyperostosis often is used as an all-encompassing term for both cribra orbitalia and porotic hyperostosis, and will be used in this paper to refer to both cribra orbitalia and porotic hyperostosis. Here, cribra orbitalia will be used to refer to only the upper orbital lesions, reserving porotic hyperostosis for the remaining cranial lesions.

History of Porotic Hyperostosis Research

Initially, the link between porotic hyperostosis, cribra orbitalia, and anemia was established through comparing modern patients suffering from severe hemolytic anemias (e.g. thalassemia major and sickle cell anemia) and identifying the associated skeletal changes (see Walker et al 2009:112). Using clinical patients, researchers eventually noticed the relationship of anemia to cribra orbitalia (e.g. Eng 1958; Shahidi and Diamond 1960). Cooley and Lee (1925) initially described skeletal changes associated with chronic anemia, specifically the widening of the diploic space they associated with thalassemia major. Moore (1929) described similar skeletal changes he associated with sickle cell anemia. Shelden described the same skeletal changes as occurring with iron deficiency anemia in 1936 (Britton et al. 1960:621, El-Najjar et al. 1975:919). In the 1950s, iron deficiency anemia had gained acceptance in the scientific community as the most likely cause of the marrow hypertrophy that results in porotic hyperostosis and cribra orbitalia (Walker et al 2009:109).

One of the first physical anthropologists to systematically link cranial porotic lesions to anemia using an evolutionary perspective was J. Lawrence Angel. While studying ancient skeletal remains in the Anatolian region, Angel (1966) noted the presence of porotic hyperostosis and pondered its etiology. He observed that both thalassemia and sickle cell anemia were endemic in areas where malaria is a strong selective pressure, such as in the Mediterranean region, and that these areas had the highest rates of porotic hyperostosis. Angel suggested that these inherited anemias were the cause of the cranial lesions seen in his sample. Angel's work stimulated others to consider a congenital cause for porotic hyperostosis, as exemplified by Kennedy (1984) and his work on ancient South Asian collections.

Angel (1966) also suggested that acquired anemias in the New World must be responsible for porotic hyperostosis due to the absence of malaria. El-Najjar and colleagues (1976) confirmed Angel's results by studying two different Native American groups in the southwestern United States, one maize and one non-maize dependent society. They noted that the maize-dependent group experienced a

higher frequency of porotic hyperostosis and concluded that this stems from phytates in maize which inhibit the absorption of iron in the blood, leading to iron deficiency. According to modern radiographic studies, Native American populations currently living in the southwestern United States have some of the highest frequencies of these cranial lesions, however, they do not have high rates of inherited hemolytic anemia. Due to this, researchers have focused on iron deficiency anemia as the most likely explanation (El-Najjar et al. 1975, Walker 1985).

What can Cause Cribra Orbitalia and Porotic Hyperostosis?

Porotic hyperostosis can manifest in either porosity extending through the outer table or diploic expansion. The manifestation of the lesion is dependent upon the etiology. For example, anemia is thought to be the only cause of diploic expansion, while porosity can be caused by many other things, for example, vitamin deficiencies. While the cause of each crania may be attributed to either anemia or vitamin deficiencies, the two etiologies are not mutually exclusive. As vitamin deficiencies are due to an insufficient diet, multiple vitamin deficiencies can be present in the same individual. Certain anemias can also be caused by an insufficient diet, leading to concurrent anemia and vitamin deficiencies in the same individual.

Etiologies that Cause Diploic Expansion

Cranial lesions can exhibit diploic expansion or porosity. Whether the lesion is porous or exhibits diploic expansion can shed light onto what caused the cranial lesion. Diploic expansion can only be caused by anemia, while porous lesions have many possible causes.

Anemia

The most common disease causing porotic hyperostosis is anemia, usually iron deficiency anemia. In 1929, both Moore and Williams independently suggested that anemia was the causative factor for porotic hyperostosis. Initially, the focus was on congenital anemias, but Hengen (1971) asserted that iron deficiency anemia was the exclusive cause of cribra orbitalia (Stuart-Macadam 1991:36). Some researchers believe that cribra orbitalia is the earliest skeletal expression of anemia followed by porotic hyperostosis, suggesting that the relatively thin bone along the orbital roof could be more susceptible to resorption by the expanding marrow cavity (Walker 1985:141).

Anemia is the reduction of hemoglobin concentrations or packed red blood cell counts below normal parameters for a certain age and sex (Sullivan 2005:253). When hemoglobin levels drop, the body becomes oxygen-starved and triggers an increase in the rate of red blood cell production. This increase occurs in the hemopoietic marrow, which causes the expansion of the diploic space; the expansion of the diplöe causes the outer table to be resorbed and it becomes thin, which creates the porosity, while overall the cranial vault becomes thicker (White and Folkens 2005:320, Walker et al 2009:109). While the skeletal changes in anemia-related cribra orbitalia are unique, studies have shown that at most only 50-75% of clinical patients with anemia show any morphological bone changes regardless of the severity of their anemia (Stuart-Macadam 1991:37). This suggests that researchers do not know all of the reasons for skeletal changes.

Anemia can further be broken down into two broad categories: acquired and congenital. Acquired anemia results from nutritional deficiencies and includes iron deficiency anemia and megaloblastic anemia. Congenital anemias are genetically-based and include sickle cell anemia and various thalassemias. For this study, the focus will be on acquired anemia, as no evidence for congenital anemia exists in Native American populations. Causes of acquired anemia include excessive blood loss, impaired erythropoiesis (red blood cell production), and increased hemolysis (red blood cell destruction) (Walker et al 2009:110). These symptoms can be caused by inadequate dietary intake, inadequate absorption of vitamins due to chronic diarrheal diseases or intestinal parasites, or a genetic or acquired inability to process vitamin B_{12} in the intestinal tract (Sullivan 2005:255). These physiological conditions often are a response to dietary insufficiencies (such as lack of iron or prolonged breastfeeding), trauma, and disease. Anemia can lead to lowered physical performance, high rates of maternal and fetal morbidity and mortality, and small, underweight babies (Walker 1985:139). In children, anemia can cause physical growth retardation, delayed walking, lowered physical capability, and slower cognitive development (Wright and Chew 1998:925).

Iron deficiency anemia

The lack of sufficient iron in the diet is one of the common causes of iron-deficiency anemia, the most frequently reported cause of porotic hyperostosis (Walker et al. 2009:111). The body must maintain a balance between too little and too much iron. Too little iron results in the above symptoms, while too

much iron also leads to a compromised immune system, increased risk of cancers, and organ failure (Stuart-Macadam 1998:56). Iron deficiency can result from prolonged consumption of iron- poor foods, a higher demand for iron during growth and reproduction, excessive blood loss due to a parasitic infection, inadequate absorption of iron through the intestines due to chronic diarrheal diseases, low iron storage protein, low transferring protein, or an elevation in erythrocyte protoporphyrin levels (Sullivan 2005:254, Stuart-Macadam 1998: 46). In an effort to compensate for low levels of iron, the body increases the total amount of red bone marrow. The low iron levels result in red blood cells that are deficient in iron, causing these cells to be microcytic (smaller) and hypochromic (paler due to lowered amounts of hemoglobin) and thus inefficient transporters of oxygen to the various tissues in the body (Stuart-Macadam 1998:46). In addition to its role in hemoglobin production, iron also functions in cell-mediated immunity, leaving those deficient in iron susceptible to infection (Sullivan 2005:253).

In 1999 there were an estimated 2.15 billion people worldwide who were iron deficient, and 1.2 billion of those people had iron-deficiency anemia (Wright and Chew 1998:925). There are three stages of iron deficiency (Wander et al. 2009), of which only the last stage results in the observable skeletal response of cribra orbitalia or porotic hyperostosis. The first stage is iron depletion, which occurs when iron stores are mobilized in response to inadequate iron in the body. The second stage is iron deficient erythropoiesis, where iron delivery to tissues is restricted and the symptoms of iron stress are apparent. The third and last stage is iron deficiency anemia, characterized by inadequate hemoglobin synthesis due to reduced iron delivery to the bone marrow (Wander et al. 2009:173). Symptoms of iron deficiency are variable, the severity of which does not necessarily reflect the severity of anemia. Symptoms include fatigue, weakness, light-headedness, headaches, difficulty breathing, and heart palpitations. Additional gastrointestinal symptoms include loss of appetite, flatulence, diarrhea, constipation, nausea, and vomiting. When severe anemia becomes chronic, spoon-shaped nails, cracks at the corner of the mouth, sore tongue, flattening of lingual papillae, atrophic gastritis, and bone changes can occur (Stuart-Macadam 1998:46).

Iron metabolism is almost a closed system, as iron is usually obtained from the destruction of old red blood cells and recycled into new red blood cells by the reticuloendothelial system. It is only during times of increased iron requirements that the intestines will increase the percentage of iron absorbed from

the diet (Stuart-Macadam 1991:37). As noted above, iron deficiency can result from inadequate intake of iron in addition to problems with iron absorption. Iron rich foods include red meat, liver, kidney, mussels, shellfish, prunes, spinach, beans, and eggs (Sullivan 2005:254). Therefore, people with diets containing low amounts of these foods are more susceptible to iron deficiency. In addition, iron absorption is affected by diet, physiology, and genetics. For example, intestinal mucosa regulates absorption of iron, although the mechanism is unknown (Stuart-Macadam 1998:54). Many foods also can enhance or inhibit the absorption of iron. The simultaneous ingestion of iron-rich foods with vitamin A, vitamin C, ascorbic acid, citric acid, lactic acid, meat, fish, poultry, or fermented foods will enhance the body's absorption of iron. The iron found within meat does not require processing in the stomach before it is absorbed, and the amino acids that result from the digestion of meat further enhance iron absorption. On the other hand, the body's ability to absorb iron can decrease due to tannins found in tea and some wines, calcium, and phytates found in cereal crops such as maize (Sullivan 2005:254, Stuart-Macadam 1998:55, Larsen 1997:29). Anderson and colleagues (1993) observed that rates of anemia decreased with age, leading the researchers to conclude that diet is possibly more critical to anemia levels than parasite load alone. Many researchers attribute the high rate of iron deficiency anemia in the prehistoric Native American populations to problems with iron absorption and lack of high-iron foods due to their maize-intensive diet (Hutchinson 2002). One study showed that less than 5% of the total iron in the diet is absorbed if the diet consists of mainly maize (Walker 1985:147).

Anemia and chronic disease

Researchers have suggested that moderate iron deficiency may be an adaptation to infectious environments, as studies have shown an increase in iron led to an increase in malaria and diarrheal diseases. The body has a natural iron-withholding defense to starve microbial and neoplastic invaders that need iron to proliferate within the host (Sullivan 2005:254). In order to remove available protein from the invaders, iron is bound to transfer or storage proteins (Sullivan 2005:255). A chronic iron-withholding response is often called anemia of chronic disease, which reduces hemoglobin concentrations, serum iron, and overall iron absorption (Sullivan 2005:254). The chronic response decreases incidence and intensity of infections (Sullivan 2005:255). For example, Wander and colleagues' study of 314 children found that mild or moderate iron deficiency protects against acute infections. This suggests that dietary

iron deficiency may be a nutritional adaptation to infection in environments where high transmission of infections or morbidity is prevalent (Wander et al. 2009).

Chronic infections can also lead to cranial lesions. Parasites feed off of the host's blood, cause internal bleeding and loss of nutrients through chronic diarrhea. Numerous varieties of parasites have been found in coprolite studies conducted in the southwestern United States (Walker 1985:151). The mouthparts of late Archaic hookworms were found in coastal South Carolina (Rathburn et al. 1980). One study found that hookworm infestations increase both in number and intensity of infections with age.

Hemolytic and megaloblastic anemia

Researchers recently have been questioning the conventional wisdom that cribra orbitalia is synonymous with iron deficiency anemia, especially since other diseases can cause porotic lesions. According to Walker et al. (2009:112), iron deficiency anemia by itself cannot sustain the high levels of erythropoiesis necessary to cause the lesions because iron deficiency reduces hemoglobin synthesis and red blood cell maturation. Walker and colleagues (2009:109, 112) instead suggest that hemolytic and megaloblastic anemias are the most likely causes of the lesions.

Hemolytic anemia is caused by premature hemolysis that overwhelms the ability of the hemopoietic marrow to compensate for these losses, resulting in expansion of the marrow space. Megaloblastic anemia results from insufficient amounts of vitamin B_{12} and B_9 due to inadequate dietary intake, intestinal parasites, chronic diarrheal diseases, or a genetic inability to process the vitamins. It is abundant in hunter-gatherer societies where breastfeeding is prolonged and fresh meat is not consumed in abundance (Walker et al. 2009:114). Primary dietary sources of B_{12} are found in animal products, such as liver, meat, oysters, saltwater fish, milk, eggs, and cheese, while sources of B_9 include fruits, liver, and leafy green vegetables (Sullivan 2005:255). The deficiency causes some marrow cells to divide abnormally and create enlarged hemopoietic cells with large nuclei. Both hemolytic and megaloblastic anemias can produce marrow expansion in order to compensate for the increased loss of red blood cells and the enlarged red blood cells, respectively

Etiologies that Cause Porosity

In contrast to anemia discussed above, other etiologies produce porosity without the diploic expansion characteristic of an anemic response. These porous lesions can be confused with diploic

expansion. On a CT scan, the lack of diploic involvement or expansion and retention of compact bone in the orbital roof would be indicative of an etiology other than anemia.

Nutritional deficiencies

In addition to the deficiency of vitamin B_{12} as noted above, other nutritional deficiencies, such as vitamins C and D, can lead to porosity in the skull (Ortner et al. 2001:344, Roberts and Manchester 2007:230, Walker et al. 2009). Vitamin C deficiency, or scurvy, can cause skeletal lesions due to the chronic bleeding at sites where blood vessels are near the skin's surface (Ortner et al. 2001:344). Chronic vitamin D deficiency, or rickets/osteomalacia, can cause the cranial vault to expand slightly, appearing similar to porotic hyperostosis (Schultz 2001:134), while in the orbits only the outer table is affected by porosity (Larsen 1997:33). As various nutritional deficiencies can occur simultaneously, it is possible that more than one disease caused the lesions. It must be kept in mind that vitamin C, vitamin D, and iron deficiency anemia are not mutually exclusive, as all three conditions are related to malnutrition.

In 2001, Ortner and colleagues studied scurvy among subadults in North America, and found that depending upon the group, the prevalence of evident vitamin C deficiency ranged from 0-38% and was most prevalent in Native American groups. They found the most common expression of the deficiency was porous lesions on the greater wing of the sphenoid and adjacent sites, although other common manifestations included orbital roof, posterior maxilla, interior zygomatic, infraorbital foramen, palate, alveolar process of maxilla, and the coronoid process of the mandible (Ortner et al. 2001).

Other causes

In addition to the above mentioned nutritional causes of cranial lesions, many non-nutritional deficient etiologies exist. Trauma to the skull can cause subperiosteal hematomas, leading to lesions that look very similar to porotic hyperostosis and cribra orbitalia (Walker et al. 2009:115). Localized pressure on the bone, due to pressure from an enlarged organ such as the lacrimal gland, can lead to bone atrophy which can result in lesions similar to porotic hyperostosis (Wapler et al 2004, Pechenkina and Delgado 2006). Pressure from artificial cranial deformation has also been proposed to cause porotic hyperostosis, but one study by Pechenkina and colleagues (2007) found that populations with high rates of lesions had no cranial deformation and populations that practiced cranial deformation had low frequencies of porotic hyperostosis. In addition, similar porous lesions can be caused by generalized

inflammation in the skull, tumors, osteitis, hypervascularization due to periostitis, and osteoporosis (Roberts and Manchester 2007:230, Sullivan 2005). Postmortem erosion can even cause porotic lesions on the skull; the eye orbit is prone to postmortem changes due to the lamina being thinner there than in other areas on the skull (Wapler et al. 2004:338).

One of the most effective means for identifying the cause of each case of cribra orbitalia, as described below, is by histological analysis. CT scanning is an alternative but yet untested non-destructive method for analyzing the structures in the orbital roof. If the scan shows diploic expansion with concurrent outer table resorption, anemia is the most likely etiology. If the outer table is the only structure affected, then porosity due to nutritional deficiencies or trauma can be suggested as possible causes. Postmortem erosion can be suspected if only the outer table is affected, and the edges of the pores are very sharp and do not show any osseous response. While cribra orbitalia has previously been attributed specifically to iron deficiency anemia, researchers are now beginning to explore other possibilities in order to more fully understand the various etiologies of cribra orbitalia.

Age and Sex Differences in Lesion Frequencies

Cribra orbitalia rates vary between adults and subadults, and between males and females. Subadults tend to exhibit higher rates of cranial lesions, due to the formation of bone during growth and development. Females tend to exhibit slightly higher rates of porotic hyperostosis due to the extra iron required during reproduction, although in most populations the differences are not statistically significant.

Age Differences

Cribra orbitalia is thought to be active exclusively in children, and any lesions seen on adult skulls are in the process of healing (Stuart-Macadam 1985). Many studies have shown that active lesions appear in the greatest frequency before age 7 (e.g. Sullivan 2005, Ortner et al. 2001, Slaus 2000). There are many possible explanations for why subadults tend to exhibit greater frequencies of cranial lesions, mostly concerning the formation of bone during childhood growth and development (see Walker et al. 2009:111). Subadults already have erythropoietic marrow in their cranium, so when additional red blood cells are needed, diploic expansion, rather than porosity, occurs. In addition to the differences in red hematopoietic marrow distribution between adults and subadults, certain conditions, such as nutritional deficiencies and trauma, are more likely to occur as a child.

The distribution of red blood cell production (erythropoiesis) sites across the skeleton changes during growth and development. At birth, all medullary cavities are filled with hematopoietic marrow. Beginning around 4 years of age, the red hematopoietic marrow gradually is replaced by non-hematopoietic yellow marrow (Stuart-Macadam 1985:392). During childhood, the main erythropoiesis sites are in the diploic space of the cranial vault and in the medullary cavities of the long bones. In adults, erythropoiesis occurs in the spongy bone of the axial skeleton, and the majority of the marrow is yellow fatty marrow. The abundance of inactive marrow leaves room for hematopoietic marrow to expand, reducing the potential for bone alterations (Sullivan 2005:266). Since subadults have their main erythropoiesis sites in their cranium, it would follow that the expansion of these sites would leave lesions along the orbital roof and cranial bones. Several longitudinal clinical studies have shown that late onset anemia does not produce bone changes (see Stuart-Macadam 1985).

In addition, other conditions which may lead to anemia-like lesions occur more frequently during childhood. For instance, skeletal trauma often can lead to subperiosteal hematomas that transform into highly vascularized, subperiosteal new bone (Schultz 2001:131). Children are more likely to form this new bone because the periosteum is not as firmly attached at the orbital roof as it is in adulthood (Walker et al 2009:115). This new bone can appear identical to cribra orbitalia on macroexamination, but the use of a microscope can tell the difference between porosity due to highly vascularized woven bone and porosity in cortical bone.

Children also are more susceptible to certain diseases and nutritional deficiencies than adults. The higher rates of nutritional deficiencies, due to an insufficient or different diet than adults, can help explain higher rates of cranial lesions often seen in subadults. Breast-fed infants rarely exhibit iron deficiency anemia (Stuart-Macadam 1998:57) before 6 months of age, as iron stores in the liver take 4-5 months to diminish (Fairgrieve and Molto 2000:329). Weaning foods have a significant impact on the rates of cribra orbitalia, as a weaning diet rich in carbohydrates and phytates provides low amounts of iron. Weanling diarrhea can occur due to the change from sterile breast milk to solid foods often contaminated with microorganisms and can last for several months, leading to dehydration and malnutrition among infants (Walker 1985:150). When the etiology of cribra orbitalia and porotic hyperostosis is better understood, researchers can use the frequencies of each cause, such as rickets or

scurvy for example, to determine if subadults in general have higher rates of those diseases and compare them with the frequency of cranial lesions.

A study conducted by Wright and Chew (1998) compared modern rural children from Plan de Sanchez, Guatemala, with a forensic sample derived from a military massacre in 1982 on the same population. They found that the forensic archaeological sample had 5 times higher rates of porotic hyperostosis. The differences between the two samples could be due to a greater proportion of anemic children surviving childhood in the past than they do today. The decreased frequency of porotic hyperostosis seen today may be due to heavier infectious disease burden and earlier weaning leading to higher childhood mortality rates.

The expected higher frequency of cribra orbitalia among subadults did not appear among the Outer Coast Algonkian populations (Hutchinson 2002:97) in the previous study. This pattern might be expected if anemia was caused by intestinal parasites and not by iron-deficiency (Hutchinson et al. 2007:60) due to dietary differences between adults and children. This study confirmed Hutchinson's (2002) original finding of higher prevalence of cranial lesions in adults, and these lesions probably did not result from iron-deficiency anemia. It is more likely, similar to Hutchinson's conclusion, that it was nutritional deficiencies other than iron deficiency that explain the prevalence of cribra orbitalia. Proper identification of the cause of these lesions may change our interpretations of the North Carolina Algonkians, their health, diet, and adaptations to their coastal environment.

Sex Differences

Women are thought to be disproportionately affected by cribra orbitalia because of their susceptibility to iron deficiency anemia brought on by the high iron and folic acid demands of reproduction. Iron demands are higher in children and pregnant or lactating women (Stuart-Macadam 1998:55). Other explanations for frequency differences between the sexes include differences in hormones and cultural access to food (Ortner 1998:81). However, many studies have not found a statistically significant difference between the sexes (Salvadei et al. 2001, Slaus 2000, Stuart-Macadam 1998). Some researchers have suggested that females have adapted a greater immune response than men to cope with the selective pressures and hazards associated with pregnancy and childbirth (Ortner 1998:81, Storey 1998:134).

Methods for Diagnosing Orbital Lesions

As noted above, differentiating pathological process, and thus being able to identify the causes of the lesions, require methods beyond simply macroscopic observation. Under a microscope, changes in the histological structures can be seen, and the surface details are enhanced. Macroscopically, one can determine whether lesions are present and conclude that an individual has cribra orbitalia, but a definitive diagnosis and the cause of the lesions cannot be determined until microscopic methods are used.

Bone Histology

The development of microscopic techniques for analyzing histological structures has a long history. Light microscopy has been used to examine tissue in medical examinations since the end of the Nineteenth century (e.g. Schaffer 1889). It was not until the 1920s, however that attempts were made to analyze sections of bone under a microscope. In order to view a bone under a light microscope, it had to be decalcified, embedded in paraffin, and cut with a microtome (see Schultz 2001, Garland 1993, Martin 1991, and Stout 1976). This process worked well for fresh bone; however, archaeological bone cannot be decalcified, so the process did not come into archaeological use until the 1950s, when thin ground sections began to be used (see Baud and Morgenthaler 1956, Ascenzi 1969, Ortner 1976, Hackett 1976, Stout 1976).

The histological structure of the cranial vault must first be understood before undertaking the examination of cribra orbitalia and porotic hyperostosis. Bone in the skull vault has three parts: the inner and outer laminae and the diploic space. The internal and external laminae are made of hard, compact bone. The diploic space is sandwiched between the laminae and is comprised of spongy, cancellous bone, often referred to as trabeculae.

In addition to understanding the histological structures of the cranial vault, an understanding of how each disease process can change those structures is necessary. Anemia will be treated as one etiology, regardless of the various types of anemia, as all anemias affect the skeleton in a similar fashion (Sullivan 2005, Stuart-Macadam 1998). Anemia causes marrow hyperplasia, which puts pressure on the surrounding bone, eventually leading to pressure atrophy of the outer table and exposing the diplöe, causing the porous appearance in the lamellar bone (Schultz 2001:132). Schultz (2001) outlines three stages of anemic changes to the cranial vault. Stage 1 is characterized by a normal internal structure,

with slight thickening of the outer table. During Stage 2, the porosity becomes more pronounced, the vault becomes thicker in affected areas, and the external lamina start to disintegrate. Stage 3 results in a pronounced thickening of the cranial vault when trabeculae take on a parallel orientation and the external lamina is resorbed (Schultz 2001:132-133). Usually these changes are only due to severe, chronic anemia, although it has been shown that mild cases of anemia can exhibit severe bone alterations. In modern clinical studies, it has been shown that only 50-75% of anemia cases exhibit bone alterations (Stuart-Macadam 1985:397). Therefore the total number of archaeological samples showing porotic hyperostosis usually under-represents the actual number of affected individuals and the absence of lesions does not equate to an absence of anemia, unless the archaeological sample can be proven to be representative of the population as a whole (i.e. Wright and Chew 1998).

Chronic diseases other than anemia can result in lesions of similar appearance, but each have slightly different histological profiles (see Figure 1). Bone changes due to vitamin C deficiency are the result of chronic inflammation and hemorrhage, rather than marrow hyperplasia. Excessive woven bone is present, the diploic space has a normal appearance, and the porosity penetrates the existing outer table (Ortner et al. 2001:345). Vitamin D deficiency results in pathological changes mostly on the external surface, with the porous appearance due to very small squamous appositions over the outer table (Schultz 1990:187). Schultz (2001:134) describes osteomyelitis as external bone apposition with no internal changes to the bone. Inflammation only resembles porotic hyperostosis in the beginning stages, with later stages of inflammation easily distinguished from the porosity due to anemia (Schultz 1990:187). Changes that occur postmortem are easily distinguished from anemia-related changes, as there are no indications of bone reaction (Wapler et al. 2004:334).

Previous research has discovered that histological analysis of skeletal pathologies (paleohistology) can lead to a better understanding of the skeletal response and the etiology of the pathology. The multivariate analysis of cranial lesions, for instance, can help researchers make better inferences about the populations they are studying because the hypotheses about those populations are constantly being tested and fine-tuned. Several authors have conducted histological studies on skeletons with porotic hyperostosis previously attributed to anemia and found that the actual prevalence of anemia was much lower than expected. For instance, Schultz (1990) found many misdiagnosed cases in a

sample from early Bronze Age Anatolia. Using only macroscopic examination, the researchers determined that out of 144 infants 7% of the lesions were due to anemia, none were due to rickets, 8.5% due to osteomyelitis, and 6.8% was due to meningeal irritation. After microscopic examination, these frequencies changed dramatically. It was found that only 4.7% of the lesions could be attributed to anemia, 3.9% due to rickets, only 4.7% were due to osteomyelitis, and 9.5% were due to meningeal irritation. Schultz conducted a similar study again in 2001 and found that most of the macroscopically diagnosed anemia was actually pseudopathology or postmortem damage (Schultz et al. 2001:222).Wapler and colleagues (2004) examined the histological structures of bones from Missiminia in North Sudan (N=333) that displayed the macroscopic features of cribra orbitalia using thin-ground sectioning. Of the 93 (28%) specimens that had cranial lesions, only 43.5% previously identified as having iron-deficiency anemia actually had anemic hypertrophy. The other 29.4% were merely inflammation, and 20% had postmortem erosion. These studies illustrate that by analyzing cases of previously diagnosed cribra orbitalia using histological methods, researchers may be able to more accurately determine what caused each case of cribra orbitalia.

The histological analysis of thin-ground sections therefore remains an extremely effective means for properly diagnosing cranial porosity. However, the passing of NAGPRA in 1990 had a significant impact on the use of destructive methods such as thin sectioning to study Native American skeletons. The law determined that any Native American remains excavated must be affiliated with the closest living Native American group; the tribe can file an “intent to repatriate” claim in order to have the skeleton returned back to them. Those skeletons curated by federal agencies had to inventory their collections and obtain permission from the affiliated group in order to perform any scientific research on the population. NAGPRA sought to eliminate the view of skeletons as solely objects for scientific research, and to bring scientists and Native American groups together.

Due to NAGPRA, researchers must first contact the Indian Affairs Council and gain permission before proceeding with any study. In most instances, NAGPRA has not hindered the ability for scientific research on Native American populations. However, in the present case, permission was not granted by

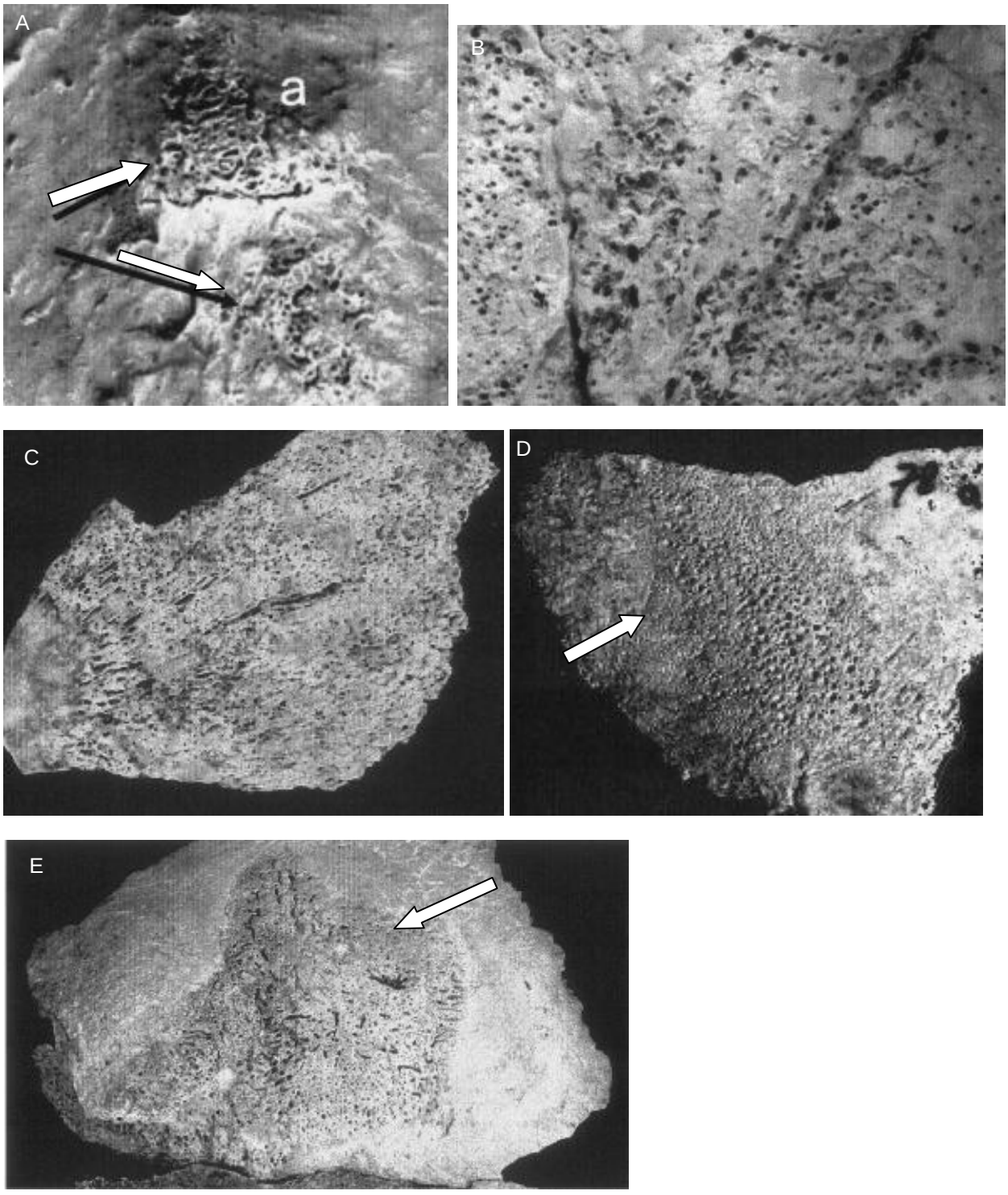

Figure 1. Porosity caused by different etiologies (see Schultz 2001for original photographs). (A) Porosity of the inner cranial vault due to hemorrhaging (B) Pseudopathology caused by postmortem erosion (C) Porotic hyperostosis due to vitamin D deficiency (D) Porosity due to osteomyelitis (E) Porosity due to subperiosteal hematoma caused by vitamin C deficiency

the Indian Affairs Council for thin section analysis to be done on the Algonkian samples curated at East Carolina University. Therefore, in order to comply with these restrictions, this study used computed tomography to see the internal bone structure. The use of computed tomography has only recently been applied to bioarchaeology and forensic anthropology. Computed tomography has been used to visualize cribra orbitalia, develop new identification techniques, and to reassess the accuracy of age and sex estimation methods (e.g. Kahana et al. 1998, Pasquier et al. 1999, Taleb-Ahmed et al. 2003, Exner et al. 2004, Telmon et al. 2005, Moskovitch et al. 2010).

Interpreting Disease

In order to study cribra orbitalia and understand its causes, researchers must understand the various biases that exist when studying paleopathology. During excavation, bones can be damaged, missing, lost, or never excavated. Preservation, recovery, and sampling biases will all be discussed in detail below. In addition, researchers must keep in mind the osteological paradox, and be watchful for pseudopathologies.

There are several external limitations to the study of paleopathology. The first of these is how well the population was preserved prior to excavation and curation. The archaeological context, including soil and weather patterns surrounding the body, highly impacts the rate of skeletal deterioration. No single factor determines the quality of preservation; rather it is the combination of the various factors that impacts how well a skeleton is preserved. There are both intrinsic and extrinsic factors that influence the level of preservation.

Intrinsic factors that determine how well a skeleton preserves include the nature of the bone itself, the complexity, chemistry, shape, density, size, and age of the bone, and the presence of antemortem injury. Henderson (1987) found that the rates of decay are inversely proportional to the size of the bone. In another study, Waldron (1987) found that the bones that survived most frequently were the dense, heavy bones- petrous portion and mastoid of the temporal, acetabulum and sciatic notch in the pelvis, and the long bones.

It is a general rule that archaeological samples are fragile, brittle, and often poorly preserved. This is due to the variety of taphonomic conditions to which they are exposed. Extrinsic variables such as the pH level of the soil, the permeability of the soil, moisture level, temperature, humidity,

microorganisms, and human activity all influencing how quickly skeletal material can be destroyed (White and Folkens 2005:52, Henderson 1987:45). Soil pressure can warp the bones, while acidic soil can dissolve the inorganic matrix, leaving the organic material susceptible to water leaching. Water and soil type affect how quickly diagenesis, the removal of proteins and minerals and absorption of ions into the bones, occurs. Bacteria and fungi can destroy the bone, leaving it unstable and not easily preserved. Bones tend to preserve best when they are located in a temperate, well-drained area with low water tables and a neutral or slightly alkaline soil pH. Humans also have a profound effect on the preservation of a skeleton, as they determine who is buried as well as when, how, and where they are buried (Henderson 1987:44-49).

The likelihood of finding pathological alterations in a population is directly related to the number of bones that survived. Therefore, it is very important for archaeologists to understand how the various postmortem processes affect each bone. There is a bias in which skeletal elements will be recovered from the field (i.e., recovery bias). Many factors influence the recovery bias, including: the size of mesh screen used, excavator's knowledge, mortuary practices of the people being studied, taphonomy, and other archaeological recovery methods (Graesch 2009, Adams and Konigsberg 2004).

Sampling bias can also impact assessment of disease in an archaeological sample. The population sample may not be representative of the group living at the time because the archaeological sample is always comprised of deceased individuals. Sampling bias may also occur during excavation. Researchers often do not excavate an entire cemetery, or an entire town; usually only a small portion of the cemetery is excavated, leaving the researchers with a sample of a sample population. The samples that have been excavated are often fragmentary, poorly preserved, and have postmortem damage. The portion of the cemetery that was excavated may not be representative of the cemetery as a whole.

There are additional limitations from the original population being studied. For example, selective mortality issues must be taken into account, along with the biocultural factors that can influence an individual's risk of disease and health, cultural factors associated with burial, and the prehistoric populations tended to not be sedentary (Roberts and Manchester 2007:12, Boddington 1987:181). While these limitations may not always be overcome, it is important to recognize that there are certain limitations that must be taken into account when analyzing paleopathologies.

Researchers must keep in mind that it could be the case that the individuals with bone pathologies may actually be the stronger and healthier individuals who survived the condition long enough for it to affect the bone (i.e., the "osteological paradox"). Conversely, those without bone lesions may be the weaker individuals who died quickly before skeletal lesions could form (Wood et al. 1992). When pathological alterations are found, researchers must determine whether the alteration is an actual pathology or pseudopathology. For example, fungi can destroy compact bone, leaving lesions that are similar to osteoporosis or a metastasizing tumor. Thickened woven bone can be mistaken for a periosteal reaction if not microscopically examined; during diagenesis, crystals can aggregate along the bone's surface leaving a pseudopathological lesion (Schultz 2001:117). When studying paleopathology, there are certain biases that exist that researchers must be aware of, even if the biases are often not able to be overcome. The recognition of these biases allows researchers to make more accurate interpretations of their data.

Prehistoric Algonkian Groups

The region in North Carolina that is referred to as the tidewater coast extends from the major sounds, including Currituck and Albemarle, to the barrier islands (Figure 2) (Hutchinson 2002:17). Most archaeologists agree that there are two main traits that define the Algonkian culture present in this region- the construction of longhouses and the use of collective ossuaries (Ward and Davis 1999:216). However, the Algonkian identity is based entirely on linguistics. During the Contact period (ca 1600), there were three linguistic groups along the coast: Iroquoian, Siouan, and Algonkian. The Algonkian language group reached north into New England and occupied the tidewater area, including most of the offshore islands. Iroquoian and Siouan populations occupied the coastal plains (Feest 1978). Several tribes combined to form the Algonkian language group, including: Chowanoke, Weapemeac, Poteskeet, Moratoc, Roanoke, Secotan, Pomuik, Neusiok, and the Croatan (Goddard 1978). The exact boundaries of the different Algonkian groups is difficult to establish because the Europeans often counted allied groups as single tribes, rather than the independent groups they were (Feest 1978).

Most of our knowledge about Algonkian culture is derived from written sources of settlers at contact. John White drew several maps of the Carolina coast and the Algonkian environment. Thomas Harriot wrote reports describing various aspects of the native cultures he encountered (Ward and Davis

1999:213). The Algonkians were a ranked society, led by a "werowance", or ruler, who was chosen from a matrilineal ruling lineage. Each ruler had their own symbol which was painted or tattooed on the backs of the men to show their affiliation (Feest 1978). Within each of the societies listed above, there was a capital town, smaller towns, and dispersed farmsteads. An average town at the time of contact had a population of 150 individuals living in 12-18 longhouses (Ward and Davis 1999:211). The largest tribe encountered by the English was the Chowanoke, which had 18 villages and a population of 2,500 (Feest 1978). John White's drawings show two different village arrangements that were common. The Secotan village was an open settlement, with nobilities' houses placed along a wide central path that connected public and ceremonial areas, and the commoners' houses scattered around the rest of the settlement. The other plan shown by White is the settlement of Pomeioc, which shows 18 longhouses forming a concentric circle around a central open plaza surrounded by a stockade (Ward and Davis 1999:213). A passage written by Barlow during his expeditions also sheds some light on a smaller fortified town: "at the north end therof was a village of nine houses, built of cedar, and fortified round about with sharpe trees to keep out their enemies, and the entrance into it made like a turne pike very artificially" (Feest 1978).

Archaeology of Algonkian Populations

Excavations of these sites have produced skeletal remains that can provide information on the health and adaptations of these groups to their environment (see Hutchinson 2002). Skeletal remains from five North Carolina coastal Algonkian sites dating to the Colington phase of the Late Woodland period (Hutchinson 2002:17, 19) were included in this research: Hollowell (31CO5), Baum 1 and 7 (31CK9), Hatteras Village (31DR38), West 2 (31CK22), and Knott's Island (31CK24). These remains are held in the bioarchaeology laboratory at East Carolina University. The dates for the sites range from AD 360 to 1460.

The Middle to Late Woodland periods (AD 200-1650) are viewed by archaeologists as the time when a gradual shift toward agriculture with more complex societies and permanent settlements was taking place. The beginning of the Woodland period is defined by the appearance of pottery (Ward and Davis 1999:3). David Phelps developed a detailed chronology for the Woodland period using the various ceramic types which allows researchers to date sites more accurately. The Colington phase of the Late Woodland period (AD 800-1650) is marked by the introduction of shell-tempered pottery (Phelps

1983:36). Due to the sandy nature of the soil, clear stratigraphic separation of temporally distinct cultural levels is difficult as pottery sherds can easily migrate between strata (Ward and Davis 1999:226), and archaeologists must be careful to notice the fine details that delineate each different strata.

During the Late Woodland period, most of the burials were collective ossuaries, where the majority of the skeletons are disarticulated. A typical Colington Algonkian ossuary contains between 20 and 60 people, usually commingled, with no cultural biases for age or sex (Hutchinson 2002:23). After death, the deceased were placed in burial houses, where they decomposed. Periodically, the bodies were removed from the burial houses and buried in the ossuaries (Ward and Davis 1999). The ossuaries contained few grave goods, as the dead were honored during the initial storage phase of the burial process, rather than in the burial phase (Hickerson 1960). The total size and shape of the burial pits varied. Ossuaries were usually located on the edges of settlements (Hickerson 1960). Not all skeletons were excavated from ossuaries, or secondary burials. At the West site, some of the skeletons were found individually in primary burials.

The coastal region contains numerous ecological niches ranging from freshwater, brackish water, to saltwater marine areas, along with wooded swamps and maritime forests (Hutchinson 2002:1). The coastal Algonkians took advantage of both the marine and non-marine food sources available to them. The evidence for subsistence patterns of the outer coastal Algonkian groups comes from carbonized plants, animal bones found in archaeological deposits, stable isotope analysis, and John Lawson's journals (Hutchinson 2002:23-26, 201). The Algonkians relied heavily on foraging plants such as amaranth, chenopodium, sumpweed, knotweed, little barley, maygrass, hickory nuts, acorns, grape, maypop, sumac, and sugarberry. They also exploited marine resources, such as sturgeon, catfish, bowfin, American eel, carp, longnose gar, sunfish, redhorse, sheepshead, sea trout, atlantic croaker, black drum, red drum, spot, yellow perch, oysters, clams, scallops, whelk, and crabs. Reptiles such as turtles, alligators, cooters, and sliders were also consumed. The Algonkians furthermore hunted birds and mammals, including wild turkey, green-winged teal, blue-winged teal, mallard, herons, redhead, canvasback, snow goose, lesser scaup, bobwhite quail, passenger pigeon, beaver, opossum, white-tailed deer, raccoon, gray squirrel, fox squirrel, eastern cottontail rabbit, marsh rice rat, marsh rabbit, and black bear (Hutchinson 2002:27). Unlike the inner coastal areas, which adopted agriculture around 1000, the

outer coastal areas did not begin intensification of maize agriculture until after 1400 (Hutchinson 2002:160). In addition to maize, the Algonkians planted melons, pumpkins, squash, beans, and sunflowers (Feest 1978:273). Hollowell, the only inner coastal site being used in this study, mostly supplemented maize production with hunting riverine and terrestrial fauna, while the outer coastal sites exploited little maize and relied mainly on marine fauna (Hutchinson 2002:138). Dental microwear analysis and isotope signatures suggest that the diet of both inner and outer coastal populations was heterogeneous with local variation (Hutchinson et al. 2007:55).

Previous Analysis

In addition to analyzing their subsistence patterns, Hutchinson (2002) completed an extensive health and disease analysis on these sites. Hutchinson separates his analysis into inner and outer coastal sites, as this influenced their diet, and thus their overall health. Of the total sample of outer coastal adults, Hutchinson found that 10% had periostitis or osteomyelitis, 16% of males and 12% of females had systemic infections, and 10% had degenerative joint disease (Hutchinson 2002:103, 118-119). The dental analysis for diet and disease patterns showed that 14% had carious lesions, 16% had one or more chipped teeth, and 10% had enamel hypoplasias, which are indicators of metabolic stress due to malnutrition or disease (Hutchinson 2002:88-89, 92). Hutchinson found that the outer coastal populations had cribra orbitalia and porotic hyperostosis rates that averaged 44%, while the inner sites averaged 20% (Hutchinson 2002:97). Usually maize diets, such as those in the inner coastal sites, are associated with high levels of anemia due to the phytates present in maize that block the absorption of iron (Stuart-Macadam 1987). This would lead to the assumption that the inner coastal sites would have higher rates of anemia, and thus higher rates of cribra orbitalia and porotic hyperostosis, than the outer coastal populations, who rely mainly on foraging. Hutchinson and colleagues (2007:62) have interpreted the anomalous pattern seen in the coastal sites as due to parasitic intestinal bleeding from outer coastal populations' mainly marine diets. Similar studies of Peruvian populations found higher rates of porotic hyperostosis in coastal populations than in terrestrial populations. The higher coastal rates of cranial lesions were explained by higher rates of intestinal parasites, as evidenced in coprolite analysis (Verano 1992).

In addition, the inner coastal adults and subadults exhibited the same prevalence of porotic hyperostosis. However, subadults in the outer coastal populations exhibit a lower frequency of cribra orbitalia and porotic hyperostosis (9%) than do adults (36%) in their community. The unusual pattern led Hutchinson to conclude that possibly subadults were given starchy cereal as weaning foods, rather than seafood that the adults were consuming (Hutchinson et al 2007:55). None of the Middle Woodland individuals exhibited porotic hyperostosis, which may be due to the lack of maize during this period. The lack of European contact also may explain this pattern, as several studies have shown that rates of porotic hyperostosis increased after contact (Larsen et al. 1992, Klaus and Tam 2009). Understanding the etiology of the cranial lesions can shed light onto the reasons behind the unexpected patterns of cribra orbitalia.

Summary

This study reassessed cribra orbitalia in coastal North Carolina Algonkian populations using non-destructive methods to view the internal structure of orbital bone. Although the Algonkian populations have previously been studied and cribra orbitalia documented, it was only performed using macroscopic techniques. By reassessing this population using more a more advanced methodology, a more accurate representation of the population was achieved. The use of CT scanning in this study not only added to the field of Native American history, it also helped to increase the applicability of this method to the field of paleopathological research.

The results from this study answered a number of important methodological and archaeological questions. First is: how different are the frequencies of cribra orbitalia using only macroscopic analysis versus analysis using CT scans? Second, is nutritional deficiency the main cause of cribra orbitalia in this sample, as proposed by Hutchinson (2002), and what frequency of orbital lesions had other causes? Additionally, what are the new demographic profiles in terms of age and sex of the individuals with a confirmed diagnosis? Are the unexpected demographic patterns noted by Hutchinson (2002) still seen?

I expect that the use of non-destructive microscopic methods, such as CT scanning will work as well as more traditional histological examination methods, such as thin-ground sectioning. Due to the use of computed tomography rather than visual observation used in the previous study, I believe that the

frequencies of lesions that show diploic expansion versus porosity, or lesions attributed to anemia versus nutritional deficiencies will vary from the previously reported frequencies.

By more accurately understanding the various etiologies of cribra orbitalia among this population, we can better understand their diet, their interaction with their marine environment, their quality of life, their susceptibility to diseases and nutritional deficiencies, and their overall health status. Currently, not much is known about the health and diseases experienced by these populations, but this research can add to the overall idea of how the Algonkian people lived their lives along the North Carolina coast. The results of this study have implications for the current interpretations of malnutrition and infectious disease in prehistoric human populations, in addition to the fields of paleopathological methodology, archaeology, and Native American history.

CHAPTER 3: MATERIALS

This study examined five North Carolina coastal Algonkian ossuaries. The five sites that were used include: Baum (31CK9), Hatteras Village (31DR38), West 2 (31CK22), Knott's Island (31CK24), and Hollowell (31CO5) (Figure 2). All of the ossuaries from these sites date to the Colington phase of the Late Woodland period (AD 800-1600) and were excavated between 1972 and 1987. The preservation of the skeletal remains is poor, with most of the sample being highly fragmented and fragile. The ossuary sample should be representative of the population as a whole, as all ages and sexes are represented at all of the sites used. It is possible that subadults are underrepresented in this sample, as only 36 crania were identified as subadults, although these 36 crania are 20% of the total sample.

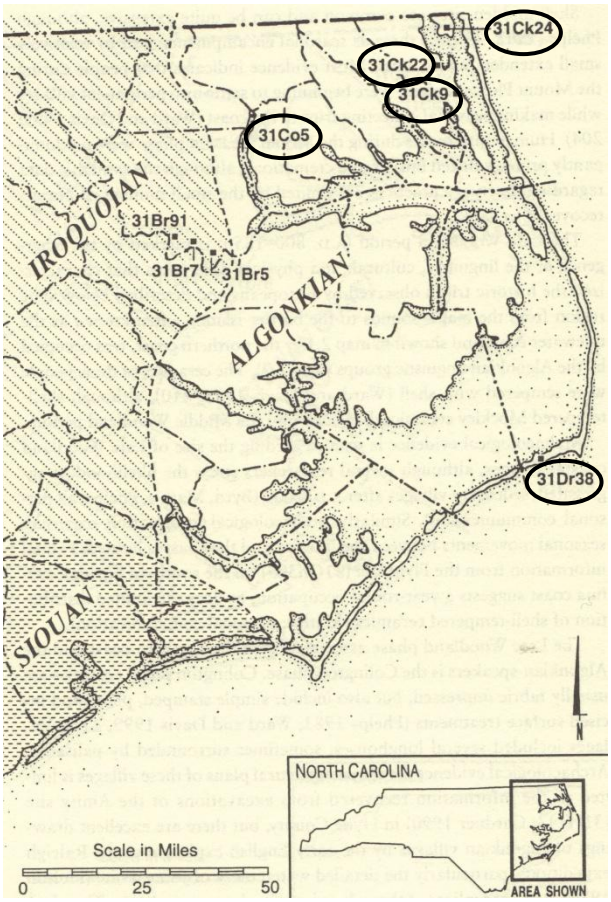

Figure 2. Map of North Carolina coastal Algonkian sites used in this study (Hutchinson 2002).

Sites Used in the Current Study

The Baum site includes five separate ossuaries in addition to several single and multiple burials. The majority of the burials were excavated by David Phelps between 1972 and 1987, with the exception

of Burial 8 which was excavated in 1987 by Mark Mathis. Multiple radiocarbon dates from bone were obtained from the various burials and range from AD 360 +/- 65 for Burial 2, to 1400 +/-60 for Burial 1 (Hutchinson 2002:32-33). The entire Baum site is comprised of Norfolk loamy fine sand and is about five acres in size, although it is estimated that 3-6 meters immediately around the ossuaries have been lost to erosion. It was during the Colington phase of the Late Woodland period that Baum reached its maximum population size as a permanent village, although occupation extends prior to 300 BC. Artifacts that have been found throughout the site include: Colington ceramic shell-tempered ware, Roanoke projectile points, lithic tools, pipes, beads, milling stones, and bone implements.

Burial 1 was the first ossuary to be positively identified as Algonkian in coastal North Carolina when test pits were dug in 1972. This burial included 58 individuals, including eight fully articulated skeletons. Associated artifacts include a panther mask, bone awls, and bone pins. In 1974, the rest of the ossuary, which was three meters from the edge of a bank along Currituck Sound, was excavated. In 1980, it was discovered that the edges of the excavation had become eroded, exposing another separate ossuary (Burial 5) that was then excavated. Burial 2 was a Middle Woodland cremation excavated in 1973 and was not included in this analysis. Burial 3 was located underneath Burial 2 and is an individual flexed burial dating to the Middle Woodland period (Hutchinson 2002:34). Burial 4 was not included in this study, but is a fragmentary subadult burial also excavated in 1974. Burial 5 had approximately 30 individuals, including three articulated skeletons, although more individuals were likely lost due to the erosion of an estimated one-third of the ossuary. In addition to the skeletons, a small necklace, 15 marginella shells, and one disc-shaped copper bead were found near a group of subadult skeletons. Food remains, including 47 fish bones, 37 mammal bones, six bird bones, six turtle bones, and 50 unidentifiable fragments, were also found within Burial 5 (Hutchinson 2002:32-33). Burial 6 was salvaged in 1983 after the ossuary eroded onto the beach. It is radiocarbon dated to AD 1310 +/- 40. Burial 7 is another Late Woodland ossuary that was exposed due to erosion. Four of the crania from this burial show evidence of red-staining. Burial 8 was dated to the Middle Woodland period based on the Mount Pleasant ceramics found within the burial fill (Hutchinson 2002:36).

The site of Hatteras Village was discovered when skeletons began washing out of the beach. Fifteen individuals, along with pottery fragments, were surface collected from the beach below the high

tide line and the water in early 1974. It was estimated at that time that over half of the burial pit was still present (Site Notes, 1974) under the water. Beginning in April 1974, the remaining ossuary was excavated. Burial 1 was approximately two by three meters and the skeletal remains were located approximately 40 cm below the surface (Site Notes, 1974), and collected an estimated 18 individuals. The total MNI for this site is 38. A single radiocarbon date of AD 1350 +/-70, calibrated to AD 1395, was obtained from human bone (Hutchinson 2002:36).

The initial notification of the West 2 ossuary came in late 1991 from the landowners who discovered the skeletal remains of approximately 2-3 individuals eroding out of a 12-foot high bank after a storm. When Mark Mathis went out to inspect the ossuary, it was determined that further excavation would likely exacerbate the severe erosion problem along the bank. To avoid this problem, the property owners were instructed to collect the skeletal remains as they eroded out of the bank rather than excavating the ossuary. However, after it was discovered that several other people had been taking the bones from the beach, excavation was deemed necessary. The first excavation took place in May 1994, with the conclusion of the salvage excavation occurring in October (Mathis 1994). During the initial excavation, approximately 50 cm of the west two-thirds of the ossuary collapsed onto the bank, after which several individuals were collected along the bank (Mathis 1994). It was noted by the excavators that 13 partially articulated bundles were placed in the south area of the burial pit, while the remains located in the center of the ossuary were highly commingled. They proposed that the bundles were placed in the ossuary first, followed by the disarticulated remains. Another possibility is that the center of the ossuary was “stirred”, that is, that the bones were placed in the ossuary and then mingled together (Mathis 1994).

This ossuary is actually the second burial pit for the West site. Burial 1 is located approximately 100 m west of Burial 2. Burial 1 was excavated in October 1984 by the Office of State Archaeology after remains were discovered eroding out of the bank. The remains of at least five individuals deposited as discrete bundles were recovered, although the actual number of remains is unknown due to the bulk of the ossuary having already eroded into the sound.

Red cordage stains were present on several crania from this ossuary. In addition to the skeletal remains, bone pins were found within the ossuary. These pins are similar to those found within other

coastal ossuaries, such as the Baum site (Hutchinson 20002:37). Colington ceramics were recovered from above the burial pit (memorandum 1984), which dates the ossuary to the Colington phase of the Late Woodland period.

The Knott's Island ossuary was exposed during road construction near the town of Knott's Island in 1989. At least 29 individuals were excavated by Mark Mathis in 1989. While no radiocarbon dates were obtained from this site, shell-tempered pottery sherds indicate a Late Woodland Algonkian cultural affiliation and date (Hutchinson 2002:37).

Hollowell is the only inner coastal site used in this study. It is located on a 30-foot high bluff along the Chowan River. The site was first reported in July 1974 when the landowner noticed skeletal remains eroding out of the surface (Phelps 1982: 25). Excavation was conducted by David Phelps of East Carolina University between June 23 and July 17, 1975. The burial pit was roughly rectangular, and measured 3.6 m east-to-west and 2.5 m north-to-south. The original depth of the ossuary could not be determined because of continual topsoil grading and removal, however, the remains began 48 cm below subsoil level (Phelps 1982: 28). Approximately 40 individuals were excavated in nine different groups. Phelps suggested that the separate groups within the ossuary represent different family units, thus estimating that the village contained at least nine longhouses during the Colington phase. Phelps also suggested that the other coastal ossuaries probably represent a similar pattern, but the overall size of the burial pits were too small and masked the familial pattern. In addition to the skeletons, the ossuary contained only a small necklace of four conch columella beads associated with an infant (Phelps 1982: 38).

Using the prevalence of certain artifacts, occupation began in the Middle Archaic period, when the site probably functioned as a seasonal base camp, and it continued through the Middle Woodland period. The higher frequency of Colington phase ceramics suggests that the most intensive use of the site occurred during this period. Phelps suggested that the site functioned as a small permanent village during this time as evidenced by a midden deposit, Cashie ceramics which he interpreted as extensive trade with the nearby Tuscarora, and the ossuary (Phelps 1982:27). A single radiocarbon date obtained from bone dates the heaviest occupation of the site to AD 1460 +/- 60 years (Hutchinson 2002:39).

Demography of the Sample

A total of 232 crania from all five sites were initially observed in this study. Forty-nine of these 232 crania (20%) have non-observable orbital regions and thus could not be included in this analysis. The assessment of the remaining 183 crania discovered that 46 had active cribra orbitalia (20%), 42 had healed lesions (19%), and 3 had both healing and active lesions at the time of death (1%). Ninety-two of the crania did not show any signs of porosity on the orbital roof (40%).

The demographic patterns of the total sample of 183 crania are shown in Table 1. Twenty percent of the total sample were subadults, 14% were young adults aged 20-34 years of age, 47.5% were middle aged adults 35-50 years of age, only one cranium (0.005%) was over 50 years of age. Among the adults, 8.5% were not complete enough for a precise age estimation beyond "adult". Sixty-two of the crania (34%) were female, 19% were male, 34% were subadults and thus sex estimation was not completed, and 28% were adults of indeterminate sex. The range of variation within the demographic profile should not affect the outcome of the results as all age ranges are adequately represented, except for the 50+ category. The sex distribution of the sample is also representative of the population as a whole, and should not affect the interpretations of the data.

Of the 183 remaining crania from all five sites, 50 were randomly chosen for further analysis using CT scanning. The 50 crania sampled were a mix of healing, active, and no lesions and crania from all five sites were included. This subsample has similar demographic patterns to the overall sample (see Table 2). The results of the CT scanning will be discussed further in later chapters.

Table 1**.** Age and sex of the total sample

Age	Male	Female	Indeterminate	Total
<15 years	--	--	36	36 (19.5%)
20-34	2	15	8	25 (14%)
35-50	23	35	29	87 (47.5%)
50+	0	1	0	1(0.005%)
Adult	10	10	14	34 (19%)
Total	35 (19%)	62 (34%)	86 (47%)	183 (100%)

Table 2. Age and sex of the CT-scanned crania

Age	Male	Female	Indeterminate	Total
<15 years	--	--	13	13 (26%)
20-34	0	5	3	8 (16%)
35-50	6	12	8	26 (52%)
50+	0	0	0	0
Adult	0	1	2	3 (6%)
Total	6 (12%)	18 (36%)	26 (52%)	50 (100%)

CHAPTER 4: METHODS

A number of macroscopic techniques were utilized in this study to assess orbital roof pathologies in the Late Woodland Algonkian coastal North Carolinian assemblage. Preliminary macroscopic analysis was conducted in order to determine age, sex, and the presence and severity of cribra orbitalia and porotic hyperostosis. After the initial macroscopic analysis, 50 samples were chosen to be explored further using computed tomography (CT) scans. The CT scan slices of the pathological areas were digitally enhanced and analyzed visually and patterns of radiopacity were quantified in order to identify clustering or patterning based on changes in the internal structure of the orbital roof.

Demography Collection

Age estimation for each of adults among the 232 crania was based on suture closure scores as described in Buikstra and Ubelaker (1994). Cranial sutures generally fuse with increasing age, although there is considerable individual variability (Buikstra and Ubelaker 1994:32) which leads to large possible age ranges. This high degree of variability reduces the value of suture fusion rates for estimating age, but the method has still proven useful in cases where other methods are not available (Masset 1989). While there are many more accurate methods for aging adult skeletons, cranial suture closure was used because no associated post-cranial remains were available. Subadult skeletons were aged using tooth eruption patterns when present, and through epiphyseal union if associated postcranial remains existed (Buikstra and Ubelaker 1994). Teeth typically follow a similar eruption pattern, which makes this method highly accurate for aging subadult skeletons. The sequence of tooth eruption used in this study follows Ubelaker (1989) as shown in Buikstra and Ubelaker (1994).

Some error in the estimation of age is inherent with archaeological populations, as the rates of degenerative change are unassociated with chronological age and vary with individuals. In addition, age-estimation techniques are strongly influenced by the age distribution of the sample used, regardless of actual degeneration rates (Meindl and Russell 1998). Boddington (1987:190) also asserts that age estimation techniques are often based on known modern samples, and researchers must assume that ancient populations both developed and degenerated at the same rates as modern populations. Sex estimation of adults was accomplished by assessing the degree of robusticity found in the superior

margin of the eye orbit, glabella, mental eminence, nuchal crest, and mastoid process when present. The degree of robusticity was determined by the standards laid out in Buikstra and Ubelaker (1994).

Initial Visual Analysis

The orbital lesions first were categorized based on visual observation into four categories: non-observable, not present, healing, and active. Each active or healing lesion was then scored for degree of porosity and diploic expansion. In addition, lesions in the cranial vault often referred to as porotic hyperostosis were scored. The scoring system from Buikstra and Ubelaker (1994), adapted from Stuart-Macadam (1985), was used for the initial macroscopic analysis, although other scoring methods have been developed (e.g. Hengen 1971, Schultz 1988, Mittler and van Gerven 1994). This system uses a 4-point scale to determine the severity of the lesions (Figure 3). A score of 1 indicates indistinct porosity, or a barely discernable lesion. A score of 2 indicates a true porosity of the orbital roof. A score of 3 occurs when the foramina have begun to coalesce, but the bone has not begun to thicken. A score of 4 also occurs when the foramina have coalesced, but this occurs in association with the thickening of the cranial vault (Buikstra and Ubelaker 1994). In addition, there are three codes for the lesions that describe the degree of healing: 1) sharp edges and woven bone of active lesions, 2) remodeled, sclerotic changes, and 3) mixed reactions (Buikstra and Ubelaker 1994:121).

Computed Tomography

After the macroscopic examination was complete, 50 frontal bones were chosen to be examined further using computed tomography. Five of the sampled orbits did not have any lesions present, and the remaining 45 orbits represented the initial visual observation severity scores ranging from 1 to 4. The computed tomography scans were completed free of charge by the Department of Cardiovascular Sciences at the Brody School of Medicine, East Carolina University using a Siemens SOMATOM definition scanner. The scans were executed using the “inner ear” protocol, which was then modified by the scanning technician and the consulting radiologist. The spiral scan settings were set to 1 pitch, 180 milliamps per second, which is the number of electrons put out by the scanner, and 80 kv, which is the amount of force the electrons have. The medial-lateral (M-L) slices were 0.6mm thick as this is the thinnest setting available while still maintaining clarity of the images with this scanner.

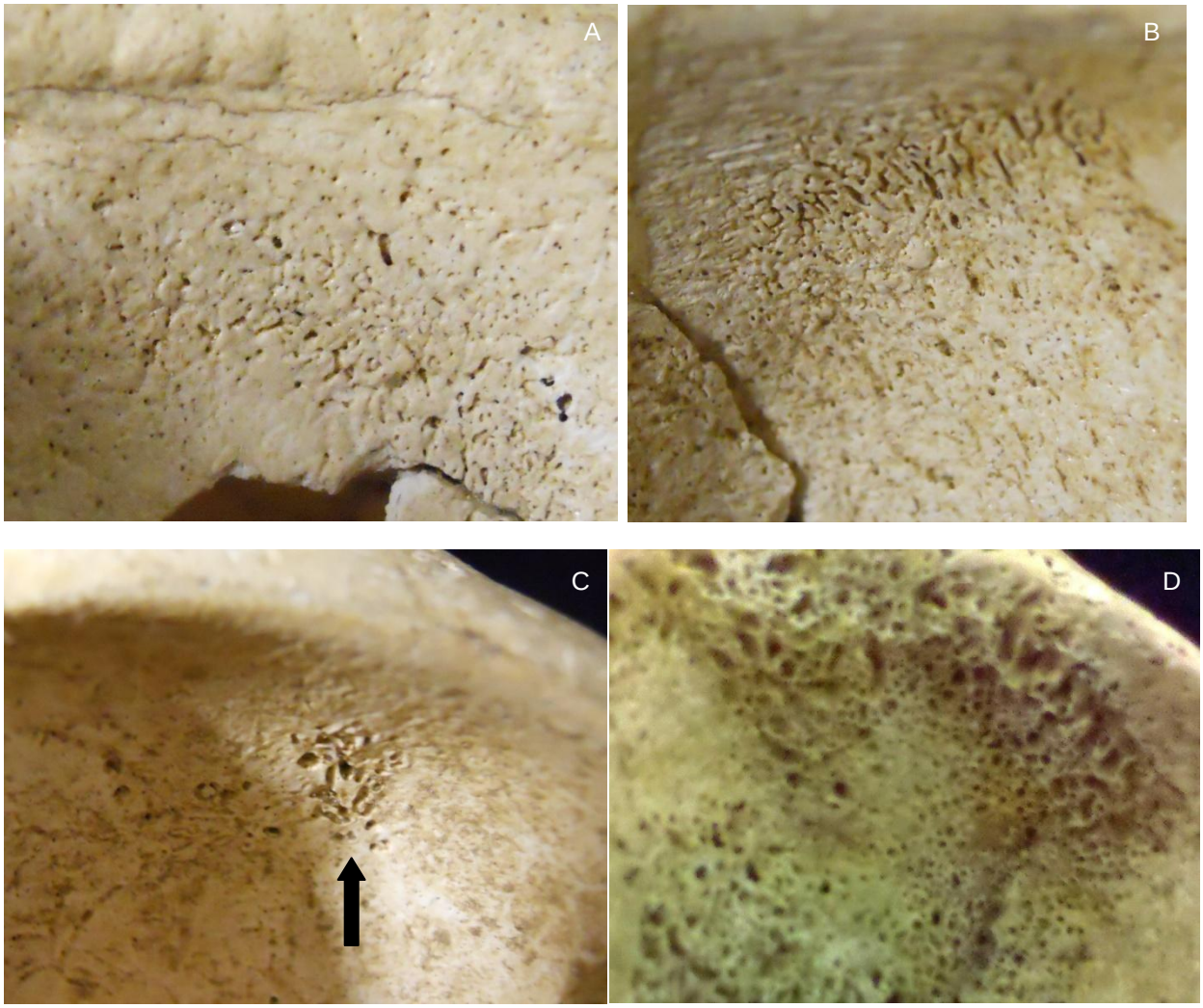

Figure 3. Initial macroscopic cribra orbitalia scores. (A) 31CO5 1Q left orbit showing indistinct porosity- score 1(B) 31CK9 1RR right orbit showing true porosity, score 2 (C) 31CK9 1E left orbit showing coalescing foramina without concurrent diploic expansion, score 3 (D) 31CO5 1A left orbit showing diploic expansion, score 4

Each skull was placed into the scanner in Frankfurt horizontal plane to produce M-L slices of the skull, and depending upon the size of the fragment, cushions were used to help stabilize the sample. A topogram, or a lateral x-ray of the sample was first taken in order to align the scanner and set up the axial slices. After the scanner was properly oriented, the computed tomography scan was taken. A recon kernel- u75uverysharpasa- was developed for use in these scans in order to reconstruct the data into meaningful images.

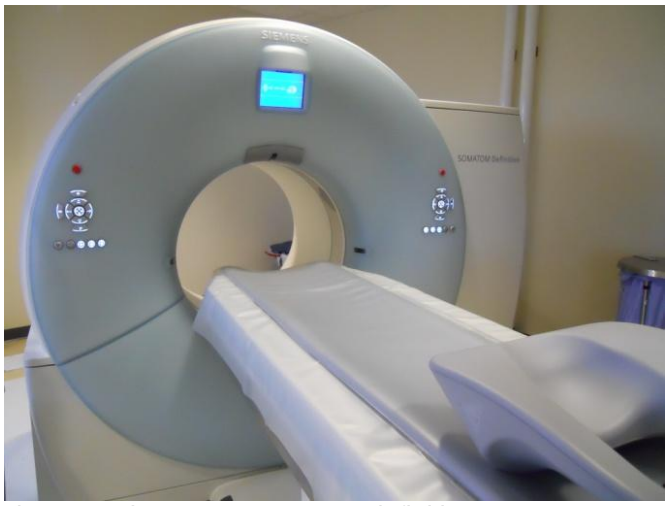

Figure 4. Siemens SOMATOM definition scanner

Figure 5. Sample 31CK9 1PP sample placed on scanner bed.

Following the completion of all 50 CT scans, a single M-L slice was selected from each orbit for analysis and saved as a .jpg file. The images were selected using Syngo Viewer software while referencing the cranium itself in order to ascertain the best axial slice image of the pathology (if present). Due to the relatively small area of the orbital region compared to the rest of the skull, each image was magnified 5 times for a total of 6.25 magnification in order to have the best image of the orbital area itself. While referencing the crania, each sample was given another visual observation code between 0 and 4 for statistical comparison. These codes differ from the initial visual observation codes in order to assess whether the diploic expansion was healing or active and to account for postmortem erosion. The initial observation scores were meant to determine degree of porosity, whereas these scores for statistical analysis merely determine whether porosity or diploic expansion is present, regardless of degree. A code of 0 was given for those orbits without lesions, porosity in the cortical bone was scored a 1 (similar to the above scores 1, 2, and 3), healing diploic expansion was scored a 2, a score of 3 is active diploic expansion (both diploic expansion codes are similar to the above score of 4), and postmortem erosion was scored a 4 (see Figure 6).

Medical image enhancement was then completed on the CT scan slices using Adobe Photoshop CS4 in order to complete further visual image assessment of the lesions. The .jpg images were converted to grayscale rather than the color images. The magic wand tool was used to enhance the outer table porosity; the tolerance level was set to 15 and the pixel selection criterion was set to non-

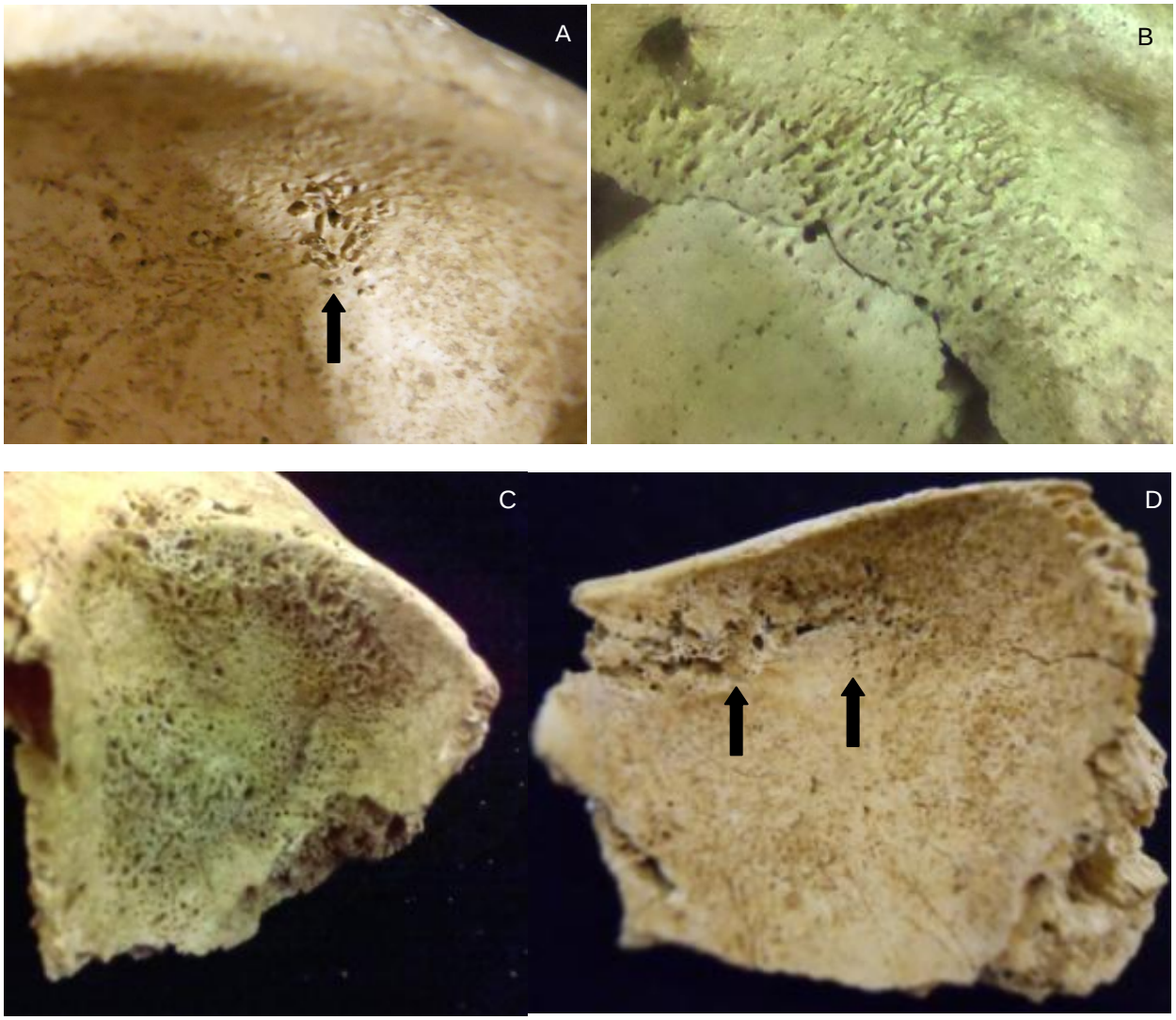

Figure 6. New visual observation scores. (A) 31CK9 1E left orbit showing porosity- score 1 (B) 31CK9 5D left orbit showing healing diploic expansion-score 2 (C) 31CO5 1A left orbit showing active diploic expansion-score 3 (D) 31CK9 1W left orbit exhibiting postmortem changes- score 4

contiguous. This means is that any pixel within 15 grayscale values of the selected pixel would be highlighted whether or not it was contiguous with the selected pixel. Then, the refine edge tool was used to enhance the contrast of the images to 32, and the brightness was set to 65. The settings were the same for each scan, and two different pixels values were selected for enhancement from each orbit.

Using the enhanced images, each sample was reassessed. A new coding system was used for these images. If the cortical bone was intact, the sample was given a score of 1. If the cortical bone had porosity, the sample was scored a 2. A score of 3 was used if the cortical bone was resorbing and diploic expansion was present (Figure 7).

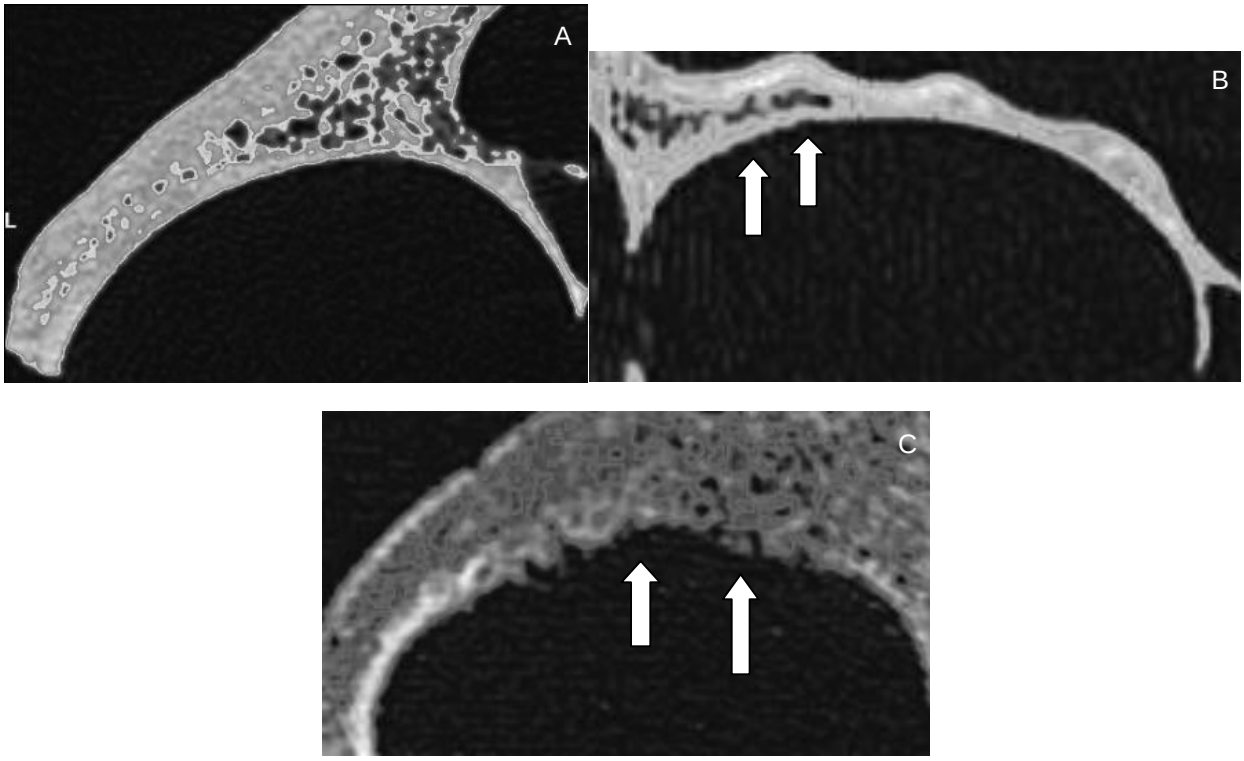

Figure 7. CT visual enhancement score examples. (A) 31CK9 1M left orbit showing intact outer table with no porosity- score 1 (B) 31CK9 1E right orbit showing porosity in cortical bone- score 2 (C) 31CO5 1A left orbit exhibiting diploic expansion- score 3

Statistical Analysis

To collect quantitative data on the density of the orbits, which reflects the intactness of the cortical bone of the outer bone layer of the upper orbit in addition to the nature of the diplöe in the internal structure, the Andromeda filter in Adobe Photoshop was used. A total of six cross sections of different sectors of each orbital roof were selected (see Figure 8 for an example) and then averaged to capture the overall internal structure of the cortical bone and diplöe of each orbit. This was done by drawing three boxes across the orbital roof in each image, which produced two cross-sections per box (the two vertical sides of the box). The Andromeda filter captured the density data of each cross section to produce a density histogram (Figure 9). The grayscale values from each cross section were imported into Microsoft Excel 2007 spreadsheet for statistical analyses.

First, the data were cleaned in Excel by deleting grayscale values reflecting the radiolucent space within the selected area (that is, just “air” instead of a solid object) and up to the actual orbital roof. A cut-off grayscale value of 30 was determined to reflect the beginning of the actual orbital roof cortical bone.

Figure 8. Screen image of Andromeda boxes on left orbit of 31CK9 1AAA.

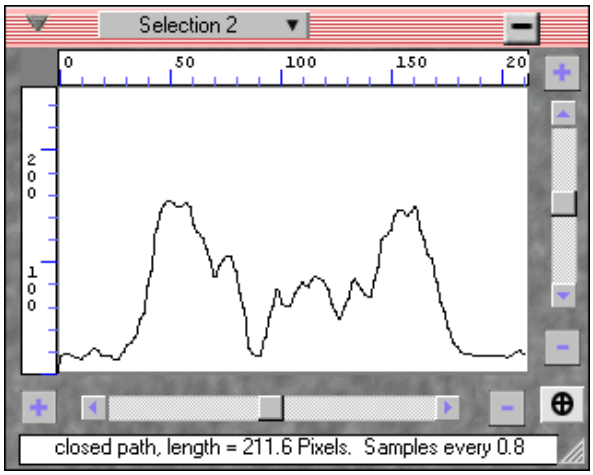

Figure 9. Screen image of outline profile of highlighted box in Figure 8.

Therefore, all values in the cross section were deleted until the values reached the "30" threshold. Finally, the first 18 grayscale values running from the outer orbital roof into the diploic space were retained for analysis for each observation.

The averaged grayscale values from the six cross-sections of each orbit were then subjected to statistical analyses using SAS version 9.2 to see if the values significantly clustered together. Adults and subadults (<15 years of age) were tested separately to control for differences in cranial thickness. First, a MANOVA test was performed to see if certain points along the orbital cross-sections varied meaningfully based on the unenhanced visual observation and the CT enhanced image codes. This information would determine where on the cross-section the greatest variation existed between the visual code categories to see if the dataset could be reduced in a meaningful way. Then, cluster analysis using Ward's method in PROC CLUSTER was run on the grayscale values to identify patterning of values across the cross-section (which presumably would indicate differences in radiopacity, and hence pathological processes, across groups). Cluster analysis was run on the entire sample of grayscale values and a trimmed sample of the first 6 values identified by the MANOVA test as varying most significantly between the visual observation codes. Based on the relationships identified by the cluster analysis, each orbit was then assigned a cluster identification. The distribution of the observations amongst the clusters was produced by the PROC SGPLOT procedure, which plots the first and second canonical coefficients of each cluster in order to visually observe the cluster groupings. Finally, a Fisher's exact test was conducted using the

PROC FREQ in SAS to assess the relationship between the cluster groupings and the visual observation codes from plain observation and observation of digitally-enhanced images. This allows the determination of whether the grayscale value patterns are similar to the patterns seen in the visual and digital observations. The Fisher's exact test is similar to a Chi-Square test, although it is a more robust test more suitable to smaller sample sizes.

Comparisons to Previous Analysis

After the results of the current study were compiled, they were compared to Hutchinson's (2002) previous analysis. The data from this study were arranged in a similar fashion to Hutchinson's compilation (i.e., combining cribra orbitalia and porotic hyperostosis into one category) in order to facilitate comparisons. The rates of cranial lesions were compared for the overall sample and broken down by adults and subadults.

CHAPTER 5: RESULTS

The multiple techniques used in this study allowed for a reassessment of orbital lesions in a Late Woodland Algonkian sample. While a previous analysis of these lesions has been completed (Hutchinson 2002), this study produced slightly different results, which will be discussed further below.

Initial Visual Observations

Half of the total sample of crania from the five sites (N=183) did not exhibit any lesions in either orbit. When broken down by adult and subadults, it was found that out of 147 adult crania, 62 (42%) had lesions, while 29 out of 36 subadult crania (80.5%) exhibited cribra orbitalia (Table 3). Of the 91 crania exhibiting orbital lesions, 32% were subadults and 68% were adults. Fourteen percent of the individuals with cribra orbitalia exhibited a score of 1, 23% had a score of 2, 11% had a score of 3, and 4% had a score of 4 (Table 5).

Table 3. Cribra orbitalia by adults vs. subadult samples

Age	with lesions		No lesions		Total	
	N	%	N	%	N	%
Adults	62	42%	85	58%	147	100%
Subadults	29	80.5%	7	19.5%	36	100%

Data on porotic hyperostosis were collected to determine how often the two pathologies coexisted in the same crania. Only 148 samples had enough cranial bones to collect information on both cribra orbitalia and porotic hyperostosis (Table 4). Twenty percent of the sample had both cribra orbitalia and porotic hyperostosis, while 30% of the sample did not. Twenty-eight percent of the sample had only cribra orbitalia without porotic hyperostosis, and 22% of the crania had porotic hyperostosis without concurrent cribra orbitalia.

Table 4. Coexistence of cribra orbitalia and porotic hyperostosis

	Cribra Orbitalia only		Porotic Hyperostosis only		Both Present		Neither Present	
	N	%	N	%	N	%	N	%
Adults	21	14%	32	22%	26	18%	39	26%
Subadults	20	14%	0	0%	4	2%	6	4%
Total crania with lesions	41	28%	32	22%	30	20%	45	30%
Total Observable	148		148		148		148	

Table 5. Cribra orbitalia and porotic hyperostosis scores in the total sample

	Cribra Orbitalia		Porotic Hyperostosis	
Score	**N**	**%**	**N**	**%**
Not observable	N/A	N/A	35	19%
No lesions present (0)	92	50%	86	47%
Indistinct porosity (1)	25	14%	35	19%
True porosity (2)	42	23%	22	12%
Coalescing foramina (3)	20	11%	4	2%
Diploic Expansion (4)	4	2%	1	1%
Total	183	100%	183	100%

Forty-seven percent of the crania did not show any signs of porotic hyperostosis. Of the 62 crania that did exhibit porotic hyperostosis, 35 showed a severity score of 1, 12% had a score of 2, 2% scored a 3, and only 1 cranium had a severity score of 4 (Table 5). In addition to the severity of the porotic hyperostosis, the degree of healing and the location of the porosity were scored (Tables 6 and 7). The majority of the lesions were either healed or in the process of healing (81%), while only 11% were active lesions, and 8% of crania had both healing and active lesions. The degree of healing can lend information about whether the deficiency that caused the lesions was still active at the time of death and how long it has been since the individual overcame the etiology. The majority of the porosity was located only along the sutures (78%), followed by lesions on the parietal and occipital bones (19%), while only 3% had lesions on the frontal, parietal, or occipital bones not along the suture. The areas where the porosity is located can sometimes help researchers determine the possible causes of the lesions. For example, if there is only one area of porosity in the middle of the occipital bone, it is possible that the pathology is due to a hematoma or some other trauma to that one area of the skull.

Visual and Digital Analysis of Subsample

For the subsample of 50 CT scanned crania, additional observations were taken visually and using digitally-enhanced CT images (Table 8). Each orbit was treated as a separate sample, as some

Table 6. Degree of healing in porotic hyperostosis lesions

Degree of Healing	Number	Percentage
1- Active	7	11%
2- Healing	50	81%
3-Mixed	5	8%
Total	62	100%

Table 7. Location of porotic hyperostosis lesions

Location of Porosity	N	%
Along suture lines	48	78%
Frontal, parietal, or occipital bones not along sutures	2	3%
Parietal or occipital bosses	12	19%
Total	62	100%

crania had only one orbit preserved while other crania had both orbits preserved. Of the 50 scanned crania, there were 73 total orbits, 36 left and 37 right.

Four percent of the 73 orbits were visually scored as having no porosity, but only 2% of the orbits showed a lack of pathology on the digital images. Fifty-five percent of the orbits showed visual porosity, which is similar to the 58% of orbits showing porosity on the digital images. For the visual assessment diploic expansion was scored as either active or healing, however healing was not possible to note on the digital images. Forty percent of the orbits using the digital images showed diploic expansion, which is the same percentage that was scored as both healing and active diploic expansion in the visual assessment. Only one orbit showed post-mortem erosion visually, but it was not coded as such in the digital observation. The similarity between the two assessments shows that in most cases visual analysis was as good as computed tomography analysis.

Table 8. Pathology codes for CT-scanned crania by orbit

Score	Visual Observation		Digitally-enhanced CT image observation	
	N	%	N	%
No pathology	3	4%	2	2%
Porosity only	40	55%	42	58%
Diploic expansion	19	26%	29	40%
Diploic expansion (active)	10	14%	N/A	N/A
Postmortem erosion	1	1%	N/A	N/A
Total	73	100%	73	100%

Analysis of Orbital Cross-Sections

The cross-sections for each eye orbit were analyzed using MANOVA, cluster analysis, and Fisher's exact tests. The results of each statistical analysis will be discussed in detail below.

MANOVA Results

The cross-section grayscale value dataset was subjected to MANOVA to find where along the orbital cross section (i.e., the distance from the orbital roof outer table) the greatest variation in grayscale values occurred between the visual observation scores and digitally-enhanced CT image scores. This assessment found that significant variation across the visual observation groups in adults existed in the 5th and 6th grayscale values from the orbital roof outer table. No significant variation in grayscale values along the cross-section was found comparing the digitally-enhanced CT scores in adults. The results among the subadult sample paralleled the adult sample, with notable variation in the 3rd - 5th grayscale values from the orbital roof edge between the visual scores and no significant variation in grayscale value patterns between the digitally-enhanced image scores.

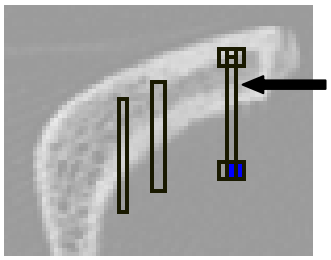

Figure 10. Location of the 5th grayscale value (estimated) on digital image of sample 31CK9 1AAA

Cluster Analysis

To organize the grayscale values into significant groups, cluster analysis was run using Ward's method in PROC CLUSTER. Both the adult and subadult samples were run twice, once with all of the grayscale values (variables) and again with a trimmed dataset of only the first six variables based on the results of the MANOVA test. Cluster analysis will group observations together based on similarities and/or differences between a series of numerical values associated with each observation. Based on the similarities of their grayscale value patterns, each orbit was assigned to a cluster. However, the cluster analysis for both datasets did not meaningfully explain the variation found within this sample.

For the adult sample, the number of suggested clusters for the complete dataset was five, and six clusters were suggested for the trimmed dataset. This means that the grayscale values are best

described by separating them into five different groups when all of the variables are taken into account. When only the first six variables are grouped, six different clusters best explain the variation. For the subadult sample, three clusters were suggested for the complete dataset, and only two clusters best described the variation within the trimmed dataset.

The PROC SGPLOT procedure produced plots of the canonical coefficients by cluster to visually show how the grayscale values group together (Figures 11 and 12). It is clear that none of the different datasets – adult or subadult, trimmed or complete – fit into well-defined clusters. This may indicate that the grayscale values cannot discriminate between different pathological processes, which can be tested partially by comparing the clusters to macroscopic and digitally-enhanced CT image scores.

Comparing Grayscale Clusters to Macroscopic Observation Scores

Fisher's exact tests were used to compare the cluster identifications based on the grayscale values with the codes derived from visual means. The Fisher's exact test found no significant relationship between the grayscale clusters and either the visual observation or the digital image codes for adults or subadults at the $p<0.05$ level. However, there is a significant relationship between the two means of visual observation, the macroscopic-based codes and the digital image codes, in the adult samples ($p= 0.0283$) (Tables 8 and 9). Skulls visually coded as porosity also tended to be scored as having porosity using the digital CT images (44% of the sample). Twenty-percent of the sample was scored visually and digitally as having diploic expansion. However, 18% of the sample was visually coded as diploic expansion, but digitally coded as porosity. For subadults, there were no statistically significant patterns seen at the $p<0.05$ level.

Table 9. Comparison of CT enhanced and visual observation scores

	Digitally-Enhanced CT Image Scores			
Visual Scores	1	2	3	Total
0	1 (1.69%)	1 (1.69%)	1 (1.69%)	3 (5.08%)
1	1 (1.69%)	26 (44.07%)	8 (13.56%)	35 (59.32%)
2	0	8 (13.56%)	9 (15.25%)	17 (28.81%)
3	0	1 (1.69%)	3 (5.08%)	4 (6.78%)
Total	2 (3.39%)	36 (61.02%)	21 (35.59%)	59 (100%)

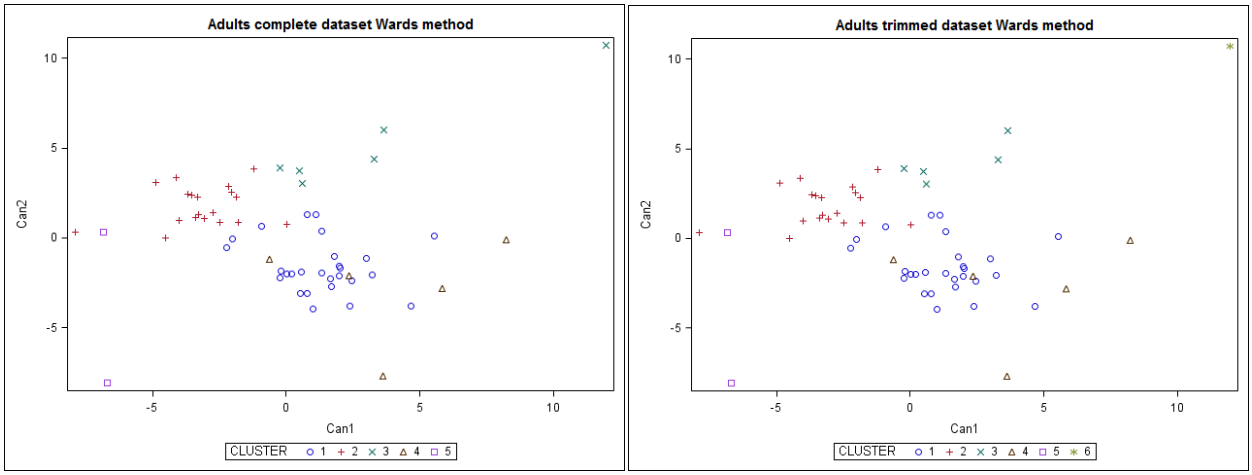

Figure 11. Plots of the canonical coefficients for each cluster in the adult samples (a) complete data set (b) trimmed dataset

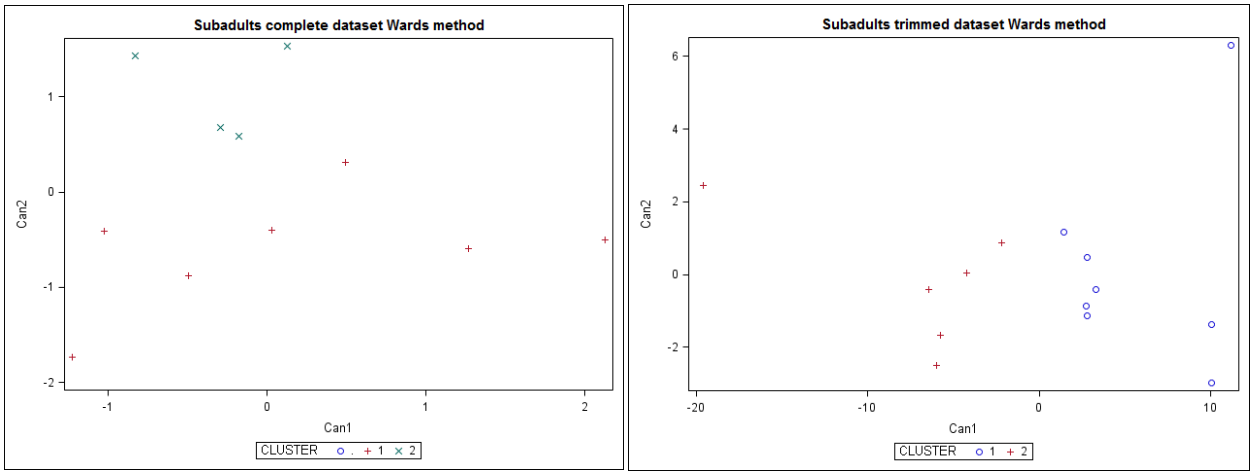

Figure 12. Plots of the canonical coefficients for each cluster in the subadult samples (a) complete data set (b) trimmed dataset

Summary

Half of the total sample exhibited cribra orbitalia- 42% of adults and 80.5% of subadults- while 42% of the total sample showed porotic hyperostosis. Twenty-eight percent of the sample had only cribra orbitalia, 22% had only porotic hyperostosis, and 20% exhibited both cribra orbitalia and porotic hyperostosis. There is a statistically significant relationship between the visual and digital observation codes, with the majority of the discrepancy between the two observations being visually coded as diploic expansion but digitally coded as porosity.

The results of the grayscale value patterning were less clear, however. The MANOVA test showed that the 5th and 6th grayscale values from the orbital roof for adults and variables 3-5 for subadults

were significant with the visual observation codes, while no variables were significantly correlated with the digital observations for adults or subadults. Cluster analysis ran to organize the grayscale values together found that five clusters best described the whole adult dataset, while six clusters described the trimmed adult dataset. Three clusters best described the variation found within the subadult grayscale values, and two clusters best organizes the trimmed subadult dataset. None of these clusters can be clearly identified via a plot of the canonical coefficients, and the cluster analysis did not meaningfully explain the variation found within this sample. Fisher's exact test showed no significant relationship between the grayscale value clusters and either the visual or the digital observations for the adults and subadults. What these results mean in the context of this study will be discussed in the next chapter.

CHAPTER 6: DISCUSSION

This preliminary study aimed to determine the applicability of computed tomography as a method of diagnosing cranial lesions in prehistoric Native American populations. While there is a significant relationship between the visual and the digital observation scores, this study was unable to prove that CT scanning images showing the internal structure outweighed simple macroscopic visual observation. In some instances, it was shown that the digital images showed merely porosity rather than the visually assessed diploic expansion. In the majority of cases, however, the two observations were the same. Further studies using computed tomography must be conducted in order to determine the accuracy and applicability of computed tomography to the diagnosis of cranial lesions, as discussed below.

Interpretations of the Statistical Analyses

The statistical analysis returned some unexpected results. The statistically significant relationship between the visual and digital assessments of the lesions shows there was minimal inter-methodological difference, although the positive correlation, or similarity, between the codes was unexpected. The expectation was that the use of computed tomography would show more detail in the orbits than simple visual observation, and thus would provide a different diagnosis.

In some cases, computed tomography did show more detail than simple visual observation. In 44% of the subsample, both the visual and the digital observations scored as porosity and 20% of the subsample had observations using both methods scored as diploic expansion. The high rate of agreement between the scores shows that the CT images are capturing what researchers are seeing visually. However, in 18% of the sample, the lesions were visually coded as diploic expansion, but the digital observation showed only porosity. It is not clear which observational method is picking up the correct pathological expression. This difference between the two observation methods shows that in some instances CT images show a different manifestation of the lesions than simple visual observation. Thus, computed tomography should be explored further as a viable alternative to simple visual assessment.

The determination that the most variation in the grayscale values occurred in variables 5 and 6 for the visual observations was expected, as they are located in the diplöe space in close proximity to the

outer table. Since diploic expansion affects the internal surface of the outer table first, it would be expected that those variables in this area would show the most variation across the sample, and thus differentiate between porosity and diploic expansion.

Cluster analysis groups together observations differently for the entire set of grayscale values versus the trimmed set of grayscale values (using only the first six values). However, the clusters did not explain the variation found within the grayscale values in a meaningful way. The fact that the visual and digitally-enhanced observation scores tended not to cluster together was unexpected. While some clusters did tend to have more of a certain observation than other clusters, almost all of the clusters had all observation scores represented. For example, in the trimmed adult sample, cluster 6 had visual scores 0-3 represented, while the 3rd cluster was a much smaller cluster comprised of only codes 1 and 2. It is possible that the grayscale values are measuring something in the pathology other than what is visually detected and thus would account for the differing values within each cluster. It is possible that the subadult sample had fewer clusters than the adult samples due to less overall variation in the grayscale values. A significant percentage (67%) of the subadults had porosity due to cribra orbitalia and 14% exhibited diploic expansion based on visual observation.

The lack of statistical significance between the grayscale values and the digital observations scores was unexpected. Since the digital observations were based on the CT images, which were quantified using the grayscale values, it was expected that the relationship between the observations and the grayscale values would be significant. It is probable that the grayscale values are identifying something in the pathology that is not determined by the researcher.

Possible Etiologies of the Orbital Lesions

The goal of this study was to determine whether CT scanning could be used to diagnose orbital lesions. Using the results from both the visual and digital observations, conclusions about the most likely cause of cribra orbitalia in this population can be drawn. In addition to using the current study's findings, comparisons with Hutchinson's (2002) initial analysis will be made. The main cause of cribra orbitalia in adults is vitamin deficiencies, and in subadults the main cause is anemia. The vitamin deficiencies could be a result of intestinal parasites introduced by the adults' mainly marine diet, as proposed by Hutchinson.

Of the total population sampled in this study, 67% had either cribra orbitalia, porotic hyperostosis, or both. This sample of skulls with lesions includes 94 adult crania (N=147, 64%) and 29 subadult crania (N=36, 81%). The combination of porotic hyperostosis and cribra orbitalia allows for comparisons to be made with Hutchinson's (2002) previous analysis. Hutchison found that for the inner and outer coastal sites, an average of 49% of the adult crania (N=340) exhibited cribra orbitalia or porotic hyperostosis and only 9% (N=98) of subadult crania showed cranial lesions (Hutchinson 2002:98, 206).

Several possibilities may account for the differences in the rates seen in the two studies. Different samples were used in each study. In the current study, only those crania with intact orbits were used, regardless of the remaining cranial bones. However, in Hutchinson's analysis all crania present were used, and therefore those skulls with porotic hyperostosis but a non-intact orbital region were included. Another possibility is inter-observer error between the scoring of minimal porosity. The current study was probably more sensitive to including those orbits with more minute amounts of porosity, whereas the previous analysis may have used a higher threshold when scoring porosity. Additionally, subadults may be underrepresented in the current study compared to the previous analysis, which could have introduced sampling bias.

A major difference between the two analyses is the sites used. Hutchinson separates his analysis based on inner and outer coastal sites, while the current analysis does not seek to do so as Hollowell is the only inner coastal site used. Hutchinson (2002) included 3 other inner coastal sites (Jordans Landing 31BR7, Sans Souci 31BR5, and Dickerson 31BR91) in his analysis. Exclusion of these sites from this study could have introduced some sampling bias, as the inner coastal sites had slightly lower overall rates of cribra orbitalia. The inclusion of Hollowell and the exclusion of the other inner coastal sites into the overall dataset for this research may explain the higher rates seen in this study than in the previous analysis.

Hutchinson and colleagues (2007:62) attributed the main cause of porotic hyperostosis and cribra orbitalia to parasitic intestinal bleeding from the population's mainly marine diet. They based this interpretation on two unique patterns seen upon their assessment of the population. The maize-dependent populations (inner coastal sites) had lower rates of orbital lesions than the marine-dependent outer coastal sites. Although the current study did not compare inner and outer coastal sites, this pattern

noted by Hutchinson is contrary to the usually high rates of anemia seen in maize-dependent societies. Another unique pattern within this population noted by Hutchinson is the lower frequency of lesions seen in subadults than adults. As noted above, cribra orbitalia and porotic hyperostosis are usually interpreted as childhood manifestations of iron-deficiency and subadults often have much higher rates of cranial lesions than do adults (e.g. Stuart-Macadam 1985). This unusual pattern led Hutchinson and colleagues (2007) to conclude that possibly the outer coastal subadults were given weaning foods different from the primary seafood-based diet of adults, therefore decreasing their exposure to intestinal parasites. The combination of the higher rates of lesions in the non-maize-dependent societies and the unusually lower subadult rates of lesions suggests that the lesions are not due to iron deficiency anemia.

As diploic expansion is only caused by anemia, comparisons between the rates of diploic expansion versus porosity can also shed some light onto possible etiologies. Of the total sample initially visually assessed, only 13% showed diploic expansion and 37% had porosity. In the subsample of scanned crania, it was found that 40% (N=29) showed diploic expansion (scores 2 and 3) and 55% (N=40) showed porosity (score 1) using visual observation. The results were similar using the digitally-enhanced CT images, 58% of the orbits had porosity (score 1), while 40% showed diploic expansion (score 2). If we correlate diploic expansion with anemia, it is obvious that more individuals had lesions caused by etiologies other than anemia as proposed by Hutchinson. When broken down by adults and subadults, the percentages change. The visual assessment of adults showed that 59% had porosity (score 1) while 36% had diploic expansion (scores 2 and 3). Thirty-three percent of subadults had porosity, and 60% had diploic expansion. The digital observations for adults showed 61% had porosity and 36% exhibited diploic expansion. The subadults showed 43% had porosity and 57% had diploic expansion.

Based on the above rates of orbital lesions that exhibited diploic expansion versus porosity, it can be seen that the majority of lesions present in adults were caused by something other than anemia. The majority of the subadult lesions show diploic expansion rather than porosity, which can be equated with anemia rather than vitamin deficiency. Even though the Algonkians were eating seafood which is high in iron content (Sullivan 2005), they are still exhibiting high rates of cribra orbitalia. One explanation for this phenomenon could be the introduction of parasites through their marine diet. These intestinal parasites

could cause nutritional deficiencies and bleeding, which can lead to anemia. While the lesions take on two different appearances of porosity and diploic expansion, it is possible that both are caused by parasites introduced by the mainly marine diet of the coastal Algonkians. Depending on which parasite, or parasites, present in an individual, they suffered from either anemia due to intestinal bleeding or malnutrition due to the parasite taking nutrients from the body. The lower rates of porotic hyperostosis in subadults than adults noted by Hutchinson was not the pattern seen with the subsample of CT scanned crania, rather the subadults had higher frequencies of diploic expansion than the adults.

Does CT Scanning Work?

This preliminary study on the use of computed tomography to examine the internal structures of the crania was inconclusive. While in some instances the scans showed only porosity where diploic expansion had visually been assumed, it was not proven that CT scanning is a viable alternative to the more destructive microscopic analysis. Without having a sample that has been studied using methods of proven accuracy (i.e. thin section analysis), it was difficult to determine whether CT scanning is as accurate accuracy rate, and is specific enough to warrant the time and money involved in scanning. The inconclusive results of the non-destructive method shows the need for histological analysis, as it is unsure whether the non-destructive methods are as accurate as the destructive thin sectioning analysis. Further testing should be completed to determine the accuracy of computed tomography as it applies to paleopathological research.

The digitally-enhanced CT scan images and regular visual observation seemed to provide similar pathology presentations, with the greatest power in correlating porosity versus diploic expansion. Significant correspondence between the visual observation scores and the digitally-enhanced CT image scores suggests that perhaps the time and money involved in CT scanning may not be more accurate than simple macroscopic assessment.

In addition, the grayscale values along cross-sections of the orbital roof had little to do with the scores the orbit received using either visual or digital assessments. The lack of correspondence between the grayscale value clusters and the scores from both the visual and digital observations indicate that the grayscale variation from the edge of the orbital roof into the diplöe is picking up a different presentation of the pathology. It is possible that the grayscale values are measuring something different than the

expected density of the bone, and therefore would explain why the values did not group neatly together into separate clusters. In fact, the variations in the internal structure of the bone could more accurately represent the pathological changes, which can only be confirmed through comparison with thin-sections of the same areas. Unfortunately, this is not possible using this sample.

The separate analyses of adult and subadult samples corrected for any differences in cranial thickness present. However, the subadult samples showed no significant relationships between the visual or digital observations and the cluster analysis. It is likely that subadult crania had less overall variation and thus the grayscale values could not be significantly related to the visual or digital observations. A more robust subadult sample could ascertain whether the lack of variation was due to a small sample size, or if it has to do with the thickness of the cranial vault.

Future Research

Further testing must be done using computed tomography to determine its applicability to paleopathological research. CT scanning showed some samples with diploic expansion not seen macroscopically, and it was not inferior to visual assessment. This study was meant as a preliminary test for this use of computed tomography, and should serve as a basis for future studies.

The best way to determine the accuracy of CT scanning as a diagnostic tool is to complete an additional study on a sample population where microscopic analysis has already been completed or is possible. As histologic analysis has previously been shown to be a highly accurate method for assessing cribra orbitalia (see Schultz 2001), the direct comparison to the proposed new methodology of CT scanning would allow for a more thorough assessment of the CT scanning's accuracy. The cluster analysis based on grayscale values and the visual observations could be compared with the pathology identified by the microscopic method to determine which non-invasive technique best reflected the actual histological structure of the orbit. The future study of such a population would allow for the direct comparison of visual assessment, histologic, and computed tomography analyses.

The future study should also attempt to quantify the CT images in a different form. As the grayscale values that were used for this study were not significantly correlated with either the visual or the digital assessment values, perhaps a different quantification method would provide a more accurate picture of what the image is showing. A different software program made for measuring radiopacity

specifically would also be helpful. While the use of Adobe Photoshop was adequate for this preliminary study, future studies should use specialized software for any analyses.

Summary

This study has attempted to determine the reliability of CT scanning as a method for studying the internal bony structures of the crania. However, further testing is needed before a definitive determination can be made on the accuracy of this method. Comparison with a sample in which histologic analysis can be conducted would be ideal to determine the accuracy of computed tomography as a diagnostic tool. As the more traditional methods of diagnosing cribra orbitalia are not always available for use on Native American populations, testing new non-destructive methodologies can increase our knowledge of how Native American groups lived their lives and adapted to their environment. The reanalysis of samples using computed tomography can increase our understanding of Native American populations and what malnutrition and infectious diseases they were susceptible to by determining not only how many people had cranial lesions, but what caused those lesions.

CHAPTER 7: CONCLUSION

This study aimed to reassess the rates of cribra orbitalia in a coastal Algonkian population using computed tomography. Cribra orbitalia is most often attributed to anemia, but has many other possible causes such as vitamin deficiencies, trauma, and inflammation in the orbital region. Invasive methods, such as thin-sectioning, have previously been proven quite accurate in diagnosing cribra orbitalia. However, lack of permission by Indian Affairs Councils has limited the use of histological analysis of Native American collections. The methods tested in this study attempted to reveal the internal structures of the crania without the use of destructive analysis by using computed tomography. CT scanning has only recently begun to be used in a forensic and bioarchaeological context (see Exner et al. 2004, Telmon et al. 2005, Moskovitch et al. 2010 for examples). The use of computed tomography in this study allowed both the reanalysis of cribra orbitalia rates and further determination of the applicability of this methodology as a diagnostic tool in paleopathology.

Using five coastal North Carolina Algonkian sites (N=183) dating to the Late Woodland period, it was found that about half of the crania exhibited cribra orbitalia. After the initial visual assessment of the total sample, 50 crania were chosen for further analysis using computed tomography. Of this subsample, it was found that 55% had porosity, 40% exhibited diploic expansion, and 5% had either no pathology or pathology due to postmortem erosion. Using the CT images, the rates are similar: 40% still exhibited diploic expansion, while 58% had porosity, and only 2% showed no pathology. While most of the visual and digital observation scores tended to equate with each other, 18% of the sample was visually scored as having diploic expansion, which was not evident on the CT image that showed only porosity through the outer table. This discrepancy shows that in some instances CT images allow researchers a more accurate picture of the internal structure of the orbit than can be inferred from visual observation.

Using the Andromeda filter in Adobe Photoshop, the linear cross-sections of the orbital roof were selected on the CT scan images to collect grayscale values from the orbital roof edge into the internal structures. These grayscales values, representing radiopacity, were hypothesized to represent differences in the cortical bone structure and diploic space due to different pathological processes Variation in these grayscale values were compared to between the codes derived from the two

observation techniques to see if any significant variation existed at certain points along the cross section (and thus at different points of the cortical bone and the internal diploic structure) The 5th and 6th grayscale values from the orbital roof had significant variation across the visual observation codes. Subadults exhibited a similar pattern, with the 3rd-5th values having the most variation with the visual observation. However, no significant variation existed between the digital observations and the grayscale values. The Fisher's exact test found no significant relationship between the grayscale pattern clusters, which presumably represent different pathological processes, and either set of observation scores at the $p<0.05$ level. However, among the adult samples a significant relationship exists between the visual and digitally enhanced observations.

The goal of this study was to determine whether CT scanning was a viable alternative method to destructive analysis in diagnosing orbital lesions. While the results do suggest in some instances CT scans revealed diploic expansion not evident visually, further testing must be done in order to definitively determine whether the accuracy of computed tomography is greater than visual analysis alone. By using a sample that had previously been analyzed, comparisons between the previous analysis and the current visual and CT analysis could be made.

The previous study (Hutchinson et al. 2007:62) proposed that parasitic intestinal bleeding from the population's mainly marine diet was responsible for the cranial lesions. These intestinal parasites could explain both the anemia related lesions (diploic expansion), due to the loss of blood caused by the parasites, and also the vitamin deficiency related lesions (porosity). If we directly correlate diploic expansion with anemia, as previous researchers have asserted, then only 13% of the entire sample suffered from anemia (although the fact that not all individuals with anemia exhibit cranial lesions must be kept in mind, as the actual rate of anemia in this population may be much higher). Thirty-seven percent of the total sample had porosity in the orbits, which would suggest a cause other than anemia, such as vitamin deficiencies. Even when the rates of cribra orbitalia in the subsample are taken into account, more individuals show porosity (56%) than show diploic expansion (40%), further suggesting that the cranial lesions in this population cannot simply be explained by anemia alone. The more detailed current study found different demographic patterns than those noted by Hutchinson. Previously the subadults had lower overall rates of porotic hyperostosis, however, with the more comprehensive analysis it was

determined that the subadults had much higher rates of diploic expansion than the adults. This alters the interpretation of subadult diet in these Algonkian populations.

Continued testing of computed tomography will allow researchers to determine the applicability of CT scanning as a methodology in paleopathological diagnosis. By testing a population where histological examination is also possible, direct comparisons can be made between visual, CT scanning, and histological analyses. A more robust subadult sample size is necessary and will allow for further statistical comparisons to determine whether the images can pick up enough detail in the thin outer table present in subadults to be a successful diagnostic tool. Software designed specifically for quantifying radiopacity is necessary in future research, as this may more accurately measure the density of the orbital bone than the Andromeda filter did. It is possible that the inconclusive results seen with the digital images are due to the grayscale values measuring a different aspect of the pathology than intended.

By reassessing the rates of cribra orbitalia using new, and possibly more accurate, methodologies a better picture of how the prehistoric people lived their lives in North Carolina can be discovered. The better understanding of what causes their cranial lesions shows not only their overall health and disease patterns, but also their overall quality of life and how the Algonkian people adapted to their marine environment. Due to NAGRPA restrictions, the more traditional histological methods are not used to study Native Americans, and therefore not as much is known about them as other populations around the world. By applying a new non-destructive method for analyzing their health patterns, a more holistic picture of who the Algonkians were and how they lived their lives along the North Carolina coast can be seen.

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APPENDIX A: DATA COLLECTION FORM

Site: _____ Burial: _____ **Skull: _____ Box: _____ Initials: _____ Date: _____**

Age: _____

Sex: _____

Cribra: _____

Porotic: _____

Age (Adult): _____

Vault	Midlambdoid	___	Nuchal Crest	___
	Lambda	___	Mastoid Process	___
	Obelion	___	Sup.orb. Margin	___
	Ant Sagittal	___	Glabella	___
	Bregma	___	Mental Eminence	___
Lat-Ant	Midcoronal	___		
	Pterion	___		
	Sphenofrontal	___	**Cribra Orbitalia: present? _____**	
	Inf Sphenotemporal	___	Degree	___
	Sup Sphenotemporal	___	Activity	___

Vault Score: _____
Age Range: _____
Lat-Ant Score: _____
Age Range: _____

Sex: _____

Porotic Hyperostosis: Present? _____
_____ frontal _____ parietal _____ temp _____ occipital
Degree ___
Location ___
Activity ___

Age (Sub-Adult): _____

Suture Closure

Spheno-occipital synchondrosis	_____
Occipital-lat to squama	_____
Occipital-basilar to lat	_____

Tooth eruption: _____

Post-cranial fusion: _____

Misc. Notes

Fragmentary? _____

APPENDIX B: INITIAL MACROSCOPIC DATA

										Cribra		Porotic Hyperostosis						
															affected			
Site	Bur #	Box	Sample	Scan	CO	PH	sex	SYMO	age	deg	Activity	deg	loc	activity	fro	tem	par	occ
31CK9	1	10	A		no	yes	F	M	30-40	0	none	1	2	heal	x		x	x
		6	AAA	x	yes	no	sub	S	N/O	3	active	0	0	none				
		6	B		yes	no	sub	S	N/O	2	active	0	0	none				
		8	BB		no	yes	M	M	35-50	0	none	1	2	mixed	x		x	x
		4	BBB	x	yes	no	sub	S	2--3	3	active	0	0	none				
		alone	C		yes	no	sub	S	9--15	1	healing	0	0	none				
		2	DDD		no	yes	F	M	30-40	0	none	1	2	heal	x		x	x
		7	E	x	yes	yes	F	Y	20-35	3	healed	2	4	heal	x		x	x
		3	EE		no	no	ind	M	35-50	0	none	0	0	none				
		10	FF		no	no	ind	M	34-40	0	none	0	0	none				
		10	G		yes	yes	M	M	35-45	1	healed	1	5	healed	x		x	x
		9	GG	x	yes	no	ind	Y	30-35	3	healed	0	0	none				
			GG2 (subadult)		yes	N/O	sub	S	N/O	2	active			N/O				
		2	H		yes	no	F	Y	30	2	active	0	0	none				
		4	I		yes	no	ind	y	20-30	1	healed	0	0	none				
		3	J		no	yes	M	M	35-45	0	none	1	2	active	x		x	x
		7	K	x	yes	yes	sub	s	9--13	2	healed	2	2	mixed				x
		alone	KK		no	yes	M	M	35-45	0	none	1	2	heal	x		x	x
		4	LL	x	yes	yes	ind	Y	20-40	3	healed	1	5	heal	x		x	x
		5	LL		yes	yes	sub	S	N/O	1	active	3	2	heal		x		
		3	M	x	yes	yes	M	M	45	3	heal	2	2	heal	x		x	x
		4	N	x	no	no	F	M	30-40	0	none	0	0	none				
		2	O		no	yes	ind	M	30-50	0	none	1	2	heal	x		x	x
		1	OO		yes	yes	ind	M	30-40	2	healed	1	4	heal	x		x	x
		2	P		no	no	F	M	40-60	0	none	0	0	none				
		10	PP	x	yes	yes	F	M	30-40	3	healed	2	2	heal	x		x	x

	5	QQ		no	no	sub	S	N/O	0	none	0	0	none				
	1	R		yes	yes	ind	M	30-40	2	healed	1	4,5	active	x		x	x
	7	RR	x	yes	yes	M	M	35-55	2	healed	1	5	mixed	x		x	x
	7	S		no	no	sub	S	4--8	0	none	0	0	none				
	3	T		no	yes	M	M	35-55	0	none	2	2	heal	x	x	x	x
	5	TT	X	yes	yes	sub	S	N/O	4	active	4	3	active			x	x
	4	U		no	yes	M	M	35-55	0	none	2	5	active	x	x	x	x
	1	UU	x	yes	yes	M	M	30-40	2	healed	2	4	heal	x		x	x
	4	V		no	yes	F	M	35-45	0	none	1	2	heal	x		x	
	9	VV		no	yes	M	M	35-55	0	none	2	2	active	x		x	x
	7	W	x	yes	no	sub	S	9--13	2	active	0	0	none				
	10	X	x	yes	no	F	S	9--13	1	healed	0	0	none				
	5	Y		yes	yes	F	M	30-40	1	healed	2	4	mix	x		x	x
	alone	Z		yes	yes	ind	M	30-40	2	healing	1	2	heal	x		x	
3	11	only individual		no	yes	F	Y	30-35	0	none	2	2	heal	x		x	x
5	14	B	x	yes	yes	ind	M	30-45	2	healed	1	2	heal	x		x	
	14	C		no	no	ind	M	35-40	0	none	0	0	none				
	14	D	x	yes	no	F	M	30-40	2	healed	0	0	none				
	14	E	x	yes	yes	ind	M	35-50	3	healed	1	2	heal	x		x	x
	13	F	x	yes	no	F	Y	20-40	2	healed	0	0	none				
	12	G		no	yes	ind	Y	25-35	0	none	2	2	heal	x		x	x
	14	H		no	no	F	Y	30-35	0	none	0	0	none				
	14	J		yes	no	sub	S	N/O	2	active	0	0	none				
	12	K	x	yes	no	sub	S	N/O	2	active	0	0	none				
	12	N	x	yes	yes	ind	M	30-40	2	heal	3	2	heal	x		x	x
	14	S	x	yes	no	ind	M	35-40	2	healed	0	0	none				
	14	U/V OLDER		no	n/o	ind	A	N/O	0	none			N/O				
	14	U/V younger		yes	no	sub	S	N/O	2	active	0	0	none				
	13	X		no	no	F	M	30-40	0	none	0	0	none				
	14	Unknown		no	no	F	M	35-40	0	none	0	0	none				
6	15	bag (just orbits)		yes	no	sub	S	N/O	1	active	0	0	none				

		15	bag (w/arrows)		yes	yes	ind	Y	25-35	1	heal	2	3	heal	x			x
		15	orbits only	x	yes	no	ind	M	30-40	3	heal	0	0	none				
	7	18	A		no	yes	M	M	35-45	0	none	1	2	heal	x		x	x
		19	AA	x	yes	no	sub	S	8--12	3	active	0	0	none				
		18	B		no	yes	M	M	30-45	0	none	3	5	heal	x		x	x
		19	BB		yes	no	sub	S	N/O	2	active	0	0	none				
		18	C		no	yes	ind	M	30-45	0	none	1	2	heal	x		x	
		19	CC		no	no	F	M	30-40	0	none	0	0	none				
		18	D		no	no	F	Y	30-35	0	none	0	0	none				
		17	E		no	no	F	M	30-40	0	none	0	0	none				
		17	G		no	no	M	M	35-45	0	none	0	0	none				
		17	I		no	yes	F	M	35-45	0	none	1	2	heal	x		x	x
		20	J		no	no	M	M	40-50	0	none	0	0	none				
		20	K		no	no	F	M	35-45	0	none	0	0	none				
		20	L	x	yes	yes	M	M	30-45	3	mix	1	2	heal	x		x	x
		16	M		no	yes	ind	Y	25-35	0	none	3	2	heal	x		x	x
		16	N		no	no	F	M	35-45	0	none	0	0	none				
		16	O		no	no	F	M	30-40	0	none	0	0	none				
		16	P		no	yes	ind	M	30-40	0	none	1	2	heal	x		x	
		17	Q		no	yes	M	Y	25-35	0	none	2	5	heal	x		x	x
		17	S		no	no	M	M	30-40	0	none	0	0	none				
		4	U		no	yes	M	M	35-50	0	none	1	2	heal	x		x	x
		4	V		no	no	F	M	30-40	0	none	0	0	none				
		19	W	x	no	no	F	M	30-40	0	none	0	0	none				
		17	Y		no	yes	ind	M	30-40	0	none	1	2	heal	x		x	
		19	Z		no	no	F	M	35-40	0	none	0	0	none				
	8	21	A	x	yes	no	F	M	30-40	3	active	0	0	none				
	17	shelf	R		no	yes	F	Y	20-40	0	none	1	2	heal	x		x	x
31CK22	2	28	5		no	yes	F	Y	15-25	0	none	1	2	Heal	X			
		28	5a	x	yes	no	sub	S	9--13	2	active	0	0	none				
		28	6		no	no	F	M	35-45	0	none	0	0	none				

	26	7	x	no	no	F	M	40-50	0	none	0	0	none					
	30	8		no	no	F	A	N/O	0	none	0	0	none					
	28	10		no	no	F	M	30-40	0	none	0	0	none					
	30	12		no	no	sub	S	8--12	0	none	0	0	none					
	22	13		no	no	F	M	30-40	0	none	0	0	none					
	26	15	x	yes	no	F	M	30-40	2	active	0	0	none					
	26	20		yes	no	F	M	30-40	1	active	0	0	none					
	24	21	x	yes	yes	F	M	30-40	2	heal	2	5	heal			x	x	
	29	22		yes	no	sub	S	N/O	2	active	0	0	none					
	26	24	x	no	no	ind	M	35-45	0	none	0	0	none					
	30	25		no	no	sub	S	N/O	0	none	0	0	none					
	30	27	x	yes	yes	sub	S	N /O	2	active	1	2	active	x		x	x	
	30	28		no	no	sub	S	1--2	0	none	0	0	none					
	26	29	x	no	no	F	M	30-40	0	none	0	0	none					
		30		yes	N/O	sub	S	N/O	2	active			N/O					
		31		no	N/O	sub	S	N/O	0	none			N/O					
	26	34	x	yes	no	sub	S	4--8	4	active	0	0	none					
	31	34		yes	no	sub	S	N/O	1	active	0	0	none					
	26	35		yes	N/O	sub	S	N/O	3	active			N/O					
	25	36		no	no	F	M	30-40	0	none	0	0	none					
	31	38	x	yes	no	F	Y	15-20	1	active	0	0	none					
	31	40		no	no	F	O	40-60	0	none	0	0	none					
	30	41		no	no	sub	S	0-2	0	none	0	0	none					
	24	no number	x	yes	yes	M	M	30-40	3	active	1	2	heal					
	31	no number		no	no	ind	M	35-45	0	none	0	0	none					
bank	29	Ind 1		no	no	IND	A	N/O	0	none			N/O					
bank	30	F-28		no	yes	ind	M	30-40	0	none	1	2	heal			x		
bank	27	F-565	x	yes	no	sub	S	N/O	4	active	0	0	none					
beach	30	F-19		no	no	ind	M	30-40	0	none	0	0	none					
beach	30	F-19 (lighter)	x	yes	no	ind	M	30-40	1	heal	0	0	none					
		beach, F-15		yes	N/O	SUB	S	N/O	2	active			N/O					

	beach	30	F-3		no	no	M	M	35-45	0	none	0	0	none				
	beach	32	hb-21	x	yes	yes	ind	M	30-40	2	heal	0	0	none				x
	beach	23	F346- 24- sunbleached	x	yes	no	IND	A	N/O	2	active	0	0	none				
	beach	23	F346- 24- darker	x	yes	no	F	A	N /O	2	heal	0	0	none				
	beach	30	no # (darker)		no	yes	ind	A	N/O	0	none	2	2	mix	x			
	beach	30	no # (lighter)		no	no	F	M	30-40	0	none	0	0	none				
31 CK 24	1	33	7	x	yes	no	ind	Y	25-35	3	active	0	0	none				
		33	24	x	yes	yes	M	M	30-40	2	mixed	1	1	heal	x		x	
			25		yes	NO	IND	A	N/O	0	none	0	0	none				
31 DR 38	1	36	1		no	yes	M	M	30-45	0	none	1	5	heal	x		x	x
		34	B	x	yes	no	F	Y	25-35	3	active	0	0	none				
		34	C		no	yes	M	Y	20-30	0	none	2	5	heal	x		x	x
		36	D		no	yes	ind	Y	25-35	0	none	2	2	heal	x		x	x
		35	F		yes	yes	F	M	30-40	1	heal	1	2	heal	x		x	x
		35	G	x	yes	yes	F	Y		2	active	2	5	heal	x		x	x
		36	K		yes	no	M	M	35-45	1	heal	0	0	heal				
		34	L		yes	yes	M	M	35-45	2	active	2	2	heal	x		x	x
		36	Bag 5-lightest		yes	no	F	A	N/O	2	active			N/O				
		36	Bag 5- lighter (right orbit)		yes	N/O	M	A	N/O	2	heal			N/O				
		36	Bag 5- right orbit, med color		no	N/O	F	A	N/O	0	none			N/O				
		36	Bag 5-darkest, small		no	N/O	F	A	N/O	3	Heal			N/O				
		36	Bag 5-right orbit w/sinus		no	N/O	M	A	N/O	0	none			N/O				
		36	Bag 5-small, nasal, lighter		no	N/O	F	A	N/O	0	none			N/O				
		36	Bag 5-med color, glabella		no	N/O	ind	A	N/O	0	none			N/O				
		34	bag 6- broken halves		yes	no	F	M	35-45	3	active	0	0	none				
		34	bag 6-bigger both orbits		yes	no	ind	M	30-40	2	active	0	0	none				
		34	bag-darkest		yes	yes	ind	M	30-40	2	mix	2	5	heal	x			
		37	Bag of Right orbits		no	N/O	M	A	N/O	0	none			N/O				
		37	Bag of Right orbits		yes	N/O	M	A	N/O	2	active			N/O				
		37	Bag of Right orbits		no	N/O	M	A	N/O	0	none			N/O				

		37	Bag of Right orbits		yes	N/O	M	A	N/O	1	heal			N/O				
		37	Bag of Right orbits		no	N/O	F	A	N/O	0	none			N/O				
		37	Bag of Right orbits		yes	N/O	F	A	N/O	1	heal			N/O				
		37	Bag of Right orbits		yes	N/O	F	A	N/O	1	heal			N/O				
		37	Bag of Right orbits		yes	N/O	M	A	N/O	2	heal			N/O				
		37	Bag of Left orbits		no	N/O	ind	A	N/O	0	none			N/O				
		37	Bag of Left orbits		no	N/O	ind	A	N/O	0	none			N/O				
		37	Bag of Left orbits		yes	N/O	M	A	N/O	2	heal			N/O				
		37	Bag of Left orbits		yes	N/O	M	A	N/O	1	heal			N/O				
		37	Bag of Left orbits		yes	N/O	ind	A	N/O	1	active			N/O				
		37	Bag of Left orbits		yes	N/O	ind	A	N/O	3	active			N/O				
		37	Bag of Left orbits		no	N/O	M	A	N/O	0	none			N/O				
		37	Bag of Left orbits		yes	N/O	F	A	N/O	3	active			N/O				
		37	Bag of Left orbits		no	N/O	ind	A	N/O	0	none			N/O				
31CO5	1	40	2		no	yes	ind	M	35-45	0	none	2	2	heal	x		x	
		41	3		yes	no	F	Y	25-35	2	heal	0	0	none				
		43	A (group B)	x	yes	no	sub	S	8--12	4	active	0	0	none				
		43	A (group G)		yes	no	sub	S	5--9	2	active	0	0	none				
		47	B, ind 2 (group B)		no	no	ind	M	30-40	0	none	0	0	none				
		47	E, ind 1 (group B)		no	no	F	Y	18-25	0	none	0	0	none				
		45	G (group A)	x	no	no	F	M	40-50	0	none	0	0	none				
		41	J		yes	yes	F	Y	20-3-	1	active	1	2	active	x		x	
		48	K (group D)	x	yes	yes	F	M	30-40	1	active	2	2	heal				
		44	O (Group E)		no	no	F	Y	25-35	0	none	0	0	none				
		46	Q, ind 1 (Group E)	x	yes	no	F	M	35-50	1	heal	0	0	none				
			R		no	yes	ind	M	30-40	0	none	1	2	heal	x		x	x
		40	U (Group F)		no	yes	ind	M	30-45	0	none	1	2	heal			x	
		46	X (Group F)		no	no	ind	M	35-45	0	none	0	0	none				
			Y (Group G)		yes	no	sub	S	9--15	1	active	0	0	none				
		41	Z		no	yes	ind	M	30-40	0	none	1	2	heal	x		x	
		46	AA (Group G)		yes	yes	F	Y	15-25	2	active	1	2	heal				

CC		yes	N/O	IND	A	N/O	1	heal			N/O					
no number		yes	N/O	IND	A	N/O	1	heal			N/O					
burial 4, group G		yes	N/O	IND	S	N/O	2	active			N/O					
group G		no	N/O	IND	A	N/O	0	none			N/O					