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Contributory Factors of Extraneous New Bone Growth on the Endocranial Surfaces of Human Infant and Sub-Adult Skeletal Remains

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CONTRIBUTORY FACTORS OF EXTRANEOUS NEW BONE GROWTH ON THE

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ABSTRACT

This study investigates the factors that may contribute to endocranial bone changes in human sub-adult skeletal remains in an effort to differentiate bone changes, or lesions, caused by pathological processes and those caused by growth and development. The contributory factors investigated included the presence of endocranial bone lesions, the age-at-death of the individual, the precise location of the bone change, the dynamic nature of the locations, and the presence (or absence) of postcranial and/or ectocranial indicators of pathology within the individual. The sample population used for this research was comprised of 129 individuals from 15 different Native American groups over five states and two cultural regions, ranging in age from birth to 15 years. Age-at-death was determined and macroscopic examinations of all bone surfaces were completed for all individuals. Results of the Chi-square tests performed on the data show that endocranial changes have a statistical association with lesion location, the dynamism of the location, and postcranial and/or ectocranial indicators of pathology. It can be stated that there is a correlation that can be established between certain specific factors and lesions caused by pathological processes whereas other factors showed a correlation to growth and developmental processes, or were found not to be contributory to either process.

CHAPTER I

INTRODUCTION

An important concern within paleopathology is the distinction between
extraneous new bone growth that can be linked to pathological processes, caused by
disease or malformation, and extraneous new bone growth that is within the range of
"normal" bone morphology. Physical traces of abnormal bone change, or bone changes
appearing near one end of the "spectrum of the normal range" (Aufderheide &
Rodriguez-Martin, 1998, p. 11), are commonly referred to as lesions. These can be
caused by pathological processes and can result from different types of infectious and
noninfectious diseases, nutritional stressors, trauma, congenital abnormalities, and
malformations. Bone changes of unknown etiology can also be called lesions, but may
be due to growth or development (Pfeiffer, 2000; Ortner, 2003).

Differentiating between lesions caused by pathological processes and the presence
of extraneous new bone growth linked to non-pathological processes is particularly
difficult in juveniles. For instance, the scientific community has become aware of
appositional growth that appears as a "deposition of immature disorganized bone"
(Lewis, 2000, p. 42) at the epiphyseal ends of juvenile long bones (Scheuer & Black,
2000). These bone alterations have been confused with pathological lesions when, in
fact, they represent normal new bone growth (Malmberg, 1944; Shopfner, 1966; Mann &

Murphey, 1990; Lewis, 2000; Lewis, 2004). Researchers have also been noting the presence of bone changes on the endocranial surface of juvenile skeletons. As Lewis (2000, 2004) states, determining the etiology of endocranial new bone growth has become a relatively new area of study, not only as to what disease or pathology may have caused the lesion(s) in question, but as to whether a disease or pathology is at all responsible.

There are two reasons why the determination of the etiologies of juvenile endocranial lesions is difficult to ascertain. The first is that in juveniles (and, in fact, humans of all ages) there are only two types of bone formed by osteoblasts, or bone-forming cells, during bone deposition. Those types are woven bone and lamellar bone. Woven bone, also called primary bone or immature bone, is the initial bone type that is laid down by osteoblasts during pathological processes, repair, or growth of bone tissue. Woven bone is highly vascular and quickly produced by osteoblasts, characteristically trapping the blood vessels within the matrix. Bone matrix is composed of collagen fibers laid down primarily in an unorganized orientation that will then become mineralized (Lovell, 2000; Schultz, 2001). Schultz (2001) notes that newly formed primary bone "shows no lamellar structure" (p. 115) and "the osteocytes [bone cells] seem to be more randomly scattered throughout the ground substance [i.e., the bone matrix]" (p. 115). Subsequent to the formation of woven bone, osteoclasts will begin promoting the destruction of bone tissue and its resorption into the blood stream. Osteoblasts will then lay down newly organized bone matrix, which will form the Haversian systems that constitute normal cortical and trabecular bone tissue, or lamellar bone. Woven bone and

lamellar bone are the only types of bone tissue formed no matter what the underlying

cause. This renders the determination of the cause of bone formation complicated and

creates an additional set of difficulties in determining the etiology of endocranial lesions.

Lewis (2000, 2004) attests that woven bone deposited as a response to a pathological

condition is indistinguishable from appositional woven bone due to growth and

development. The second reason it is difficult to determine the etiology of endocranial

lesions is the limited number of ways that bone tissue can react in response to a stimulus.

Living bone tissue reacts to stimuli by either depositing new bone through osteoblastic

activity, resorbing bone tissue through osteoclastic activity, or a combination of the two.

The determination of etiologies then can be accomplished only by making distinctions

between the different patterns of bone deposition and bone destruction throughout the

skeleton (Steele & Bramblett, 2000). The authors also note that "in many instances the

patterns are similar in one or more disorders, making positive diagnosis difficult" (p. 15).

Exploring the etiology of endocranial extraneous bone growth in this thesis will

require the recognition and isolation of important variables. The first variable is whether

or not there are any indicators of disease present within the postcranial and/or ectocranial

skeletal elements of the individual. This information may indicate that some extraneous

new bone growth located on the endocranial surface might be attributed, at least in part,

to a pathological process. The second variable is the age at death of the individual under

examination. The age of the individual may be an important factor in the determination

of the general etiology of lesions, especially when dealing with individuals who are still

in the early stages of growth and life. Lewis (2000) maintains that age is specifically

important when discerning between proliferative new bone growth due to development or due to a pathological condition. The third variable requiring investigation is the physical location of the lesions on the bone(s) within the endocrania. Location can be a specific bone feature or an anatomic or systemic locale within the endocrania, or the lesions may have no association with any feature or system located on the endocranial surface. The importance of recording the exact locations of these lesions is for the purpose of correlating them to the areas of the endocrania that are functional or undergoing morphological changes due to bone growth and development remains underemphasized. The variables, when examined in conjunction with one another, will assist in the determination of the etiology of endocranial lesions.

The goal of this project is to provide a basis upon which other researchers can understand the presence of extraneous new bone growth found within juvenile endocrania. It is expected that the analysis of variables will not only assist in determining which of the variables are instrumental in the production of new bone growth, but will also assist in the determination of the etiology of endocranial lesions. It is this distinction, pathological from non-pathological, that must serve as the initial first step in the process of differential diagnosis. And only through careful differential diagnosis can skeletal biologists, bioarchaeologists, and paleopathologists begin to understand growth and development, and the presence of disease in the past.

CHAPTER II

REVIEW OF RELATED LITERATURE

Bone Development and Growth

No other region in the human skeletal system has as much variation as the cranium. The cranium consists of three separate but equally important components that make up the whole of the skull (Moss & Young, 1960; Moore & Persaud, 1998; Pick & Howden, 2003; Scheuer & Black, 2000). The first of these is the neurocranium, also termed the cranial vault, calvaria, or braincase. The neurocranium comprises the roof, walls, and floor of the bony structure that surrounds and protects the brain. The second aspect is the viscerocranium, splanchocranium, or the facial aspect of the cranium. This comprises the most anterior and anterior/inferior aspect of the skull, and consists of two components, the upper and lower facial regions. The chondrocranium, or cranial base, consists of the superior and posterior aspect of the viscerocranium, and the inferior aspect of the neurocranium. The cranial base houses the organs of special sense - the eyes, nose, and inner ears - and forms a barrier between the braincase and the facial skeleton while allowing communication between the two through the use of foramina and fissures within the bone.

Osteogenesis of the cranial base is endochondral in origin and less susceptible to variations in morphology than the other two components of the skull. In brief,

endochondral bone formation is the secondary ossification of a primary cartilaginous precursor. The hyaline cartilage model undergoes a transformation allowing it to ossify. This is accomplished through a series of events beginning with the hypertrophy, or enlargement, of the chondrocytes, promoting the calcification of the surrounding matrix, which in turn promotes the cartilage cell's eventual death. Concurrently, the perichondrium, the fibrous layer surrounding the cartilage tissue, develops into the periosteum, the fibrous layer that surrounds the bone. The periosteum is highly vascularized connective tissue whose blood vessels will invade the interior of the cartilage model (which was left with cavities by the numerous cartilage cell deaths) to deposit hemopoietic cells responsible for the formation of blood cells (i.e., the bone marrow) and osteoblasts. These osteoblasts then lay down a bony matrix on the areas of calcified cartilage, forming spicules from which the bone will eventually radiate (Moore & Persaud, 1998; Moore & Dalley, 2006).

The neurocranium and viscerocranium are formed through intramembranous ossification, which can be defined as the direct mineralization of a highly vascular connective tissue membrane, according to Pick & Howden (2003). The membrane being referred to is mesenchyme, an embryonic connective tissue. The mesenchyme cells cluster and condense while becoming highly vascularized. Some of these mesenchyme cells differentiate into osteoblasts and will begin to deposit intercellular substances called the bone matrix, or osteoid. The osteoid becomes mineralized as calcium phosphate is deposited, trapping some of the osteoblasts within the newly calcified matrix. These

osteoblasts become osteocytes and are utilized to communicate from within the bone to various systems external to the bone.

New bone has no organized pattern and is called woven bone. The extensive deposition of new bone forms bony projections called spicules. In turn, these spicules coalesce to become trabeculae that form the basic building blocks of the cancellous, or spongy, bone that surrounds the future marrow cavity. Concurrently, as more bone tissue is deposited, mesenchyme condenses on the surface of the newly formed trabeculae and differentiates into the periosteum. The periosteum contains a layer of osteoblasts that lays down bone tissue surrounding the trabeculae to form a plate of compact bone. As more layers of calcified bone matrix are laid down, the newly formed woven bone becomes more organized into lamellar bone. It is in this lamellar bone that the typical Haversian systems develop (Marieb & Hoen, 2008; Moore & Persaud, 1998; Pick & Howden, 2003).

The sites of initial bone formation are termed ossification centers (Marieb & Hoehn, 2008; Moore & Dalley, 2006; Pick & Howden, 2003; Scheuer & Black, 2000). New bone growth radiates outward from the center of ossification toward the "major [dural] bands" (Smith & Tondury, 1978, p. 665), which represent the future site of the cranial sutures. The sutures of the crania, which separate the growing bones of the skull, are not equivalent to the epiphyses that are found in the postcranial skeleton. Sutures do not push growing bones apart; they merely allow for the addition of new bone to take place "as the cerebral capsule expands and passively carries the bones outward" (Moss & Young, 1960, p. 282). Growth of the vault is accomplished through ossification at the

appositional margins of the flat bones of the skull (Pick & Howden, 2003), eventually closing in upon the sutural areas and the fontanelles, the "membranous intervals" (Moore & Dalley, 2006, p. 903) at the angles, or corners, of the bones of the growing cranial vault (Moore & Dalley, 2006).

Bone tissue is living tissue and undergoes a continuous process of remodeling and homeostasis. During growth and development, bone remodeling occurs at a rapid rate and there is a high turnover of bone tissue (Aoki et al., 1987; Lewis, 2000; Scheuer & Black, 2000). Remodeling consists of the balancing of osteoclastic activity and osteoblastic activity in order to modify the shape of the growing bones to better suit the extrinsic and/or intrinsic forces that act upon it (Pick & Howden, 2003; Scheuer & Black, 2000). Remodeling occurs for different reasons throughout an individual's lifespan. Bone tissue is remodeled as a result of growth and developmental processes, i.e., the maturation of both cortical and trabecular bone tissue; as a result of functional stresses placed on bone; and also in response to a disease or injury (Lovell, 2000).

Bone tissue develops and remodels in response to the demands placed upon it while changes in the function of bone are represented by changes in its structure, as stated by Wolff in 1870 (Marieb & Hoehn, 2008; Sperber, 1989; Wolff, 1870). Marieb & Hoehn (2008) state that due to mechanical stresses and gravity placed on bone, "bone is stressed whenever weight bears down on it" (p. 170). The stress is typically off center and is apt to bend the bone. Bending of bone causes compression on the aspect of the bone that is bearing the load while the other side of the bone will be subject to tension, or stretching. There are virtually no forces acting upon the center of the bone, and the bone

can be hollow as a result without compromising the integrity of the bone. Wolff's law, as it has come to be known, also puts forth that (1) long bones are thickest midway along the diaphyses where mechanical stresses are at their greatest, (2) curved bones are thickest where they would have the greatest tendency to buckle, and (3) the trabeculae of cancellous bone forms support along the lines of greatest compression (Marieb & Hoehn, 2008).

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Pathological Conditions as a Factor in

Determining the Etiology of Endocranial Bone Changes

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There is no doubt that pathological conditions can leave vestiges on the endocranial surfaces of individuals, especially sub-adults, whose skeletal systems have not had enough time to undergo remodeling at the time of death. Pathological conditions will leave only traces of their existence on the bone tissue when there has been a sufficient amount of time for the skeletal system to react. This usually occurs during chronic diseases when the skeletal system has had time to respond, and not during acute diseases or the initial phases of chronic diseases that may take the life of the individual before a bony response is attained (Lewis, 2000; Ortner, 2003).

Two pathological conditions, periostitis and meningitis, can manifest a bony response in the individuals that they affect. Periostitis is an inflammation of the periosteum (Lewis, 2000 & 2004; Mann & Murphey, 1990; Mensforth, Lovejoy, Lallo, & Armelagos, 1978; Schultz, 1999; Ribot & Roberts, 1996). The periosteum is the fibrous outer layer of connective tissue that surrounds the bone surfaces of the skeleton and houses the osteoblasts. During pathogenic infections the separation of the periosteum

and the bone surface is a common occurrence and can result in proliferative new bone growth. Meningitis on the endocranial surface is comparable to periostitis, in that it is an inflammation or reaction of the periosteal layer of the meninges (Lewis, 2004; Ortner, 2003; Roberts & Manchester, 2005; Schultz, 2001). It should be mentioned again that the periosteal layer of the meninges takes on the osteogenic properties of the periosteum and can react as such (Schultz, 2001; Ortner, 2003).

The lifting away of the periosteum or periosteal layer of the dura mater can have a markedly different affect in a sub-adult than the same set of circumstances has in an adult. Lewis (2000) notes that because of the loosely attached periosteal layer in sub-adults the effects can be much more severe than in adults, where the periosteal layer is more tightly adhered. Because of this, Lewis (2000) also asserts that the "diagnosis of periostitis in non-adult skeletal remains is more problematic [than the diagnosis of other pathological conditions]" (p. 42). In fact, many authors who have observed and studied the endocrania of sub-adults will attest to the fact that rapid new bone formation, due to the growth and development of the cranium, can mimic the appearance of periostitis (Lewis, 2000; Mann & Murphey, 1990; Scheuer & Black, 2000).

Growth and Age as Factors in

Determining the Etiology of Endocranial Bone Changes

The growth of the separate bones of the neurocranium develops independently of each other, but also as a functional unit in constant relation to one another (Humphrey, 1998; Moss, Noback, & Robertson, 1956). Moss et al. (1956) discovered that all of the bones that comprise the cranial vault grow at a steady rate while maintaining a constant

relation to one another. The authors determined this by measuring specific dimensions on the bones of the cranial vault and observed that in each, an interphase, or period of change in the rate of growth, occurred. The interphase occurred during the same period of development (when the crown-rump length reached 85mm) on each bone studied, thus showing that a constant rate of growth for the skeletal neurocranial unit occurs.

Young (1957), in his work with X-rays of juvenile skulls, shows that the cranial vault expands rapidly within the first two years of life. This was established by examining the relationship of three anatomical points of the skull's outer surface: nasion, which is the point of intersection between the internasal and nasofrontal sutures; bregma, which is the point of intersection between the frontal and parietal bones on the coronal suture at the midline; and lambda, which is the point of intersection between the occipital and parietal bones on the lambdoid suture at the midline. As an individual develops over time, it can be seen that the arcs - the curved line that follows the outer surface of the skull between two anatomical points of the frontal and parietals - increase in depth, as do the chords - the straight lines that traverse the bones from one anatomical point to another. The expansion of these arcs and chords indicate that the volume of the braincase is increasing as well. It should also be noted that nasion, bregma, and lambda do not change position relative to one another in any noteworthy way either. They remain in a consistent relative association as the cranial vault expands, which would indicate that the bones are growing at a steady pace as a functional unit, as shown previously by Moss et al. (1956).

Louise Humphrey, in 1998, composed a study in which the frontal and occipital bones of developing individuals were observed in order to determine when adult size is attained in postnatal skeletal elements. It was found that specific aspects of the frontal and occipital bones, the orbital breadth and foramen magnum, respectively, attain more than 70% of their adult size by birth; unfortunately, it was not stated in her paper when the entire bone reaches approximate adult size. Humphrey's research comes as a direct result of R. E. Scammon's research from 1930. Scammon determined that different rates of growth occur for the different functional systems of the body. This was accomplished by ascertaining the relative weights of each bodily system during growth and plotting those growth rates as a percentage against its weight when maturity is reached. The skeletal, digestive, excretory, and respiratory systems followed the same growth curve as the body overall, which showed that these systems remain constant relative to body size during growth. However, the neural growth curve, that of the central nervous system, organs of special sense, and the skull follow a different growth pattern. It was found that the rate of growth of the neural system, and the skeletal elements associated with it, is extremely rapid, so much so that it reaches 60% of adult size by two years of age, 80% by four years, and 90% by approximately six years.

The growth of the neurocranium is directly associated with the growth of the brain and of the organs of special sense. Humphrey (1998) points out that the growth of the cranial bones (and the skeletal system in general) is "a response to the requirements of the associated non-skeletal tissues" (p. 57). She justified this by determining that "the earliest growing variables are measured on the frontal and occipital bones" (p. 69), which

correspond to the early, rapid development of the eyes, brain, and spinal cord (Humphrey, 1998; Pick & Howden, 2003; Sperber, 1989). This is in concurrence with the findings of O'Rahilly & Muller (1986) that the frontal and interoccipital bones are the first to ossify at eight postovulatory weeks.

The braincase therefore continues to grow as such, in relation to the rapidly growing brain and developing meninges, and to a lesser degree to the sense organs as they are enclosed in their own specific environments during childhood (Humphrey, 1998; Pick & Howden, 2003). The growth of the brain is the primary force in the growth of the calvarium (Humphrey, 1998; Moss, 1959; Moss et al., 1956; Moss & Young, 1960; Scheuer & Black, 2000) as research on hydrocephaly, microcephaly (Young, 1959), and anencephaly shows (Moore & Dalley, 2006; Moore & Persaud, 1998).

There seems to be a general consensus that the most rapid period of growth of the human neurocranium occurs postnatally within the first two years of life. Although Lewis (2004) claims that this occurs only during the first year, this discrepancy is not of the utmost significance, as it still conveys the understanding that this time period within an individual's skeletal development, especially the neurocranial element, is precarious, in that the growth of the calvarium needs to be very precise in its developmental stages or certain abnormalities may occur. After this two-year period, the growth of the skull vault begins to decrease until it reaches adult proportions (or approximate adult proportions). Kabanni & Raghuveer (2004), Pick & Howden (2003), and Lewis (2004) believe this age to be approximately seven years, whereas Scheuer & Black (2000) believe the age to be ten, Humphrey (1998) and Sinclair (1986) believe it to coincide with the age that puberty

is reached, and Moore & Persaud (1998) believe the age of adult proportions is reached at 16 years.

Extraneous bone growth due to non-pathological processes will materialize at those times in human skeletal development when bone growth occurs. The incidence of such growth should be greater in times of rapid development, the occurrence of bone lesions should also lessen as the rate of bone growth and development slows, and the appearance of extraneous bone growth should cease as bone growth and development subsides. Determining at what ages in an individual's sub-adulthood these periods (rapid growth, deceleration, and the cessation of growth) take place should shed light on the age-at-death of the individual and how it relates to the presence of non-pathological endocranial bone growth.

Age-at-death, as it relates to bone growth due to pathological processes, is not as discernable as the relationship of bone growth to developmental processes. Pathological lesions, in general, can occur during any period of an individual's lifespan (Roberts & Manchester, 2005) and on any feature of the endocranial surface. However, Mensforth et al. (1978) contend that some pathological lesions that form as a direct result of infectious diseases are, in fact, age-specific. Their research shows that as the age of the individual increases, the number of pathological lesions decreases. There may be specific pathological conditions that cause bone changes that may arise during the time frame of being a sub-adult, yet those conditions should not overshadow the fact that pathologies, in general, are not age-specific and can occur throughout an individual's lifespan.

Location as a Factor in Determining

the Etiology of Endocranial Bone Changes

The location of extraneous new bone growth may also provide key information that can help discern between lesions due to pathological conditions and normal bone growth. Examining regions of the braincase by location will allow future researchers to take into consideration the placement of bone lesions when determining their etiology. Locations of bone lesions can be allocated to three groups. The first is anatomical region of the braincase -those features that share a common area. The second is systemic location -those features that share a common function. And the third is dynamic locations - which undergo a morphological change.

Anatomic Location

The intramembranously formed cranial vault and facial skeleton are more prone to variations during the growth of the individual bones, whereas the cranial base, formed from a cartilage predecessor, is much more stable. Sperber (1989) attributes this occurrence to several factors. The basicranium forms around the already present and developing brain, cranial nerves, blood vessels, and organs of special sense. The cranial base has established an early relationship with the organs and tissues it will surround and protect and this gives the basicranium a relative stability as compared to the cranial vault. This relationship begins to form in the sixth week of embryonic life when the cartilage models begin to form the base of the skull (Pick & Howden, 2003). At this early stage of life, it is noted that the cartilaginous precursors are already forming about the otic

capsules, orbits, and nasal aperture (Sperber, 1989). The centers of ossification of the neurocranium, however, do not appear until the eighth or ninth week of embryonic life (O'Rahilly & Muller, 1986; Pick & Howden, 2003). This two- to three-week period of time between the initiation of cartilage of the base and the bone formation of the vault may not seem substantial, but, in terms of structure, it can mean a great deal. The unossified cranial base is formed in a rigid cartilage model, whereas the vault is formed in a pliable membranous tissue. In addition, the base is a slow, constantly growing region as compared to the vault, which is highly volatile the first two years of postnatal life.

Sperber (1989) also states that the enlargement of the skull base can come only from the elongation of the skull and the lateral, or outward, expansion through the synchondroses, whereas expansion of the cranial vault occurs through displacement both laterally, superiorly, and fronto-posteriorly at the sutures.

Young (1959) states that in rats exhibiting the congenital abnormality of craniosynostosis, the premature fusion of cranial sutures, the cranial base is affected only to a minor degree. He observed animals afflicted with either microcephaly or hydrocephaly and determined that, while the vault was highly affected (not only as a whole, but on the level of individual bones as well), only the fossae of the basicranium and the foramen magnum "showed slight compensatory changes in the overall morphology" (p. 401).

Systemic Locations

The endocranium not only surrounds and protects the brain, but also interacts with several other systems of the body. It is in constant contact with the meninges, which

surround the brain and provide an encasement for the cerebrospinal fluid. The endocranial surface also interacts with the circulatory system, which supplies blood to the brain, meninges, and bones of the skull. Nerves also pass through certain areas of the bones of the braincase to allow communication from the brain to the organs of special sense and from the brain to the spinal cord.

The function of the meninges of the brain is twofold for the purposes of this paper. The outermost layer of the meninges is termed the dura mater. It is the only meningeal layer that comes in direct contact with the endocranial surface. Thus, its outermost layer acts as the periosteum, which surrounds the other bones of the skeletal system; it is highly vascularized and houses the osteoblasts (Sperber, 1989; Smith & Tondury, 1978). Because the dura mater acts as the periosteum, there is continuous interaction with the surface of the bones of the neurocranium, and in some areas that interaction is greater than in others. The dura mater, along with having osteogenic properties, also has tenacious attachments at specific points on the base of the cranium anchoring the dural processes that form the membranous separations between different aspects of the brain (Moore & Dalley, 2006; Pick & Howden, 2003). These attachment sites and processes include the falx cerebri, which separates the hemispheres of the cerebrum, and attaches to the crista galli and frontal crest anteriorly and the internal occipital protuberance and occipital crest posteriorly; the tentorum cerebelli, which separates the cerebrum and the cerebellum, and attaches to the internal occipital protuberance posteriorly and the superior/posterior angle of the petrous portions; the falx cerebelli, which separates the hemispheres of the cerebellum and attaches to the occipital

crest; and the diaphrama sellae, which covers the pituitary gland and attaches to the anterior and posterior clinoid processes (Moore & Dalley, 2006; Pick & Howden, 2003).

Other areas of the dura mater adhere quite strongly as well. These areas follow the majority of the suture lines of the cranial vault, some of which follow certain dural processes of the cranium (Moss, 1959; Smith & Tondury, 1978). They are the metopic and sagittal sutures, which align themselves with the flax cerebri, and both the left and right sutura mendosas, which align themselves with the tentorum cerebelli. The other suture lines that do not follow the dural processes but still have a strong union of the dura mater to the surface of the endocrania are the coronal and lambdoid sutures. These sutures are formed, not by the dural processes, but by tensile or biomechanical forces that restrict osteogenic progression at the site of the suture (Smith & Tondury, 1978). It should be noted that this mechanism of hampering osteogenesis is the main factor at work concerning the sutures that follow the dural processes as well; the dural processes themselves are merely coincident to the sutures. The sutures of the crania, while determined by the dura mater, are not necessarily considered a part of the meningeal system. Sutures are areas where two or more bones are separated from one another to allow for the outward expansion of osteogenesis within that bone, or bones. The suture is also the eventual site where the bones of the skull will articulate with one another.

The endocranial surface of the skull also has interactions with the circulatory system. As with the sutural system, the dura mater incorporates itself into this system as well. The dura mater consists of two layers, a periosteal layer and a meningeal layer. These two layers are fused together very strongly by connective tissue and are separated

from one another only where the venous sinuses are located (Moore & Dalley, 2006; Sperber, 1989). The venous sinuses contain deoxygenated blood that is collected from the veins of the dura mater and of some cranial bones (Pick & Howden, 2003). Two of these sinuses, the sagittal sinus and the transverse sinuses, run along the anterior aspect of the falx cerebri and posterior to the tentorum cerebelli, respectively (Pick & Howden, 2003; Moore & Persaud, 1998). This means, naturally, that these venous sinuses also run along the line of the sagittal suture and the sutura mendosas. In addition to the venous sinuses, there are blood vessels that supply blood to the dura mater and some cranial vault bones. The mid-meningeal arteries and the branches of vessels that stem from them, known as the mid-meningeal vessels, form a network of depressions within the endocranial surface of the walls and roof of the skull (Scheuer & Black, 2000). Also, the many foramina located within the cranial base and vault are utilized for the purpose of allowing blood vessels to pass through from the braincase to other areas of the body.

The circulatory system, which is known to have a direct association with the transference of blood borne pathogens throughout the body (Aufderheide & Rodriguez-Martin, 1998; Ortner, 2003), may be indirectly associated with the occurrence of bone lesions on the endocranial surface of the skull by transporting pathogens to the braincase; it may also be the primary cause of the lesions as well. Hemorrhages, which are the breaking of blood vessels (i.e., the mid-meningeal arteries and vessels), and subsequent leakage of blood between the periosteal layer of the dura mater and the endocranial surface causing their separation (Moore & Dalley, 2006; Marieb & Hoehn, 2008), are also a source of bone lesions (Schultz, 2001; Ortner, 2003). Whereas some hemorrhages

may definitely be the result of trauma to the skull, Schultz (2001) contends that birth itself may be a cause of that trauma.

Dynamic Locations

Another topic that requires consideration is that of specific anatomical features located within the neurocranium as compared to the different anatomical regions or features associated with different systemic aspects. These features are those that change over the period of time it takes for the skull, and more specifically the neurocranium, to reach maturity. These features are dynamic in nature and occur where two bones are fusing to become one, such as at the petromastoid suture in the temporal bones and the sutura mendosa in the occipital bone; where one bone is continuing to grow and expand, such as the sagittal, coronal, lambdoid, and squamosal sutures of the parietal bones; or in areas where both processes are taking place concurrently, such as the metopic suture of the frontal bone(s). There are approximately 60 dynamic bone features located on the seven substantial bones that comprise the endocranial surface.

Extraneous new bone growth, no matter the etiology, can manifest on any location of the endocranial surface; however, there may be certain locations that may be more susceptible to new bone growth. Dynamic features and features associated with the sutures (which have both been shown to be locations where the dura mater is very tightly adhered to the endocranial surface) may have a stronger association to non-pathological extraneous new bone growth than other areas or features of the braincase, while circulatory features may have a stronger association to lesions due to pathologies than other areas of the neurocranium.

To date, the determination of whether extraneous new bone growth located within the endocrania has a pathological etiology remains difficult to ascertain. This may be due to a lack of understanding of bone development in a clinical setting, or the lack of information detailing where, and when, in the human body non-pathological lesions may arise. In order to understand the presence and occurrence of extraneous new bone growth due to pathological conditions and non-pathological processes on the endocranial surface of the skull, each variable must be examined in turn. The variables, age-at-death of the individual, location of the bone lesions (anatomic, systemic, and dynamic) and the presence of postcranial and/or ectocranial pathological indicators, may specify when, where, and under what circumstances extraneous new bone growth may occur.

CHAPTER III

EXPERIMENTAL APPROACH AND METHODS

Premise

This study seeks to differentiate between extraneous new bone growth caused by developmental processes and bone growth caused by pathological processes. This research will provide insight into the etiology of endocranial lesions by determining which variables – such as the presence of postcranial and/or ectocranial pathological conditions, the age-at-death of the individual, and the location of extraneous new bone growth according to region, functionality, or morphological area - have a statistical association with the presence of lesions on the endocranial surface of the skull. These associations will then be used to determine if the variables, either singly or in conjunction with one another, are contributing factors in the formation of new endocranial bone growth.

The Sample

The sample population used for this research was comprised of 129 individuals ranging from birth to 15 years in age. The individuals belonged to 15 different Native American groups from five states and two cultural regions. This sample population diversity will overcome one very important concern in this type of research; that any or all bone lesion patterns observed on the skeletal material would be skewed due to a lack

of sample heterogeneity. In addition, a diverse sample population would thereby

eliminate the possibility that any results obtained could be due to the distinctive aspects

of a microenvironment or single population.

An inventory of all skeletal elements associated with each individual was

conducted to determine if sufficient remains were present to conduct further research

(Appendix 1). The requirements selected were that at least 75% of each individual

cranial bone was present on at least one side, and at least 50% of a paired bone was

present. If, for example, an unpaired bone was examined, 75% of the bone must be

present, which was also the condition for paired bones of which the bone from the

opposing side was missing. For postcranial remains the requirement was less stringent; at

least 50% of the postcranial skeletal elements needed to be present. In addition, because

pathological lesions are usually bilateral (Shopfner, 1966; Mensforth et al., 1978), only

one of each paired bone needed to be present.

Only those individuals who exhibited bone changes that were formed as a direct

result of an osteoblastic reaction of the periosteum, and not as a secondary reaction of the

periosteum (i.e., any pathological bone changes that may occur as a consequence of

malformations or congenital defects), were included in the sample.

Of the nine North American prehistoric populations curated at the Field Museum

of Natural History in Chicago, Illinois, 30 individuals met the criteria for inclusion in this

study (Table 1), and, of the six prehistoric North American populations curated at the

Illinois State Museum in Springfield, Illinois, 99 individuals met those criteria (Table 2).

Table 1. Sites and Number of Skeletal Remains
Analyzed from the Field Museum of Natural History

Site	Pop. Size	Sub-Adults	With Crania
AZ (Canyon de Chelly)	25	7	5
AZ (Mineral Creek Pueblo)	4	1	1
CO (Ackmen)	10	2	1
CO ("Cliff Dweller")	10	1	1
NM (Higgins Flat Pueblo)	15	9	7
NM (Oak Springs Pueblo)	8	3	1
NM (S.U. Site)	33	8	1
OH (Fort Ancient)	48	17	11
OH (Taylor's Farm, Oregonia)	29	2	2
Total	**182**	**50**	**30**

Table 2. Sites and Number of Skeletal Remains
Analyzed from the Illinois State Museum

Site	Pop. Size	Sub-Adults	With Crania
IL (Albany Mounds)	262	71	3
IL (Dickson Mounds)	1219	652	54
IL (East St. Louis Stone Quarry)	121	46	4
IL (Elizabeth Mounds)	277	101	26
IL (Kuhlman Mounds)	273	116	11
IL (Tree Row)	122	42	1
Total	**2274**	**1028**	**99**

Methods and Techniques

The age-at-death was assessed through the use of postcranial long bone lengths

(Scheuer & Black, 2000; Black & Scheuer, 1996; Saunders, Hoppa, & Southern, 1993;

Maresh, 1970; Gindhart, 1973; Anderson, Messner, & Green, 1964), ossification patterns

of secondary ossification sites (Scheuer & Black, 2000), fusion rates of primary/primary

and primary/secondary ossification centers (Scheuer & Black, 2000), and dental

development and eruption patterns (Moorees, Fanning, & Hunt, 1963a, 1963b; Ubelaker,

1989). Age categories were established by utilizing research previously published on

skeletal growth and development and the occurrence of pathological processes. The age

interval of birth to 1.9 years was assigned because the most rapid stage of neurocranial

growth occurs within that age range and also appears to coincide with a period of greater

amounts of endocranial lesions possibly due to chronic diseases put forth by Gordon,

Wyon, & Ascoli (1967) and Mensforth et al. (1978). The age interval of 2 years to 3.9

years was assigned due to the correspondence between this age range and a lowering of

immune functions within sub-adults and an increase in certain age-related pathologies as

mentioned previously (Gordon et al., 1967; Mensforth et al., 1978). The age interval of 4

to 6.9 years was assigned for the reason that seven years of age is when some authors

argue that the volumetric growth of the cranium ceases (Kabanni & Raghuveer, 2004;

Lewis, 2004; Pick & Howden, 2003). This was also the reason for the assignment of the

next age grouping of 7 to 9.9 years; as stated previously, ten years of age is also when

some authors argue that the volumetric growth of the cranium ceases (Scheuer & Black,

2000). The age category consisting of individuals ten years of age and older, holding that

braincase growth ceases by the lower margin of this category, is a period of no

neurocranial growth. Of the 129 individuals examined for this study, 39 were between

the ages of birth and 1.9 years, 44 were between the ages of 2 and 3.9 years, 17 were

between the ages of 4 and 6.9 years of age, seven were between the ages of 7 and 9.9

years of age, and 22 were ten years of age or older (Figure 1).
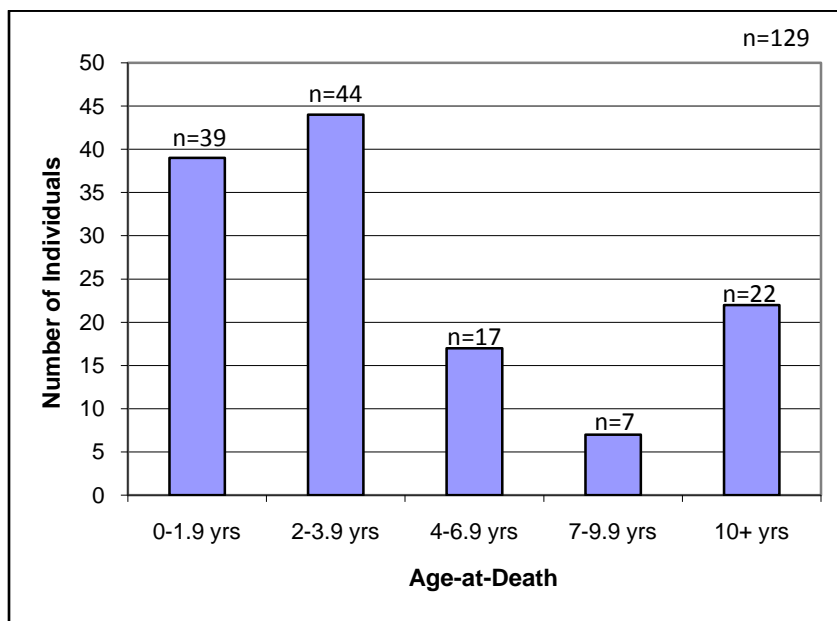
**Figure 1. Distribution by Age-at-Death
of the Individuals in the Sample Population**

All endocranial surfaces of the neurocranium were macroscopically examined for the presence of extraneous new bone growth. Bone growth that was composed of woven bone (Figure 2) was identified by having a raised appearance, striations, a pitted or porous surface, a rough texture, and definitive margins. This type of new bone growth has been described as "small, loosely attached patches of tree bark" (Mann & Murphey, 1990, p. 109), "spiculated new layers of bone," and "fibrous, vascular, somewhat irregular…and gives the appearance of a 'scab' over new bone" (Mensforth et al., 1978, p. 9).
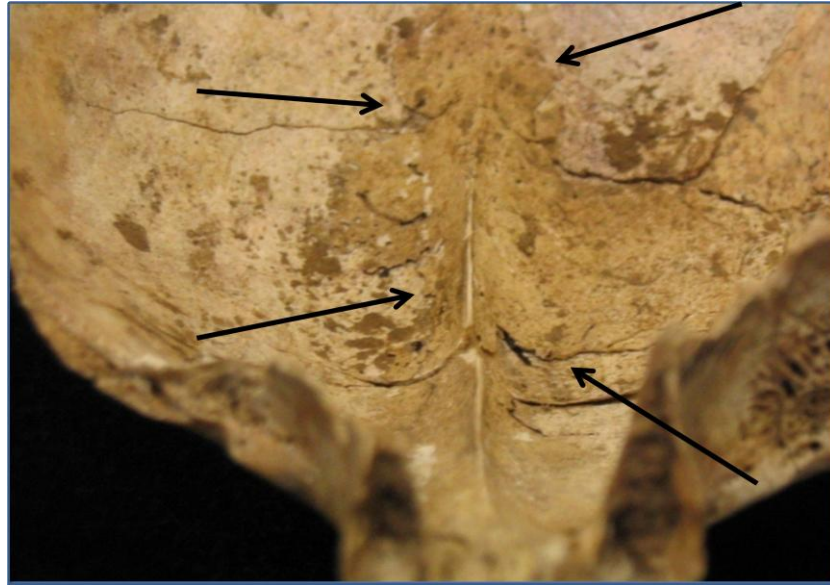
**Figure 2. Woven Bone on the Endocranial Surface
of the Frontal Bone (arrows indicate the borders of the woven bone)**

Lamellar bone (Figure 3) on the endocranial surfaces was identified by its raised appearance with definitive margins, but having a smooth surface and texture. It appears notably more solid and compact, and less porous (Mensforth et al., 1978; Roberts & Manchester, 2005).
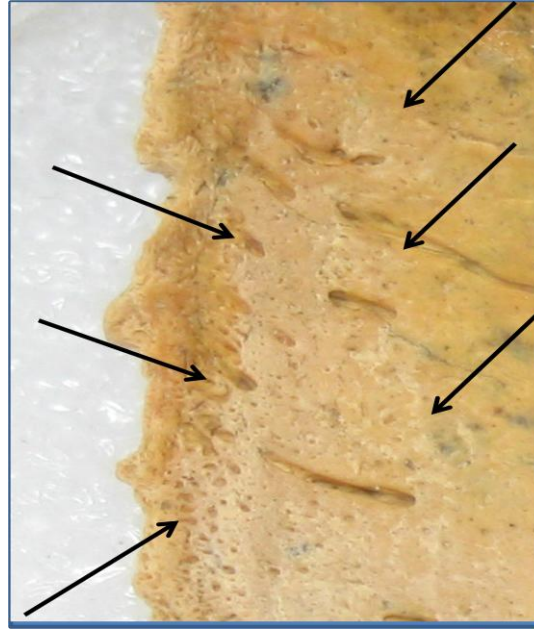
**Figure 3. Lamellar Bone on the Endocranial Surface
of the Parietal Bone (arrows indicate the borders of the lamellar bone)**

The areas of the braincase where new bone growth was found were placed into four separate groups and further placed into discrete categories; they are anatomic locations, which are areas on the cranial vault, base, or intermediate area between the two: systemic locations, which are areas adjacent to the sutures (Figure 4), areas associated with circulatory features (Figure 5), areas with an overlap of both the suture and circulatory features (Figure 6), or areas with no association to the sutural and/or circulatory features (hereafter referred to as "non-systemic features"; dural adherence locations, which are features with a tighter association with the dura mater (Figure 7), and features with a normal association; and features that are dynamic at some point within the life span of a sub-adult, as well as those features that are non-dynamic.

Figure 4. Examples of Sutural Areas on the Endocranial Surface of the Skull.
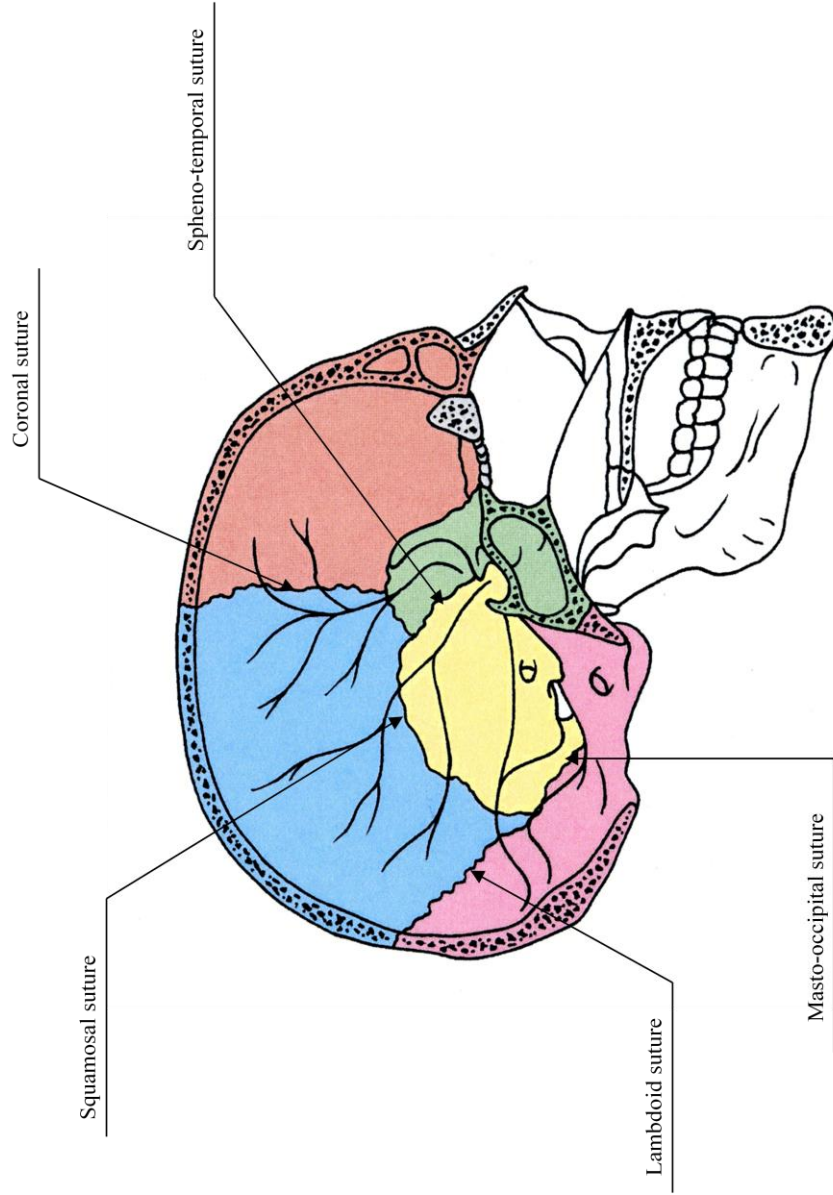
Figure 5. Examples of Circulatory Systemic Features on the Endocranial Surface of the Skull.
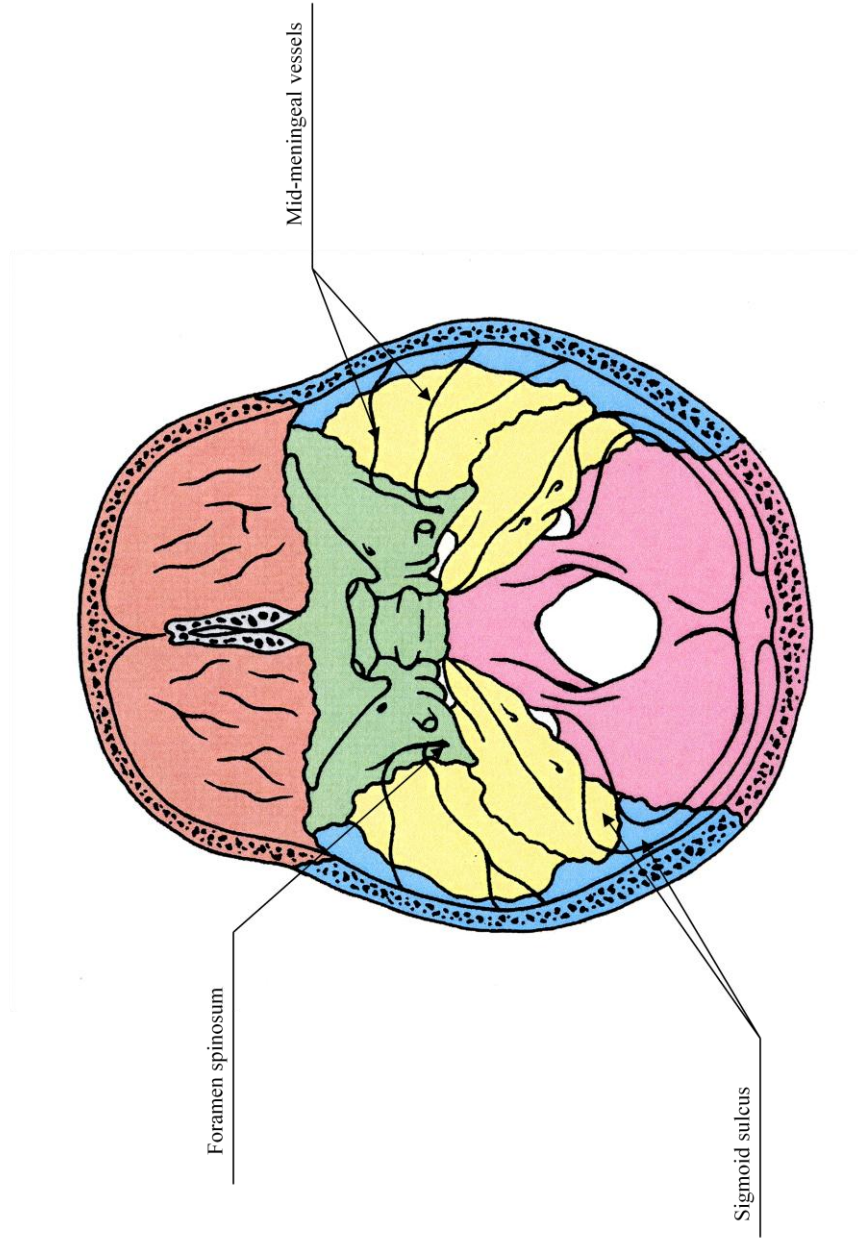
Figure 6. Examples of Overlapping Sutural and Circulatory Areas on the Endocranial Surface of the Skull.

Superior sagittal sulcus/Metopic suture

Sagittal sinus/Sagittal suture
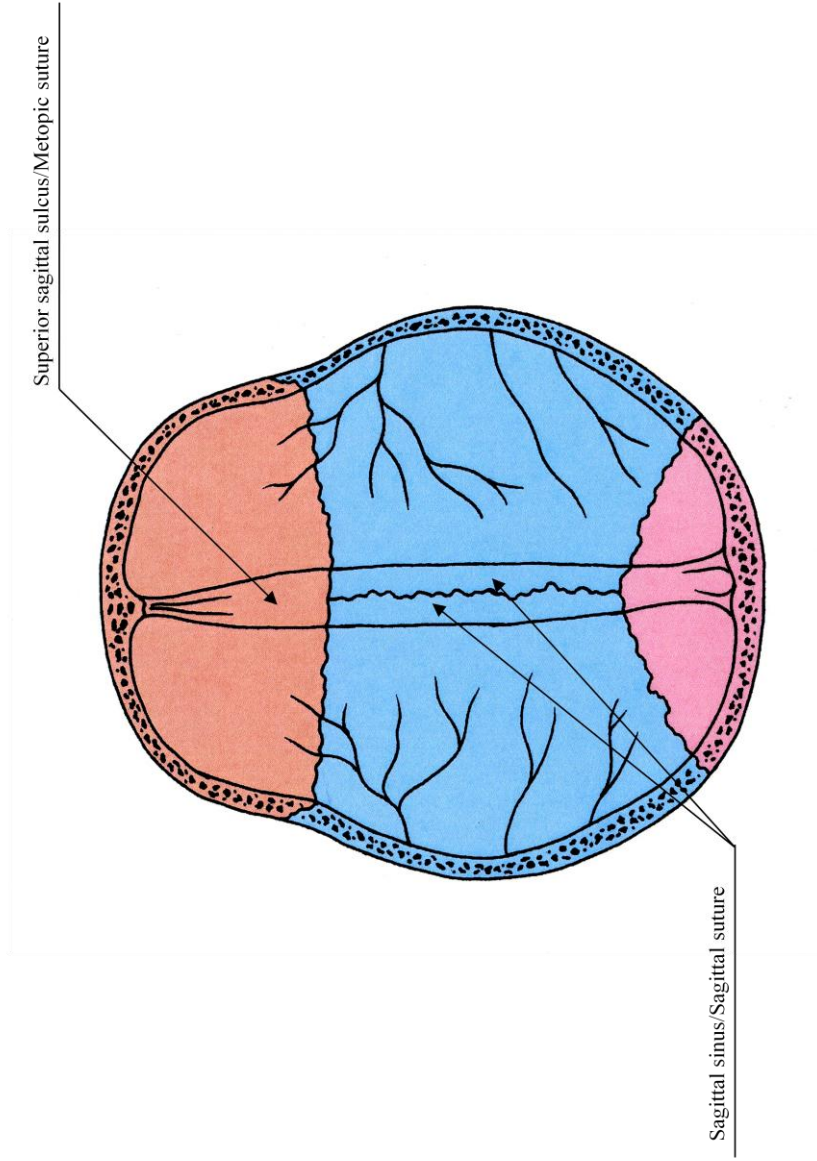
Figure 7. Examples of Tight Dural Attachment Sites on the Endocranial Surface.
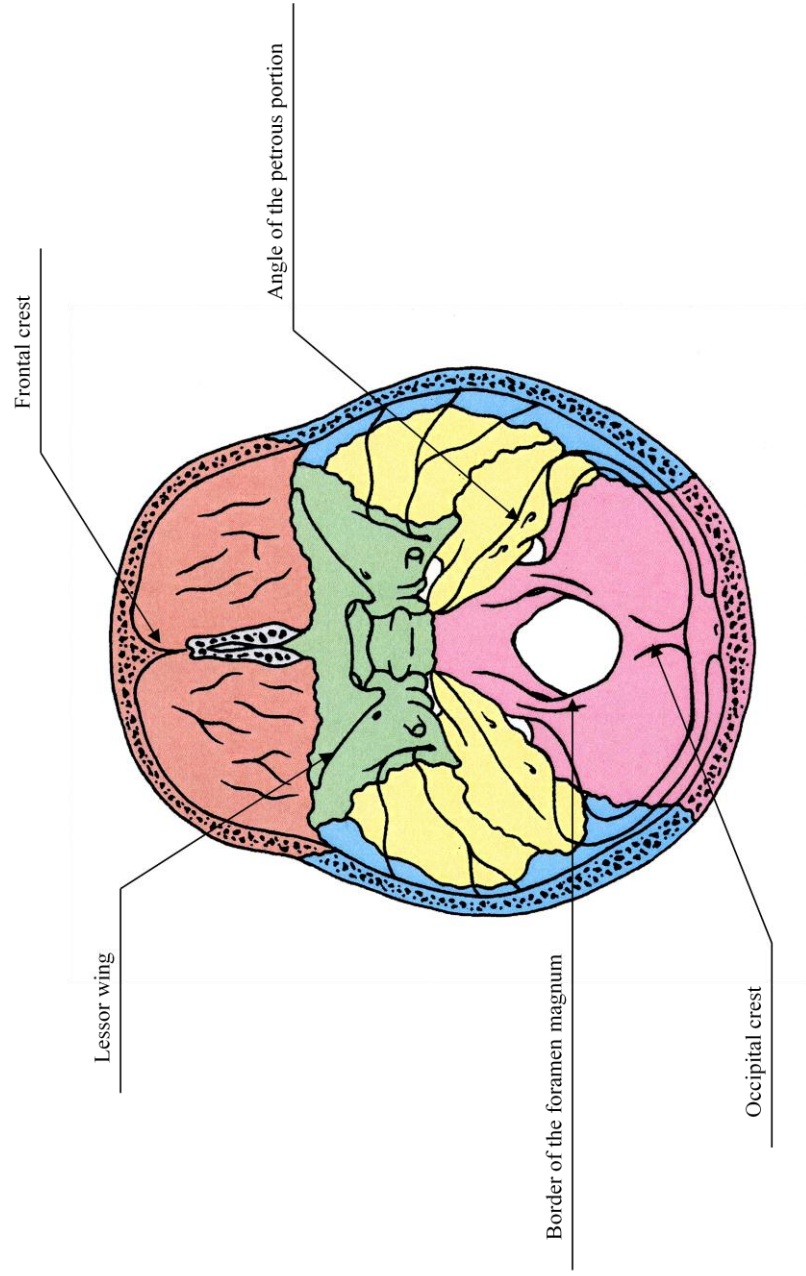
Ectocranial and postcranial bones were macroscopically examined for the presence of pathological lesions. Pathological lesions were either identified by their location on the bone(s), such as being in conjunction with certain features like nutrient foramina, the extent of the surface area the lesion covered, or a combination of the two. The locations of all lesions were recorded by individual bone and feature and the side from which the bone came. The extent of affected bone tissue was also recorded to determine if bone changes were localized to a specific area on the surface of the bone(s) or if they were large enough to encompass multiple bone features.

The lesion location, extent of bone involvement, and age-at-death assessments were recorded on data collection sheets for each individual (Appendices 2 and 3). This information was entered into a computer spreadsheet in Microsoft Excel 2007, and subsequent statistical tests were performed. Chi-square tests were conducted on the data to explore the strength of the associations between the variables.

CHAPTER IV

RESULTS

To understand the presence of extraneous new bone growth on the endocranial surface of the skull, data on the presence of postcranial and/or ectocranial indicators of pathology, age of the individual, and the location of endocranial lesions must be collected and statistically tested both singularly and in conjunction with one another.

Frequency of Endocranial Bone Changes (ECB)

ECB were found in 59.7% (77 of 129) of the individuals examined. Further analysis indicates that less than half, 47.8% (32 of 67), of the individuals examined who did not have postcranial/ectocranial pathological indicators (PI) had ECB whereas almost three-fourths of those individuals who did have PI had ECB (72.6%, or 45 of 62 individuals). Chi-square analysis of independence between the presence of ECB and the presence of PI proved statistically significant (Table 3).

Table 3. Association Between ECB and PI

PI	ECB Present	ECB Absent	Total
Present	45	17	62
Absent	32	35	67
Total	77	52	129

$$X^2_{1,0.05}=3.84, X^2=8.24$$

Frequency of Endocranial Bone Changes by Age

Table 4 shows the number and frequency of individuals with ECB by age-at-death. The data in Table 4 were further broken down by individuals with or without PI. Tables 5 and 6 show the number and frequency of individuals with ECB, both without and with PI, respectively. A graphic representation of the data found within Tables 5 and 6 is depicted in Figure 8.Note that the frequency of individuals with EBC decreases with each consecutive age category for individuals both with, and without, PI. No other statistical tests could be conducted concerning the age-at-death of the individuals with this data as there were too many cell values less than 5.

Table 4. Frequency of ECB by Age-at-Death of the Individual

Age	ECB Present	ECB Absent	Total
0-1.9	34 (87.2%)	5 (12.8%)	39 (100%)
2-3.9	31 (70.5%)	13 (29.5%)	44 (100%)
4-6.9	7 (41.2%)	10 (58.8%)	17 (100%)
7-9.9	2 (28.6%)	5 (71.4%)	7 (100%)
10+	3 (13.6%)	19 (86.4%)	22 (100%)
Total	77	52	129

Table 5. Frequency of ECB in Individuals Not Displaying PI

Age	ECB Present	ECB Absent	Total
0-1.9	18 (81.8%)	4 (18.2%)	22 (100%)
2-3.9	8 (47.1%)	9 (52.9%)	17 (100%)
4-6.9	3 (30.0%)	7 (70.0%)	10 (100%)
7-9.9	1 (25.0%)	3 (75.0%)	4 (100%)
10+	2 (14.3%)	12 (85.7%)	14 (100%)
Total	32	35	67

Table 6. Frequency of ECB in Individuals Displaying PI

Age	ECB Present	ECB Absent	Total
0-1.9	16 (94.1%)	1 (5.9%)	17 (100%)
2-3.9	23 (85.2%)	4 (14.8%)	27 (100%)
4-6.9	4 (57.1%)	3 (42.9%)	7 (100%)
7-9.9	1 (33.3%)	2 (66.7%)	3 (100%)
10+	1 (12.5%)	7 (87.5%)	8 (100%)
Total	45	17	62

	0-1.9 yrs	2-3.9 yrs	4-6.9 yrs	7-9.9 yrs	10+ yrs
Individs with no PI	81.82%	47.06%	30.00%	25.00%	14.29%
Individuals with PI	94.12%	85.19%	57.14%	33.33%	12.50%
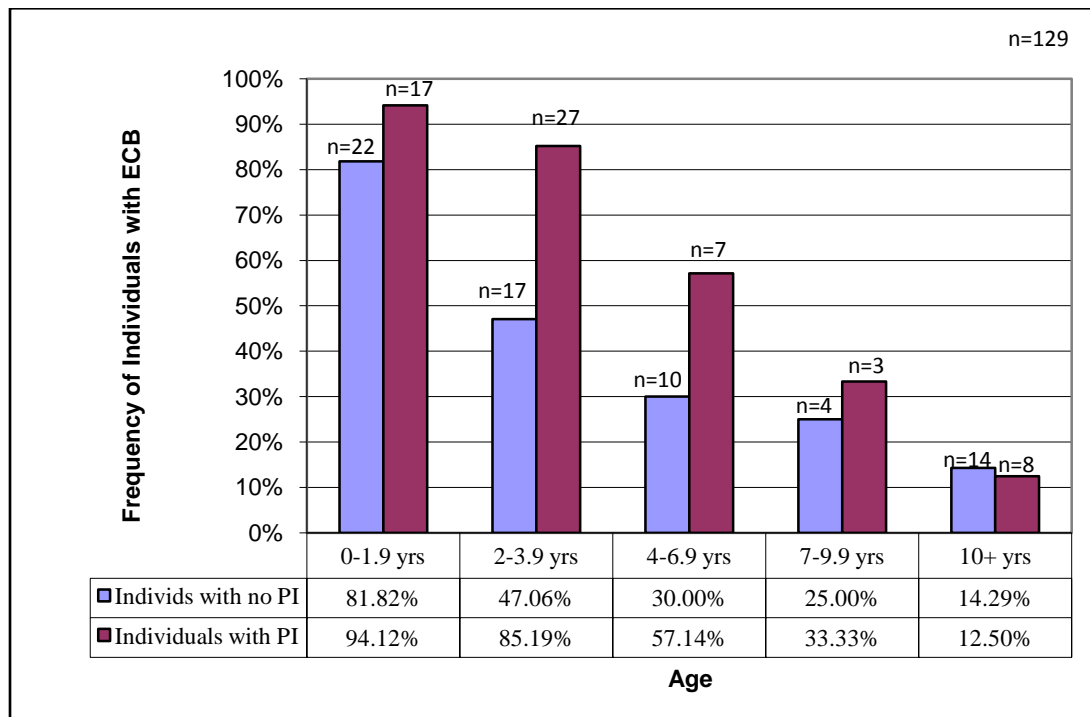
Figure 8. Frequency of Individuals with ECB and PI.
Percentages were calculated by dividing the number of individuals with ECB
by the number of total individuals, in each age category.

Frequency of Endocranial Bone Changes by Location

The results of the analyses of the systemic locations indicate that ECB were found on features associated with the sutural system in 30.2% (39 of 129 individuals), on features associated with the circulatory system in 31.8% (41 of 129 individuals), on features that are considered to be a part of both the sutural and circulatory systems in 28.7% (37 of 129 individuals), and on features that are a part of neither system in 29.5% (38 of 129) of the individuals examined. The frequency of endocranial bone changes on systemic locations in individuals with, and without, pathological indicators is represented in Figure 9. Figure 9 shows that individuals with ECB and PI on features of the circulatory system and features not associated with any systemic locale are significantly higher in frequency than those individuals with ECB but no PI, whereas individuals with ECB and PI on features of the sutural system and features that overlap both the sutural and circulatory systems are not significantly higher in frequency than those individuals with ECB but no PI.
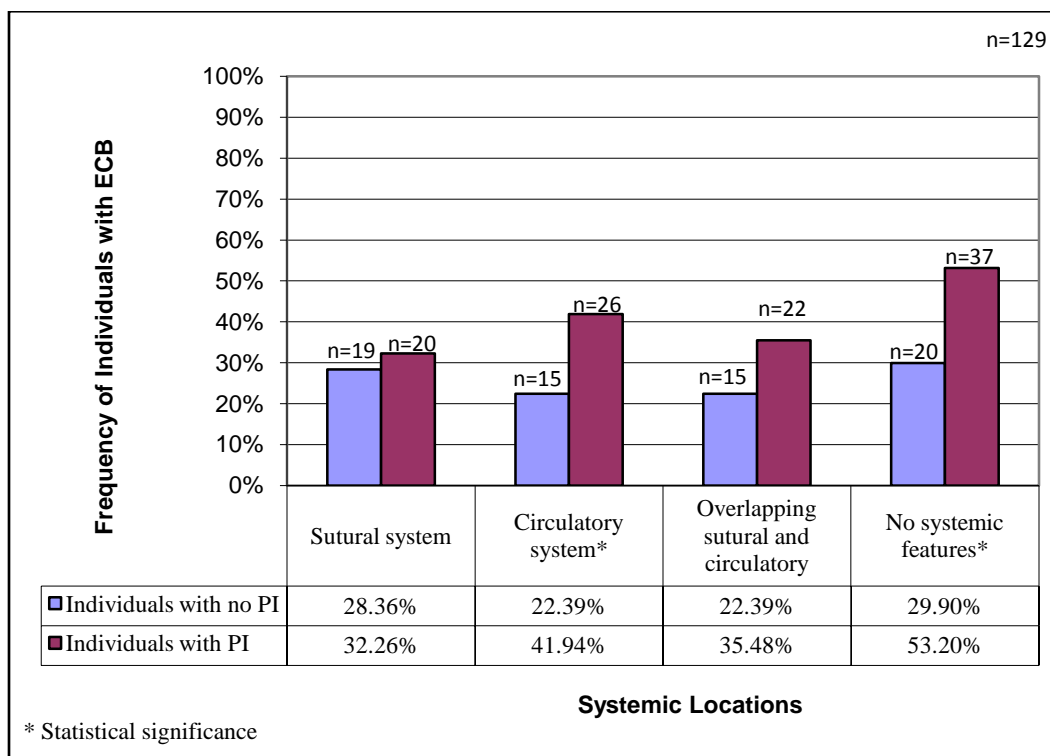
Figure 9. Frequency of Individuals with ECB by Systemic Location, and PI. Percentages were calculated by dividing the number of individuals with ECB by the total number of individuals, in each category. The features associated with the circulatory system were statistically significant at the p=.05 level.

Chi-square analysis of this data indicates that the presence of ECB found on features of the sutural system, and the features of the overlapping area of the sutural and circulatory systems, are not statistically significant (Tables 7 & 8), whereas the presence of ECB found on just the circulatory systemic features and the non-systemic features is statistically significant with the presence of PI (Tables 9 & 10).

Table 7. Association Between ECB and PI on the Sutural Systemic Features

PI	ECB Present	ECB Absent	Total
Present	20	42	62
Absent	19	48	67
Total	39	90	129

$$X^2_{1,0.05}=3.84, X^2=0.23$$

Table 8. Association Between ECB and PI on the Overlapping Sutural and Circulatory Systemic Features

PI	ECB Present	ECB Absent	Total
Present	22	40	62
Absent	15	52	67
Total	37	92	129

$$X^2_{1,0.05}=3.84, X^2=2.70$$

Table 9. Association Between ECB and PI on the Circulatory Systemic Features

PI	ECB Present	ECB Absent	Total
Present	26	36	62
Absent	15	52	67
Total	41	88	129

$$X^2_{1,0.05}=3.84, X^2=5.67$$

Table 10. Association Between ECB and PI on the Non-systemic Features

PI	ECB Present	ECB Absent	Total
Present	35	27	62
Absent	22	45	67
Total	57	72	129

$$X^2_{1,0.05}=3.84, X^2=7.27$$

When the dural attachment sites are observed, the results of the analyses of ECB at these locations indicate that lesions were found on 48.1% (62 of 129) of the individuals at strong attachment sites of the dura mater; also 52.7% (68 of 129) of the individuals have ECB at sites that do not have as strong an attachment. The frequency of endocranial bone change on dural adherence sites in individuals with, and without, pathological indicators is represented in Figure 10. Figure 10 shows that individuals with ECB and PI on both types of dural attachment sites are significantly higher in frequency than those individuals with ECB but no PI.
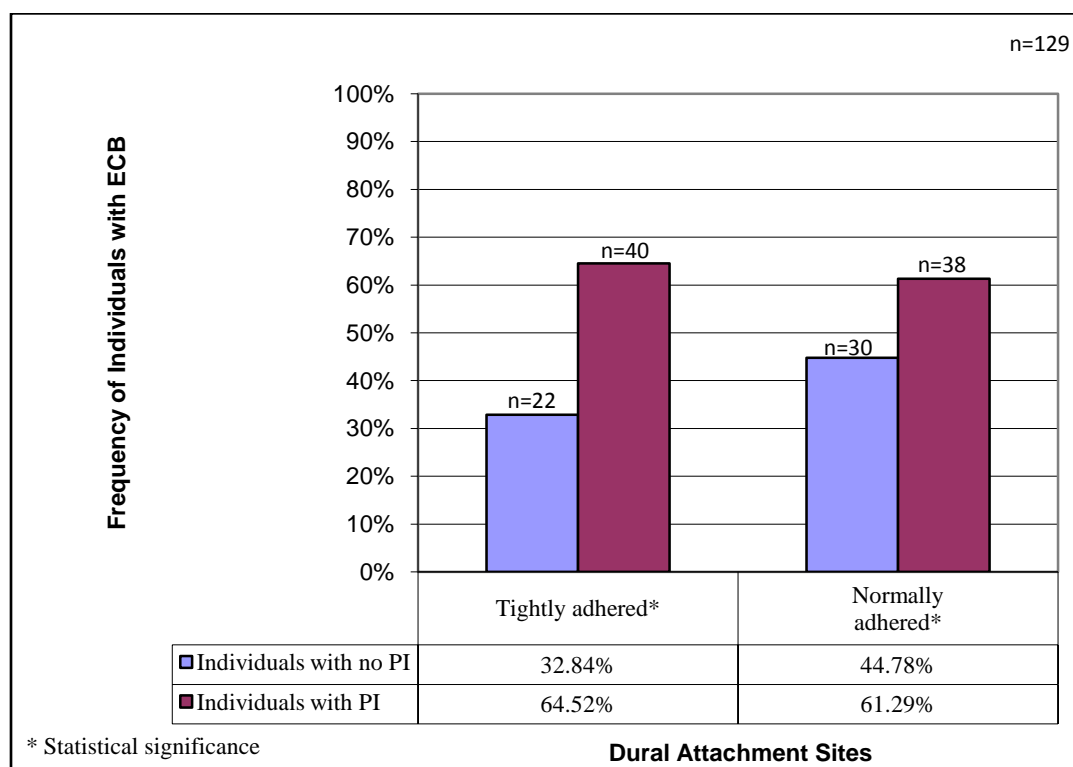
Figure 10. Frequency of Individuals with ECB by Dural Attachment Sites, and PI. Percentages were calculated by dividing the number of individuals with ECB by the total number of individuals, in each category. Those features associated with areas tightly adhered were statistically significant at the p=.05 level.

Chi-square analysis of the above data indicates that the presence of ECB is statistically significant on tight dural attachment sites (Table 11) and is also significant on normal dural attachment sites (Table 12). A chi-square test was also conducted on tightly adhered dural features with no circulatory features included. The result of that test showed no association between the presence of ECB and pathological indicators (Table 13). Another chi-square test was conducted on circulatory features with no tightly adhered dural features included. The result of that test showed that there was an association between the presence of ECB and pathological indicators (Table 14).

Table 11. Association Between ECB and PI on Tight Dural Attachment Sites

PI	ECB Present	ECB Absent	Total
Present	40	22	62
Absent	22	45	67
Total	62	67	129

$$X^2_{1,0.05}=3.84, X^2=12.94$$

Table 12. Association Between ECB and PI on Normal Dural Attachment Sites

PI	ECB Present	ECB Absent	Total
Present	38	24	62
Absent	28	39	67
Total	66	63	129

$$X^2_{1,0.05}=3.84, X^2=4.90$$

**Table 13. Association Between ECB and PI on Tight Dural Attachment Sites
with No Circulatory Systemic Features Included**

PI	ECB Present	ECB Absent	Total
Present	22	40	62
Absent	15	52	67
Total	37	92	129

$$X^2_{1,0.05}=3.84, X^2=2.68$$

**Table 14. Association Between ECB and PI on
Circulatory Systemic Features with No Tight Dural Attachment Features Included**

PI	ECB Present	ECB Absent	Total
Present	6	56	62
Absent	1	66	67
Total	7	122	129

$$X^2_{1,0.05}=3.84, X^2=4.22$$

Two more chi-square tests were conducted on this data; the first tested the

statistical significance of normally adhered dural features with no circulatory features

included (Table 15), and the second tested the significance of circulatory systemic

features with no normally adhered dural features included (Table 16). The results

showed that normally adhered dural features were no longer statistically significant

between the presence of ECB and PI when circulatory features were removed, but that

circulatory systemic features were statistically significant when normally adhered dural

features were removed. Tests could not be performed on tightly adhered dural features

with non-systemic features removed, or vice-versa, because non-systemic features have

only dural attachment sites that are normally and none that are tightly adhered; also tests

could not be performed on normally adhered dural features with non-systemic features

removed, or vice versa, for the same reason. There are only non-systemic features that

have a normal adherence to the surface of the endocrania, and none that are tightly

adhered.

Table 15. Association Between ECB and PI on
Normal Dural Attachment Sites with No Circulatory Features Included

PI	ECB Present	ECB Absent	Total
Present	36	26	62
Absent	28	39	67
Total	64	65	129

$$X^2_{1,0.05}=3.84, X^2=3.41$$

Table 16. Association Between ECB and PI on
Circulatory Features with No Normal Dural Attachment Features Included

PI	ECB Present	ECB Absent	Total
Present	25	37	62
Absent	15	52	67
Total	40	89	129

$$X^2_{1,0.05}=3.84, X^2=4.85$$

The results of the analyses of the anatomic locations and those ECB associated

with them indicate that ECB were found on features of the cranial vault in 50.4% (64 of

129) of individuals, on features of the cranial base in 45.0% (58 of 129) of individuals,

and on those features that are intermediate between the cranial vault and base in 16.3%

(21 of 129) of the individuals examined. The frequency of endocranial bone change on

different anatomical locations in individuals with, and without, pathological indicators is

represented in Figure 11. Figure 11 shows that individuals with ECB and PI on features

of the cranial vault are significantly higher in frequency than in those individuals with

ECB but no PI, whereas individuals with ECB and PI on features of the cranial base and

intermediate area are not higher in frequency than in those individuals with ECB but no

PI.
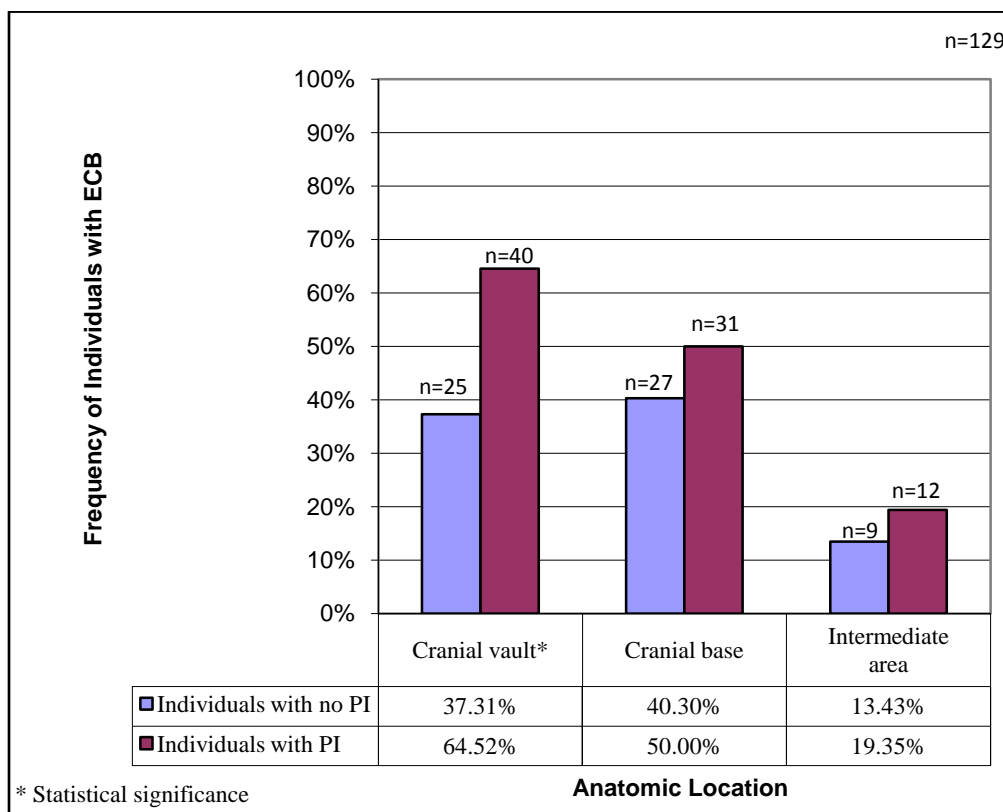
**Figure 11. Frequency of Individuals with ECB by
Anatomic Location, and PI. Percentages were calculated by dividing the number of
individuals with ECB by the total number of individuals, in each category. Data
derived from the cranial vault were statistically significant at the p=.05 level.**

Chi-square analysis of these data indicates that the presence of ECB found on the

cranial vault and the presence of pathological indicators are statistically significant for

their location (Table 17), whereas the presence of ECB and the presence of pathological

indicators found on the cranial base and intermediate area are not statistically significant

for their location (Tables 18 & 19).

Table 17. Association Between ECB and PI on the Cranial Vault

PI	ECB Present	ECB Absent	Total
Present	40	22	62
Absent	25	42	67
Total	65	64	129

$$X^2_{1,0.05}=3.84, X^2=9.53$$

**Table 18. Association Between ECB and PI
on the Intermediate Area of the Cranial Vault**

PI	ECB Present	ECB Absent	Total
Present	12	50	62
Absent	9	58	67
Total	21	108	129

$$X^2_{1,0.05}=3.84, X^2=0.83$$

Table 19. Association Between ECB and PI on the Cranial Base

PI	ECB Present	ECB Absent	Total
Present	31	31	62
Absent	27	40	67
Total	58	71	129

$$X^2_{1,0.05}=3.84, X^2=1.22$$

In addition, a chi-square test was conducted on the association between ECB and the presence of pathological indicators in the vault, this time with no circulatory features added. The test statistic showed that there was still an association (Table 20). It is also necessary to perform a chi-square test on the vault excluding any features that are not associated with any particular bodily system. This test showed that there is still an association between the presence of ECB and indicators of pathology (Table 21). A third test was conducted on the vault, this time removing those features that have a tight dural attachment to the underlying bone, and again, the test result is that there is still statistical significance between the presence of ECB and pathological indicators (Table 22).

Table 20. Association Between ECB and the Cranial Vault
with No Circulatory Systemic Features Included

PI	ECB Present	ECB Absent	Total
Present	30	32	62
Absent	16	51	67
Total	46	83	129

$X^2_{1,0.05}=3.84$, $X^2=8.42$

Table 21. Association Between ECB and PI on the Cranial Vault
with No Non-systemic Features Included

PI	ECB Present	ECB Absent	Total
Present	32	30	62
Absent	21	46	67
Total	53	76	129

$X^2_{1,0.05}=3.84$, $X^2=5.47$

**Table 22. Association Between ECB and PI on the Cranial Vault
with No Tight Dural Attachment Features Included**

PI	ECB Present	ECB Absent	Total
Present	27	35	62
Absent	11	56	67
Total	38	91	129

$X^2_{1,0.05}=3.84, X^2=11.42$

Three more chi-square tests were conducted on the cranial vault: the first

excluded the features of both the circulatory system and those with no systemic

association, the second excluded both the circulatory systemic features and the tightly

adhered dural features, and the third excluded both the non-systemic features and the

tightly adhered dural features. Of the three tests, only the test that checked the

association between the presence of ECB and pathological indicators on the vault with no

features from the circulatory system and features that are tightly adhered to the

endocranial surface showed significance (Tables 23, 24, & 25). No statistical test could

be conducted on the cranial vault in which all three groups of features (i.e., the

circulatory systemic, the non-systemic, and the tightly adhered dural features) were

excluded as that would have left no bone features on the entire cranial vault to be tested.

**Table 23. Association Between ECB and PI on the Cranial Vault
with No Circulatory and/or Non-systemic Features Included**

PI	ECB Present	ECB Absent	Total
Present	23	39	62
Absent	15	52	67
Total	38	91	129

$X^2_{1,0.05}=3.84, X^2=3.39$

**Table 24. Association Between ECB and PI on the Cranial Vault
with No Circulatory and/or Tightly Adhered Dural Features Included**

PI	ECB Present	ECB Absent	Total
Present	25	37	62
Absent	11	56	67
Total	36	93	129

$X^2_{1,0.05}=3.84$, $X^2=9.15$

**Table 25. Association Between ECB and PI on the Cranial Vault
with No Non-systemic and/or Tightly Adhered Dural Features Included**

PI	ECB Present	ECB Absent	Total
Present	12	50	62
Absent	7	60	67
Total	19	110	129

$X^2_{1,0.05}=3.84$, $X^2=-1.34$

Frequency of Endocranial Bone Changes by Dynamism

The results of the analyses of the dynamic locations and ECB associated with

them indicate that endocranial bone changes were found on dynamic features in 49.5%

(53 of 129) of individuals, and on non-dynamic features in 52.5% (69 of 129) of

individuals examined. The frequency of endocranial bone change on dynamic locations

in individuals with, and without, pathological indicators is represented in Figure 12.

Figure 12 shows that individuals with ECB and PI on non-dynamic features are

significantly higher in frequency than those individuals with ECB but no PI, whereas

individuals with ECB and PI on dynamic features are not significantly higher in
frequency than those individuals with ECB but no PI.
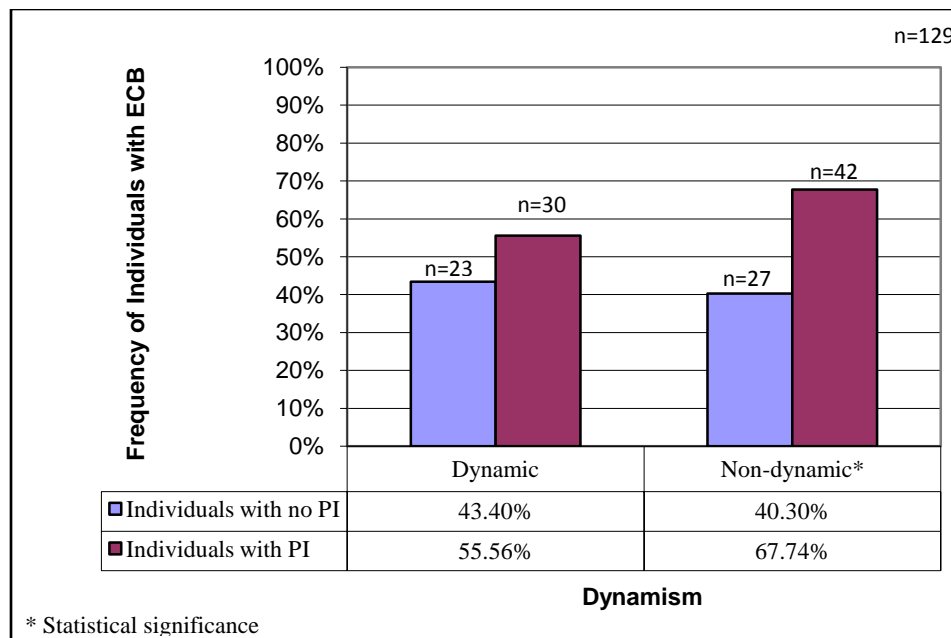
Figure 12. Frequency of Individuals with ECB by Dynamism and PI. Percentages were calculated by dividing the number of individuals with ECB by the total number of individuals, in each category. Non-dynamic features were statistically significant at the p=.05 level.

Chi-square analysis of these data shows that the presence of ECB is statistically
significant to pathological indicators on dynamic features (Table 26), whereas it is not
significant to pathological indicators on non-dynamic features (Table 27).

Table 26. Association Between ECB and PI on Dynamic Features

PI	ECB Present	ECB Absent	Total
Present	30	24	54
Absent	23	30	53
Total	53	54	107

$X^2_{1,0.05}=3.84$, $X^2=1.58$

Table 27. Association Between ECB and PI on Non-dynamic Features

PI	ECB Present	ECB Absent	Total
Present	42	20	62
Absent	27	40	67
Total	69	60	129

$X^2_{1,0.05}=3.84$, $X^2=9.75$

In addition, three chi-square tests were conducted on the association between ECB and the presence of postcranial/ectocranial indicators of pathology on non-dynamic features, this time with no exclusively circulatory features included. The test statistic showed that there was still no association (Table 28). It was also necessary to perform a chi-square test on the non-dynamic features with no features that were not associated with either the sutural and circulatory features. This would give the exact result as the chi-square test conducted only non-systemic features and the presence of PI (Table 10). In addition, two more statistical tests were done, one with no circulatory, and the other with no non-systemic features included. Both of these tests showed the result of no association (Tables 29 & 30).

**Table 28. Association Between ECB and PI on
Non-dynamic Features with No Circulatory Systemic Features Included**

PI	ECB Present	ECB Absent	Total
Present	37	25	62
Absent	24	43	67
Total	61	68	129

$X^2_{1,0.05}=3.84$, $X^2=7.35$

**Table 29. Association Between ECB and PI on
Non-dynamic Features with No Non-systemic Features Included**

PI	ECB Present	ECB Absent	Total
Present	35	27	62
Absent	22	45	67
Total	57	72	129

$X^2_{1,0.05}=3.84$, $X^2=7.27$

**Table 30. Association Between ECB and PI on Non-dynamic Features
with No Circulatory Systemic or Non-systemic Features Included**

PI	ECB Present	ECB Absent	Total
Present	37	25	62
Absent	22	45	67
Total	59	70	129

$X^2_{1,0.05}=3.84$, $X^2=36.82$

Frequency of Endocranial Bone Changes by Location and Dynamism

The results of the analyses of the anatomic locations and dynamic features and

ECB indicate that lesions were found on the dynamic features of the cranial vault in

27.9% (36 of 129) of individuals; lesions on dynamic features of the cranial base show

the same frequency as in the cranial vault and on dynamic features of the intermediate

areas in 3.9% (5 of 129) of individuals examined. The frequency of endocranial bone

change on dynamic locations within the endocranial vault in individuals with, and

without, pathological indicators is represented in Figure 13. Figure 13 shows that

individuals with ECB and PI on dynamic features of the cranial vault, cranial base, and

intermediate area are not significantly higher in frequency than those individuals with

ECB but no PI.
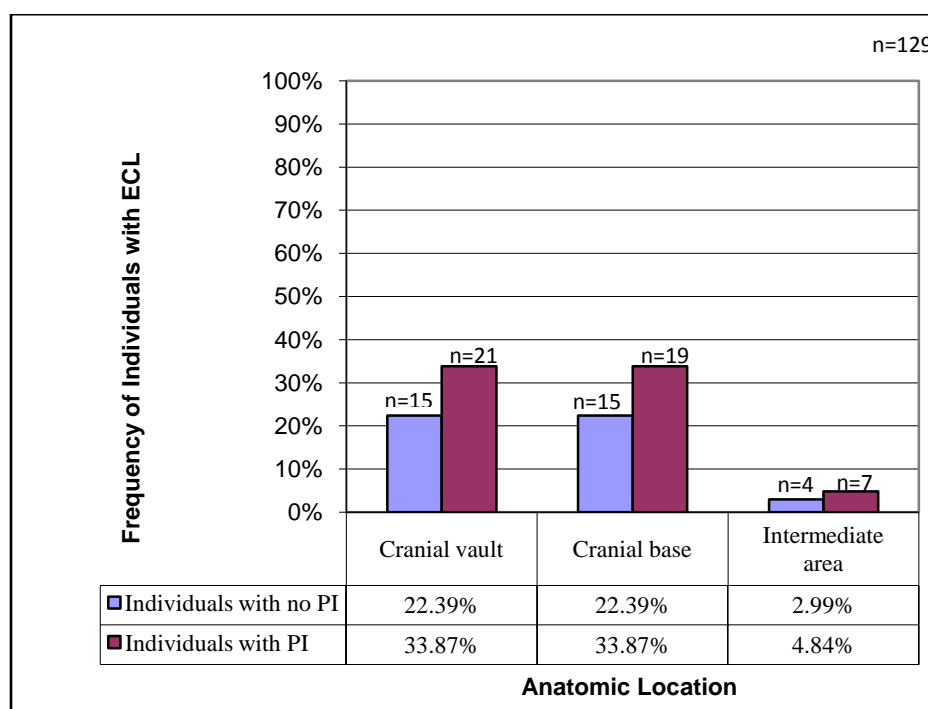
**Figure 13. Frequency of Individuals with ECB by
Anatomic Location on Dynamic Features, and PI. Percentages were calculated by
dividing the number of individuals with ECB by the total number of individuals, in
each category.**

Chi-square analysis of the above information revealed that the distribution of

ECB was independent of pathological indicators on the cranial vault, and the cranial base

(Tables 31 & 32). The chi-square test for the intermediate area was impossible due to too
many cell values under five.

**Table 31. Association Between ECB and PI
on the Dynamic Features of the Cranial Vault**

PI	ECB Present	ECB Absent	Total
Present	21	33	54
Absent	15	38	53
Total	36	71	107

$X^2_{1,0.05}=3.84$, $X^2=1.34$

**Table 32. Association Between ECB
and the Dynamic Features of the Cranial Base**

PI	ECB Present	ECB Absent	Total
Present	19	35	54
Absent	15	38	53
Total	34	73	107

$X^2_{1,0.05}=3.84$, $X^2=0.58$

The results of the analyses of the anatomic locations and non-dynamic features
and ECB indicate that lesions were found on the non-dynamic features of the cranial
vault in 42.6% (55 of 129) of individuals; lesions on non-dynamic features of the cranial
base show that 37.2% (48 of 129) of individuals have ECB, and on non-dynamic features
of the intermediate area 15.5% (20 of 129) of individuals are affected. The frequency of
endocranial bone change on non-dynamic locations within the cranial vault in individuals
with, and without, pathological indicators is represented in Figure 14. Figure 14 shows
that individuals with ECB and PI on non-dynamic features of the cranial vault are

significantly higher in frequency than those individuals with ECB but no PI, whereas

individuals with ECB and PI on the features of the cranial base and intermediate area are

not significantly higher in frequency than those individuals with ECB but no PI.

n=129

	Cranial vault*	Cranial base	Intermediate area
Individuals with no PI	31.34%	31.34%	11.94%
Individuals with PI	54.84%	43.55%	19.35%

* Statistical significance
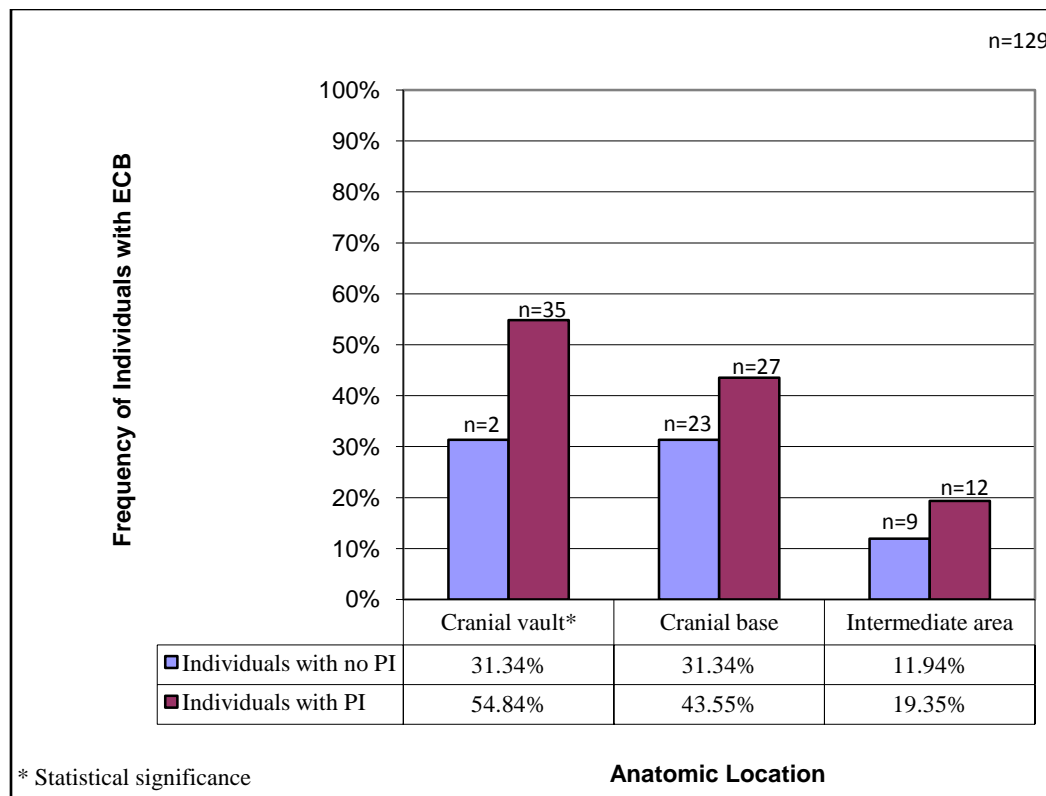
Anatomic Location

Figure 14. Frequency of Individuals with ECB by Anatomic Location on Non-Dynamic Features, and PI. Percentages were calculated by dividing the number of individuals with ECB by the total number of individuals, in each category. The cranial vault was found to be statistically significant at the p=.05 level.

Chi-square analysis of the above information revealed that the distribution of

ECB was statistically significant between pathological indicators and bone changes on

the cranial vault and intermediate area (Tables 33 & 34) but was not significant on the

cranial base (Table 35).

Table 33. Association Between ECB and PI
on Non-dynamic Features of the Cranial Vault

PI	ECB Present	ECB Absent	Total
Present	35	27	62
Absent	21	46	67
Total	56	73	129

$X^2_{1,0.05}=3.84$, $X^2=8.27$

Table 34. Association Between ECB and PI on the
Non-dynamic Features of the Intermediate Area of the Cranial Vault

PI	ECB Present	ECB Absent	Total
Present	12	50	62
Absent	9	58	67
Total	21	108	129

$X^2_{1,0.05}=3.84$, $X^2=0.83$

Table 35. Association Between ECB and PI
on Non-dynamic Features of the Cranial Base

PI	ECB Present	ECB Absent	Total
Present	27	35	62
Absent	23	44	67
Total	50	79	129

$X^2_{1,0.05}=3.84$, $X^2=1.15$

The results of the analyses of the systemic locations indicate that ECB were found

on dynamic features of the sutural system in 29.5% (38 of 129 individuals), on dynamic

features of the circulatory system in 15.5% (20 of 129 individuals), and on those dynamic

features that are considered to be a part of both the sutural and circulatory systems in 18.6% (24 of 129) of the individuals examined. There were no individuals with ECB on both dynamic features and features not associated with either the sutural or circulatory systems, as those two categories have no overlap. The frequency of endocranial bone changes on dynamic locations in conjunction with systemic features in individuals with, and without, pathological indicators is represented in Figure 15. Figure 15 shows that individuals with ECB and PI on dynamic features of any systemic location are not significantly higher in frequency than those individuals with ECB but no PI.
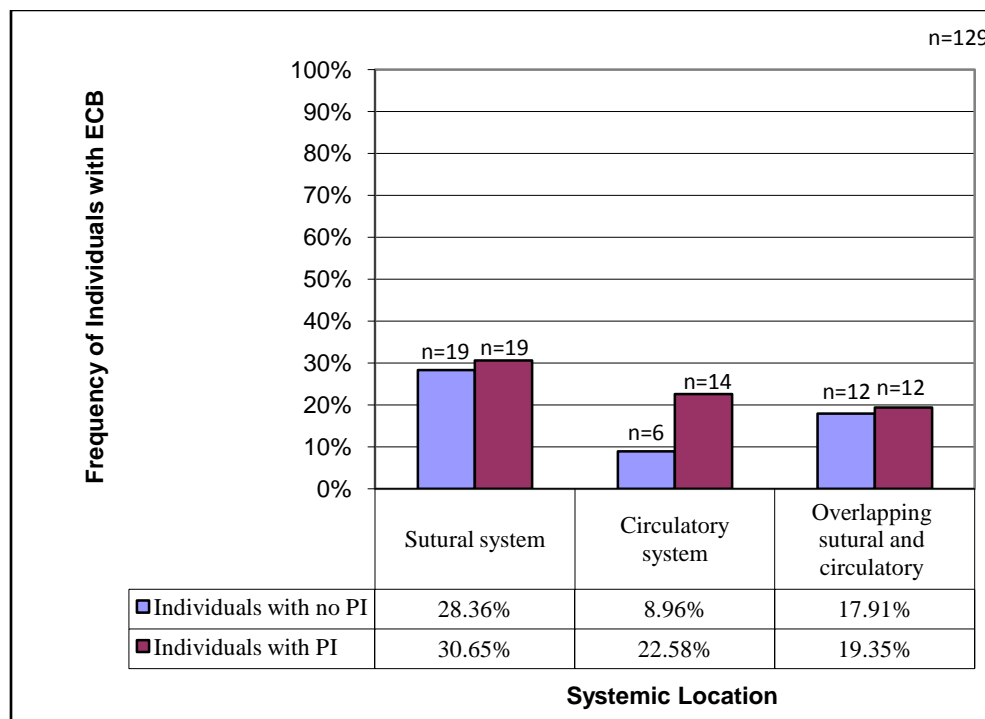
Figure 15. Frequency of Individuals with ECB by Systemic Location on Dynamic Features, and PI. Percentages were calculated by dividing the number of individuals with ECB by the total number of individuals, in each category.

Chi-square analysis of the above information revealed that the distribution of ECB was not statistically significant between pathological indicators and lesions located on the sutural, circulatory, and combined sutural and circulatory systemic features (Tables 36, 37, & 38).

Table 36. Association Between ECB and PI on Dynamic Features of the Sutural System

PI	ECB Present	ECB Absent	Total
Present	19	35	54
Absent	19	34	53
Total	38	69	107

$$X^2_{1,0.05}=3.84, X^2=0.01$$

Table 37. Association Between ECB and the Dynamic Features of the Circulatory System

PI	ECB Present	ECB Absent	Total
Present	14	35	49
Absent	6	39	45
Total	20	74	94

$$X^2_{1,0.05}=3.84, X^2=3.24$$

Table 38. Association Between ECB and PI on Dynamic Features of Overlapping Sutural and Circulatory Systemic Features

PI	ECB Present	ECB Absent	Total
Present	12	42	54
Absent	12	41	53
Total	24	83	107

$$X^2_{1,0.05}=3.84, X^2=0.00$$

The results of the analyses of the systemic locations and ECB indicate that bone changes were found on non-dynamic features of the sutural system in 1.6% (2 of 129 individuals), on non-dynamic features of the circulatory system in 22.5% (29 of 129 individuals), and on those non-dynamic features that are considered to be a part of both the sutural and circulatory systems in 15.5% (20 of 129) of the individuals examined. The frequency of endocranial bone changes on non-dynamic locations in conjunction with systemic features in individuals with, and without, pathological indicators is represented in Figure 16. Figure 16 shows that individuals with ECB and PI on non-dynamic features of the circulatory system are significantly higher in frequency than those individuals with ECB but no PI, whereas those individuals with ECB and PI on features associated with no systemic locales are not significantly higher in frequency than those individuals with ECB but no PI.
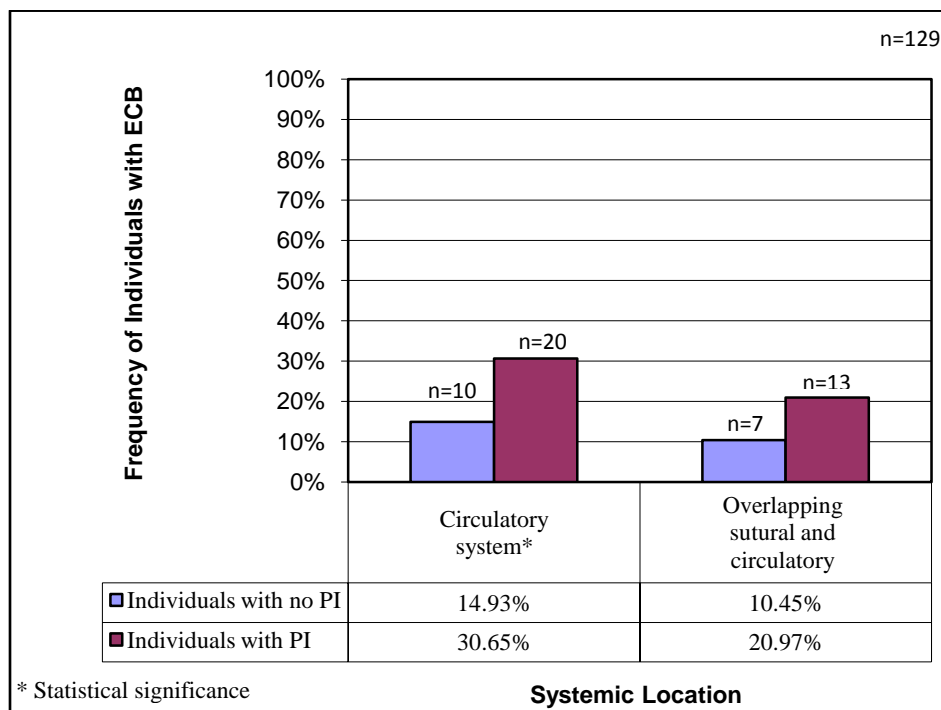
Figure 16. Frequency of Individuals with ECB by Systemic Location on Non-dynamic Features, and PI. Percentages were calculated by dividing the number of individuals with ECB by the total number of individuals, in each category. Features associated with the circulatory system were statistically significant at the p=.05 level.

Chi-square analysis of the above information revealed that the distribution of ECB was statistically significant on the circulatory systemic features (Table 39) but was not significant of pathological indicators on the combined sutural and circulatory systemic features (Table 40). The chi-square test could not be run on the non-dynamic features of the sutural system due to too many cell values under five.

Table 39. Association Between ECB and PI
on Non-dynamic Features of the Circulatory System

PI	ECB Present	ECB Absent	Total
Present	20	42	62
Absent	10	57	67
Total	30	99	129

$X^2_{1,0.05}=3.84$, $X^2=5.42$

Table 40. Association Between ECB and PI on Non-dynamic Features
of the Overlapping Sutural and Circulatory Systemic Features

PI	ECB Present	ECB Absent	Total
Present	13	49	62
Absent	7	60	67
Total	20	109	129

$X^2_{1,0.05}=3.84$, $X^2=2.72$

The results of the analyses of the dural attachment locations and ECB indicate

that lesions were found on dynamic features associated with tightly adhered dural sites in

36.4% (47 of 129 individuals), and on dynamic features associated with normally adhered

dural sites in 17.1% (22 of 129 individuals). The frequency of endocranial bone changes

on dynamic locations and dural attachment sites in individuals with, and without,

pathological indicators is represented in Figure 17. Figure 17 shows that individuals with

ECB and PI on dynamic features of tightly adhered dural features are significantly higher

in frequency than those individuals with ECB but no PI, whereas individuals with ECB

and PI on normally adhered dural features are not significantly higher in frequency than

those individuals with ECB but no PI.
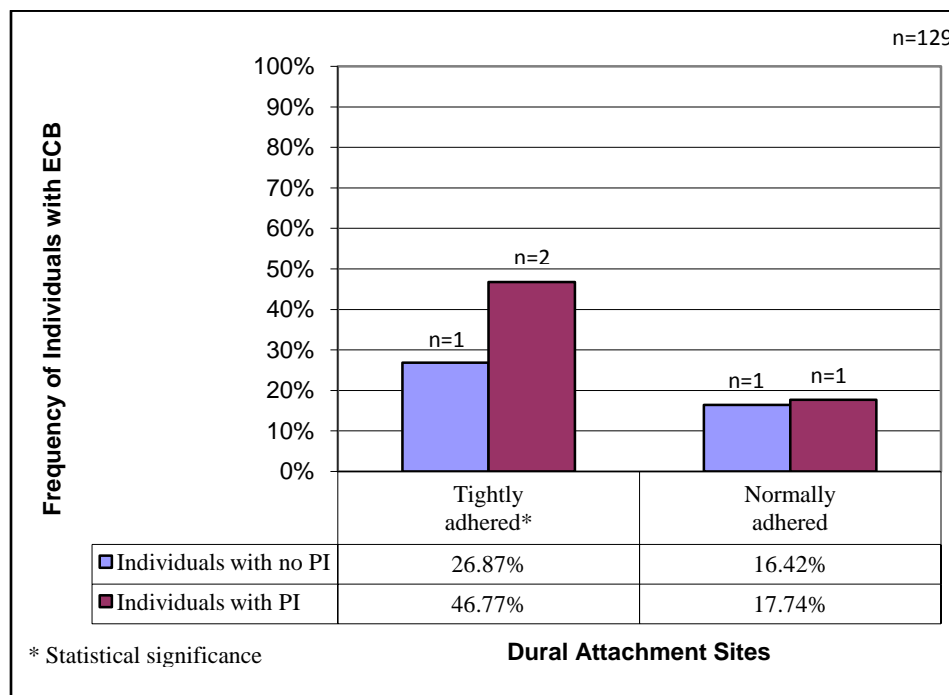
Figure 17. Frequency of Individuals with ECB by Dural Attachment Sites on Dynamic Features, and PI. Percentages were calculated by dividing the number of individuals with ECB by the total number of individuals, in each category. Areas of tight adherence were statistically significant at the p=.05 level.

Chi-square analysis of the above information revealed that the distribution of

ECB was statistically significant of pathological indicators on the areas of tightly adhered

dura mater (Table 41), but was not significant on the areas of normally adhered dura

mater (Table 42).

Table 41. Association Between ECB and PI
on Dynamic Features and Tight Dural Attachment Sites

PI	ECB Present	ECB Absent	Total
Present	29	25	54
Absent	18	35	53
Total	40	67	107

$$X^2_{1,0.05}=3.84, X^2=4.23$$

Table 42. Association Between ECB and PI
on Dynamic Features and Normal Dural Attachment Sites

PI	ECB Present	ECB Absent	Total
Present	11	40	51
Absent	11	38	49
Total	22	78	100

$$X^2_{1,0.05}=3.84, X^2=0.01$$

The results of the analyses of the dural attachment locations and ECB indicate that lesions were found on non-dynamic features associated with tightly adhered dural sites in 31.0% (40 of 129 individuals) and on non-dynamic features associated with normally adhered dural sites in 46.5% (60 of 129 individuals). The frequency of endocranial bone changes on non-dynamic locations and dural attachment sites in individuals with, and without, pathological indicators is represented in Figure 18. Figure 18 shows that individuals with ECB and PI on non-dynamic features of normally adhered dural features are significantly higher in frequency than those individuals with ECB but no PI, whereas individuals with ECB and PI on tightly adhered features are not significantly higher in frequency than those individuals with ECB but no PI.
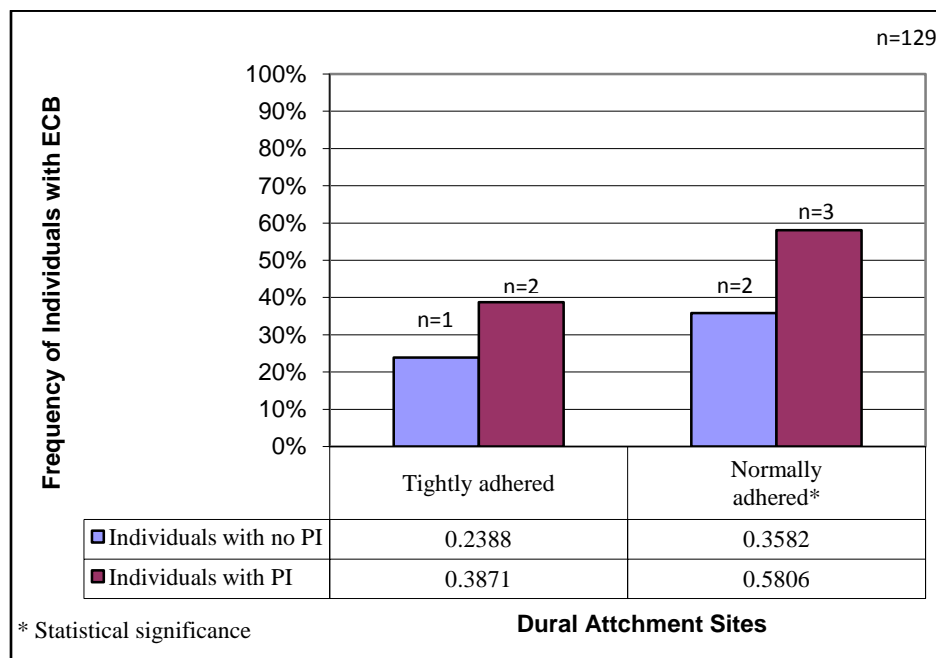
**Figure 18. Frequency of Individuals with ECB by Dural Attachment
Sites on Non-dynamic Features, and PI. Percentages were calculated by dividing
the number of individuals with ECB by the total number of individuals, in each
category. Less tightly adhered sites were statistically significant at the p=.05 level.**

Chi-square analysis of the above information revealed that the distribution of
ECB was not statistically significant of pathological indicators on the areas of tightly
adhered dura mater (Table 43) but was significant on the areas of normally adhered dura
mater (Table 44).

**Table 43. Association Between ECB and PI
on Non-dynamic Features and Tight Dural Attachment Sites**

PI	ECB Present	ECB Absent	Total
Present	24	38	62
Absent	16	51	67
Total	40	89	129

$$X^2_{1,0.05}=3.84, X^2=3.32$$

**Table 44. Association Between ECB and PI
on Non-dynamic Features and Normal Dural Attachment Sites**

PI	ECB Present	ECB Absent	Total
Present	36	26	62
Absent	24	43	67
Total	60	69	129

$X^2_{1,0.05}=3.84$, $X^2=6.40$

CHAPTER V

DISCUSSION

The basis of this thesis was to provide a foundation upon which other researchers could determine the etiology of juvenile extraneous new bone growth on the endocranial surface. Clinical research and case studies provided general information about this topic, but specific data as to where on the endocranial surface, and when in a juvenile's life span, new bone growth occurs, was lacking. The ultimate goal of this study was to contribute to the understanding and determination of the etiology of endocranial extraneous new bone growth by conducting physical examinations on archaeological specimens.

The first objective of this study was to isolate specific variables that may contribute to the formation of new bone growth on the endocranial surface. This was accomplished by gathering information that consisted of the presence of endocranial new bone growth, the location of the new bone, the dynamism of the new bone's location, the age-at-death of the individual, and whether or not any postcranial/ectocranial indicators of pathology were present within the individual. This information provided general frequencies and distribution patterns of endocranial lesions.

For all frequencies and distributions discussed, it should be noted that individuals were divided into two groups, one group having postcranial/ectocranial indicators of

pathology, and the other not. Of 129 individuals, 67, or 51.94%, had no indicators of postcranial/ectocranial pathology, whereas 62, or 48.06%, did have indicators of postcranial/ectocranial pathology. By comparing these two groupings of individuals to one another it should begin to become apparent how endocranial new bone growth, either caused by development or pathology, affect the surface of the endocrania, which was the second objective of this study.

Statistical testing showed that there was an association between the presence of endocranial lesions and the presence of postcranial/ectocranial indicators of pathology (Table 3). This result implies that the presence of a pathological condition elsewhere within the individual is correlated to the formation of some endocranial bone growth. However, extraneous new bone growth was also present when no indicators of pathology were present as well. Although this does not definitively mean that all of the extraneous new bone growth within individuals with no indicators of postcranial/ectocranial pathology is developmental in origin, it does seem to present evidence that there is a possibility that this bone growth is growth related.

In order to either confirm or refute the above-mentioned premise concerning the etiology of extraneous new bone growth as being caused by development or pathology, the data must be correlated with other information that has been gathered, i.e., the age-at-death of the individuals, the location of the new bone growth, and the dynamism of those locations. Questions can be asked of the resulting data that comes from those correlations. For instance, can an association between the presence of endocranial new bone growth and the age-at-death of the individual be found, between the presence of

extraneous new bone growth and anatomical locations, or between the presence of new bone growth and areas of the cranium that are dynamic? The subsequent sections within this chapter will attempt to provide answers to these questions.

<div align="center">Endocranial Bone Changes and Age</div>

The presence of extraneous new bone growth when examined in conjunction with age-at-death displayed a negative correlation in both groups of individuals, those who had indicators of pathology and those individuals who did not. The frequency of new bone growth in individuals with no indicators of pathology is greatest in the birth to 1.9-year age range, drops by almost 40% by the second category of 2 to 3.9 years, and then decreases steadily over the next three age intervals (Figure 8). This trend showed that, indeed, the majority of extraneous new bone growth is correlated to the period of rapid growth of the skull vault within the first two years of life, and that as growth rates decrease, so does the frequency of new bone growth.

The data (Figure 8) for individuals with postcranial/ectocranial indicators of pathology support the research of Mensforth et al. (1978), which states that endocranial lesions, linked to pathological processes, are also age-specific and decrease as age increases. However, Mensforth et al. attributed the endocranial lesions to pathological processes and did not take into account that extraneous new bone growth could be caused by developmental processes as well. It is a definite possibility that the location of bone changes on the surface of the endocrania caused by non-pathological processes was also present in the individuals. If that is indeed the case, then the negative correlation between pathological lesions and age may not be as clear-cut as put forth by the authors.

Endocranial Bone Changes and Location

The location of extraneous new bone growth on the endocranial surface can provide vital information regarding whether or not a pathological condition may be present, as well as what type of developmental processes may be taking place within the cranial vault and how these two factors have an effect on the presence of new bone growth. Locations of new bone growth may be found on general anatomic locations, such as the vault, base, or intermediate area between the two, or more specific locations, such as groups of systemic features that traverse the anatomic regions of the endocrania.

Extraneous new bone growth associated with specific areas of the braincase was grouped by the different types of features on which it was located. Features were either grouped by their association with specific systems that traverse the endocrania, such as the sutures and circulatory systemic features, or those features of the dura mater that encompass the surface of the endocrania.

The sutural and circulatory systemic features were tested in their own set of separate data from the features of the dura mater. Some of the features in these two categories overlap, and for that reason a third category was created that included those features that are a part of both systems. A fourth category was also created that consisted of non-systemic features that did not have an association with either the sutural or circulatory systems. This provided for independence of the data for statistical testing. The dura mater, on the other hand, encompasses the brain and is attached to all areas of the endocranial surfaces; these features, for that very reason, had their own set of statistical tests performed on them as well.

Of the three systemic categories and one non-systemic group, only the exclusively circulatory and the non-systemic features showed a statistical significance between the presence of extraneous new bone growth and indicators of pathology (Tables 9 & 10). The fact that circulatory features expressed lesions when pathological indicators were present coincides with research put forth by Aufderheide & Rodriguez-Martin (1998) and Ortner (2003) that indicated that there is a circulatory bond to many diseases. The association between endocranial lesions and pathological indicators on non-systemic features may not be apparent; however, this result may be explainable. Non-systemic areas of the braincase are usually squamal portions of the bones of the skull, such as the parietals, frontal(s), temporals, and occipital. Squamal portions of bone are, at times, featureless; however, depending on the age-at-death those features may not be in a developed and visible state on the surface of the bone. Features that usually occur on the squama are circulatory in nature, such as the mid-meningeal arteries and vessels, and may therefore have the same results as statistical tests performed on the circulatory systemic features.

The features associated exclusively with the sutures and the overlapping sutural and circulatory features did not have a statistical association between the presence of extraneous new bone growth and pathological indicators (Tables 7 & 8). The new bone that is located on these features is most likely to be caused by developmental processes given that all sutures (and most overlapping sutural and circulatory features) are dynamic from birth until the braincase reaches approximate adult proportions. The data collected from these two areas of the braincase indicated that new bone growth on the endocranial

surface seems to subside after the age of 6.9 years on exclusively sutural features, as there were no lesions observed after the 4 to 6.9-year age category, and after 1.9 years on overlapping sutural and circulatory features as there were only three lesions observed between the ages of 2 and 9.9 years. The overlapping sutural and circulatory features, as stated, did not have a statistical association between new bone growth and pathological indicators either (Table 9). This is in contrast to the information put forth by Aufderheide & Rodriguez-Martin (1998) and Ortner (2003) concerning the circulatory system and its interaction with pathological conditions that may cause endocranial lesions. There may, however, be an explanation for this result. The features that are associated only with the circulatory system are relatively small features: foramina, fissures, canals, and small sulci, whereas circulatory features that overlap sutural features are relatively larger, such as the superior sagittal sulci and sagittal sinus. The sinus and sulci are formed from a division of the two layers of the dura mater, the periosteal and meningeal (Moore & Dalley, 2006; Ortner, 2003; Schultz, 2001; Sperber, 1989). Moore & Dalley (2006) state that "except where the dural sinuses and infolding occur, the internal meningeal layer is intimately fused with the periosteal layer and cannot be separated from it" (p. 908). This would suggest that bone lesions seen in conjunction with the overlapping sutural and circulatory features are not pathological in origin.

Dural features were subdivided into two categories as well: those that adhere tightly to the surface of the endocrania and those that have a normal adherence. Features with a tight association to the dura were found to have a statistical association between the presence of extraneous new bone growth and postcranial/ectocranial indicators of

pathology (Table 11), and those features not as tightly bound to the dura were also found to have an association (Table 12). Both types of dural attachment sites include circulatory features, and as stated previously, circulatory features (when not in conjunction with the sutures) are known to have a statistical association between the presence of new bone growth and pathological indicators. Two chi-square tests were done on both the tightly adhered and normally adhered dural features with the circulatory systemic features removed. Both show that there was no statistical association between the presence of endocranial new bone growth and pathological indicators on either type of dural attachment site (Tables 13 & 15). These results indicate that it is, at least in part, due to the inclusion of features associated with the circulatory system that new bone growth is present on both types of dural adherence features.

Since it was determined that features that are tightly and/or normally adhered to the endocrania are significant in terms of extraneous new bone growth, as are circulatory systemic features, two more chi-square tests were conducted on circulatory systemic features. The first test included only circulatory features with no tightly adhered dural features, and the second test included circulatory features with no normally adhered dural features (Tables 14 & 16). The results confirmed that there is still an association between circulatory systemic features and endocranial new bone when either tightly adhered features were removed or normally adhered features are removed.

The three anatomic areas of the braincase (i.e., the cranial vault, intermediate area, and base) are composed of features associated with specific systemic areas and locales, such as the sutures, circulatory system, and dural attachment sites. For that

reason, the data on the anatomic areas of the skull will not be evaluated in depth, as the data gathered on these areas of the endocranial surface would not contribute any information on the etiology of endocranial new bone growth.

Endocranial Bone Changes and Dynamism

Dynamic features are those features that will undergo a morphologic change during the years an individual is considered a sub-adult. Dynamic features, when tested against the presence of extraneous new bone growth and postcranial/ectocranial indicators of pathology, showed no association between the two (Table 26), whereas features that are non-dynamic in nature did show an association (Table 27). This may be explained, in part, because the number of non-dynamic features increases as age progresses within the individual, while the number of dynamic features decreases.

In addition, 43% of individuals with no pathological indicators have new bone growth on dynamic features. New bone growth due to development should be present on dynamic features undergoing a morphological process as compared to those features that do not. That seems to be what is occurring here.

Endocranial Bone Changes, Location, and Dynamism

To further break down analyses as to what endocranial new bone growth may be deemed pathological and which may be deemed non-pathological, combinations of factors must be analyzed. Previously, only two variables were tested at a time: the presence of extraneous new bone growth and the presence of postcranial/ectocranial indicators of pathology, determined by one category, either age, location, or dynamism.

By utilizing the same five variables and adding two categories, location and dynamism of features, more information can be obtained.

As each systemic location was separated again by the dynamism or non-dynamism of features and re-tested for statistical significance, some locations exhibited somewhat different results than previously recorded in this study when only one variable was taken into account. The circulatory systemic features, which were found to show a relation between endocranial new bone growth and pathological indicators, for example, now show no association between the two variables when only dynamic features were tested (Table 37). An explanation for this can be due to there being almost three and a half as many circulatory features that coincide with non-dynamic features as with dynamic.

Non-dynamic features were found to be significantly associated with extraneous new bone growth on the surface of the endocrania. Three more chi-square tests were conducted in order to determine if non-dynamic features were the sole cause for this test result. It is possible that other accompanying variables (i.e., locations, dural effects) could have an effect on the results. To determine if this was indeed the case, new tests on non-dynamic features, without circulatory systemic features, tightly adhered dural features, and a combination of the two, were performed. On all tests conducted, the results remained the same: a statistical association was found between non-dynamic features and endocranial new bone growth (Tables 28, 29, & 30). Circulatory features make up only 25% to 27% of the non-dynamic features at any given one-year age interval throughout the time span of the individuals in this study. Also, the majority of dural

features with a tight adherence to the endocranial surface in the younger age categories

were found on dynamic features. Only as the individual's age increases do the tight

adherence features become more associated with non-dynamic features; however, as age

increases the amount of endocranial bone growth decreases, and therefore only a small

number of tight dural attachment sites affects non-dynamic features.

Normally adhered dural features also displayed results that differed from those

obtained after the original chi-square test was performed and before the second variable,

dynamism, was added. Whereas previously these features did not have a statistically

significant association between the presence of extraneous new bone growth and

pathological indicators (Table 44), they did show a statistically significant association

when dynamism (or non-dynamism) was taken into account. In addition, 83% to 100%

of the features associated with less tightly adhered dura mater are non-dynamic in nature,

which may explain this result.

CHAPTER VI

CONCLUSIONS

There has been a long-standing problem in bio-anthropological research with regard to the etiology of extraneous new bone growth on the endocranial surface of juvenile skeletal remains: How can new bone growth caused by pathological processes be distinguished from bone growth caused by normal skeletal development when bone's reaction to stimuli is limited to deposition, resorption, or a combination of the two? The solution to this problem, to date, is to compare the patterns of bone tissue response in skeletal remains to patterns of bone change discussed in the clinical literature in order to determine the etiology of new bone growth. As a way to potentially determine the etiology of extraneous new bone growth, this thesis proposed that individual variables, argued to be contributory factors in new bone growth formation, be isolated and analyzed in order to establish their significance. The variables examined were the presence of pathological indicators in the postcranial and/or ectocranial skeletal remains, the age-at-death of the individual, the location of extraneous new bone growth, and the dynamism of that location. The results of the chi-square tests conducted on these variables led to conclusions as to their importance, either singularly or in conjunction with one another, and to the etiology of extraneous new bone growth.

The presence of pathological indicators does appear to be an important contributory variable to help ascertain the etiology of extraneous new bone growth because it was found to be statistically significant when tested against the presence of new bone growth on the endocranial surface of the skull. This would indicate that, indeed, the presence of postcranial/ectocranial pathological indicators is a factor in the presence of some endocranial new bone growth.

The age-at-death of the individual and its relationship to the presence of endocranial extraneous new bone growth could not be statistically tested as there were too many cells with values under five. That notwithstanding, generalizations can be made concerning the etiologies of new bone growth on the neurocranial surface of the skull. By first examining those individuals who had no postcranial/ectocranial indicators of pathology, it is clear that the incidence of extraneous new bone growth tends to follow a negative correlation with age. The amount of new bone growth on the endocranial surface of the skull decreases drastically between the first two years of life and the successive two years. This decrease corresponds to the cessation of the period of rapid growth that the braincase undergoes in early childhood. In individuals with pathological indicators, it can be seen that new bone growth also follows a negative correlation with age. This may signify that lesions due to pathological processes decrease in number as age increases, as Mensforth et al. (1978) state, or it may suggest that developmental bone changes within the endocrania are skewing the data in these individuals by masking those lesions due to pathological processes.

Systemic locations on the endocranial surface, i.e., sutural, circulatory, those that overlap both categories, and those that are not associated with either, are an important contributory factor in determining the etiology of extraneous new bone growth on the endocranial surface of the skull. The information gathered from the sutural and the overlapping sutural and circulatory features are important indicators affirming this conclusion. Lesions located on the sutural systemic features, whether or not associated with the circulatory system, were shown to have no statistical association between endocranial lesions and postcranial/ectocranial indicators of pathology. It is on these systemic features where growth will occur and new bone growth due to development will be found. The circulatory features and those features that do not have an affiliation with any system, however, did show statistical significance between the presence of endocranial new bone growth and indicators of pathology. The correlation between the circulatory system and bone growth due to pathological processes has been discussed regarding hemorrhages and the circulation of some pathogens to different areas of the body. The features that do not have any systemic association may also be connected with the circulatory systemic features and would have a corresponding statistical test result. These features would have new bone growth due to pathological processes, and not development as the sutural and overlapping features did. This is also supported by the fact that once pathological indicators were removed from testing, no statistical association was observed on circulatory and non-systemic features.

Locations on the endocranial surface that coincide with dural attachment sites and a location's dynamism, however, are not key variables, in and of themselves, in helping

to determine the etiology of extraneous new bone growth. This is due to the fact that these locations, and the dynamism or static nature of these locales, are directly correlated with the four systemic locations discussed previously. For instance, normally adhered dural features are not associated with the sutural features (with or without circulatory features included), whereas tightly adhered features are. Only a small percentage of tightly adhered features are associated with circulatory systemic features, and none are associated with squamal, or non-systemic features. Only a small amount of tightly adhered dural features are related with non-dynamic features but are fully interrelated with dynamic features. Less tightly adhered features, alternatively, have a total association with non-dynamic features but no association with dynamic. Dynamic features are only associated with tightly adhered dural features, and subsequently with those systemic features that have an association with tightly adhered features as well. Non-dynamic features, conversely, are only associated with normally adhered dural features and those systemic features associated with the normally adhered features discussed above, whereas only a small amount of non-dynamic features are associated with tight dural features.

Tight dural features were found not to be statistically significant between the presence of extraneous new bone growth on the endocranial surface and the presence of pathological indicators except when in conjunction with circulatory features. However, when circulatory features were removed, the tight dural features showed no statistical significance. This result would be expected, as tightly adhered dural features are closely associated with both sutural and overlapping sutural and circulatory features. In this

regard, extraneous new bone growth found on tightly adhered dural features would be due to growth and development as well. This can also be said concerning the new bone growth found on dynamic features due to their association with tightly adhered dural features and the systemic features associated with them, as stated above. However, this may not hold true for the dynamic features that are associated with the circulatory systemic features, as the circulatory features are most likely to have new bone growth attributable to pathological processes.

New bone growth located on less tightly adhered dural features was found to be statistically significant with the presence of pathological indicators. This would be expected, as these locations are associated with the circulatory systemic and non-systemic features. Non-dynamic features were also found to be statistically significant; however, while it may be that these features were found to be statistically significant due to their association with circulatory, non-systemic, and normally adhered dural features, it must be stated again that as age increases the number of non-dynamic features increases as well, and this fact must play a role in the significant chi-square test result.

The data provided above support the claim that there are certain variables, such as the presence of postcranial/ectocranial indicators of pathology and systemic locations, that contribute valuable information with regard to differentiating between extraneous new bone growth that can be attributed to pathological processes versus new bone growth attributable to developmental processes, more so than the remaining variables, i.e., the dural adherence locations and a location's dynamism. However, it must be stated that all information gathered and observed for this study contributed to the understanding of, and

possible etiologies of, extraneous new bone growth. Although definitive etiologies could not be established through this study, this research presents information as to what future researchers need to be cautious of and also provides an important initial step toward attaining that ultimate goal of determining distinct etiologies for extraneous new bone growth. Further research is required to understand the true association between each variable and how it relates to age-at-death, the presence of pathological indicators, and the presence of new bone growth on the endocranial surface of the skull.

APPENDIX A

SKELETAL INVENTORY RECORDING FORM

(BUIKSTRA & UBELAKER, 1994)

INVENTORY RECORDING FORM
FOR COMPLETE SKELETONS

Site Name/Number _____/_____   Observer _____

Feature/Burial Number _____/_____   Date _____

Burial/Skeleton Number _____/_____

Present Location of Collection _____

CRANIAL BONES AND JOINT SURFACES

	L(left)	R(right)		L	R
Frontal	___	___	Sphenoid	___	___
Parietal	___	___	Zygomatic	___	___
Occipital	___	___	Maxilla	___	___
Temporal	___	___	Palatine	___	___
TMJ	___	___	Mandible	___	___

POSTCRANIAL BONES AND JOINT SURFACES

	L	R		L	R
Clavicle	___	___	Os Coxae		
Scapula			Ilium	___	___
Body	___	___	Ischium	___	___
Glenoid f.	___	___	Pubis	___	___
Patella	___	___	Acetabulum	___	___
Sacrum	___	___	Auric. Surface	___	___

VERTEBRAE (individual)

	Centrum	Neural Arch
C1	___	___
C2	___	___
C7	___	___
T10	___	___
T11	___	___
T12	___	___
L1	___	___
L2	___	___
L3	___	___
L4	___	___
L5	___	___

VERTEBRAE (grouped)
#Present/# Complete

	Centra	Neural Arches
C3-6	___/___	___/___
T1-T9	___/___	___/___

Sternum: Manubrium ___ Body ___

RIBS (individual)

	L	R
1st	___	___
2nd	___	___
11th	___	___
12th	___	___

RIBS (grouped)
#Present/# Complete

	L	R	Unsided
3-10	___/___	___/___	___/___

CHAPTER 2: Attachment 1

Series/Burial/Skeleton_____

Observer/Date_____

LONG BONES

	Proximal Epiphysis	Diaphysis			Distal Epiphysis
		Proximal Third	Middle Third	Distal Third	
Left Humerus	____	____	____	____	____
Right Humerus	____	____	____	____	____
Left Radius	____	____	____	____	____
Right Radius	____	____	____	____	____
Left Ulna	____	____	____	____	____
Right Ulna	____	____	____	____	____
Left Femur	____	____	____	____	____
Right Femur	____	____	____	____	____
Left Tibia	____	____	____	____	____
Right Tibia	____	____	____	____	____
Left Fibula	____	____	____	____	____
Right Fibula	____	____	____	____	____

Left Talus_____
Right Talus_____
Left Calcaneus_____
Right Calcaneus_____

HAND (# Present/# Complete)

	L	R	Unsided
# Carpals	___/___	___/___	___/___
#Metacarpals	___/___	___/___	___/___
#Phalanges	___/___	___/___	___/___

FOOT (# Present/# Complete)

	L	R	Unsided
#Tarsals	___/___	___/___	___/___
#Metatarsals	___/___	___/___	___/___
#Phalanges	___/___	___/___	___/___

Comments:_____

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Attachment 1: CHAPTER 2

APPENDIX B

SUB-ADULT AGE RECORDING FORMS

(BUIKSTRA & UBELAKER, 1994)

IMMATURE REMAINS RECORDING FORM:
BONE UNION AND EPIPHYSEAL CLOSURE

Site Name/Number _____/_____   Observer _____

Feature/Burial Number _____/_____   Date _____

Burial/Skeleton Number _____/_____

Present Location of Collection _____

Stage of Union: blank = unobservable; 0 = open; 1 = partial union; 2 = complete union

EPIPHYSEAL FUSION

Bone	Epiphysis	Stage of Union	
Cervical Vertebrae	superior	_____	
	inferior	_____	
Thoracic Vertebrae	superior	_____	
	inferior	_____	
Lumbar Vertebrae	superior	_____	
	inferior	_____	
		L	R
Scapula	coracoid	_____	_____
	acromion	_____	_____
Clavicle	sternal	_____	_____
Humerus	head	_____	_____
	distal	_____	_____
	medial epicondyle	_____	_____
Radius	proximal	_____	_____
	distal	_____	_____
Ulna	proximal	_____	_____
	distal	_____	_____
Os Coxae	iliac crest	_____	_____
	ischial tuberosity	_____	_____
Femur	head	_____	_____
	greater trochanter	_____	_____
	lesser trochanter	_____	_____
	distal	_____	_____
Tibia	proximal	_____	_____
	distal	_____	_____
Fibula	proximal	_____	_____
	distal	_____	_____

PRIMARY OSSIFICATION CENTERS

Bone	Area of Union	Extent
Os Coxae	ilium-pubis	_____
	ischium-pubis	_____
	ischium-ilium	_____
Sacral Segments	1-2	_____
	2-3	_____
	3-4	_____
	4-5	_____
Cervical Vertebrae		
	neural arches to each other	_____
	neural arches to centrum	_____
Thoracic Vertebrae		
	neural arches to each other	_____
	neural arches to centrum	_____
Lumbar Vertebrae		
	neural arches to each other	_____
	neural arches to centrum	_____
Cranium		
	spheno-occipital synchondrosis	_____
Occipital		
	lateral part to squama	_____
	basilar part to lateral part	_____

CHAPTER 4: Attachment 12

Series/Burial/Skeleton_____

Observer/Date _____

Estimate of chronological age based on postcranial maturation

Fetal   _____

b-5   _____

5-10   _____

10-15   _____

15-20   _____

20+   _____

Comments:_____

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Attachment 12: CHAPTER 4

IMMATURE MEASUREMENTS RECORDING FORM

Site Name/Number _____/_____   Observer _____

Feature/Burial Number _____/_____   Date _____

Burial/Skeleton Number _____/_____

Present Location of Collection _____

CRANIAL MEASUREMENTS

	L	M	R
1. Lesser Wing of the Sphenoid			
(a) Length:	_____		_____
(b) Width:	_____		_____
2. Greater Wing of the Sphenoid			
(a) Length	_____		_____
(b) Width:	_____		_____
3. Body of the Sphenoid			
(a) Length:		_____	
(b) Width:		_____	
4. Petrous and Mastoid Portions of the Temporal			
(a) Length:	_____		_____
(b) Width:	_____		_____
5. Basilar Part of the Occipital			
(a) Length:		_____	
(b) Width:		_____	
6. Zygomatic			
(a) Length:	_____		_____
(b) Width:	_____		_____
7. Maxilla			
(a) Length:	_____		_____
(b) Height:	_____		_____
(c) Width:	_____		_____
8. Mandible			
(a) Length of the Body:	_____		_____
(b) Width of the Arc:	_____		_____
(c) Full Length of Half Mandible:		_____	

CHAPTER 4: Attachment 13

Series/Burial/Skeleton_____

Observer/Date _____

POSTCRANIAL MEASUREMENTS

	L	R			L	R

9. Clavicle
    (a) Length: _____ _____
    (b) Diameter: _____ _____

10. Scapula
    (a) Length (height): _____ _____
    (b) Width: _____ _____
    (c) Length of the Spine: _____ _____

11. Ilium
    (a) Length: _____ _____
    (b) Width: _____ _____

12. Ischium
    (a) Length: _____ _____
    (b) Width: _____ _____

13. Pubis
    (a) Length: _____ _____

14. Humerus
    (a) Length: _____ _____
    (b) Width: _____ _____
    (c) Diameter: _____ _____

15. Ulna
    (a) Length: _____ _____
    (b) Diameter: _____ _____

16. Radius
    (a) Length: _____ _____
    (b) Diameter: _____ _____

17. Femur
    (a) Length: _____ _____
    (b) Width: _____ _____
    (c) Diameter: _____ _____

18. Tibia
    (a) Length: _____ _____
    (b) Diameter: _____ _____

19. Fibula
    (a) Length: _____ _____
    (b) Diameter: _____ _____

Comments:

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Attachment 13: CHAPTER 4

DENTAL INVENTORY RECORDING FORM
DEVELOPMENT, WEAR, AND PATHOLOGY: PERMANENT TEETH

Site Name/Number _____/_____   Observer _____

Feature/Burial Number _____/_____   Date _____

Burial/Skeleton Number _____/_____

Present Location of Collection _____

Tooth presence and development: code 1-8. For teeth entered as "1" (present, but not in occlusion), record stage of crown/root formation under "Development." **Occlusal surface wear:** use left teeth, following Smith (1984) for anterior teeth (code 1-8) and Scott (1979) for molars (code 0-10). If marked asymmetry is present, record both sides. Record each molar quadrant separate in the spaces provided (+) and the total for all four quadrants under "Total." **Caries:** code each carious lesion separately (1-7); **Abscesses:** code location (1-2). **Calculus:** code 0-3, 9. Note surface affected (buccal/labial or lingual).

		Tooth Presence	Development	Wear /Total		Caries			Abscess	Calculus/Affected	
Maxillary Right	1 M³	____	____	____ ____		____ ____ ____			____	____ ____	
	2 M²	____	____	____ ____		____ ____ ____			____	____ ____	
	3 M¹	____	____	____ ____		____ ____ ____			____	____ ____	
	4 P²	____	____	____ ____		____ ____ ____			____	____ ____	
	5 P¹	____	____	____ ____		____ ____ ____			____	____ ____	
	6 C	____	____	____ ____		____ ____ ____			____	____ ____	
	7 I²	____	____	____ ____		____ ____ ____			____	____ ____	
	8 I¹	____	____	____ ____		____ ____ ____			____	____ ____	
Maxillary Left	9 I¹	____	____	____ ____		____ ____ ____			____	____ ____	
	10 I²	____	____	____ ____		____ ____ ____			____	____ ____	
	11 C	____	____	____ ____		____ ____ ____			____	____ ____	
	12 P¹	____	____	____ ____		____ ____ ____			____	____ ____	
	13 P²	____	____	____ ____		____ ____ ____			____	____ ____	
	14 M¹	____	____	____ ____		____ ____ ____			____	____ ____	
	15 M²	____	____	____ ____		____ ____ ____			____	____ ____	
	16 M³	____	____	____ ____		____ ____ ____			____	____ ____	

CHAPTER 5: Attachment 16

Series/Burial/Skeleton_____

Observer/Date_____

	Tooth Presence	Development	Wear /Total	Caries	Abscess	Calculus/Affected
Mandibular Left 17 M₃	____	____	─┼─ ____	____ ____ ____	____	____ ____
18 M₂	____	____	─┼─ ____	____ ____ ____	____	____ ____
19 M₁	____	____	─┼─ ____	____ ____ ____	____	____ ____
20 P₂	____	____	____ ____	____ ____ ____	____	____ ____
21 P₁	____	____	____ ____	____ ____ ____	____	____ ____
22 C	____	____	____ ____	____ ____ ____	____	____ ____
23 I₂	____	____	____ ____	____ ____ ____	____	____ ____
24 I₁	____	____	____ ____	____ ____ ____	____	____ ____
Mandibular Right 25 I₁	____	____	____ ____	____ ____ ____	____	____ ____
26 I₂	____	____	____ ____	____ ____ ____	____	____ ____
27 C	____	____	____ ____	____ ____ ____	____	____ ____
28 P₁	____	____	____ ____	____ ____ ____	____	____ ____
29 P₂	____	____	____ ____	____ ____ ____	____	____ ____
30 M₁	____	____	─┼─ ____	____ ____ ____	____	____ ____
31 M₂	____	____	─┼─ ____	____ ____ ____	____	____ ____
32 M₃	____	____	─┼─ ____	____ ____ ____	____	____ ____

Estimated dental age (juveniles only)_____

Supernumerary Teeth:	Position between teeth	Location (1 - 4)	Position between teeth	Location (1 - 4)	Position between teeth	Location (1 - 4)
	____/____	____	____/____	____	____/____	____
	____/____	____	____/____	____	____/____	____

Comments:

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Attachment 16: CHAPTER 5

DENTAL INVENTORY RECORDING FORM
DEVELOPMENT AND PATHOLOGY: DECIDUOUS TEETH

Site Name/Number _____ / _____   Observer _____

Feature/Burial Number _____ / _____   Date _____

Burial/Skeleton Number _____ / _____

Present Location of Collection _____

Tooth presence and development: code 1-8. For teeth entered as "1" (present, but not in occlusion), record stage of crown/root formation under "Development." **Caries:** code each carious lesion separately (1-7); **Abscesses:** code location (1-2). **Calculus:** code 0-3, 9. Note surface affected (buccal/labial or lingual).

	Tooth	Presence	Development	Caries			Abscess	Calculus/Affected	
Maxillary Right	51 m^2	____	____	____ ____ ____			____	____ _____	
	52 m^1	____	____	____ ____ ____			____	____ _____	
	53 c	____	____	____ ____ ____			____	____ _____	
	54 i^2	____	____	____ ____ ____			____	____ _____	
	55 i^1	____	____	____ ____ ____			____	____ _____	
Maxillary Left	56 i^1	____	____	____ ____ ____			____	____ _____	
	57 i^2	____	____	____ ____ ____			____	____ _____	
	58 c	____	____	____ ____ ____			____	____ _____	
	59 m^1	____	____	____ ____ ____			____	____ _____	
	60 m^2	____	____	____ ____ ____			____	____ _____	
Mandibular Left	61 m^2	____	____	____ ____ ____			____	____ _____	
	62 m^1	____	____	____ ____ ____			____	____ _____	
	63 c	____	____	____ ____ ____			____	____ _____	
	64 i^2	____	____	____ ____ ____			____	____ _____	
	65 i^1	____	____	____ ____ ____			____	____ _____	
Mandibular Right	66 i^1	____	____	____ ____ ____			____	____ _____	
	67 i^2	____	____	____ ____ ____			____	____ _____	
	68 c	____	____	____ ____ ____			____	____ _____	
	69 m^1	____	____	____ ____ ____			____	____ _____	
	70 m^2	____	____	____ ____ ____			____	____ _____	

CHAPTER 5: Attachment 17

Series/Burial/Skeleton_____

Observer/Date_____

Estimated dental age (juveniles only)_____

Supernumerary Teeth:	Position between teeth	Location (1 - 4)	Position between teeth	Location (1 - 4)	Position between teeth	Location (1 - 4)
	____/____	_____	____/____	_____	____/____	_____
	____/____	_____	____/____	_____	____/____	_____

Comments:

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Attachment 17: CHAPTER 5

APPENDIX C

PALEOPATHOLOGY RECORDING FORMS

(BUIKSTRA & UBELAKER, 1994)

PALEOPATHOLOGY RECORDING FORM I
SHAPE, SIZE, BONE LOSS, FORMATION, FRACTURES, AND POROTIC HYPEROSTOSIS

Site Name/Number _____/_____   Observer _____

Feature/Burial Number _____/_____   Date _____

Burial/Skeleton Number _____/_____

Present Location of Collection _____

1.0 SHAPE

Bone_____	Bone_____	Bone_____	Bone_____	Bone_____	Bone_____
Side_____	Side_____	Side_____	Side_____	Side_____	Side_____
Bone_____	Bone_____	Bone_____	Bone_____	Bone_____	Bone_____
Side_____	Side_____	Side_____	Side_____	Side_____	Side_____
Obs1_____	Obs1_____	Obs1_____	Obs1_____	Obs1_____	Obs1_____
Obs2_____	Obs2_____	Obs2_____	Obs2_____	Obs2_____	Obs2_____

2.0 SIZE

Bone_____	Bone_____	Bone_____	Bone_____	Bone_____	Bone_____
Side_____	Side_____	Side_____	Side_____	Side_____	Side_____
Obs_____	Obs_____	Obs_____	Obs_____	Obs_____	Obs_____

3.0 BONE LOSS

Bone_____	Bone_____	Bone_____	Bone_____	Bone_____	Bone_____
Side_____	Side_____	Side_____	Side_____	Side_____	Side_____
Section_____	Section_____	Section_____	Section_____	Section_____	Section_____
Aspect_____	Aspect_____	Aspect_____	Aspect_____	Aspect_____	Aspect_____
Obs1_____	Obs1_____	Obs1_____	Obs1_____	Obs1_____	Obs1_____
Obs2_____	Obs2_____	Obs2_____	Obs2_____	Obs2_____	Obs2_____
Obs3_____	Obs3_____	Obs3_____	Obs3_____	Obs3_____	Obs3_____
Obs4_____	Obs4_____	Obs4_____	Obs4_____	Obs4_____	Obs4_____
Obs5_____	Obs5_____	Obs5_____	Obs5_____	Obs5_____	Obs5_____
Obs6_____	Obs6_____	Obs6_____	Obs6_____	Obs6_____	Obs6_____
Obs7_____	Obs7_____	Obs7_____	Obs7_____	Obs7_____	Obs7_____
Obs8_____	Obs8_____	Obs8_____	Obs8_____	Obs8_____	Obs8_____

4.0 FORMATION

Bone_____	Bone_____	Bone_____	Bone_____	Bone_____	Bone_____
Side_____	Side_____	Side_____	Side_____	Side_____	Side_____
Section_____	Section_____	Section_____	Section_____	Section_____	Section_____
Aspect_____	Aspect_____	Aspect_____	Aspect_____	Aspect_____	Aspect_____
Obs1_____	Obs1_____	Obs1_____	Obs1_____	Obs1_____	Obs1_____
Obs2_____	Obs2_____	Obs2_____	Obs2_____	Obs2_____	Obs2_____
Obs3_____	Obs3_____	Obs3_____	Obs3_____	Obs3_____	Obs3_____
Obs4_____	Obs4_____	Obs4_____	Obs4_____	Obs4_____	Obs4_____
Obs5_____	Obs5_____	Obs5_____	Obs5_____	Obs5_____	Obs5_____
Obs6_____	Obs6_____	Obs6_____	Obs6_____	Obs6_____	Obs6_____

CHAPTER 10: Attachment 25

Series/Burial/Skeleton_____

Observer/Date_____

5.0 FRACTURES

Bone____	Bone____	Bone____	Bone____	Bone____	Bone____
Side_____	Side_____	Side_____	Side_____	Side_____	Side_____
Section_____	Section_____	Section_____	Section_____	Section_____	Section_____
Obs1_____	Obs1_____	Obs1_____	Obs1_____	Obs1_____	Obs1_____
Obs2_____	Obs2_____	Obs2_____	Obs2_____	Obs2_____	Obs2_____
Obs3_____	Obs3_____	Obs3_____	Obs3_____	Obs3_____	Obs3_____
Obs4_____	Obs4_____	Obs4_____	Obs4_____	Obs4_____	Obs4_____
Obs5_____	Obs5_____	Obs5_____	Obs5_____	Obs5_____	Obs5_____
Obs6_____	Obs6_____	Obs6_____	Obs6_____	Obs6_____	Obs6_____
Obs7_____	Obs7_____	Obs7_____	Obs7_____	Obs7_____	Obs7_____
Obs8_____	Obs8_____	Obs8_____	Obs8_____	Obs8_____	Obs8_____
Obs9_____	Obs9_____	Obs9_____	Obs9_____	Obs9_____	Obs9_____
Obs10_____	Obs10_____	Obs10_____	Obs10_____	Obs10_____	Obs10_____
Obs11_____	Obs11_____	Obs11_____	Obs11_____	Obs11_____	Obs11_____

6.0 POROTIC HYPEROSTOSIS

Bone_____	Bone_____	Bone_____	Bone_____	Bone_____	Bone_____
Side_____	Side_____	Side_____	Side_____	Side_____	Side_____
Obs1_____	Obs1_____	Obs1_____	Obs1_____	Obs1_____	Obs1_____
Obs2_____	Obs2_____	Obs2_____	Obs2_____	Obs2_____	Obs2_____
Obs3_____	Obs3_____	Obs3_____	Obs3_____	Obs3_____	Obs3_____

Comments:

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Attachment 25: CHAPTER 10

PALEOPATHOLOGY RECORDING FORM II
VERTEBRAL PATHOLOGY, ARTHRITIS, AND MISCELLANEOUS

Site Name/Number _____/_____   Observer _____

Feature/Burial Number _____/_____   Date _____

Burial/Skeleton Number _____/_____

Present Location of Collection _____

7.0 VERTEBRAL PATHOLOGY

Bone____	Bone____	Bone____	Bone____	Bone____	Bone____
Side_____	Side_____	Side_____	Side_____	Side_____	Side_____
Aspect_____	Aspect_____	Aspect_____	Aspect_____	Aspect_____	Aspect_____
Bone____	Bone____	Bone____	Bone____	Bone____	Bone____
Side_____	Side_____	Side_____	Side_____	Side_____	Side_____
Aspect_____	Aspect_____	Aspect_____	Aspect_____	Aspect_____	Aspect_____
Obs1_____	Obs1_____	Obs1_____	Obs1_____	Obs1_____	Obs1_____
Obs2_____	Obs2_____	Obs2_____	Obs2_____	Obs2_____	Obs2_____
Obs3_____	Obs3_____	Obs3_____	Obs3_____	Obs3_____	Obs3_____

8.0 ARTHRITIS

Bone____	Bone____	Bone____	Bone____	Bone____	Bone____
Side_____	Side_____	Side_____	Side_____	Side_____	Side_____
Section/	Section/	Section/	Section/	Section/	Section/
Aspect_____	Aspect_____	Aspect_____	Aspect_____	Aspect_____	Aspect_____
Bone____	Bone____	Bone____	Bone____	Bone____	Bone____
Side_____	Side_____	Side_____	Side_____	Side_____	Side_____
Section/	Section/	Section/	Section/	Section/	Section/
Aspect_____	Aspect_____	Aspect_____	Aspect_____	Aspect_____	Aspect_____
Obs1_____	Obs1_____	Obs1_____	Obs1_____	Obs1_____	Obs1_____
Obs2_____	Obs2_____	Obs2_____	Obs2_____	Obs2_____	Obs2_____
Obs3_____	Obs3_____	Obs3_____	Obs3_____	Obs3_____	Obs3_____
Obs4_____	Obs4_____	Obs4_____	Obs4_____	Obs4_____	Obs4_____
Obs5_____	Obs5_____	Obs5_____	Obs5_____	Obs5_____	Obs5_____
Obs6_____	Obs6_____	Obs6_____	Obs6_____	Obs6_____	Obs6_____
Obs7_____	Obs7_____	Obs7_____	Obs7_____	Obs7_____	Obs7_____
Obs8_____	Obs8_____	Obs8_____	Obs8_____	Obs8_____	Obs8_____
Obs9_____	Obs9_____	Obs9_____	Obs9_____	Obs9_____	Obs9_____

MISCELLANEOUS

Bone____	Bone____	Bone____	Bone____	Bone____	Bone____
Side_____	Side_____	Side_____	Side_____	Side_____	Side_____
Obs_____	Obs_____	Obs_____	Obs_____	Obs_____	Obs_____
Bone____	Bone____	Bone____	Bone____	Bone____	Bone____
Side_____	Side_____	Side_____	Side_____	Side_____	Side_____
Obs_____	Obs_____	Obs_____	Obs_____	Obs_____	Obs_____

CHAPTER 10: Attachment 26

Series/Burial/Skeleton_____

Observer/Date_____

COMMENTS

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Attachment 26: CHAPTER 10

BIBLIOGRAPHY

Anderson, M., Messner, M. B., & Green, W.T. (1964). Distribution of lengths of the normal femur and tibia from one to eighteen years of age. Journal of Bone and Joint Surgery, 46A, 1197-1202.

Aoki, J., Yamamoto, I., Hino, M., Kitamura, N., Sone, T., Itoh, H., Torizuka, K. (1987). Reactive endosteal bone formation. Skeletal Radiology, 16, 545-551.

Aufderheide, A. C., & Rodriguez-Martin, C. (1998). The Cambridge encyclopedia of human paleopathology. Cambridge, NY: Cambridge University Press.

Black, S. & Scheuer, L. (1996). Age changes in the clavicle: From the early neonatal period to skeletal maturity. International Journal of Osteoarchaeology, 6, 425-434.

Buikstra, J. E. & Ubelaker, D. H. (1997). Standards for data collection from human skeletal remains: Proceedings of a seminar at the Field Museum of Natural History. Fayetteville, AR: Arkansas Archeological Survey.

Fazekas, I. G., & Koza, F. (1978). Forensic fetal osteology. Budapest, Hungary: Akademiai Kiado.

Gindhart, P.S. (1973). Growth standards for the tibia and radius in children aged one month through eighteen years. American Journal of Physical Anthropology, 39, 41-48.

Gordon, J. E., Wyon, J. B., & Ascoli, W. (1967). The second year death rate in less developed countries. The American Journal of the Medical Sciences, Sept, 121-144.

Humphrey, L. T. (1998). Growth patterns in the modern human skeleton. American Journal of Physical Anthropology, 105, 57-72.

Kabbani, H. & Raghuveer, T. S. (2004). Craniosynostosis. American Family Physician, 69 (12), 2863.

Lewis, M. E. (2000). Non-adult paleopathology: Current status and future potential. In: M. Cox & S. Mays (Eds.) Human osteology in archaeology and forensic science (pp.39-53). London, UK: Greenwich Medical Media LTD.

Lewis, M. E. (2004). Endocranial lesions in non-adult skeletons: Understanding their aetiology. International Journal of Osteoarchaeology, 14, 82-97.

Lovell, N. C. (2000). Pathological description and diagnosis. In: M. A. Katzenberg & S. R. Saunders (Eds.) Biological anthropology of the human skeleton (pp. 217-248). New York, NY: Wiley-Liss.

Malmberg, N. (1944). Occurrence and significance of early periosteal proliferation in the diaphyses of premature infants. Acta Paediatrica, 32, 626-33.

Mann, R. W., & Murphey, S. P. (1990). Regional atlas of bone disease: A guide to pathologic and normal variation in the human skeleton. Springfield, IL: Charles C. Thomas.

Maresh, M. M. (1970). Measurements from roentgenograms. In: R. W. McCammon (Ed.) Human Growth and Development (pp. 157-200). Springfield: C.C. Thomas.

Marieb, E. N., & Hoehn, K. (2008). Anatomy and physiology (3rd ed.) San Francisco, CA: Pearson/Benjamin Cummings.

Mensforth, R. P., Lovejoy, C. O., Lallo, J. W., & Armelagos, G. J. (1978). The role of constitutional factors, diet, and infectious disease in the etiology of porotic hyperostosis and periosteal reactions in prehistoric infants and children. Medical Anthropology. 2 (1), 1-59.

Moore, K. L., & Dalley, A. F. II (2006). Clinically oriented anatomy (5th ed.) Philadelphia, PA: Lippincott, Williams & Wilkins.

Moore, K. L., & Persaud, T.V.N. (1998). The Developing human: Clinically oriented embryology (6th ed.) Philadelphia, PA: WB Saunders.

Moorees, C.F.A., Fanning, E.A., & Hunt, E.E. (1963a). Age formation by stages for ten permanent teeth. Journal of Dental Research, 42, 1490-1502.

Moorees, C.F.A., Fanning, E.A., & Hunt, E.E. (1963b). Formation and resorption of three deciduous teeth in children. American Journal of Physical Anthropology, 21, 205-213.

Moss, M. L. (1959). The pathogenesis of premature cranial synostosis in man. Acta Anatomica, 37, 351-70.

Moss, M. L., Noback, C. R., Robertson, G. G. (1956). Growth of certain human fetal cranial bones. American Journal of Anatomy, 98, 191-204.

Moss, M. L. & Young, R. W. (1960). A functional approach to craniology. American Journal of Physical Anthropology, 98, 191-204.

O'Rahilly, R. & Muller, F. (1986). The meninges in human development. Journal of Neuropathology and Experimental Neurology, 45 (5), 588-608.

Ortner, D. J. (2003). Identification of pathological conditions in human skeletal remains. Washington, DC: Smithsonian Institution Press.

Pfeiffer, S. (2000). Paleohistopathology: Health and disease. In: M. A. Katzenberg & S. R. Saunders (Eds.) Biological anthropology of the human skeleton (pp. 287-302). New York, NY: Wiley-Liss.

Pick, T. P., & Howden, R. (2003). Gray's anatomy (16th ed.) East Molesy, UK: Longmans, Green and Co.

Ribot, I. & Roberts, C. (1996). A study of non-specific stress indicators and skeletal growth in two mediaeval subadult populations. Journal of Archaeological Science, 23, 67-79.

Roberts, C. & Manchester, K. (2005). The Archaeology of Disease (3rd ed.). Ithaca, NY: Cornell University Press.

Saunders, S., Hoppa, R., & Southern, R. (1993). Diaphyseal growth in a nineteenth-century skeletal sample of subadults from St. Thomas' Church, Belleville, Ontario. International Journal of Osteoarchaeology, 3, 265-281.

Scammon, R. E. (1930). The measurement of the body in childhood. In: J. A. Harris, C. M. Jackson, D. G. Paterson, & R. E. Scammon (Eds.) The measurement of man (pp. 171-215). Minneapolis, MN: University of Minnesota Press.

Scheuer, L., & Black, S. (2000). Developmental juvenile osteology. San Diego, CA: Academic Press.

Schultz, M. (1999). The role of tuberculosis in infancy and childhood in prehistoric and historic populations. In: G. Palfi, O. Dutour, J. Deak, & I. Hutas (Eds.) Tuberculosis Past and Present (pp. 502-507). Hungary: Golden Book Publishers.

Schultz, M. (2001). Paleohistopathology of bone: A new approach to the study of ancient diseases. Yearbook of Physical Anthropology, 44, 106-47.

Shopfner, C. E. (1966). Periosteal bone growth in normal infants. American Journal of Roentgenology, 97, 154-63.

Sinclair, D. (1986). Human Growth After Birth (4th ed.). New York, NY: Oxford University Press.

Smith, D. W., & Tondury, G. (1978). Origin of the calvaria and its sutures. American Journal of Diseases of Children, 132, 662-666.

Sperber, G.H. (1989). Craniofacial embryology (4th ed.). London, UK: Wright, Butterworths.

Steele, D. G., & Bramblett, C. A. (1988). The Anatomy and biology of the human skeleton. Collage Station, TX: Texas A&M University Press.

Ubelaker, D. H. (1989). Human Skeletal Remains (2nd ed.). Washington, DC: Taraxacum Press.

Wolff, J. (1870). Ueber die innere architectur der knochen und ihre bedeutung fur die furge vom knochenwachsthum. Virchows Arch. f. path. Anat. L, 389.

Young, R. W. (1957). Postnatal growth of the frontal and parietal bones in white males. American Journal of Physical Anthropology, 15, 367-386.

Young, R. W. (1959). The influence of cranial contents on postnatal growth of the skull in the rat. American Journal of Anatomy, 105, 383-415.

VITA

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