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Modelling Suture Ossification: A View from the Cranial Capsule

Hugh Bryson Matternes
University of Tennessee, Knoxville

To the Graduate Council:

I am submitting herewith a thesis written by Hugh Bryson Matternes entitled "Modelling Suture Ossification: A View from the Cranial Capsule." I have examined the final electronic copy of this thesis for form and content and recommend that it be accepted in partial fulfillment of the requirements for the degree of Master of Arts, with a major in Anthropology.

Richard L. Jantz, Major Professor

We have read this thesis and recommend its acceptance:

Lyle W. Konigsberg, William M. Bass

Accepted for the Council:
Dixie L. Thompson

Vice Provost and Dean of the Graduate School

(Original signatures are on file with official student records.)

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MODELING SUTURE OSSIFICATION:
A VIEW FROM THE CRANIAL CAPSULE

A Thesis

Presented for the

Master of Arts

Degree

The University of Tennessee, Knoxville

Hugh Bryson Matternes

August, 1992

DEDICATION

This thesis is dedicated to by wife,

Jennifer H. Matternes

and the late

Mrs. Hazel Modine.

Without their encouragement and support, I could not have completed this project.

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Perhaps as important as the people whose support enabled the data to be gathered, are those who served as my sounding boards - the poor souls who endured endless hours of babble and still found something relevant to say about sutures. My thanks go to Phil

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ABSTRACT

A review of the development, anatomy, and physiology of the human cranial vault suggests that post-adolescent ossification of the suture margins is dictated by interactions between tissues of the cranial capsule and forces deriving from ectocranial, endocranial and diploid sources. A model viewing suture physiology as sensitive to changes in the cranial capsule's environment is tested to identify whether several processes stimulate connective tissue transformation in the suture area. Correlation and factor analysis of the suture-to-suture interactions produced results indicating that when age is controlled, endocranial and ectocranial surfaces independently respond to pressures placed on the anterior and posterior portions of the cranial vault. Post-adolescent suture changes appear to be responses to age change in the structures around the cranial capsule. Modeling the suture in terms of external pressures provides a clearer understanding of how the cranial capsule is affected by age.

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LIST OF ABBREVIATIONS

Endocranial Coronal Suture at Bregma - Right.....ICBRR

Endocranial Coronal Suture at Bregma - Left.....ICBRL

Endocranial Coronal Suture at Complicata - Right.....ICCOR

Endocranial Coronal Suture at Complicata - Left.....ICCOL

Endocranial Sagittal Suture at Bregma.....ISBR

Endocranial Sagittal Suture at Vertica.....ISVE

Endocranial Sagittal Suture at Obelica.....ISOB

Endocranial Sagittal Suture at Lamdica.....ISLA

Endocranial Lambdoidal Suture at Lamdica - Right.....ILLAR

Endocranial Lambdoidal Suture at Lamdica - Left.....ILLAL

Endocranial Lambdoidal Suture at Intermedia - Right.....ILINR

Endocranial Lambdoidal Suture at Intermedia - Left.....ILINL

Endocranial Lambdoidal Suture at Asterion - Right.....ILASR

Endocranial Lambdoidal Suture at Asterion - Left.....ILASL

Ectocranial Coronal Suture at Bregma - Right.....OCBRR

CHAPTER I.

INTRODUCTION

One of the methods used in Anthropology to explain complex relationships is the model. This hypothetical abstraction allows the researcher to incorporate several different theoretical elements to learn how the object of study is affected by the world around it. Its implementation in Physical Anthropology simplifies research by isolating critical variables and helps the researcher deduce how these variables may be measured. Models typically imply that a mathematical equation can be drawn to illustrate the proposed relationship (Zar 1984). A useful model must incorporate a good fit between current theory and the data set utilized, enable testable hypotheses to be drawn, and point out areas needing additional research (Pelto and Pelto 1978).

Models guide and outline how researchers perceive the operation of a world they cannot scientifically control. In an aggressive academic paradigm, scientific models are critically evaluated from time to

time to see whether other bodies of knowledge shed relevant information on the inter-workings of the modeled phenomena. Many researchers contend that the biology dictating human skeletal morphology cannot be comprehensively explained by passively treating bones as biologically independent elements. A more holistic approach models the skeleton as an integral component of a biological system working to maintain homeostasis. This paradigm suggests that the investigation of skeletal dynamics should not be limited to the skeletal structure, rather research must encompass the effects of other physiological complexes which interact to form the observed state. This broader approach to the human skeleton may be a more powerful tool to explain what induces morphological change than the more traditional viewpoints, particularly when looking at how the skeleton ages. Unfortunately, these models have seen little use in the study of gerontological morphology (Rowe and Kahn 1987).

When a skull is viewed, one of the most striking surface features is the complex system of cranial sutures. This network of gaps separating the various osseous components of the skull has been the subject of considerable attention in Anthropology as an analytical tool, but has received almost no regard as the subject of study.. The discipline relies on the suture as an

age predictor and to define landmarks for orientation and identification. Suture morphology is very unstable. Because it is subject to frequent, unexplainable change, many researchers feel that the utility of a suture is highly questionable until its biological dynamics are better understood. Recently, Reichs noted:

> Ultimately, explanations of abnormal closure must derive from an understanding of the processes governing normal calvarial growth. (1989:271).

Physical Anthropology has traditionally considered cranial suture closure as a phenomenon associated with the hard tissues of the skull. This approach models ossification as an event isolated from other physiological processes in the body. Examinations have typically documented shifts in skeletal morphology without considering how these changes relate to the surrounding soft structures. This approach limits an understanding of how a mature skull changes form solely to the effects occurring within bone.

A review of the academic literature identifies that most of what is known about the suture margin cannot be adequately applied to post-adolescent human cranial physiology. What if post-adolescent suture closure is not controlled by drives within bone, but is dictated by interaction with the surrounding tissues in much the same way as found in developing sutures?

Perceiving the suture from its anatomical context and addressing why biomechanical demands shift with age requires an evaluation of a new model of closure. The focus of this investigation is to learn whether other biological processes could be influencing sutures to change form. Data gathered from the R.J. Terry and Armed Forces Institute of Pathology's Civil War Collections are tested to see whether relationships between closure and age involve interaction with additional biological influences. The results of this investigation will serve to shed light on why suture form is erratic and identify specific avenues of inquiry needed to further understand why sutures take such seemingly inexplicable forms.

CHAPTER II.

THE CRANIAL CAPSULE AND THE SUTURE AREA

Anthropological literature usually identifies sutures as one of the features found in facial and neurocranial bones. Sutures are defined as serrated, interlocking margins that separate bones of the skull (Bass 1987). Twenty-three sutures are recognized in the average, healthy, young adult human skull; ten are found in the calvaium and the other thirteen articulate aspects of the face (Baker 1984). All sutures act to maintain the structural and protective support of the skull as a unit. They unify discrete hard tissue surfaces, allowing the functional demands from one bone to interdigitate with those of another. Sutures, therefore, must be responsive to the demands of the different components which form cranial bone. Despite the seeming uniformity in the osseous aspect's function, alleviation of structural demands require a considerable amount of morphological variation within the hard tissue. These variations largely fall within the confines of the cranial elements, suture junctions,

and sutural margins.

1. Cranial Elements

The bones of the skull are composed of three distinct layers, a hard outer table, a middle diploic layer, and a hard inner table (Figure 1). The outer table forms an ectocranial surface of cortical bone. It is functionally responsive to the demands of the scalp musculature (Moss and Young 1960). The inner table is also composed of cortical bone and lines the skull's endocranial aspect. The inner table is responsive to changes in the neural environment. Bone is removed or deposited on its surfaces to accommodate age related changes in the size of the brain (Moss and Young 1960). This table is usually thinner than the outer table (Miroue 1975). The different environmental stressers placed on the inner and outer tables translate into different functional demands imposed on each osseous tissue. Their adaptive responses bear little relation to the form taken by the opposing hard surface (Baer and Harris 1969).

The diploic space isolates the inner and outer tabular environments. During development, increased muscle tension on the ectocranial surface pulls the outer table away from the brain (Ohtsuki 1977). Through remodelling, a space filled with trabecular

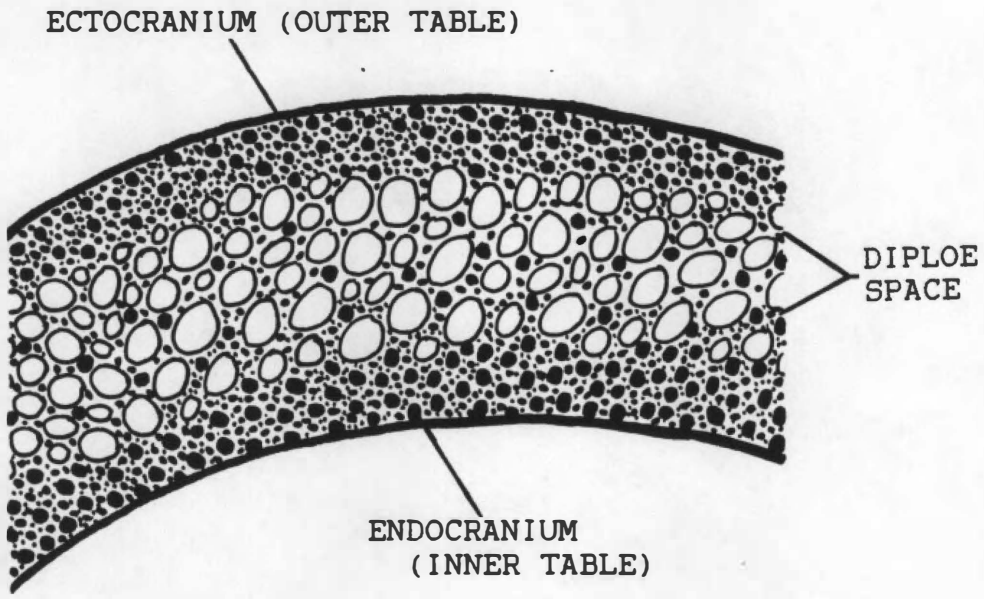


Figure 1.

Cranial Bone in Profile

bone emerges between the two bone surfaces. Diploe formation in this space commences after opposing bone surfaces grow close enough for suture inter-digitation (Young 1959). Young (1959) noted that consequential to this stage in development, the outer table, scalp tissues, and diploe become functionally independent of changes occurring inside the cranial vault. The diploic space also serves as a site of hematopoiesis, a major venous drainage route, and helps alleviate the weight of the skull by replacing cortical with trabecular bone. Its fulfillment of these activities can be related to the thickness of the skull (Ohtsuki 1977). Miroue (1975) has observed that the diploe bordering a suture area becomes progressively thicker as examination of the skull progresses posteriorly.

2. Suture Junctions

Sutures normally exhibit either flat or squamous junctions. The Flat Junction occurs where two opposing bones meet end to end, forming a butt joint (Figure 2). These can be observed in the human sagittal and medial aspects of the coronal and lambdoid sutures. They form as a response to tensile forces acting to pull these bones apart. In young rats, this type of suture morphology develops from growth in the cerebellum and in the nuchal musculature (Moss 1957). The flat

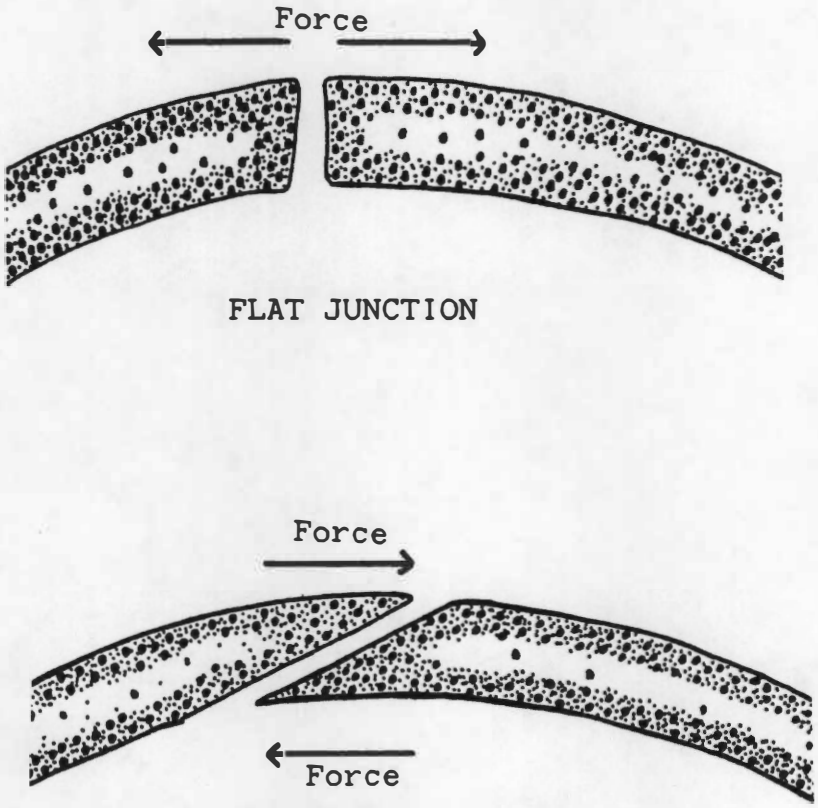


Figure 2.

Types of Suture Junctions in the Cranial Vault

junction, prior to interdigitation is the intrinsic embryonic form (Moss 1957).

The Squamous Junction is observed in the human temporal, spheno-temporal, and sphenofrontal sutures. It relieves compressive stress by allowing the adjacent sutural margins to slide past one another (Roberts 1979). The squamous junction forms during development; directional force applied to two opposing bones cause them to overlap, forming a bevelled junction. If pressure is removed from the squamous junction during the initial growth phase, the suture will revert to a flat junction morphology (Moss 1957). This juncture should be viewed as an adaptive response of the flat junction suture to its environment. The genetic contribution to suture morphology is more reflective of the soft tissue surrounding the suture area than of the suture itself.

3. The Suture Margins

The suture juncture is not simply a bone's edge, rather it possesses a unique structure which varies according to where the margin is viewed. From the ectocranial aspect, a flat junction suture is highly serrated. It forms sets of regular patterns commonly used to subdivide the suture into definable parts (Singer 1953). In profile, a flat junction suture

continues into the margin, forming the Pars Externa, a complex series of interdigitating projections and depressions running roughly parallel to the ectocranial surface (Figure 3).

The endocranial aspect is much less serrated than its counterpart. It appears as an irregular line separating the bone surfaces. There are no definable morphological features to provide subdivisions; traditionally the suture has been split into metrically equal parts (Perizonius 1984). The internal morphology is referred to as the Pars Interna.

The pars interna and externa also reflect adaptive responses to bio-mechanical forces at the suture site. Oudhof (1978) noted that the distinction between these divisions allows a suture to operate much like a hinge. Internal and external forces passed to the hard tissue aspects of the cranial capsule are directed towards its weak point - the suture margin. Most of this pressure is absorbed and dissipated in the form of movement within the pars externa's enhanced surface area. The remaining force is channeled away from the brain by compression of the pars interna's edges. This reduces its surface area and serves as a fulcrum to channel force back towards the pars externa. This morphology effectively reduces the amount of tensile and compressive force directed towards the internal aspects

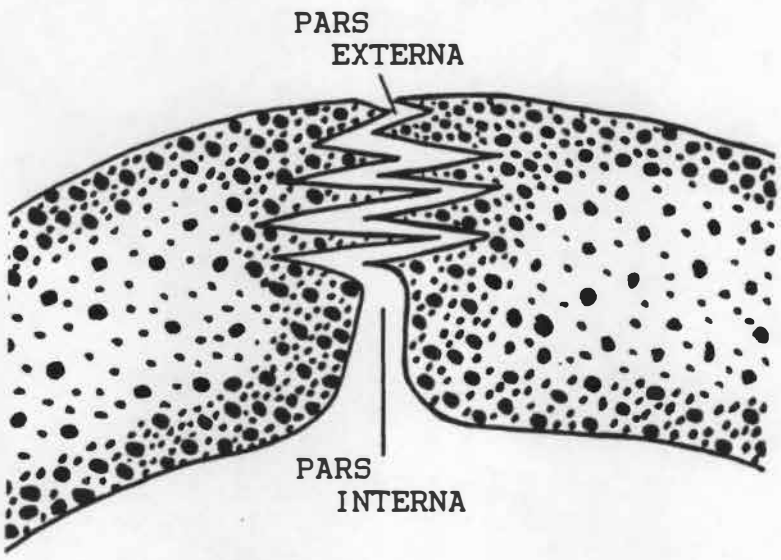


Figure 3.

Morphology of the Flat Junction Suture

of the skull.

4. The Cranial Capsule

Sutures are not features visible only in dry bone, nor are they composed exclusively of osseous materials. They contain a considerable amount of other soft tissue. Sutures are areas, not features. Moss (1957) suggested that the suture area encompassed the edges of opposing bones and the surrounding layers of connective tissue. These tissues work in conjunction to provide a mechanical union that firmly holds both cranial bone and the brain to prevent any movement. The suture area also allows bone growth (Ranly 1988; Babler and Persing 1982). Sutures represent one aspect of the skull's functional organization, the addition of soft tissue to the suture's composition define it as part of a larger, more complex system (Ranly 1988). Moss and Young (1960) argue that cranial morphology must be perceived according to how these structural components fulfill functional demands. These soft tissues serve an integral part in shaping cranio-facial structure, making it necessary to define the skeletal morphology in terms of bio-mechanical units (Watzek, et al. 1982).

The suture area is but one aspect of the bio-mechanical unit referred to as the Cranial Capsule. The

cranial capsule contains numerous membranes surrounding, protecting, nourishing, and supporting the brain. Moss and Young (1960) identify the cranial capsule's composition as both the hard, ossified tissue in the calvarium and the soft connective tissues making up the dura mater and periosteum. The cranial capsule model views the suture area's hard and soft tissue components as simply as an extension of the brain's dura mater. The **Dura Mater** is composed of dense fibrous connective tissue which surrounds the cerebro-spinal network (Junquiera, et al. 1989). In the skull, it underlies the major calvarial sutures (Moss and Young 1960). These ligaments, oriented towards attachment sites surrounding the sella turcica, respond to the growing brain's demands through gradual controlled expansion (Moss and Young 1960; Jackson 1925). The dura mater serves as a protective sheath and a circulatory barrier, reducing the amount of capillary flow between the endo-dural and ecto-dural environments. Although collagen fibers and cells are noted to grade into the internal periosteum, the scarcity and orientation of blood vessels in the dura mater has serves to define it as a seperate anatomical unit from other periosteal tissues (Bennett 1967; Ham and Cormack 1979).

The suture is an important aspect of the cranial

capsule; it allows opposing demands on the capsular structure to be met. The continuity of the connective tissue between the dura mater and the ectocranial periosteum maintains protective immobility by firmly adhering all elements together and mediates expansive brain growth, by allowing the skull to increase in size (Moss and Young 1960). Sutures do not represent primary growth sites; they undergo compensational growth much the same as other calvarial surfaces.

2. Tissue Components of the Cranial Capsule

An essential aspect of the cranial capsule concept is that all tissues operate as a single functioning unit. Researchers do not agree about what soft tissues compose the cranial capsule's suture area. Differences in the age of the specimen (Kokich 1974; Miroue 1975), staining technique (Pritchard, et al. 1956), growth model applied (Enlow 1975), and study site (Kokich, et al. 1979; Miroue 1975) create observation biasesand partially account for this lack of agreement. Other sources of anatomical confusion stem from differences in tissue sectioning and subsequent naming discontinuities. In general, however, there is agreement that the cranial capsule's soft tissue component consists of several varieties of connective tissue.

The periosteal membrane is a connective tissue layer encapsulating the ectocranial, endocranial, and endosteal bone surfaces (Figure 4). Like the dura, it is composed primarily of dense fibrous connective tissue (Junqueira, et al. 1989). It possesses an extensive vascular plexus that anastomoses overlying blood vessels with those found in bone (Whiteside 1984). The periosteum supplies the blood to the bone, making nourishment one of the its principal functions.

The periosteum is divided into the cambial and capsular layers. The **Cambial Layer** is in direct contact with the bone surface. It consists of a membrane of osteoblasts and osteogenic precursor cells surrounded by a matrix of dense collagen bundles (McLean and Urist 1968). The cambial layer is the area of active bone formation; in the suture area, it serves as the site of expansive calvarial growth (Moss 1954). The morphology of the cambial layer shifts with age. Pritchard, et al. (1956) observed that it is thick and well defined in immature specimens.

With increased maturation, the layer shrinks in size and composition. In adult human sutures, this membrane is very difficult to detect. It is suggested by Miroue (1975) that the mature cambial layer may be only a single cell layer thick.

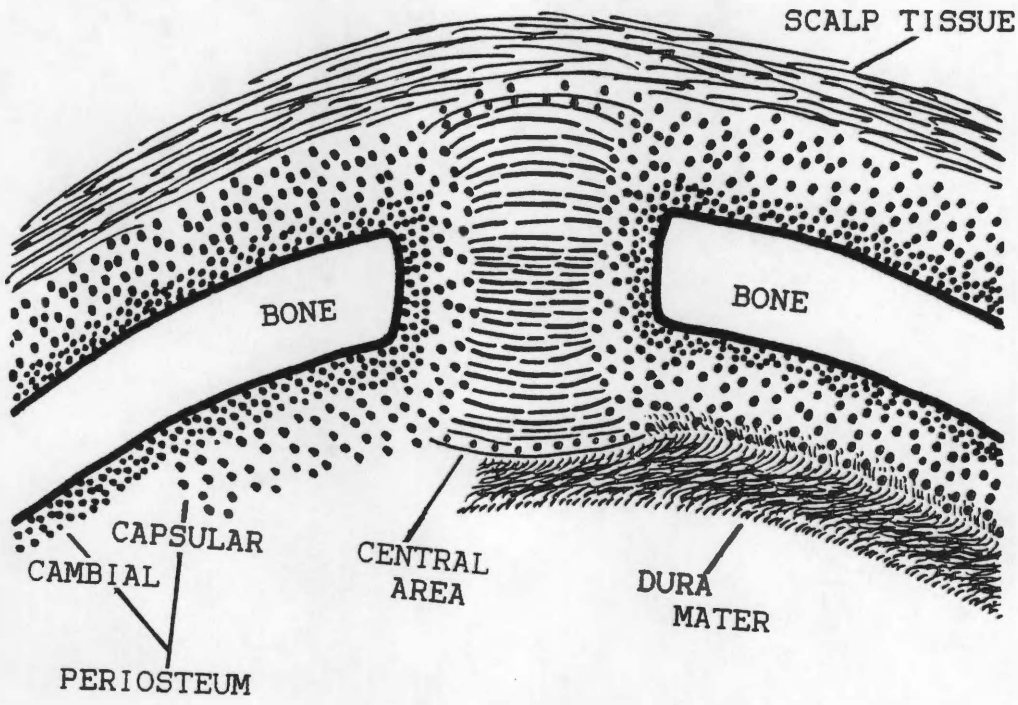


Figure 4.

Soft Tissue Anatomy of the Flat Junction Suture Margin

The **Fibrous Layer** (or Capsular Layer) is differentiated from the cambial layer by virtue of its higher ratio of fibroblasts to osteoblasts (McLean and Urist 1968). The collagen fiber orientation changes gradually from parallelling the bone surface, in the cambial periphery, to the fibrous layer's perpendicular arrangement (Pritchard, et al. 1956). The principal functions of the fibrous layer are to support the vascular plexus and anchor it to the bone (Ham and Cormack 1979).

The **Central Area** between bone margins is composed of dense fibrous connective tissue. Collagen fibers bundles are oriented perpendicular to the osseous rim and stretch across the sutural gap (Kokich 1976). This structure allows some bone movement without a loss of the tight protective seal around the brain. The arrangement of collagen fibers into bundles gives the middle zone a less fibrous texture (Pritchard, et al. 1956). Vascular networks, supplying blood to periosteal layers between the bone surfaces, interlace through the ligamental fibers (Kokich 1974; Miroue 1975). Anastomosis and transfer of blood between endocranial and ectocranial periostea occur in the central area. All tissues in the central area are continuous with the tissues surrounding it.

The fibrous and cellular network of the periosteum

is not strictly limited to the surface of the bone, rather continues into the cortical structure as the **Haversian System** (Enlow 1962). This network serves to nourish and replace cellular elements within the bone matrix (Ranly 1988). It also functions to remove mineral stores for consumption (Frost 1964). Unlike the periosteal membrane, which surrounds bone, the haversian system is encompassed by bone, limiting it to a plexus of canals. A single canal, the osteon, contains concentric layers of collagen fibrils, osteoblastic and osteogenic cells, epithelial cells, several blood vessels, a complement of capillaries, and unmyelinated nerve cells (Cooper, et al. 1966). The osteon anastomoses freely with other osteons, forming concentric spirals, running in the general orientation of the bone (Cohen and Harris 1958). These structures are rarely found more than two hundred microns apart and are believed to help arrest microstructural damage (Martin and Burr 1982; Vaughn 1970). Osteons form the greatest balance between functional demands, from the cells for nutrition and metabolic needs, the organ (bone) for structural support, and the body as a means of access to materials and reviving structurally weak areas.

6. Cellular Components of the Cranial Capsule

All substrates in the Cranial Capsule are composed of connective tissue, allowing them to function as a single unit. Despite differences in tissue morphologies, histologists agree that these structures include all varieties of proper connective tissue, bone, cartilage, and blood-marrow-lymph cells (Ham and Cormack 1979; Junqueira, et al. 1989).

These cells occupy only a portion of the tissue volume, the rest is filled with an extracellular matrix of fibers, a ground substance, and tissue fluid (McLean and Urist 1968). The ratio of these cellular secretions, as they work in conjunction with the cell to fulfill an environmental need, is the foundation of the Connective Tissue Continuum concept. Each extracellular element provides certain properties needed to maintain homeostasis. Fibers and ground substance determine the strength and flexibility of the tissue and the tissue fluid content serves to absorb pressure and allow materials to pass through the tissue (Junqueira, et al. 1989). The number of cells in a unit of tissue reflect how much biological intervention is necessary to maintain homeostasis. Influences determining the composition of the connective tissue continuum are:

1). The Basic Genetic Component of the Cell. All

tissues in the continuum originate in the embryonic mesodermal layer. Cells following the metameric pattern differentiate into stem cells; these are capable of forming any one of the connective tissue forms (Noden 1982). At the stem cell stage, all connective tissue cells are influenced by the same portions of the genetic sequence. Cell form is limited to that expressible in these sequences; as a result, all connective tissue cellular output possess numerous compositional similarities, varying mostly in structural arrangement.

2). **The Maturation Sequence.** All connective tissue cells undergo some form of maturation. These cells change their size, shape, and appearance from the generalized stem cell to a more specialized configuration, capable of producing a specific extracellular matrix. The resulting progenitor cell's output is limited by selection of specific genetic sequences to produce a narrower range of connective tissue. In adult organisms, mitotic division of the progenitor cell is the source of renewed cells (Ham and Cormack 1979; Junqueira, et al. 1989). When a cell reaches maturity, it loses its mitotic capability. Because of the common genetic background and the adaptability of the progenitor cell, it is difficult to predict the final form of an adult cell (Caplan 1988).

3). **The Environmental Context**. Most structures in the cranial capsule are composed of bone, cartilage, and dense regular connective tissue. Mechanical and nutritional factors outside the cellular environment act in concert to determine what type of connective tissue is formed (Bassett 1964). Bassett and Herrmann's (1961) work with bone cultures identified that oxygen availability, compressive and tensile forces are the primary agents governing whether bone, cartilage, or dense connective tissue is differentiated.

Determination of the final tissue product is not based purely on the integral components of the cell, rather whether environmental conditions fall within the range of certain pre-determined responses. What dictates the range of potential responses are those genetic decisions which were made prior to the connective tissue cell reaching maturity. Since the entire cranial capsule possesses a common underlying sequence of genetic codes, which limit the range of expressible morphology, whether stem and progenitor cells differentiate or remain inherent components of the tissue matrix cannot be used to distinguish which discrete biomechanical trajectory a cell will follow. It is the environmental situation inwhich a stem cell finds itself that determines whether the end product

will be bone, cartilage, or some other form.

The suture area is only a single component of a larger unified system - the cranial capsule. It can be morphologically differentiated into various cellular and extracellular components, each reflecting overall adaptive responses to help meet an extra-capsular demand. A suture cannot be truly defined strictly in terms of gross osteological phenomena because most of the components are not represented as hard tissue. The anatomy and histology of the suture area suggest that the dynamics of the suture require a study of tissue environments and how the entire continuum responds as a biomechanical unit.

CHAPTER III.

DYNAMICS OF THE IMMATURE CRANIAL CAPSULE

The cranial capsule is composed of different structures which operate in conjunction with each other. Fulfillment of an environmental need is not relegated to a single membrane, rather it involves interaction from the entire structural network. Sutures do not grow at a uniform rate. They are variable and responsive to the growth trajectories of the tissues around them. For example, rat sutures develop faster along the naso-frontal, coronal, and lambdoid complexes than regions not affected by the development of a snout (Massler and Schour 1951). This suggests that suture expansion is adaptive to maturational drives.

Responses at a suture area are not isolated, but unified across several suture sites. Any alteration or manipulation of one part of the structure produces a change in others. Persson, et al. (1979) illustrated this concept by initiating premature closure of the coronal suture in rabbit pups. All planes of the

growing skull underwent significant shifts in size and location indicating that sutures compensated for each other (Persson, et al. 1979). Using the same procedures, Babler and Persing (1982) noted that premature closure resulted in a five percent reduction in cranial length. Curtailing the growth rate of sutures parallelling the coronal plane also diminished their ability to interact. Babler and Persing's results indicated that there was less of a possibility for one suture to compensate for the inhibition of another. Sutures do not exhibit maturation drives associated with primary growth centers, which would have continued osteoblastic activity irrespective of the surrounding structures, rather they compensate for the effect of several different growth stimuli simultaneously. The cranial capsule can be seen to respond as a whole unit, not a series of isolated responses.

1. Ectocranial Stress Sources

Environmental stressors, such as muscle activity, expansion of the brain, gravity, and cultural modification from orthodontics, cradle boarding, and other cranial deforming practices apply mechanical force to the skull. These stresses substantially influence the shape of the cranium (Meikle, et al.

1979). The suture's morphological features are direct reflections of how these bio-mechanical forces affect the cranial capsule.

The tissues of the cranial suture do not, in themselves, have a very strong genetic predisposition in terms of location or morphology. Numerous researchers have demonstrated that experimental alteration can drastically change suture morphology. The suture's adaptive response to changes in the local environment was explored in Giblin and Alley's (1944) manipulation of sutures in puppies. In cores where the suture was rotated 90 degrees and allowed to heal, the coronal suture remained patent and a new suture developed, uniting to the existing suture around the core's margin. Switching the location of the suture plug to the center of the parietal resulted in all surfaces of the plug ossifying completely. Giblin and Alley concluded that premature closure in the coronal suture occured when structural rigidity became an environmental need. Compensatory growth in the surrounding tissue triggered complete ossification. It is important to recognize that mechanical stress passed from the surrounding tissues to a suture site; no growth drives within the bone governed the suture's morphology.

For complex interdigitation to arise, muscle

tension must be present. Washburn (1947) demonstrated that by clipping the temporal muscle in rat pups, there were decreases in the outer table's development and amounts of serration in the occipito-interparietal suture's Pars Externa. In some specimens, the temporal muscle was partially cut, creating abnormally complex sutures where normally simple patterns should have formed. Massler and Schour (1951) demonstrated that interdigitation is affected by how long the suture remains patent. Those which stay open longest are generally the most serrated, indicating a structural adaptation to fulfill functional demands. A lack of interdigitation occurs when other forces are greater than muscle tension. In the case of hydrocephalic crania, internal pressure continues capsular expansion. These prevent the suture margins from approximating each other until late in development (Davies 1961). Loss of margin plasticity occurs before muscle tensions can alter the suture's shape.

Suture tissue responds not only to variance in the stress applied at different locations, but will change morphologically to meet changes in functional demands. Oudhof's (1978) research has focussed on the removal of coronal suture sections from the cranial environment of rat pups. The loss of tensile forces resulted in synostosis of their ectocranial aspects (Markens and

Oudhof 1980). In the affected capsular environment, underlying tissues from the dura mater initiated osteosynthesis to accommodate the local bone loss (Markens and Oudhof 1978). Oudhof (1978) and Markens and Oudhof's (1980) work demonstrate that the cranial capsule's structures are not specialized beyond further adaptation, but will enlist cellular components from other aspects of the capsule to fulfill needs for the whole unit.

Suture morphology is not simply the result of the relationship between tension and bone; suture form is also intermediated by soft tissue. Studies of sutural fibroblasts indicate that stress triggers an accumulation of protein within the cell, followed by increases in collagen synthesis (Meikle, et al. 1979). Collagen bundles and fibers are designed to resist traction between bone and connective tissue; the greater the number and size of the fibers, the greater the resistance to stress (Enlow 1975). A bone's reaction is initiated when shifts in the tension on the collagenous sharpey's fibers create a weakened state in the cambial layer. Bone grows to realign the necessary tensile forces (Enlow 1975). Growth of cranial bone is, therefore, an adaptation to stress. Its production indicates that significant change has occurred in the soft tissue environment.

2. Endocranial Sources of Stress

Stress shaping sutural morphology is not limited to ectocranial sources, but is applied from the developmental growth of the brain. In humans and other mammals with thin cranial walls, growth and formation of the cranial capsule largely depends on the growth and morphology of the neural mass (Moss and Young 1960). In the fetal state, the cranial capsule's exterior layers are characterized by a highly fibrous, cartilagineous tissue; the desmocranium or ectomennix (Fazekas and Kosa 1979; Pritchard, et al. 1956). In the early intra-uterine state, there is no indication of the suture's presence. Intramembranous ossification proceeds from the growth centers, laying cortical bone in sheets which separate the fibrous layer into endocranial and ectocranial tissues (Weinmann and Sicher 1955). The ectomennix forms the periosteal layer upon this division (Pritchard, et al. 1956). In the fourth intra-uterine month, human fetuses form a fusiform blastema underneath the presumed location of the coronal suture (Markens 1975). Marken's work with rat pups determined that the blastema's enlargement shifts dural and pericranial collagen fiber orientation changes from parallel to perpendicular to the capsule's surface. As the approaching margins of bone encounter the blastema, they are obstructed by the course of

fiber orientation. The blastema serves to insure that a connective tissue barrier is present between bones and differentiates into the ligamentous central area (Enlow 1975).

These studies do not support the concept that cranial bone growth and maturative expansion of the brain are two separate growth processes, rather they view these processes as part of a single system. The model contends that cranial bone growth is a response to expansion of the brain displacing the surrounding tissues. It follows then, that the suture's connective tissue activity also has a functional relationship with brain growth. Outwardly directed pressure on the skull places tension on sutural membranes, triggering a fibroblastic elongation of the collagenous Sharpey's fibers. This results in a release of tensile pressure, stimulating bone matrix synthesis. Young's (1959) induction of microcephaly and hydrocephaly in rat pups demonstrated that expansive brain pressure is great enough to shift cell and fiber orientation. The result, internally induced cranial deformation, links brain and tissue growth through the soft tissue matrix.

When growth of the brain abates, activity levels in other tissues decrease. The greatest amount of human brain growth occurs in the first six years of

life and is completed by the end of adolescence (Adeloye, et al. 1975). Miroue (1975) states that the suture's tensile strength is greater during active brain growth than after maturative expansion abates. If cerebral growth initiates connective tissue activity in the suture, the cellular components would be denser during youth than in the suture of a mature brained mammal. Numerous researchers have observed that there are changes in the thickness of cambial, fibrous, and middle layers with maturation (Kokich 1974; Kokich, et al. 1979; Miroue 1975; Pritchard, et al. 1956; Scott 1954) Girgis and Pritchard (1958) report that attempts to modify suture morphology after maturation have been completely unsuccessful. Suture morphology appears to be fixed to the form present at the completion of brain maturity. Growth and the amount of sutural connective tissue can be positively associated with the amount of brain activity.

Growth does not simply occur from the accretion of bone to existing surfaces, rather it involves a considerable amount of remodeling to conform inside and outside surfaces of the bone to meet the demands placed on the new structure. Moss (1954) has demonstrated that growth of the vault is carried out through the periosteum. Remodeling is accomplished through removal of matrix by osteoclastic cells from one location and

bone formation occurring in another area. Internal remodeling occurs primarily within the diploic space (Massler and Schour 1951). Cranial bones are not limited to remodeling outward to account for increases in the brain's circumference; passive growth in the suture accounts for connective tissue changes and also adds area to the bone. Cranial growth entails some proportional amount of bone growth at the suture margins (Enlow 1966). Enlow and Hunter (1966) have demonstrated that sutures are sites of vertical facial growth through the processes of apposition and remodeling. The cambial periosteum responds to compression created by the abatement of tensile forces on any side of a suture by depositing lamellar bone. This processes counterbalances growth without a loss of the tension gradient.

3. Sutural Morphology and Genetics

At best, any direct genetic predisposition for sutural location involves osteogenic cells found in the advancing bone margins, transforming into osteoblasts and advancing the margin forward another layer (Decker and Hall 1985). This component does not appear to be very strong; indirect genetic influences from other tissues in the area probably account for at least as

much affect as found in the sutural tissues. Removing the osseous component of the suture displaces its mechanical stresses to the surrounding tissue; the sutural margin counters these environmental demands by prompting an osseous overgrowth (Moss 1954).

There are limits to tissue plasticity. Removal of both hard and soft elements of the suture prevents any bone regeneration (Moss 1954). Girgis and Pritchard's (1958) experiments demonstrated that destruction of the cambial layer eliminated all osteogenic activity. The agent behind growth and displacement is demonstrated to be in the connective tissue, not in the bone. Bennett (1967) has argued that craniostenosis, the loss of a suture's location, is not a direct genetic outcome. This condition represents a tertiary response to modifications in the cranial base's growth rate. At the very least, any genetic participation in a suture's morphology is evidenced to be heavily influenced by the environment.

4. Animal versus Human Models

The vast majority of what is known about suture dynamics is based on studies using animal models. It has been argued that since these animals are thin skulled, their crania are viable representations of the

human capsular environment (Moss 1954). There are three distinct problems which must be addressed before applying these models to humans: there is considerable variation between species and no mammal form has served as the standard for investigation; human and mammalian skulls do not possess the same developmental drives; and animal models tend to address questions of development, not conditions found in the mature form. Clarification of these points will further define their importance.

First, no species standard for suture studies has been defined. The majority of the work with sutures was done using rats, but important aspects have been explored using rabbits, pigs, and dogs as animal models. Hall (1983) and Jee (1964) have noted that there is a considerable amount of variation in the cellular interaction and physiology observed between species. Because of the inter-related nature of the cranial capsule tissues, it would be unreasonable to expect a lack of differences at other levels. In interpreting the dynamics of the suture area, only general trends from these animal models can be applied towards a central model of suture growth.

Second, major differences exist in human and animal facial growth trajectories. Growth in the skull is an interplay of various relationships between

growing subsystems. Roberts (1979) has observed that the masticatory processes are the major elements determining the morphology of the cranio-facial skeleton. Within Anthropoidea most species begin closure between the eruption of the second and third molars (Krogman 1930; Moore and LaVelle 1974). Bolk (1915) has also observed that this parallels the cessation of the brain enlargement in apes. Humans appear to be the exception to the rules of development - brain growth continues after many of the other maturation processes are complete. While suture closure can be seen as a natural event in Anthropoid biology, the variation in different growth rates among body parts will affect how the tissues interact.

In the animal models used, the tendency towards lengthening of the skull to accommodate the snout results in a different cranial configuration than found in humans. The presence of elements not found in the human cranial capsule, such as inter-parietal bones, sagittal and nuchal crests demonstrate that capsular environments are very different. There is little reason to expect a human's morphology to closely follow these animal analogues. No work using a subject species with a less prognathic growth pattern has been found.

3). The focus of most growth models has been the

developing skull. While these studies identify major drives behind suture growth and morphology, they provide little information about what happens after maturation (Behrents 1985a). The bio-functional activities of the mature suture are poorly understood. The applicability of developmental growth dynamics to the processes involved in normal adult suture fusion must reasonably be called into question.

CHAPTER IV.

THE DYNAMICS OF THE MATURE HUMAN CRANIAL CAPSULE

1. Post-Adolescent Cranial Change

During developmental growth phases, there are bursts of cellular activity geared for replication or expansion. With maturation, this proliferation slows and the tissues concentrate on maintaining their environment. Growth does not stop with the completion of the adult frame, but there are characteristic changes in its form. Aging diminishes the number of cells and shrinks their activity levels (Hayflick 1978). These changes are most pronounced in cells specialized to perform specific functions (Hayflick 1980). Shifts in the ratio of cells, ground substance, and tissue fluid in fibroblasts result in a shrunken, elongated, less responsive cell (Tonna 1977). Collagen fibers become thinner, more dispersed in the matrix, and shift from a linear to a more spiral shape (Gross 1961; Kokich, et al. 1979; Vincentelli 1978). Whiteside (1984) observed that the density of periosteal vasculature also diminished with age. These

changes engender a slower, less effective reaction in the periosteum to growth and trauma (Tonna 1977).

Within bone, the aging process prompts osteoblastic activity to drop below osteoclastic rates, resulting in a general loss of bone mineral content (Frost 1964). In the cortical structure, Kerley (1965, 1969), and Thompson (1979, 1981a) have demonstrated that internal remodeling processes replace much of the original bone with less mineralized matrixes. The total area replaced bears a linear relationship with age (Singh and Gunberg 1970). Despite their similarity, aging affects different aspects of the connective tissue continuum, as well as other organ systems, in vastly different ways. There are no hard, fast rules which describe how these tissues will age (Hall 1966).

The cellular changes with age do not rule out the presence of post-adolescent growth. Garn, et al. (1967) demonstrated that metacarpal width increased four percent between maturity and the eighth decade of life. Age related changes in skeletal morphology have been isolated to weight bearing or stress responses in the post-cranial skeleton (Rossman 1977). Unlike the post-cranial skeleton, the skull does not loose bone; rather it gains osseous components (Israel 1967). Adult bone growth can not simply be viewed as a

response to bone loss in the weight bearing elements.

Soft tissue elements of the cranial capsule also undergo senescent modification. Hooten and Dupertuis (1951) noted that an age related decline in the head circumference could be attributed to shrinkage of the scalp and temporal muscles. Upwards of eleven percent of the brain weight is lost between adulthood and old age (Appel and Appel 1942a, 1942b). Despite losses in soft tissues there are ample data demonstrating increases in cranial dimension. Behrents (1985b) observed a general increase in size, particularly in the vertical dimensions. Boersma (1974) found that elements of the nasal floor contributed to a considerable portion of age related increases in length. Israel (1977) calculated this increase to be about 3.5 percent over prior length. Conversely, Rogers (1982) reports work by Fenar and Dufresnoy (1975) establishing that Pars Bregmatica lowered and the cranial mass shifted its dimension caudally and posteriorly with age. Israel's (1967) longitudinal radiographic study found median increases in cranial size of about half a millimeter.

Cranial thickness has also been noted to expand. Rossman (1977) cites work by Buchi (1950), who noted that a mean increase of five millimeters in vault thickness was associated with increases in cranial size

and age. Israel substantiated these findings, observing that they were reflected mostly in the endocranial dimension (Israel 1973). Cranial growth occurs throughout life. This activity reflects change in the needs of the soft tissues; much like that found in the developing cranial capsule; however, at a much slower rate.

2. Post-Adolescent Ossification

The suture, like other soft tissue, undergoes morphological change associated with the aging process. Most of what is known about post-adolescent suture growth is based on change observed only in the hard structure. The association of age to closure has been utilized by Physical Anthropologists and Anatomists to estimate the biological age of crania. Since the sixteenth century, suture closure has served as a relative age indicator; a systematic inquiry of the suture change with age, however was not attempted until Gratiolet's work in 1856 (Ashley-Montagu 1938).

Not all sutures have an established age relationship. Closure of the parieto-mastoid, squamosal, and occipito-mastoid sutures show no association with age (Meindl and Lovejoy 1985). Others, like the sphenoid and fronto-nasal sutures,

remain viable throughout life. Because sutures associated with the facial aspects close very late in life, their pattern of closure has not been very thoroughly explored. (Cobb 1955; Kokich 1976). Only recently has attention turned to the maxillary sutures. These have been observed to undergo advanced closure at a predictable rate (Mann, et al. 1987, 1991; Persson and Thilander 1977).

Endocranial and ectocranial suture surfaces show vastly different closure rates. Sauvage (1850) noted that endocranial closure usually commenced first (In: Ashley-Montagu 1938). Todd and Lyon (1924) found that endocranial fusion started in the early twenties and terminated by the time one reached their early forties. While both surfaces commence closure at about the same time, there is considerable variation in the ectocranial aspect, which tends to close more slowly (Todd and Lyon 1925a). These results have been verified in numerous other study populations (Baker 1984; Miroue 1975).

Perizonius's (1984) work with Danish crania provided results indicating that older individuals tend to show less suture fusion. Suture closure may be negatively related to life expectancy; factors preventing suture closure might allow one to live longer. Perizonius compared the Danish collection to

Hungarian crania, collected fifty to seventy years later. He noted a greater degree of fusion in the more contemporaneous sample. This suggests that secular trends may also influence suture activity. Perizonius's work establishes that factors shaping the longevity and growth of the individual play a part in determining suture morphology.

A few demographic differences have been observed in fusion patterns. Brooks (1955) noted that female suture closure lags behind changes in the pubic symphysis. Baker (1984) found that narrower age ranges were present in coronal and sagittal suture closure rates. No significant differences in obliteration have been noted between the sexes (Meindl and Lovejoy 1985; Perizonius 1984). While Baker (1984) noted that Negro ectocranial fusion was more complete than among Caucasians, variation in amounts of closure and rates of closure for any given suture site do not express major differences between racial stocks (Krogman and Iscan 1986; Meindl and Lovejoy 1985; Todd and Lyon 1925b, 1925c). Baker (1984) has also reported that health can affect the way a suture closes, but the evidence supporting his assertions is not conclusive. It has been the general consensus that these demographic differences do not play an active role in suture closure.

The erratic closure behavior in sutures has hampered their use as an age estimation tool. Attempts to improve age prediction have led researchers to divide sutures into groups. For example, ectocranial suture closure, particularly in the sagittal sections, occurs in bursts of activity. They often do not fuse to the degree found in the endocranial aspect. Todd and Lyon (1924, 1925a) felt that these problems make ectocranial suture closure a less reliable age estimator. Todd and Lyon (1925a) have also suggested that the ectocranial and endocranial surfaces should not be considered as a unit, rather they must be treated as separate entities. Subsequent research has concentrated on the ectocranial surface. Meindl and Lovejoy (1985) broke the ectocranial sutures into Lateral-Anterior and Vault systems based on regional locations on the skull. They have been able to demonstrate that the Lateral-Anterior provides reliable age predictions with relatively narrow age ranges.

The predictive applications of suture closure to age have not received favorable reviews. Singer noted:

> ...With the techniques available at present, an assessment regarding the precise age at death of any individual, gauged on the degree of closure of the vault sutures of the skull is a hazardous and unreliable procedure. (1953:59)

Suture closure's use as a single measure of age is not

advocated (Dwight 1890; Krogman and Iscan 1986). Comparisons of suture closure with other skeletal age estimators, such as the pubic symphysis, establish a poor relationship between both sutural and symphyseal age prediction and morphologies (Brooks 1955). Most researchers believe that, at best, suture closure should be used as part of a battery of measurements to estimate age (Meindl and Lovejoy 1985).

While these studies have intensely explored the relationship of closure and closure rates with age, research questions have not focused on determining whether other factors are affecting suture fusion. There has been little consideration of how sutures fuse beyond the observed endocranial and ectocranial states. This lack of knowledge stems from the perception of suture closure's value primarily as a predictive tool, not as the subject of study. Knowledge of the post-adolescent suture growth has not expanded much since Todd and Lyon noted:

> It will be realized that the observations are correct only to the external and internal surfaces of the cranium and we have no accurate information regarding the condition of sutures deep within the substance of the skull wall. (1924:332).

Most of the research cited in this section has the common goal of age determination; the research methods applied are based on attributable features, which do not necessarily reflect an accurate view of the

biology. In order to improve the technology applied to suture age prediction, a more comprehensive understanding of the physiological processes surrounding closure are needed.

3. How Fusion Occurs

Ashley-Montagu (1938) observed that in the skull only dentition and sutures display any age related post-adolescent change. Unlike dental change, which can be attributed to use and attrition, the dynamics of suture closure are poorly comprehended (Lovejoy 1985). Fusion does not occur uniformly across the whole suture margin. It is progressive, beginning in several locations and gradually ossifying various regions of soft tissue with time. Kokich (1976) has observed that commencement never occurs on the periosteal surface, rather it is initiated within the sutural gap.

From the perspective of gross observation, post-adolescent suture growth appears to follow many of the same processes governing developing forms. Singer (1953) noted the presence of ossified bridges stretching across the sutural gap on both endocranial and ectocranial surfaces. In the facial complexes, the constant lengthening of sutural projections with age allows appositional surfaces to meet (Kokich, et al. 1979). Fusion results from a union of the opposing,

periosteal layers (Scott, 1954). What initiates the fusion process has not been determined.

Observed histologically, suture closure is considerably more complex. The bony projections of the pars externa increase in number and length (Kokich 1976). These surfaces become increasingly irregular. Projections parallel the ectocranial surface and tend to be longest in the center of the suture. Union of the central projections explains why the periosteal surfaces initially close first. Singer's bony bridges have their origin in these microscopic projections.

There is some confusion among researchers about the types of bone found in the sutural margin. Zoller and Laskin (1969) report that zygomaxillary sutures among piglets possess numerous resting lines, suggesting apposition of lamellar bone. Others, such as Weinmann and Sicher (1955) and Scott (1954), also note that suture margins expand through lamellar deposition Direct observation of lamellar deposits was substantiated by Miroue (1975). In seeming contradiction, he also states that bony bridging is accomplished through woven bone. Kokich (1974) observed a similar phenomenon. Since woven bone is deposited rapidly, it does not contain appositional resting lines (Frost 1964, 1960). With continued bone matrix secretion in long bones, the slower deposition

processes fill in the spaces left by woven bone fibers and gradually remodel these in to the stronger lamellar bone (Frost 1964, 1960; Kerley 1965; Vincentelli and Grigorov 1985). Miroue (1975) has pointed out that these deposition processes are the same utilized in the repair of trauma. He contends that in adults, continued growth of bone is limited only to only a few ways of gaining new osseous structures. It is important to note that lamellar and woven matrix activities are not discrete sequences, rather they may occur simultaneously.

Within the sutural ligament, collagen fiber density decreases and becomes more irregularly arranged with age (Kokich 1976). Fiber orientation changes from perpendicular/parallel arrangements to a more disorganized pattern (Miroue 1975). These losses serve to weaken the suture's tensile abilities. There is little doubt that the loss of collagen is associated with age changes in fibroblasts. It would be expected that these conditions would initiate osteoblastic activities, but observations of the ligament area suggest that this is not necessarily the case. Persson and Thilander (1977) have noted the presence of cartilagineous connective tissue forming bridges in the palatal sutures. It is unclear what role cartilage plays in the fusion process. It seems reasonable to

suggest that cartilage develops in the compressive avascular environment formed in the sutural gap and may replace the need for continued ossification. Moss (1957, In: Miroue 1975) has suggested that cartilage may be replaced by bone; Miroue, however, observed cartilage in only three of one hundred and eleven sutures exhibiting any signs of closure. It is more probable that cartilage is an environmental product and is not required for suture ossification.

The loss of fibroblastic collagen fibrils in the sutural margin does not necessarily dictate replacement by cartilage or bone. In most instances, the suture area has been observed to fill with diploic tissues. Kokich (1976, 1974) and Miroue (1975) have observed that with increased age, the diploe removes part of the suture's cortical structure and replaces it with sinisoidal tissue. Communication with the sutural ligament, particularly after bony bridges have spanned the sutural gap, also increase with age. While cellular elements within the bone structure tend to remain hematopoietic, those filling the sutural gap change over time into fatty reservoirs. Miroue (1975) has observed that the presence of diploic tissue in the suture gap indicates a major shift in function from protection and growth to storage and blood cell production.

With increased maturity, changes in morphology reveal that the biomechanical demands placed on the skull are no longer the same. Young crania display sutural patterns designed for increased brain and facial growth as well as meeting the demands of stress displaced from the cranial musculature. With the cessation of brain growth the need for expansion is lessened to a point where remodeling is able to satisfy most of these demands. With increased age, these qualities are lost. Fusion of the suture indicates that its flexibility has been exchanged for a more rigid structure and the tissues within it are replaced with those satisfying fewer structural needs. Modeling a suture's morphology should reflect some representation of how various environmental demands are met and change as an individual ages.

CHAPTER V.

THE CAPSULAR MODEL OF SUTURE CLOSURE

Why do sutures change with age? A review of the literature suggests that the theoretical constructs currently applied to human post- adolescent suture activity cannot adequately model how and why morphology varies with age. Animal models are not satisfactory because maturation in most subject-species involves the growth of additional structures not present in humans. Descriptive models are geared to explain the growing state; their application, however, is very limited beyond completion of maturation. Age-from-suture relationship models are inappropriate because their goal of age prediction does not identify why a change in structure should occur. The use of these analytical tools does not adequately account for osteoblastic activity in the mature human skull. Beyond the well documented relationship between age and suture closure, the dynamics of fusion are poorly understood. It is unclear what biological forces are responsible for

initiating tissue change in the skull. To answer this question, the suture must be perceived from its anatomical context and address the reasons why biomechanical needs of the skull shift with age.

Moss and Young's cranial capsule includes the suture as part of a hard and soft tissue complex. Sutures are not viewed as isolated cranial elements; their morphology identify that environmental influences are acting on the cranial capsule. Each tissue acts in conjunction with others to meet with the demands placed on the structure as a whole. Any alteration in the capsular environment will result in a change in the tissue complex's morphology. These transformations are identifiable in bone as shifts in cellular activity.

Most models view post-adolescent suture activity as a phenomenon isolated to the hard structure. The level of analysis and power of explanation generated by subsequent research is only capable of addressing bony tissue. The cranial capsule does not perceive bone as a separate organ, but as part of a larger system of interacting tissues. Bone morphology reflects adaptations to the environmental state placed on the capsule as a whole. By emphasizing tissue interaction, an explanation of suture fusion inherently integrates changes in suture morphology with forces

outside the bone.

One of the advantages to this perspective is the ability to separate cranial bone into its biomechanical elements. Prior research has confirmed that if disproportionate demands are placed on the cranial capsule, only portions, not the whole structure, will react. Modeling the mechanics driving suture closure require recognition, control, and study of the cranial capsule according to identifiable differences in stress. One important distinction is recognized between endocranial and ectocranial demands. Since the neural environment does not affect the cranial capsule the same way as scalp and muscular environments, morphology inside the vault should not mirror that outside. On the ectocranial surface, growth studies have confirmed that variations in mechanical demand placed on the skull influence the type of suture joint formed and the complexity of interdigitation between calvarial bones. Age variation in fusion indicate that differences in functional demands are still extant beyond maturity.

Soft elements of the skull must be divided similarly. Young sutures do not possess the same connective tissue complement found in more mature forms. These changes in the soft components result from modifications of the cranial capsule's needs.

With age, these shifts in the biomechanical forces shape the cranial tissue. Any explanation of the suture's form must recognize the existence of those forces and demonstrate how force relates to the tissue state observed. Suture morphology is modelled as the interaction of three prime forces:

1. Expansive forces from the brain and surrounding structures pushing the cranial capsule outwards. This force is directed at all aspects of the capsule and dispersed through the hard tissue via the suture. The suture must allow for expansion and still maintain a firm bond between the cranial capsule and the brain.

2. Muscles attached to the skull apply tensile force pulling the capsule away from the brain's surface. The suture must maintain sufficient structural integrity to support and provide leverage without compromising the capsule's protective shield.

3. Expansive forces, principally in the diploe, act to remodel cortical bone in order to increase hematopoietic and storage capability inside the cranial capsule. The suture must adapt to maximize diploic volume and provide enough structural integrity to meet physical demands.

Viewing sutures as a tissue interaction phenomenon allows the morphological change observed by other

paradigms to be translated into differences in functional demand placed on the cranial frame. Suture morphology is the consequence of interplay between expansive and tensile forces on the entire tissue spectrum; identifying the demands placed on the capsular environment provides a continuity between all existing sets of data. These explanations: a) are not limited by age; b) can be adapted to fit the presence or absence of non-human cranial features; and c) provide insight as to what suture-age prediction is truly testing. Simple encapsulation of existing data cannot demonstrate that one model is any better a picture of reality than others, a superior model must be able to explain observations that other forms cannot. The capsular model's validity can be suggested by testing whether morphological changes in suture form are uniformly related to age or if differences in bio-mechanical demands contribute to closure.

The cranial capsule should be capable of mathematical as well as rhetorical description. The application of this model; that observable morphology is the result of interaction between ectocranial influence and age, endocranial influence and age, diploic influence and age; can be expressed as:

$$M=(IA)+(OA)+(DA)+E$$

where,

M (Morphology) - Refers to the sequence of tissues present at a defined suture site in the cranial capsule.

I (Endocranial Influence) - The morphological responses to expansive stresses, originating principally from the neural environment, are expressed in this variable.

O (Ectocranial Influence) - This value expresses the effect tensile forces, originating from the ectocranial environment, have on morphology in the tissue structure.

D (Diploic Influence) - This variable defines how expansive force from the diploe acts to alter the observed morphological state. Assessments of D require cross sectioning the suture site. These destructive processes could not be utilized on the collections available for analysis. For the purposes of this study, error has absorbed this influence).

A (Age) - This variable recognizes that tissue states are not constant, but are influenced

by maturation and senescence. The aging process does not affect different environments the same way or at the same rate. Age cannot be considered as an independent variable rather as an affect acting on each examined aspect (Rowe and Kahn 1987). Differences in aging trajectories in each tissue require that age must be factored separately for each variable (Shock 1985).

E (Error)- This variable encapsulates influences not identified in this model.

Without testing the validity of this model, it cannot help explain how age affects morphology. One way the model's utility can be demonstrated is by how well testable hypotheses can be drawn from it. These hypotheses should be: a) able to exploit data from realistically obtainable samples; b) grounded on variables capable of statistical manipulation; and c) maximize explanation from a minimal amount of data. In the following sections, each of these requirements will be detailed according to how they relate to the capsular model. It is hypothesized that if differences in environmental pressures affect

post-adolescent suture morphology, then after age has been controlled, these demands will be reflected as groups of sutures displaying prominant high and low, regionally clustered correlations of suture activity. This hypothesis will provide evidence that either supports or dismisses the capsular model's usefulness.

CHAPTER VI.

POPULATION PARAMETERS

Collection, preparation, and analysis of data capable of testing the model had to be free of uncontrolled variation. A primary concern was whether an adequate sample could be selected from a representative population of human skulls. The sample population needed to consist of a large collection of relatively complete, undeformed crania. Study sites on the endocranial and ectocranial aspects had to be observable. Race was controlled by concentrating on subjects of Euro-American extraction.

Age was the principal demographic variable known to affect suture form. Because slight variations in the age can result in profound differences in morphology, documentation of the known chronological age was a necessity. It was noted that the practice of age estimation, based on appraisal of the biological age, has been utilized in some skeletal collections (Katz and Suchey 1989; Meindl, et al. 1990). the use of crania from age estimated skeletons would not

adequately control for this important parameter and were not used for two reasons: first, age estimation limited ages to pooled categorical variables, limiting their conversion into other forms of data; and secondly, construction of age estimates inherently added an element of error to the data. Only crania whose age was verified in the collection's documentation comprise the sample (See Comments by Meindl, et al. 1990).

These requirements were best met by using a modern population. In similar studies, Kokich (1974, 1976) and Miroue (1975) utilized medical school cadavers. Baker's (1984) analysis considered an autopsy population and McKern and Stewart's (1957) work focused on Korean War dead. Other researchers have had considerable success with established permanent anatomical collections. These assemblages have had many other aspects of their skeletal biology documented and research can be replicated or expanded to new aspects within the same group.

The R.J. Terry collection, currently housed at the National Museum of Natural History, Smithsonian Institution, Washington, D.C., was well suited to meet the needs of this project. It is comprised of human skeletal remains accumulated by the Department of Anatomy, Washington University, between 1914 and about

1965 (Thompson 1981b). This collection is extremely well documented and consists of well over 1500 individuals (Moore-Jansen 1989).

Holland (1986) noted that the Terry collection contained greater numbers of older individuals and few young adults. To supplement this sample with penicontemporaneous cohorts, crania from the Armed Forces Institute of Pathology's Civil and Indian Wars collections were used. These collections represent cranial samples obtained from casualties between 1860 and about 1870 (Sledzick - personal communication). Many of these crania are from known soldiers, representing individuals less that 45 years old. Unfortunately the nature of the collections, aimed at documenting battlefield related trauma, produced very few complete crania; relevant data were obtained and used to help boost the under-represented young adult sample.

A stringent stratified random sampling technique was implemented to maximize statistical reliability. Separate inventories of all white males and females were ordered by age. Individuals within each age were selected using a random number generation table (Ott 1988). To facilitate an individual skull's data independence, random drawing without replacement was utilized until a full age complement was constructed.

Crania unsuitable for analysis (missing or damaged features, obvious pathological or traumatic influence on closure, or like situations) were discarded and replaced with the next randomly selected individual in the age cohort. The examination of sequence of crania was randomized in order to prevent any age related scoring bias. The male portion of the collection was samples completely five times, selecting one individual each time from every age available. In ages containing less than five individuals, all observeable crania were used. Limitations on time enabled the female portion to be sampled only three times. The collection sample consists of crania from 409 individuals. Verification of age, race, and sex with the original morgue records reduced the study sample to 399 (See Appendix).

CHAPTER VII.

OBSERVATION SITES ON THE SKULL

Within the study population this investigation focused on suture areas exhibiting a flat junction morphology. Flat and bevelled sutures display fundamental morphological differences profound enough to expect morphological change associated with age to be dissimular. Concentration on a single form insured that the study's results were not confounded by pooling divergent environmental conditions.

The Capsular Model of Suture Closure recognized that morphological differences between ectocranial and endocranial surfaces stem from their relative proximity to stressing agents. Testing the validity of this observation required recording data from the same location on the interior and exterior surfaces.

On the ectocranial surface, identifiable patterns in suture serration have been linked to variations in developmental and environmental stress. Researchers have noted that these influences vary with age; this is evidence that the cranial capsule attempts to

localize activity as much as possible (See Meindl and Lovejoy 1985; Todd and Lyon 1924, 1925a-c). It cannot be assumed that physiological processes occurring at one site on the sutural margin are the same elsewhere. Sutures need to be examined according to known variance in the environmental state. Recognizing that different stresses in the maturing skull resulted in variations in sutural morphology, the ectocranial surface was separated and examined by these recognizable shifts in serration. The general lack of distinguishable features in the endocranial suture margin make it difficult to select areas on both sides of the bone which suggest variations in stress. The distinctiveness of ectocranial features was given precedence in defining examination sites. Endocranial surfaces were observed and scored in areas immediately contrasting an ectocranial landmark. For each skull, four unilateral and five bilateral sites were examined, producing a total of 28 observations. Descriptions of these observations are as follows:

A.Coronal Suture

1. Pars Bregmatica: From the point bregma, the coronal suture forms a marginally serrated line, abruptly transforming into the more complex Pars Complicata. Pars Bregmatica falls within these two

features. The regions observed are one square centimeter areas approximately 0.5 to 1.5 centimeters left and right of bregma (Figure 5).

Combined with the sagittal pars bregmatica, Meindl and Lovejoy (1985) found that average ectocranial fusion commenced in the early 40's. Todd and Lyon (1925a) have observed ectocranial fusion commenced at about 26 years of age and completed by age 29. Endocranially, they found that fusion began at 24 and ended around 36 (Todd and Lyon 1924).

2. Pars Complicata: Lateral to the pars bregmatica, this complexly serrated suture extends to the temporal line, gradually transforming into a bevelled suture. These serrations suggest a high muscle tension environment. Pars complicata is also measured bilaterally. The areas of observation are the approximate midpoints between bregma and pars stephanica (Figure 5).

Ectocranially, Meindl and Lovejoy (1985) noted that fusion initiated in the early 40's; Todd and Lyon (1925a) found 29 to be the average age of commencement. Endocranially, 25 was when closure started and complete fusion was reached by 36 (Todd and Lyon 1924).

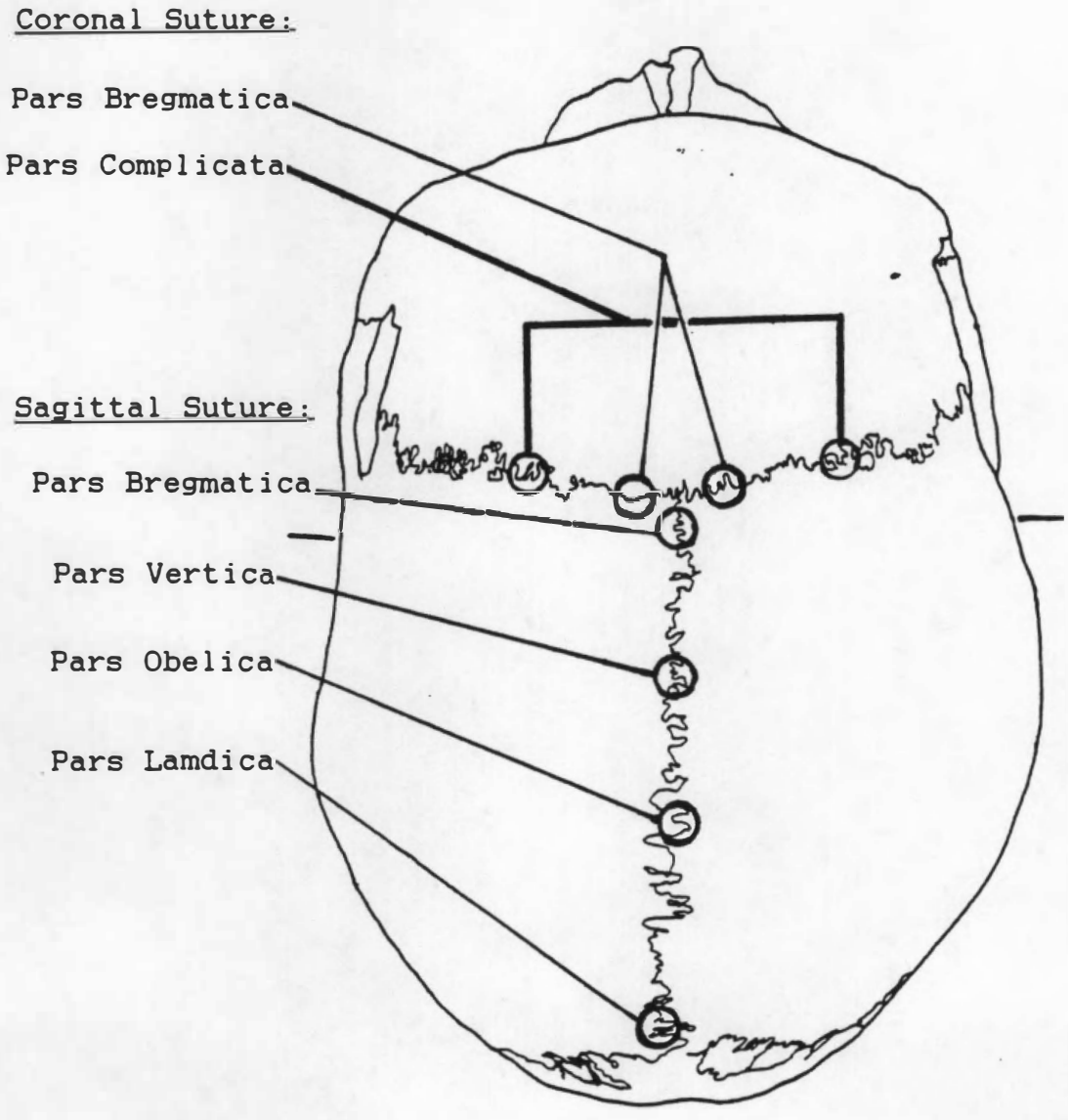


Figure 5.

Coronal and Sagittal Suture Sites

B. Sagittal Suture

3. Pars Bregmatica: At the point bregma, the sagittal suture forms a relatively uncomplicated line, composed of short, wide, simple serrations. As the suture weaves posteriorly, it becomes gradually more complex. A single observation of the sagittal pars bregmatica is made dorsally about one centimeter from bregma (Figure 5).

Todd and Lyon (1925a) noted that ectocranial fusion occured between ages 26 and 29. Endocranial fusion spanned from 23 to 31 (Todd and Lyon, 1924).

4. Pars Vertica: Moving dorsally, approximately one third the distance from bregma to lamda, the sagittal suture forms a long, narrow, and highly interdigitating line. One observation is taken in the approximate center of this region (Figure 5). The serration is indicative that high amounts of stress were prevalent at least during maturation.

Todd and Lyon (1925a) observed ectocranial fusion commencing around 21 and fusion stopping by age 29. Endocranial fusion occurs between 23 and 28 (Todd and Lyon 1924).

5. Pars Obelica: At approximately two-thirds of the dorsal length from bregma to lambda, the suture becomes long, wider, and less complicated. The parietal foramen is usually in close vicinity to this

region. A single observation is scored from the center of the suture section (Figure 5).

Todd and Lyon (1924, 1925a) noted ectocranial fusion began at about 20, with closure observed around 29 and endocranial fusion ranged from 22 to 29.

6. Pars Lambdica: This region is between pars obelica and the point lambda. It is characterized by the same long, narrow, highly interdigitated morphology present in pars vertica. One observation is made from an area approximately one centimeter superior to lambda (Figure 6).

Meindl and Lovejoy (1985) noted closure commencement for sagittal and lambdoid pars lamdica averaged at 38 years with obliteration occurring about 10 years later. Todd and Lyon (1925a) found a different pattern for the sagittal complement, indicating ectocranial closure began at about 21 and ended around 29. Endocranially, fusion ranged between 23 and 34 (Todd and Lyon 1924).

C. Lambdoid Suture

7. Pars Lamdica: From the point lambda, extending bilaterally approximately one third the distance of the lambdoid suture, this region is characterized by relatively short inter- digitations. The width and serration complexity is highly variable,

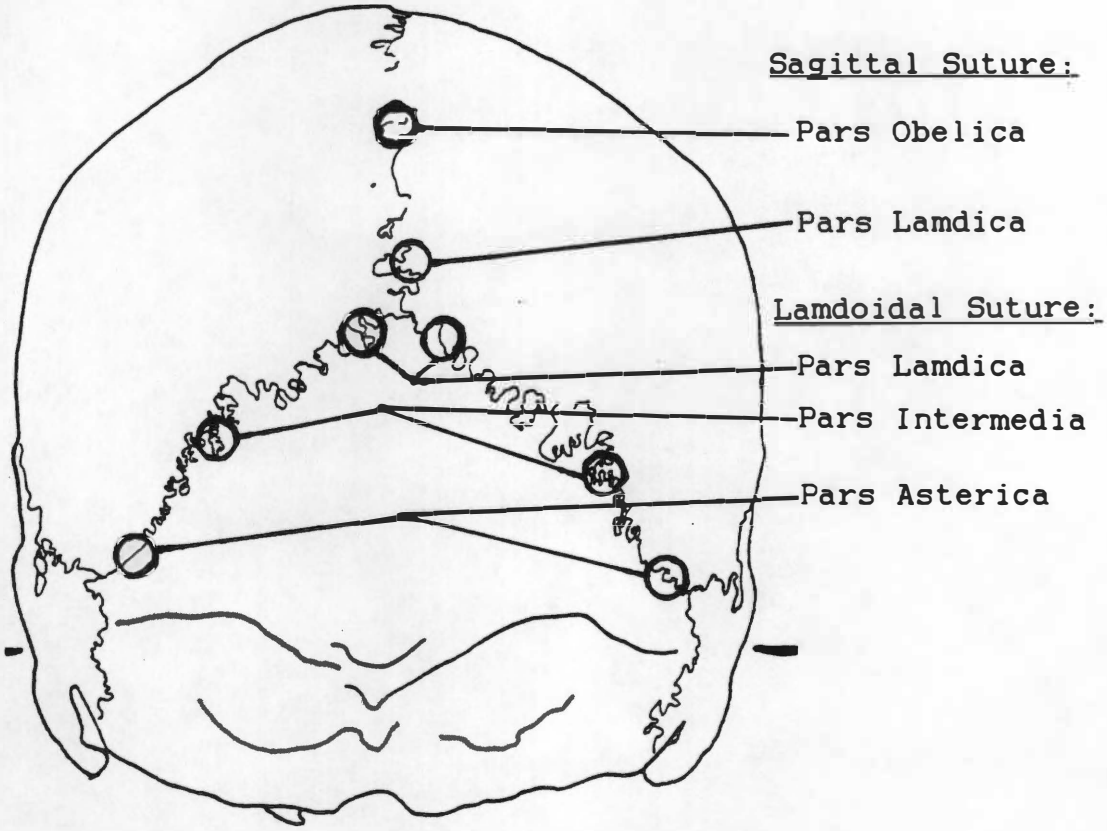


Figure 6.

Sagittal and Lambdoidal Suture Sites

but consistently less than that seen in the pars intermedia. This suture area appears to be subject to high amounts of tensile force. Bilateral observations are made from areas approximately one centimeter on either side of lambda (Figure 6).

Todd and Lyon (1925a) indicated that ectocranial closure began about 26 and terminated around 30 years of age. Endocranially, they found average closure started at 25 and ended by 41 (Todd and Lyon 1924).

8. Pars Intermedia: This region is located centrally between lambda and asterion. It is highly serrated with elongated, narrow interdigitations. Bilateral observations are taken from the central centimeter of this region (Figure 6). Todd and Lyon (1924) noted that endocranial closure commenced around age 25 and finished by 40. Meindl and Lovejoy (1985) found closure starting about age 40 and the area was obliterated around age 52.

9. Pars Asterica: Between pars intermedia and asterion, the lambdoid suture's serrations become shorter, wider, and less interdigitated. Very often this process is gradual, until the suture reached asterion; this suggests developmentally that it was a transitional environment between areas of greater and lesser tension. Bilateral observations are taken from the area one centimeter medial of asterion (Figure 6).

Todd and Lyon (1925a) noted that ectocranial closure takes place between the ages of 26 and 34. Endocranial closure started around 25 and terminated about age 47.

CHAPTER VIII.

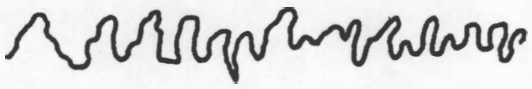
SCORING

1. Analytical Problems in Suture Scoring

Suture analysis has been plagued by several methodological barriers. There has been an inability to reliably document the true state of a given suture. Closure has been quantifiable only by conversion into ordinal or categorical variables. It is generally recognized that when mature, the adult has no osseous union;with age the skull proceeds through phases of bony bridging and gradual closure of the sutural gap, finally terminating with obliteration of the suture's presence on the skull. Early accounts have focused on describing the changes observed (e.g. Dwight 1890). The most widely used classification system was developed for Todd and Lyon's studies of the Western-Reserve Collections (Todd and Lyon 1924, 1925a-c). This system broke closure into five morphological states and assigned them numeric values (Figure 7). This scoring permitted a quantifiable relationship between closure and age to be assessed (Ashley-Montagu

Meindl and Lovejoy (1985) Scoring Method

SUTURE = 0



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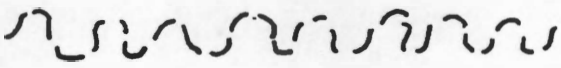
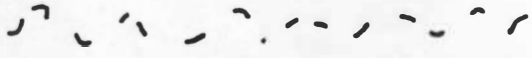


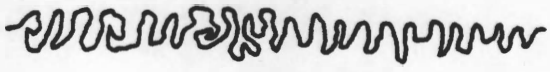
SUTURE = 2



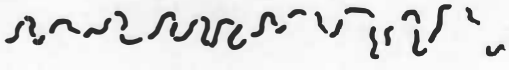
SUTURE = 3

Todd and Lyon (1924, 1925a-c) Scoring Method

STAGE 0



STAGE 1



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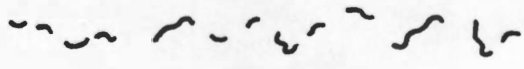
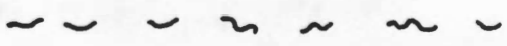


STAGE 3



STAGE 4

Figure 7.

Differences in Common Suture Scoring Methods

1938). A similar scoring system was developed, using five similar states to calculate a mean closure score (Perizonius 1984). It enabled an age assessment to be made from all sutures (Perizonius 1984).

Despite the high ability for mathematical manipulation, the reliability of these methods is based on the initial subjective interpretations involved in assigning the observed state to a category. An accurate method of measuring this type of activity as a continuous variable has not been developed.

In order to reduce the interobserver variability, Meindl and Lovejoy (1985) simplified the design to a four stage system (Figure 7). Age estimation was accomplished by adding the scores of the Lateral-Anterior System and applying these to a growth table. A second age score was obtained from the use of the Cranial Vault System. The simplest scoring method, developed by Baker (1984) to examine a Los Angeles County autopsy population, categorized a suture either as open, fusing or closed. Age estimates were based on comparison with age ranges established for each state. This technique, unfortunately does not provide an analysis indicating the predictive reliability of these ranges, making its use highly questionable.

Another observational problem has been scoring the

lapsed suture union. Lapsed unions are ectocranial regions of the suture margin which have remained patent, often displaying rim thickening. These areas are in sharp contrast with both endocranial activity and closure elsewhere on the ectocranial surface. Lapsed unions are common in mammals and are seen mostly in the lambdoidal and sagittal sutures (Todd and Lyon 1924). Cobb (1952) noted that the lapsed suture is seen in individuals around thirty years old and may be part of the regular arrest of activity associated with midlife. Lapsed unions do not reactivate; the only morphological change noted is a shift from granular to smooth surface texture, indicating that bone growth has terminated (Todd and Lyon 1924). Because the observation's identification involves a comparison with activity on the internal surface, researchers have overcome the problem by limiting observation to specific sites on one side of the skull (Krogman and Iscan 1986; Meindl and Lovejoy 1985). This practice, however does not accurately portray whether the lapsed union is a normal part of the maturation.

2. Scoring Method

Most of the methods used to score suture morphology cannot evaluate it in terms of a realistic biological

condition. All researchers agree that sutures showing no fusion ('open sutures') and those crania showing a continuous osseous surface ('closed sutures') represent fundamentally different conditions. Methodological confusion arises when sutures are observed in the process of fusing. Traditionally, the answer has been to divide partial fusion into ordinal variables, based on the amount of fusion that has occurred (see for example Baker 1984; Falk, et al. 1989; Meindl and Lovejoy 1985; Perizonius 1984; Todd and Lyon 1924, 1925a-c for various treatments of this problem). Imposing an ordinal scale to suture closure severely restricts the statistical manipulation. It requires a greater subjective interpretation, than other forms of data. This results in relatively high levels of inter-observer error (Meindl and Lovejoy 1985). Finally, ordinal interpretations imposed on the continuous process of closure force artificial segregations of the data.

While non-parametric statistical methods do not have the same power as their metric counterparts, but they are able to provide conservative approximations of difference between random and non-random distributions (Siegel 1956). The simplicity, lessened degree of intra-observer error, and uniform acceptance of the Meindl and Lovejoy three stage system (described

earlier) are arguments that led to collecting ectocranial and endocranial data according to this descriptive morphology. The scoring of suture closure is as follows:

0 - This observation contains no osseous material in the exposed surface (Figure 8) A completely open sutural gap indicates that the tissue present was clearly soft connective tissue and that demands on the cranial capsule had not dictated a transformation of tissue forms.

1 - Less than 50 percent of the suture is observed to have ossified (Figure 8). The presence of bony protuberances or bridges, as described by Kokich (1974), Miroue (1975), or Singer (1953) are fusion features demonstrating the presence of a tissue transformation.

2 - Between 50 and 99.9% of the suture appears ossified (Figure 8). The tissue concentration in this observation has substantially emphasized the need for a greater amount of bone at the expense of the softer structure.

3 - Sutural gaps that contain no connective tissue will have a morphology completely composed of bone (Figure 8). The observed surface will not have a margin, rather is a continuous plane of osseous tissue.

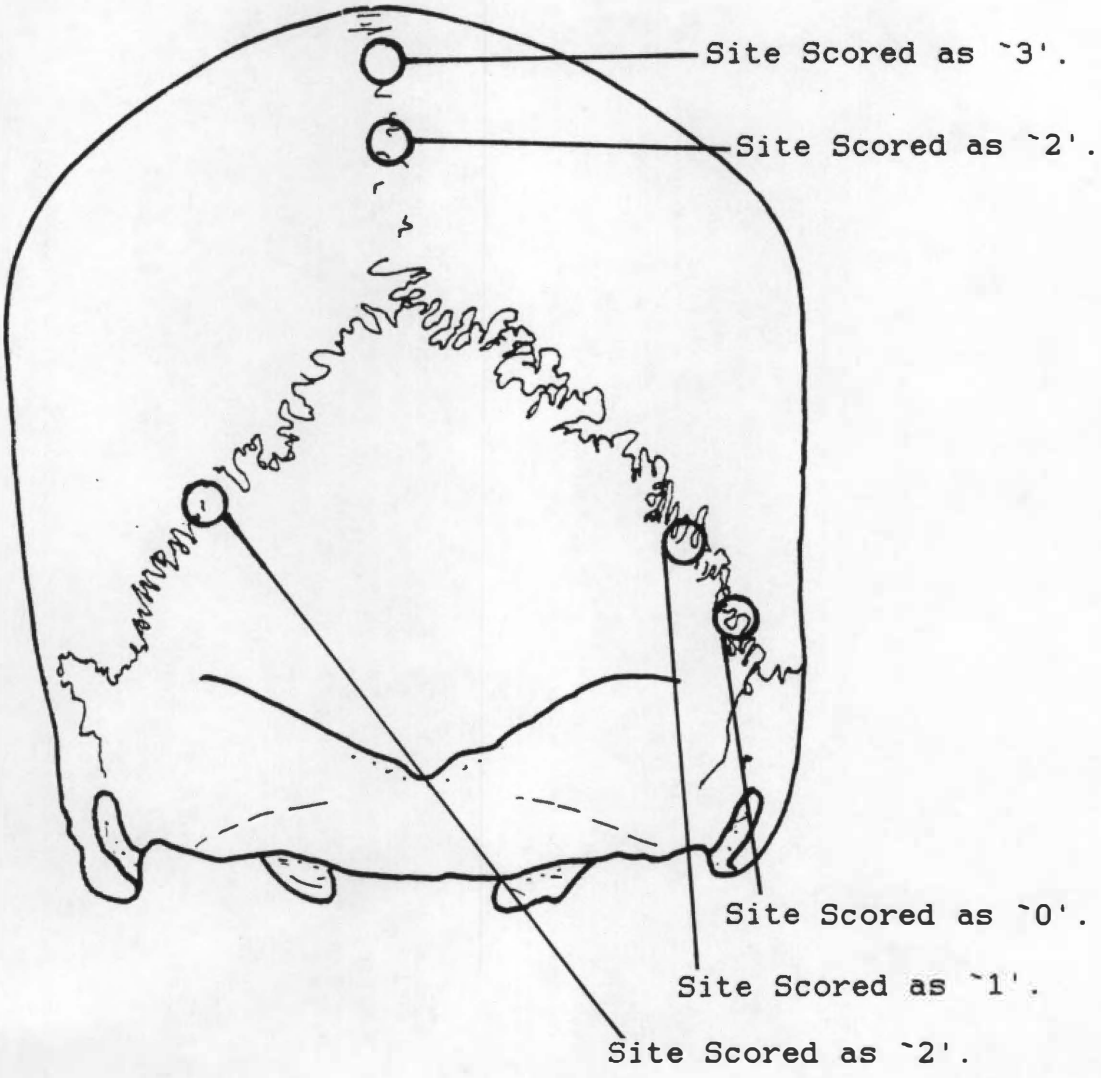


Figure 8.

Examples of Applied Suture Measurements

Following Meindl and Lovejoy (1985), sutures were measured according to conditions found in a one centimeter area of each study site described in Chapter VII. In cases where extrasutural bones were encountered, observations were made on all sides and averaged. To reduce the potential for biases from rigidly scoring sutures in the same order, suture scoring starting points were varied.

CHAPTER IX.

STATISTICAL ANALYSIS

1. The Research Hypothesis

In the post-adolescent human, there is considerable evidence that the sutural margin's composition changes from mostly soft connective tissue to bone. Research traditionally considers this process by viewing ossification as an age related drive originating mostly from within the bone's own physiological system. If the cranial capsule serves to isolate stresses to localized regions of the cranium, it would be anticipated that the structural composition of the capsule would vary according to environmental demands. This concept views the impetus for suture closure as a response to changes occurring primarily outside the capsular environment. The research hypothesis states that, with age controlled, differences in environmental affect on suture morphology, will be detected as both highly and poorly correlated cranial regions.

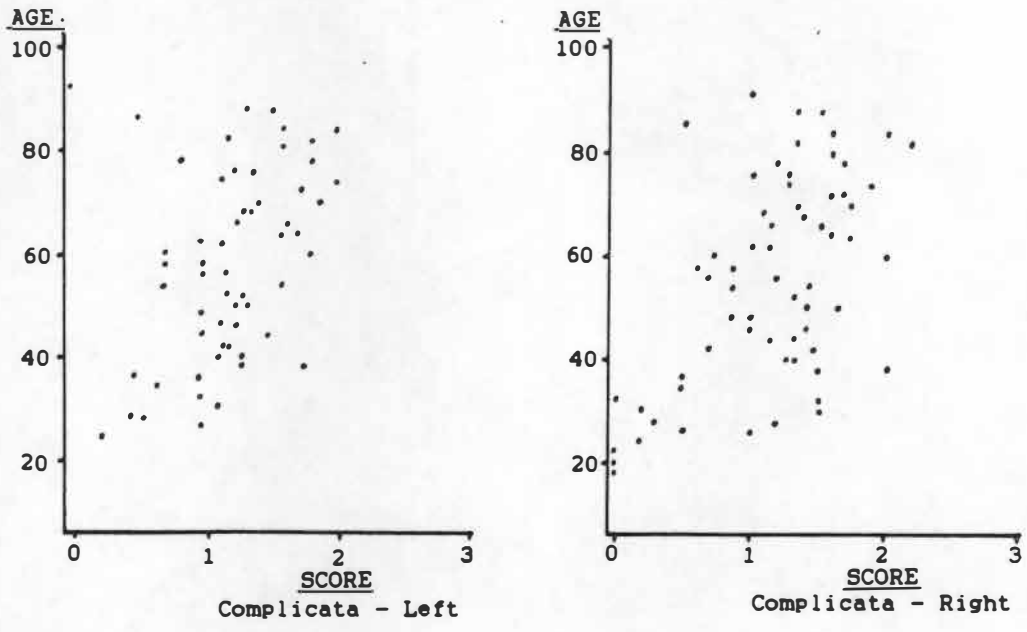
To test this hypothesis, data drawn from modern human ectocranial and endocranial suture sites are examined to learn how much osseous tissue is present in the suture gap. The data generated consists of linear (age), ordinal (tissue concentration scores) and categorical data (suture site). Confirming the capsule's influence on suture morphology requires that the parameters of tissue transformation be identified and that major forms of external variation be controlled. Each of these forms of data are critical to the results obtained from testing the research hypothesis.

2. Visual Inspection of the Data

The data collected in this project is similar to that from other related investigations, but alterations in scoring and study sample are potential sources of spurious variation. Verification of the relationship between age and suture morphology insures that data collection and the study population follow patterns suggested by previous researchers. Visual inspection of the age and score relationships for each observation site are accomplished by plotting a mean suture score for each age sample. In general, the scattered distribution and relatively large sample size obscures

any visible differences brought on by sexual dimorphism; for the purpose of this analysis, samples are pooled to clarify major trends in tissue content. Ectocranial sutures tend to follow a positive linear relationship with age.

There are numerous variations observable in the distribution between different suture locales. The clearest, strongest relationship between morphology and age is observed in sites along the coronal suture (Figure 9). Suture scores tend to fall into a fairly narrow range of variation, increasing gradually and systematically with age. In contrast, the sagittal suture exhibits the least evidence for a relationship with age (Figure 10). While increases in age can be correlated with advancement of osseous tissues, the range of variation is very wide. Plots of bregma and vertica strongly suggest that the relationship with age is curvilinear; sutures in this region gradually increase in bone content, terminating with incomplete closure at about age 35. Posterior sagittal sutures reflect similar distributions to those in the medial lamdoid surfaces. Lambdoidal sutures show a very strong positive relationship with age, however, tissue transformation appears to terminate when the site is about half closed (Figure 11). All bilateral observations show an extremely high degree of



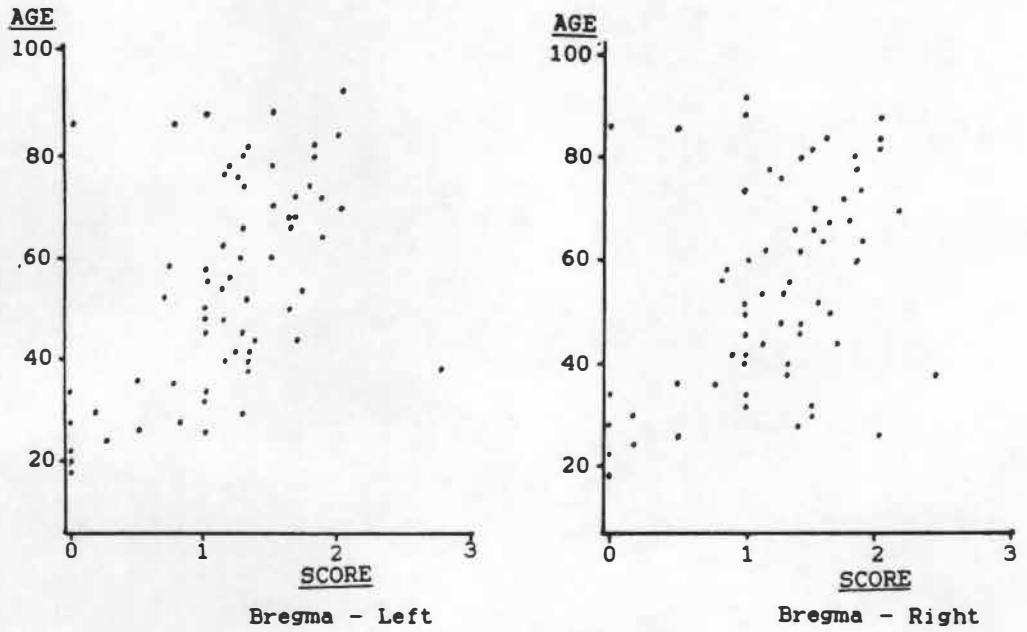


Figure 9.

The Distribution of Mean Ectocranial Suture Scores, by Age Group, for the Coronal Suture.

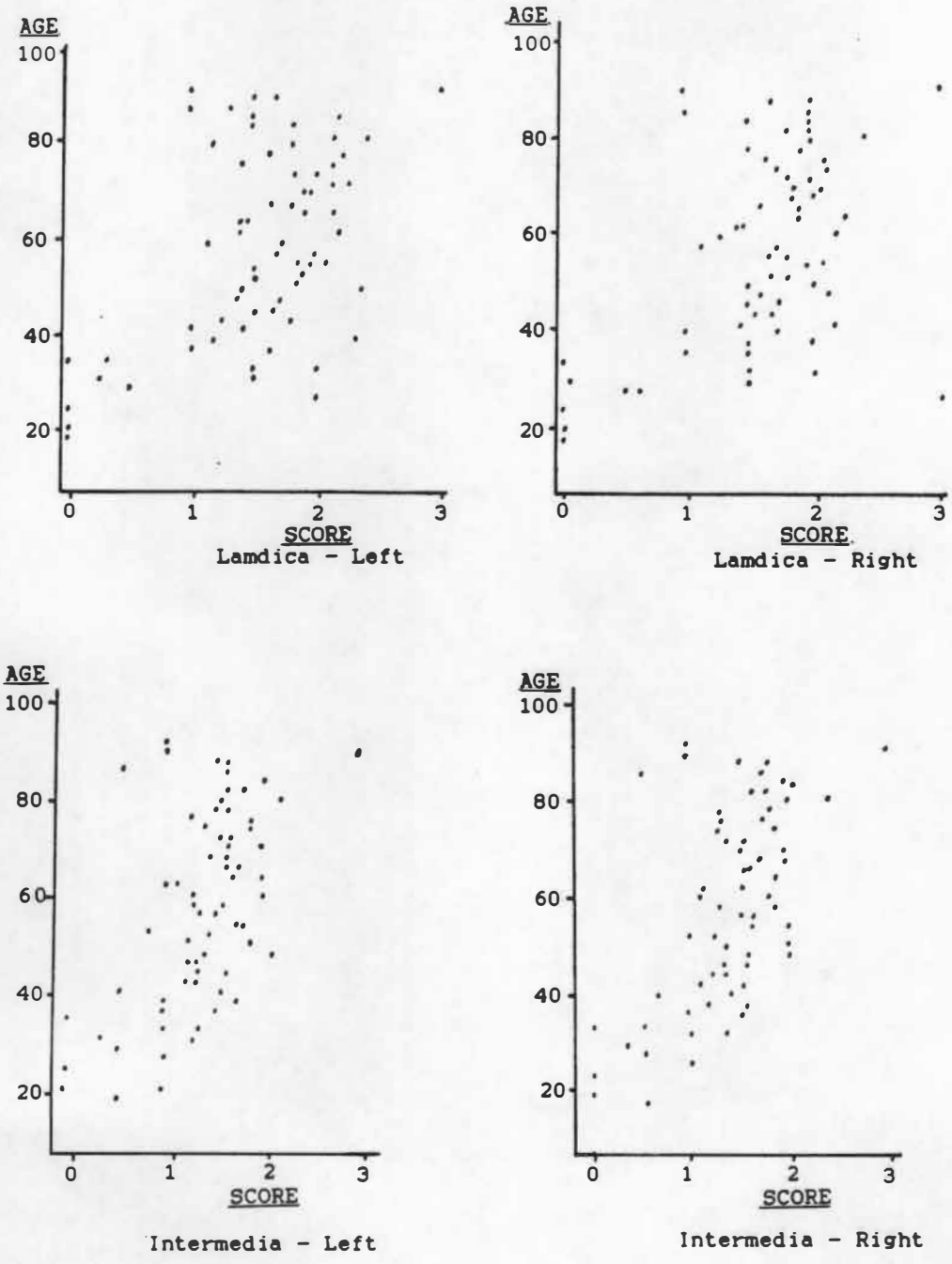


Figure 10.

The Distribution of Mean Ectocranial Suture Scores, by Age Group, for the Sagittal Suture.

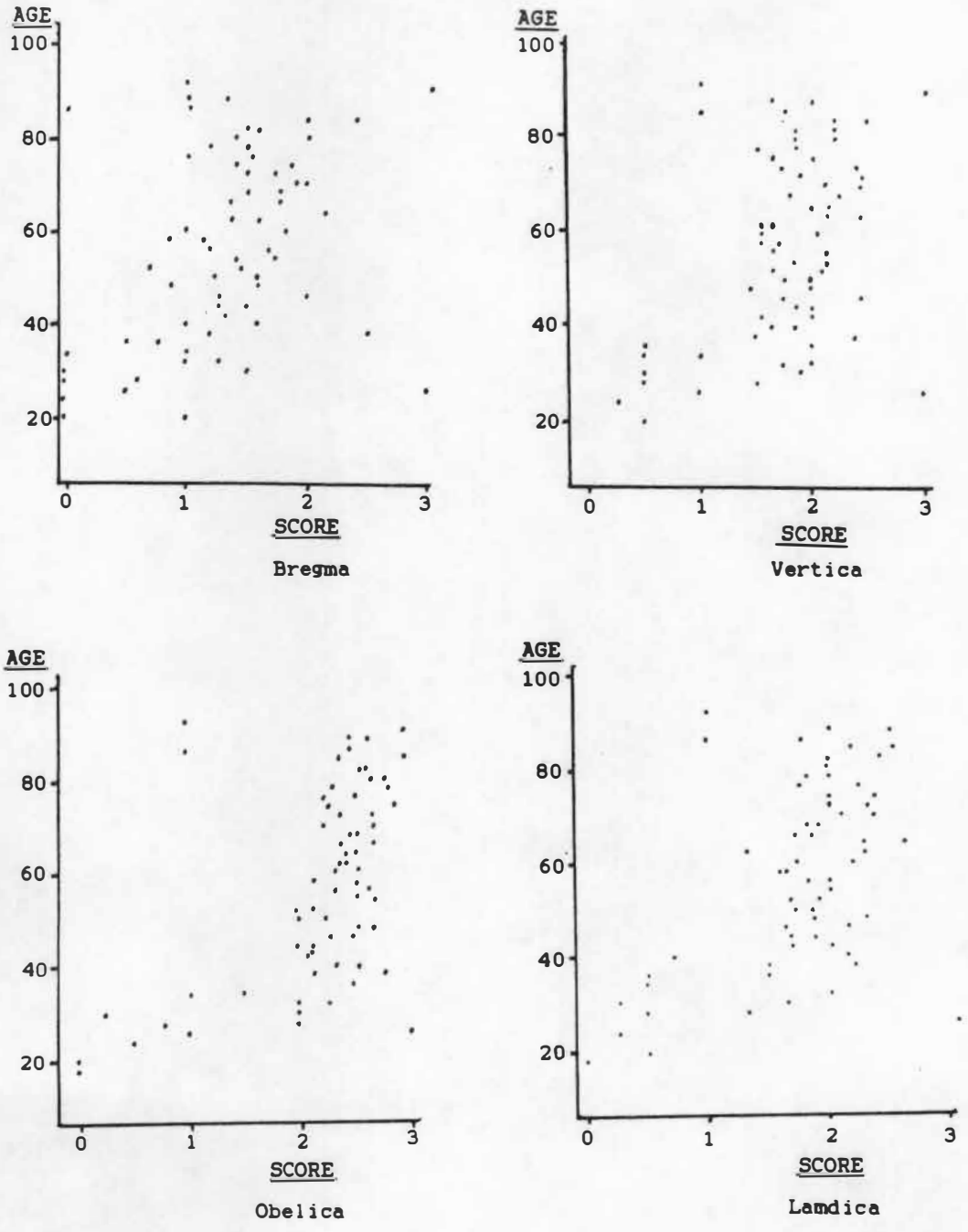
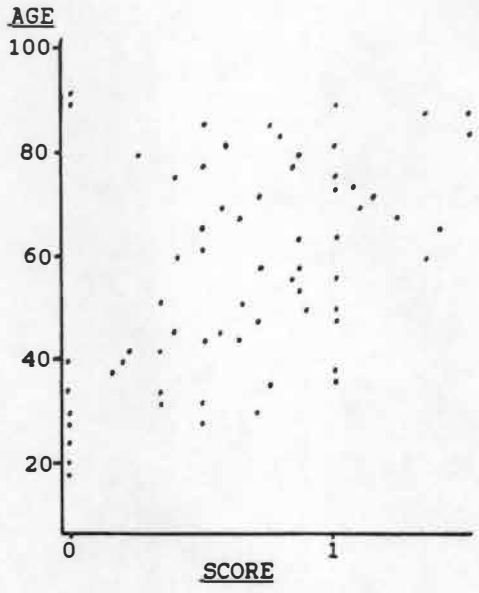
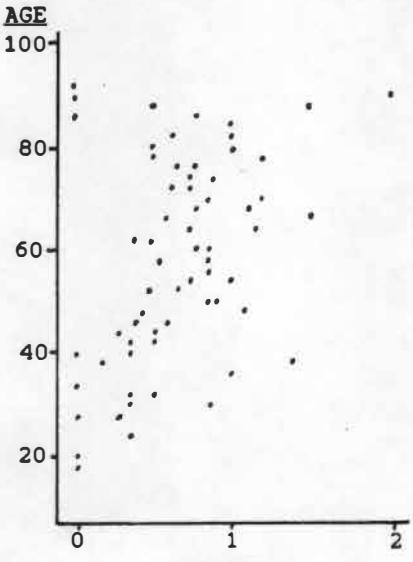


Figure 11.

The Distribution of Mean Ectocranial Suture Scores, by Age Group, for the Lambdoidal Suture.



Asterion - Left



Asterion - Right

Figure 11 (continued)

similarity. The only exception to this are the asterionic sites; while the left shows a considerable amount of variation with age, the right exhibits a distinctly narrow scatter.

Endocranial sutures do not possess as straight-forward a relationship with age. Generally, the accumulation of osseous tissue is gradual with a marked increase occurring in the 25 to 40 year. This pattern suggests the relationship is as linear as the ectocranial sample. Obliteration of non-osseous connective tissue in the sutural margin is much more pronounced than observed in the ectocranial surface. Internal surfaces tend to exhibit less variation than seen in their external counterparts. Observation of the coronal and sagittal bregmatic sites are virtually identical (Figures 12-13). All of these sites have narrow ranges of variation with complete loss of soft tissue occurring around age 40. While following the same positive relationship with age, the distribution among other sagittal sutures is more scattered. This pattern is consistant among the lamdoid sutures (Figure 14). After about age 45, the range of lambdoid closure varies considerably. Complete ossification is not as nearly pronounced among older adults as observed in more anterior sutures. With an increase in age, there tended to be a decrease in the amount of ossification.

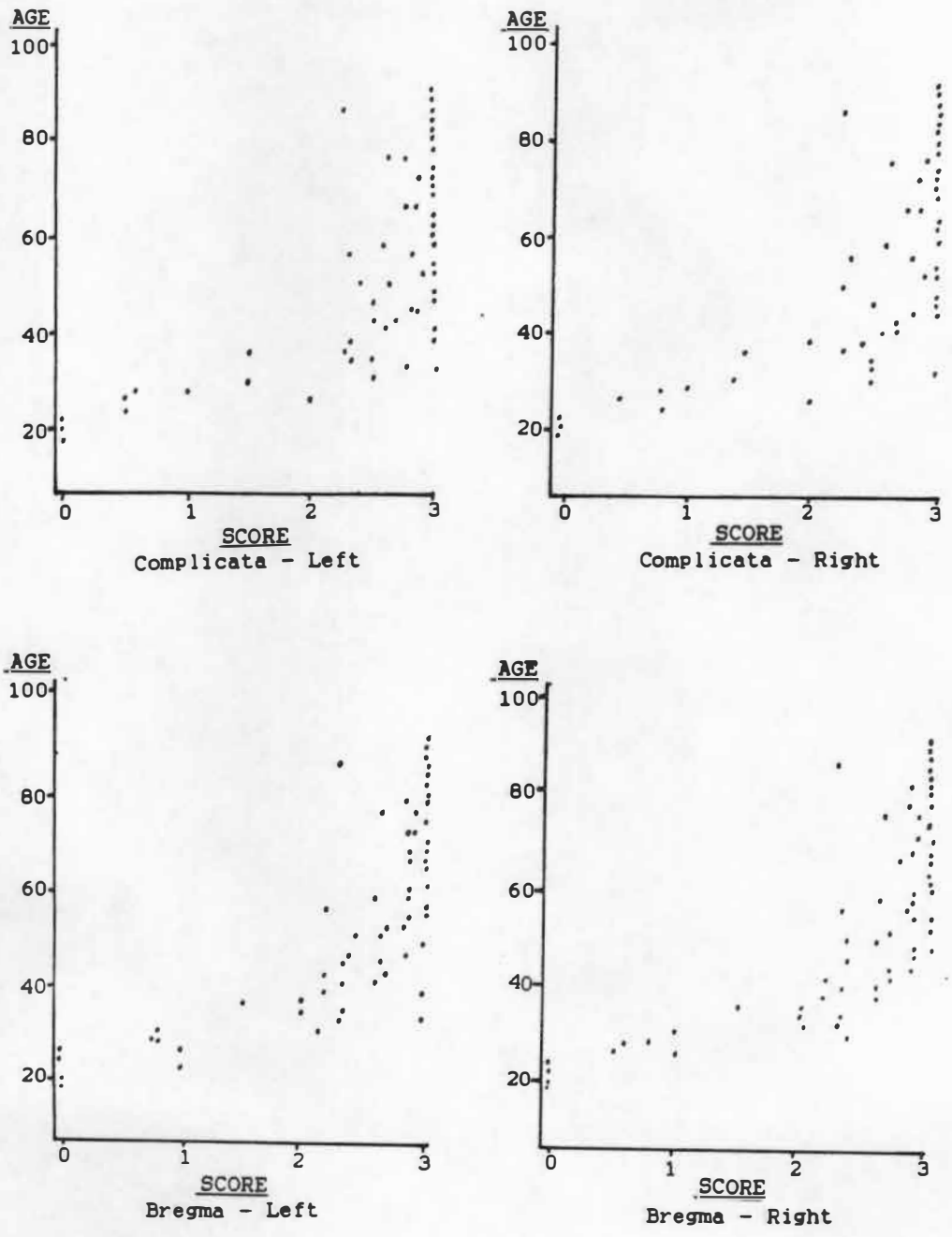


Figure 12.

The Distribution of Mean Endocranial Suture Scores, by Age Group for the Coronal Suture

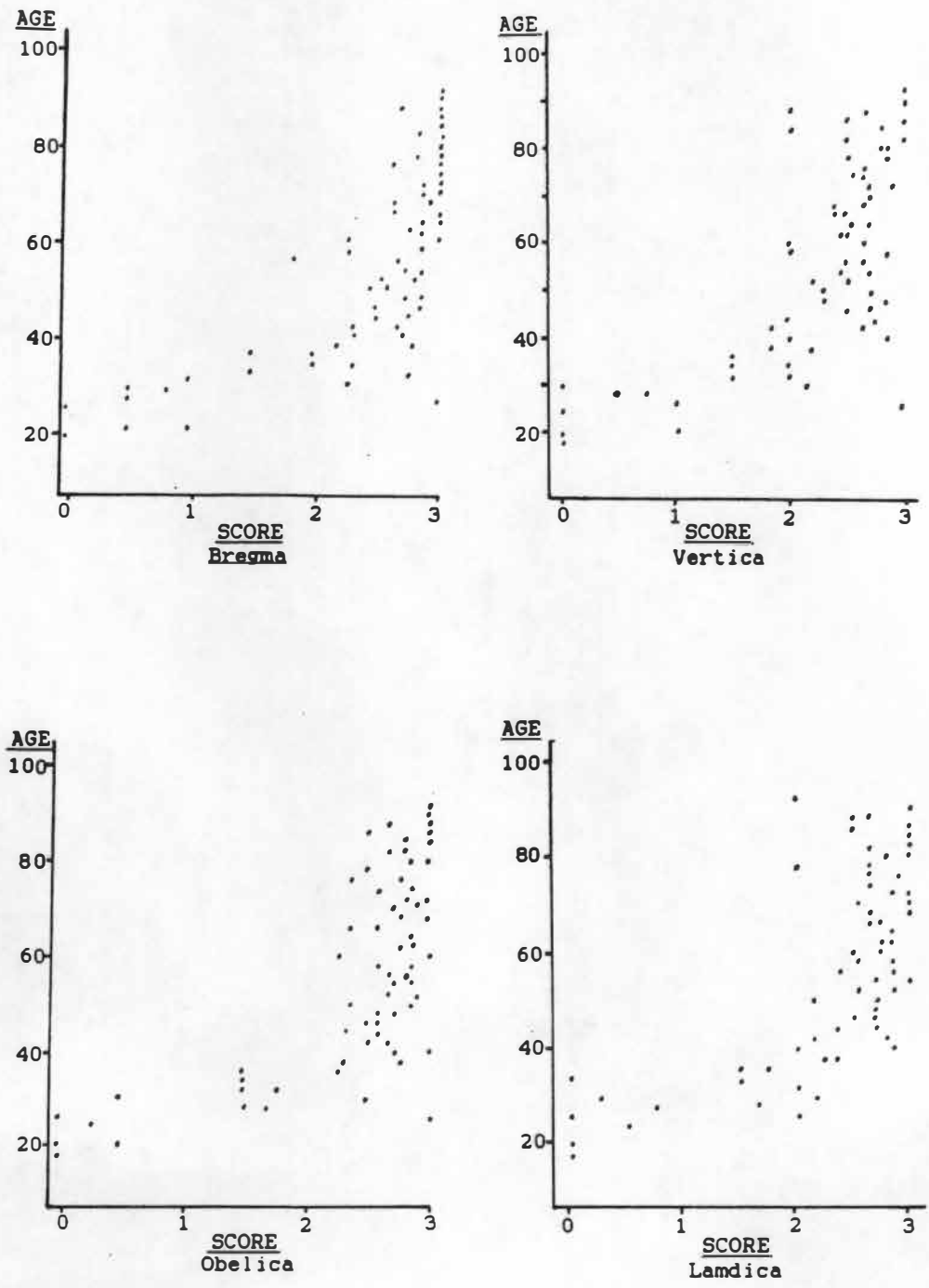


Figure 13.

The Distribution of Mean Endocranial Suture Scores, by Age Group, for the Sagittal Suture

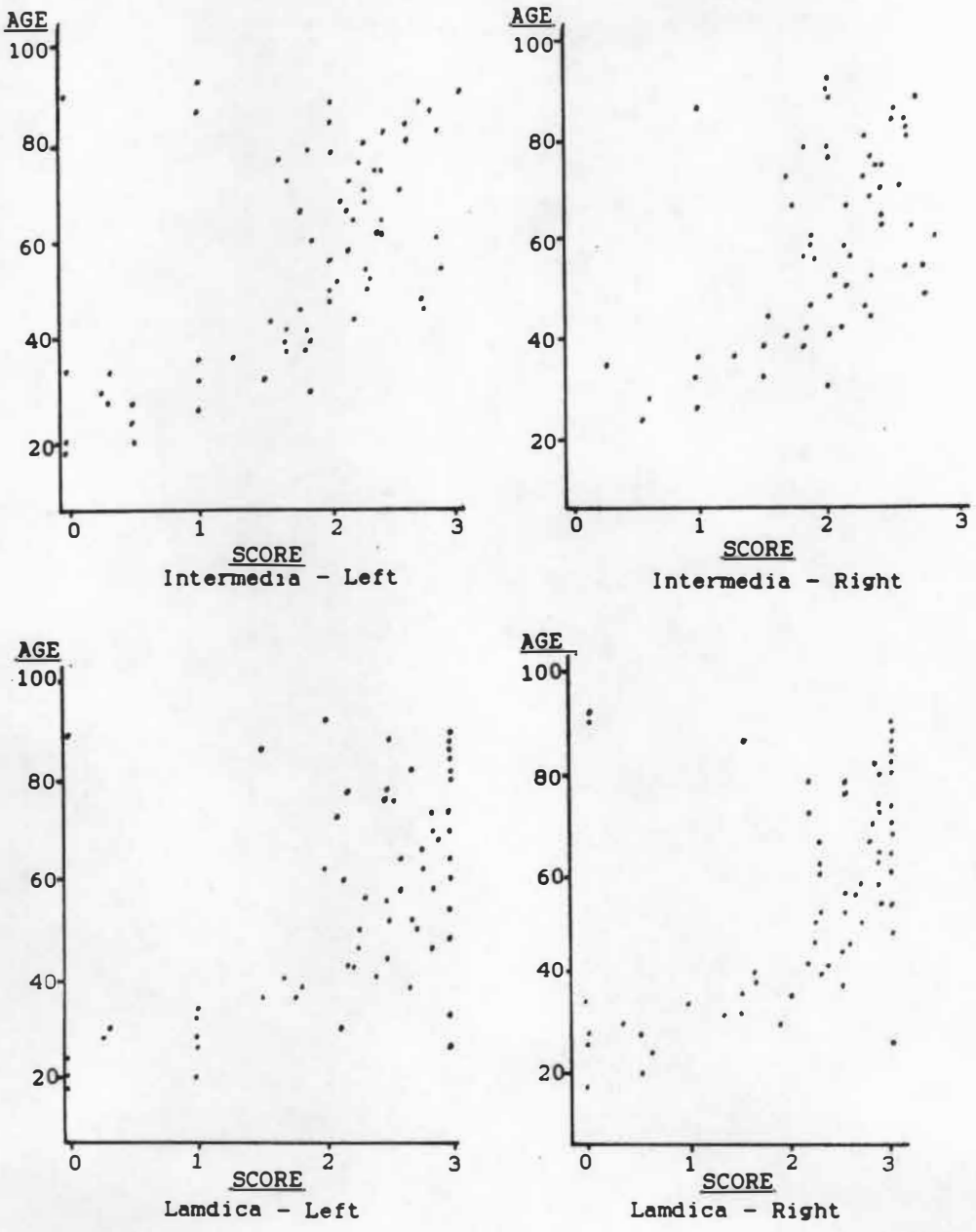
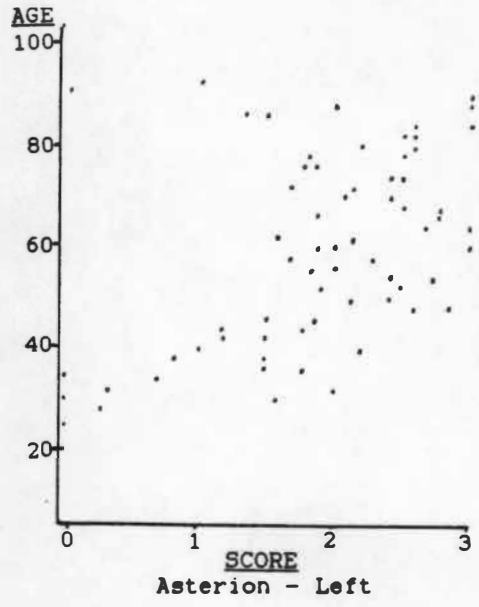


Figure 14.

The Distribution of Mean Endocranial Suture Scores, by Age Group, for the Lambdoidal Suture.



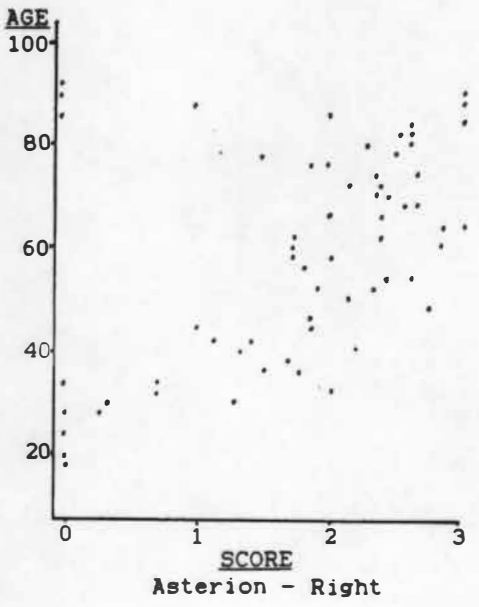


Figure 14 (continued)

This is extremely pronounced in the asterionic suture sites. Perizonius (1984) observed a similar phenomena in Danish crania and has suggested that the retention of suture tissue may be related to longevity. Bilaterally, all endocranial sutures appear to have the same distribution.

It appears that age and suture ossification possess the same positive relationships observed in other populations. Ranges of distribution fell roughly within the established physiological parameters. The details of the age-morphology interaction are unique to each suture site, particularly when comparing endocranial and ectocranial aspects.

The endocranial suture structures show a great deal of similarity. Slight differences, particularly in the `middle age and older' portions of the sample, are observeable between anterior and posterior sutures. Ectocranially, variance in ventral-dorsal distribution is much more pronounced. These observations suggest that differences in location on a particular cranial plane may have an affect on tissue transformation. Neither surface shows a great deal of variance between bilateral sutures. Compelling evidence for the capsular model can be obtained from simple observation of the data, but this visual inspection is not capable of approximating the strength of the relationship with

age or demonstrating the model's validity. More sophisticated statistical treatments are employed to further address these issues.

3. Regression Analysis of the Data

Surveying the data's form is best accomplished by treating the catagorical data (suture sites) as independent observations. This process allows an idea of what activity occurs at each discrete locale to be obtained before combining the data into larger common units. Visual inspection suggests that tissue transformation in the sutural margin is a function of age.

The capsular model, M=(IA)+(OA)+(BA)+E, assumes that independent functional relationships exist between the driving influence and age. The data available for statistically verifying this information are in the form of interval (age) and ordinal (suture morphology) data; the use of regression analysis is appropriate for verifying a functional relationship between age and morphology (Ott 1988; Zar 1984). Each endocranial and ectocranial measurement is independently tested with age (e.g. Icbr=Age or Ocbr=Age). It is necessary to verify whether age follows a linear, curvilinear, or no relationship with the degree of suture closure. Gender influence on morphology, with and without a curvilinear

age interaction, is tested to learn if these variables need statistical control. The affect of differences on museum collection have been observed by Perizonius (1984); identification and prevention of this confounding feature can be accomplished by using multiple regression. The research hypothesis in this analysis notes that age, sex, their interaction and sample unit (museum collection) have an affect on suture morphology. The Null Hypothesis states that these features will possess no relationship. The University of Tennessee's SAS package includes a PROC GLM (General Linear Models) procedure (SAS, Inc 1985). This program is the source of the presented regression results.

Modeling the amount of osseous tissue present in ectocranial sites produced statistically significant F values (Table 1). Testing to see whether the Armed Forces Institute and Terry Collections influence the data indicate that collections are not a significant contributor to the variation. On this cranial surface, suture morphology does not show a strong relationship with sex, nor could either Type I or III regression results substantiate any significant interactions with linear or curvilinear age models (Tables 2-5). Both simple linear and quadratic expressions of age identify a positive relationship with suture

Table 1.

F Values from Multiple Regression Tests

Suture Site	'F' Statistic	Probability of 'F'
ICBRR	58.70	0.0001
ICBRL	55.39	0.0001
ICCOR	40.95	0.0001
ICCOL	43.08	0.0001
ISBR	36.64	0.0001
ISVE	32.55	0.0001
ISOB	23.03	0.0001
ISLA	41.42	0.0001
ILLAR	23.73	0.0001
ILLAL	21.76	0.0001
ILINR	31.74	0.0001
ILINL	28.42	0.0001
ILASR	19.17	0.0001
ILASL	17.34	0.0001
OCBRR	7.76	0.0001
OCBRL	7.47	0.0001
OCCOR	7.80	0.0001
OCCOL	11.44	0.0001
OSBR	8.05	0.0001
OSVE	10.95	0.0001
OSOB	24.57	0.0001
OSLA	14.39	0.0001
OLLAR	12.82	0.0001
OLLAL	13.32	0.0001
OLINR	10.93	0.0001
OLINL	9.26	0.0001
OLASR	3.80	0.0001
OLASL	5.98	0.0001

Table 2.

Results of Multiple Regression Tests

Against Suture Influence (Type II)

Suture Site	Sex	Age	Age Squared	Age/Sex
ICBRR	0.08	179.79	108.96	1.00
	(0.7758)*	(0.0001)	(0.0001)	(0.3187)*
ICBRL	0.83	173.93	100.49	0.21
	(0.3641)*	(0.0001)*	(0.0001)	(0.6434)*
ICCOR	0.02	122.93	79.32	0.62
	(0.8796)*	(0.0001)	(0.0001)	(0.4306)*
ICCOL	0.03	122.22	97.31	0.77
	(0.8693)*	(0.0001)	(0.0001)	(0.3821)*
ISBR	0.12	131.22	48.17	2.70
	(0.7328)*	(0.0001)	(0.0001)	(0.1012)*
ISVE	1.62	106.33	50.82	2.99
	(0.2039)*	(0.0001)	(0.0001)	(0.0844)*
ISOB	4.64	70.57	59.21	9.31**
	(0.0319)*	(0.0001)*	(0.0001)	(0.0024)
ISLA	0.04	116.60	76.20	14.08**
	(0.8415)*	(0.0001)	(0.0001)	(0.0002)
ILLAR	1.34	70.51	45.76	0.69
	(0.2476)*	(0.0001)	(0.0001)	(0.4079)*
ILLAL	4.33	63.28	38.12	2.68
	(0.0381)	(0.0001)	(0.0001)	(0.1026)*
ILINR	11.07	87.47	59.02	1.07
	(0.0001)	(0.0001)	(0.0001)	(0.3025)*
ILINL	13.58	78.42	47.89	2.11
	(0.0003)	(0.0001)	(0.0001)	(0.1467)*
ILASR	15.06	53.26	26.68	0.24
	(0.0001)	(0.0001)	(0.0001)	(0.6258)*
ILASL	13.48	48.65	24.56	0.01
	(0.0003)	(0.0001)	(0.0001)	(0.9205)*
OCBRR	1.09	23.73	4.48	9.40**
	(0.2964)*	(0.0001)	(0.0348)	(0.0023)
OCBRL	1.73	22.44	6.32	6.81**
	(0.1887)*	(0.0001)	(0.0123)	(0.0094)
OCCOR	12.73	15.55	2.36	8.23**
	(0.0004)	(0.0001)	(0.1250)*	(0.0044)

Table 2 (continued)

Suture Site	Sex	Age	Age Squared	Age/Sex
OCCOL	14.74	22.39	7.00	12.70**
	(0.0001)	(0.0001)	(0.0085)	(0.0004)
OSBR	0.11	22.17	8.57	8.60**
	(0.7394)*	(0.0001)	(0.0036)	(0.0036)
OSVE	1.49	26.47	17.25	9.06**
	(0.2236)*	(0.0001)	(0.0001)	(0.0028)
OSOB	24.13	65.10	27.97	5.25**
	(0.0001)	(0.0001)	(0.0001)	(0.0225)
OSLA	6.90	42.72	12.01	9.28**
	(0.0090)	(0.0001)	(0.0006)	(0.0025)
OLLAR	4.43	39.84	18.89	0.92
	(0.0360)	(0.0001)	(0.0001)	(0.3380)*
OLLAL	4.60	38.17	22.92	0.62
	(0.0326)	(0.0001)	(0.0001)	(0.4320)*
OLINR	0.50	39.84	12.11	1.97
	(0.4812)*	(0.0001)	(0.0006)	(0.1615)*
OLINL	0.36	32.66	10.01	2.69
	(0.5492)*	(0.0001)	(0.0017)	(0.1016)*
OLASR	5.07	9.89	2.67	1.14
	(0.0249)	(0.0018)	(0.1032)*	(0.2855)*
OLASL	4.23	14.16	5.26	6.11
	(0.0405)	(0.0002)	(0.0224)	(0.0139)*

(0.001) - Indicates the probability of 'F'.
* - Indicates a nonsignificant F Value.
** - Indicates a Sum of Squares of Less than Means Square - Error Value.

Table 3.

Results of Multiple Regresson Tests Against Suture Influence (Type II): Age Square/Sex Interactions

Suture Site	Age Square/Sex	Suture Site	Age Square/Sex
ICBRR	3.68 (0.0557)*	OCBRR	0.10 (0.7466)*
ICBRL	1.48 (0.2246)*	OCBRL	0.04 (0.8513)*
ICCOR	1.87 (0.1717)*	OCCOR	0.12 (0.7275)*
ICCOL	1.07 (0.3006)*	OCCOL	0.36 (0.5464)*
ISBR	1.00 (0.3174)*	OSBR	0.81 (0.3691)*
ISVE	0.98 (0.3237)*	OSVE	0.49 (0.4826)*
ISOB	1.45 (0.2301)*	OSOB	0.42 (0.5179)*
ISLA	0.17 (0.6832)*	OSLA	1.03 (0.3099)*
ILLAR	0.33 (0.5634)*	OLLAR	0.00 (0.9664)*
ILLAL	0.37 (0.5421)	OLLAL	0.32 (0.5749)*
ILINR	0.08 (0.7759)*	OLINR	0.25 (0.6196)*
ILINL	0.12 (0.7312)*	OLINL	0.56 (0.4539)*
ILASR	0.62 (0.4331)*	OLASR	0.24 (0.6214)*
ILASL	0.01 (0.9182)*	OLASL	0.17 (0.6818)*

(0.001) - Indicates the probability of 'F'.
* - Indicates a nonsignificant F Value.
** - Indicates a Sum of Squares of Less than Means Square - Error Value.

Table 4.

Results of Multiple Regression Tests

Against Suture Influences (Type III)

Suture Site	Sex	Age	Age Squared	Age/Sex
ICBRR	1.82	98.35	74.07	3.04
	(0.181)*	(0.0001)	(0.0001)	(0.0820)*
ICBRL	0.86	95.12	71.15	1.28
	(0.3541)*	(0.0001)	(0.0001)	(0.2586)*
ICCOR	0.80	74.28	56.66	1.52
	(0.3706)*	(0.0001)	(0.0001)	(0.2182)*
ICCOL	0.31	86.68	67.63	0.79
	(0.5754)*	(0.0001)	(0.0001)	(0.3737)*
ISBR	0.10	54.93	37.43	0.55
	(0.7552)*	(0.0001)	(0.0001)	(0.4594)*
ISVE	0.14	55.90	4.10	0.51
	(0.7118)*	(0.0001)	(0.0001)	(0.4768)*
ISOB	0.06	64.29	50.33	0.52
	(0.8018)*	(0.0001)	(0.0001)	(0.4710)*
ISLA	2.86	102.29	77.80	0.95
	(0.0916)*	(0.0001)	(0.0001)	(0.3291)*
ILLAR	0.01	47.49	35.98	0.20
	(0.9235)*	(0.0001)	(0.0001)	(0.6565)*
ILLAL	1.87	49.41	37.25	0.73
	(0.1721)*	(0.0001)	(0.0001)	(0.3937)*
ILINR	0.91	69.75	53.12	0.19
	(0.3397)*	(0.0001)	(0.0001)	(0.6619)*
ILINL	0.28	55.54	41.40	0.01
	(0.5949)*	(0.0001)	(0.0001)	(0.4823)*
ILASR	0.91	33.53	24.32	0.49
	(0.3402)*	(0.0001)	(0.0001)	(0.4823)*
ILASL	0.08	29.60	21.34	0.01
	(0.7793)*	(0.0001)	(0.0001)	(0.9309)*
OCBRR	1.63	12.36	7.85	0.62
	(0.2024)*	(0.0005)	(0.0053)	(0.4310)*
OCBRL	0.94	13.14	8.82	0.34
	(0.3335)*	(0.0003)	(0.0032)	(0.5596)*
OCCOR	0.99	8.16	5.06	0.61
	(0.3194)*	(0.0045)	(0.0251)	(0.4356)*

Table 4 (continued)

Suture Site	Sex	Age	Age Squared	Age/Sex
OCCOL	2.09	17.73	12.26	1.30
	(0.1489)*	(0.0001)	(0.0005)	(0.2547)*
OSBR	3.33	19.14	13.67	1.78
	(0.0688)*	(0.0001)	(0.0002)	(0.1826)*
OSVE	0.23	22.89	17.20	0.05
	(0.6319)*	(0.0001)	(0.0001)	(0.8147)*
OSOB	0.41	36.16	25.42	0.09
	(0.5207)*	(0.0001)	(0.0001)	(0.7704)*
OSLA	0.11	18.80	12.08	0.30
	(0.7453)*	(0.0001)	(0.0006)	(0.5873)*
OLLAR	0.27	23.42	16.85	0.01
	(0.6064)*	(0.0001)	(0.0001)	(0.9172)*
OLLAL	0.00	23.81	17.76	0.19
	(0.9795)*	(0.0001)	(0.0001)	(0.6642)*
OLINR	0.84	19.96	13.33	0.49
	(0.3596)*	(0.0001)	(0.0003)	(0.4827)*
OLINL	1.52	18.28	12.30	0.98
	(0.2181)*	(0.0001)	(0.0005)	(0.3231*
OLASR	0.40	5.44	3.58	0.42
	(0.5253)*	(0.0202)	(0.0592)*	(0.5174)*
OLASL	0.06	9.25	6.27	0.00
	(0.8012)*	(0.0025)	(0.0127)	(0.9722)*

(0.001) - Indicates the probability of 'F'.
* - Indicates a nonsignificant F Value.
** - Indicates a Sum of Squares of Less than Means Square - Error Value

Table 5.

Results of Multiple Regression Tests

Against Suture Influences (Type III):

Age Square/Sex Interactions

Suture Site	Age Square/Sex	Site	Age Square/Sex
ICBRR	3.68 (0.0577)*	OCBRR	0.10 (0.7466)*
ICBRL	1.48 (0.2246)*	OCBRL	0.04 (0.8513)*
ICCOR	1.87 (0.1717)*	OCCOR	0.12 (0.7275)*
ICCOL	1.07 (0.3006)*	OCCOL	0.36 (0.5464)*
ISBR	1.00 (0.3174)*	OSBR	0.81 (0.3691)*
ISVE	0.98 (0.3237)*	OSVE	0.49 (0.4826)*
ISOB	1.45 (0.2301)*	OSOB	0.42 (0.5179)*
ISLA	0.17 (0.6832)*	OSLA	1.03 (0.3099)*
ILLAR	0.33 (0.5634)*	OLLAR	0.00 (0.9664)*
ILLAL	0.37 (0.5421)*	OLLAL	0.32 (0.5749)*
ILINR	0.08 (0.7759)*	OLINR	0.25 (0.6196)*
ILINL	0.12 (0.7312)*	OLINL	0.56 (0.4539)*
ILASR	0.62 (0.4331)*	OLASR	0.24 (0.6214)*
ILASL	0.01 (0.9182)*	OLASL	0.17 (0.6818)*

(0.0001) - Indicates the Probability of 'F'.
* - Indicates a nonsignificant F Value.

morphology. Endocranial suture regression produces significant F Values that are at least twice that of their ectocranial counterparts (See Table 1). lationships with collection, sex and sex interactions ith age all indicate non-significant contributions to tissue morphology. Linear and curvilinear age values are significant with morphology, but linear F Values are higher than the curvilinear form. Consistantly, F Values are stronger for the endocranial relationship than those obtained from the ectocranial surface.

From the multiple regression models, it can be determined that the effects of sex and collection are not critical to the morphology expressed in this study sample. Pooling collections and sexes can be assumed not to skew the sample's variation.

4. Spearman Partial Correlation Analysis

Given that positive, variable relationships can be established between age and morphology at different suture sites, if the source of variation is in the tissue environment (for example neural versus scalp/muscular, coronal versus sagittal, or right versus left), the source for this disparity can be implied by correlating surface relationships while controlling for age. Unlike the variables used in the age-suture regression, identifying relationships

between suture sites does not involve interval data. Several analytical methods are capable of taking advantage of the non-independent relationship present in the ordinal suture scores, the most powerful is correlation analysis (Thomas 1986). Application of the Kolomogrov D statistic to each suture site's distribution illustrate that the lack of normality is significant to at least the 0.0001 level (Table 6). Without the ability to assume what distribution form is present, only Rank Correlation Analysis can be used (Ott 1988; Zar 1984). The Spearman Rank Correlation Coefficient (Rho) compares the overall similarity between two sets of ranks and expresses this relationship as a measurement between +1 (A perfect positive correlation) and -1 (A perfect negative correlation), (Siegel 1956; Thomas 1986). Results from the Spearman Rank Correlation reflect true distributions more than those obtained from Kendall's Tau and the method of computation enables a probability for Rho to be developed (Chow, et al. 1974; Thomas 1986). The affect of age on suture morphology is controlled by applying Cholesky's Decomposition Algorithm to the Spearman Equations (SAS, Inc. 1990). This partial correlation statistically controls age's influence so that the variation measured was only that between samples (Siegel 1956). The resulting matrix of

Table 6.

Normality Distribution of Suture Scores by Site

Suture Site	Kolmogrov D Statistic	Probability
ICBRR	0.483562	0.001
ICBRL	0.475093	0.001
ICCOR	0.417146	0.001
ICCOL	0.412296	0.001
ISBR	0.527191	0.001
ISVE	0.687946	0.001
ISOB	0.56088	0.001
ISLA	0.575697	0.001
ILLAR	0.790568	0.001
ILLAL	0.801163	0.001
ILINR	0.553539	0.001
ILINL	0.556738	0.001
ILASR	0.710554	0.001
ILASL	0.695978	0.001
OCBRR	0.846749	0.001
OCBRL	0.845448	0.001
OCCOR	0.841955	0.001
OCCOL	0.843115	0.001
OSBR	0.847256	0.001
OSVE	0.841720	0.001
OSOB	0.712085	0.001
OSLA	0.841146	0.001
OLLAR	0.854048	0.001
OLLAL	0.856303	0.001
OLINR	0.853492	0.001
OLINL	0.852944	0.001
OLASR	0.763029	0.001
OLASL	0.766055	0.001

Rho correlation values identify how suture sites vary with other sites (Tables 7-9).*

Despite the removal of age, morphological similarity between several suture sites can be clearly ascertained. Correlations expressing a value of greater than or equal to 0.70 are defined as strong positive relationships. The most striking pattern is between bilateral observations. There were no exceptions; all sutures from the left correlate extremely well with their complement on the right, representing the largest values in the matrix. Another interesting observation is among the anterior endocranial sutures. Variation in the coronal suture morphology appears to respond as a unified element. The sagittal bregma changes respond in much the same manner as its coronal counterpart, but its tissue is dissimilar to the complicata sites. Bregma, regardless of which suture is involved operates as a unit. Contrasting these endocranial observations, the anterior ectocranial sutures exhibit only bilateral associations. The posterior ectocranial sagittal

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* Spearman Rho Partial Correlations were produced only on crania possessing all measurements. The sample size for this matrix is 345 individuals.

Table 7.

Spearman Rho Partial Correlation Matrix

Endocranial Sutures By Endocranial Sutures

	ICBRR	ICBRL	ICCOR	ICCOL	ISBR	ISVE	ISOB	ISLA	ILLAR	ILLAL	ILINR	ILINL	ILASR	ILASL
ICBRR	1.0	0.9392	0.7930	0.7522	0.7691	0.4817	0.3971	0.4959	0.4040	0.3939	0.4449	0.4502	0.3547	0.3312
	0.0	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001
ICBRL	1.0		0.7780	0.7657	0.7467	0.4690	0.3967	0.4925	0.3889	0.3822	0.4527	0.4585	0.3685	0.3447
	0.0		0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001
ICCOR	1.0			0.9560	0.6154	0.4165	0.3218	0.4078	0.3472	0.3520	0.4873	0.4757	0.3879	0.3717
	0.0			0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001
ICCOL	1.0				0.5961	0.4020	0.3349	0.3888	0.3336	0.3423	0.4692	0.4580	0.3852	0.3635
	0.0				0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001
ISBR	1.0					0.5980	0.3935	0.4887	0.3761	0.3876	0.3851	0.3851	0.3212	0.3016
	0.0					0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001
ISVE	1.0						0.3923	0.5603	0.3936	0.3849	0.2923	0.2983	0.2543	0.1916
	0.0						0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0003
ISOB	1.0							0.5185	0.2923	0.3086	0.2194	0.2274	0.1978	0.1613
	0.0							0.0001	0.0001	0.0001	0.0001	0.0001	0.0002	0.0027
ISLA	1.0								0.4663	0.4692	0.3868	0.3950	0.3193	0.2876
	0.0								0.0001	0.0001	0.0001	0.0001	0.0001	0.0001
ILLAR	1.0									0.8652	0.6024	0.5520	0.4798	0.4163
	0.0									0.0001	0.0001	0.0001	0.0001	0.0001
ILLAL	1.0										0.6271	0.5969	0.5020	0.4435
	0.0										0.0001	0.0001	0.0001	0.0001
ILINR	1.0											0.9013	0.6891	0.6568
	0.0											0.0001	0.0001	0.0001
ILINL	1.0												0.6710	0.6868
	0.0												0.0001	0.0001
ILASR	1.0													0.8773
	0.0													0.0001
ILASL	1.0													
	0.0													

Table 8.

Spearman Rho Partial Correlation Matrix:

Endocranial by Ectocranial Sutures

	OCBRR	OCBRL	OCCOR	OCCOL	OSBR	OSVE	OSOB	OSLA	OLLAR	OLLAL	OLINR	OLINL	OLASR	OLASL
ICBRR	0.3136	0.3412	0.3369	0.3217	0.3684	0.3330	0.3591	0.4009	0.3722	0.3943	0.3628	0.3075	0.1526	0.1974
	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0045	0.0002
ICBRL	0.3015	0.3324	0.3424	0.3216	0.3620	0.3286	0.3556	0.3713	0.3595	0.3735	0.3528	0.2980	0.1602	0.2007
	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0029	0.0002
ICCOR	0.3086	0.3295	0.2646	0.2689	0.3092	0.3002	0.3492	0.3777	0.3239	0.2960	0.3237	0.2599	0.1536	0.1817
	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0043	0.0007
ICCOL	0.3002	0.3124	0.2658	0.2827	0.2907	0.2845	0.3279	0.3581	0.3077	0.2866	0.3046	0.2282	0.1476	0.1738
	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0061	0.0012
ISBR	0.3381	0.3582	0.3635	0.3539	0.3516	0.3817	0.3729	0.4186	0.3510	0.3687	0.3519	0.3215	0.1738	0.2502
	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001
ISVE	0.2128	0.2144	0.2953	0.2249	0.3179	0.2841	0.2322	0.3309	0.3044	0.3262	0.2476	0.2327	0.1659	0.1762
	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0020	0.0010
ISOB	0.2471	0.2664	0.3437	0.2266	0.2614	0.2481	0.2810	0.2213	0.2072	0.2340	0.2138	0.1970	0.1279	0.1454
	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0002	0.0176	0.0069
ISLA	0.2310	0.2282	0.2757	0.2131	0.2894	0.3155	0.3051	0.3648	0.3494	0.3362	0.3044	0.2799	0.3059	0.2259
	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001
ILLAR	0.2042	0.2230	0.2737	0.2233	0.2704	0.2964	0.2866	0.3339	0.5100	0.4906	0.4242	0.3855	0.2467	0.2923
	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001
ILLAL	0.2099	0.2459	0.2816	0.2739	0.3139	0.3538	0.2948	0.3614	0.5216	0.5022	0.4537	0.3858	0.2576	0.3029
	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001
ILINR	0.2060	0.2513	0.1981	0.2103	0.2842	0.3231	0.3207	0.3636	0.5118	0.4563	0.4785	0.4571	0.3544	0.4173
	0.0001	0.0001	0.0002	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001
ILINL	0.2196	0.2836	0.2249	0.2368	0.3122	0.3492	0.3152	0.3617	0.4979	0.4633	0.4557	0.4565	0.3596	0.4178
	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001
ILASR	0.2226	0.2927	0.2400	0.2349	0.3252	0.3290	0.3060	0.3705	0.4354	0.4308	0.4848	0.4898	0.3989	0.4039
	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001
ILASL	0.2188	0.3038	0.2482	0.2375	0.3031	0.3000	0.2598	0.3287	0.4017	0.3891	0.4309	0.4372	0.3829	0.4195
	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001

Table 9.

Spearman Rho Partial Correlation Matrix:

Ectocranial by Ectocranial Sutures

	OCBRR	OCBRL	OCCOR	OCCOL	OSBR	OSVE	OSOB	OSLA	OLLAR	OLLAL	OLINR	OLINL	OLASR	OLASL
OCBRR	1.0	0.8489	0.6771	0.6640	0.6885	0.5714	0.3847	0.5239	0.3955	0.3856	0.4471	0.4214	0.2958	0.2870
	0.0	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001
	OCBRL	1.0	0.6552	0.6897	0.7009	0.6007	0.4006	0.5453	0.4313	0.4204	0.4689	0.4628	0.2966	0.2886
		0.0	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001
		OCCOR	1.0	0.8146	0.5911	0.5502	0.2924	0.4580	0.3974	0.3692	0.4934	0.4672	0.3225	0.3095
			0.0	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001
			OCCOL	1.0	0.5787	0.5304	0.2720	0.4619	0.3998	0.3887	0.5100	0.4630	0.3477	0.3591
			0.0	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001
			OSBR	1.0	0.6642	0.4084	0.6018	0.4813	0.4928	0.4731	0.4969	0.3069	0.2774
				0.0	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001
				OSVE	1.0	0.5921	0.7562	0.6011	0.6212	0.5615	0.5967	0.3303	0.3076
					0.0	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001
					OSOB	1.0	0.6604	0.4884	0.5048	0.4174	0.4245	0.1919	0.1943
						0.0	0.0001	0.0001	0.0001	0.0001	0.0001	0.0003	0.0003
						OSLA	1.0	0.6370	0.6667	0.5463	0.5807	0.2910	0.2913
							0.0	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001
							OLLAR	1.0	0.8591	0.7138	0.6922	0.4460	0.4235
								0.0	0.0001	0.0001	0.0001	0.0001	0.0001
								OLLAL	1.0	0.6734	0.6907	0.3953	0.3914
									0.0	0.0001	0.0001	0.0001	0.0001
									OLINR	1.0	0.8113	0.5488	0.4983
										0.0	0.0001	0.0001	0.0001
										OLINL	1.0	0.4876	0.5062
											0.0	0.0001	0.0001
											OLASR	1.0	0.7625
												0.0	0.0001
												OLASL	1.0
													0.0

sutures (vertica, and lambda) and medial right lambdoid sutures (lambda and intermedia) change form in unison.

The presence of low correlations is as important to verifying the capsular model as high Rho values. Values of less than or equal to 0.3 are defined as being poorly related. The clearest pattern to emerge is between anterior and posterior
suture sites on opposing cranial surfaces. The poorest correlations are observed between endocranial and ectocranial coronal sutures and contrasting lambdoid and posterior sagittal regions. The least correlated sutures are endocranial mid-sagittal suture sites (obelion and vertica); they appear to transform in relative independence, scoring only nominal correlations with more anterior endocranial sites. This pattern is not present in the ectocranial surfaces. Examination of the contrasts between endocranial and ectocranial aspects note how of the 14 interactions, only complicata, obelion, and vertica show very little similarity. All other sites show that some very weak relationship is present, but these values do not extend higher than the 0.51 (Illar-Ollar) level. While these values do not fall within the defined `low' category, they and other between-side observations tend to be lower than same surface between-sites correlations.

5. Factor Analysis

Spearman's Partial Correlation is able to distinguish that tissue transformations are not simply related to age, but show distinct, variable relationships between suture sites. It establishes that variability in form is either sensitive or unresponsive to conditions occurring around it. This analysis, however, cannot identify what forms operate as independent units. Subjecting the Rho correlation matrix to factor analysis identifies important continuities in suture activity. Because these values represent a partial correlation, the affect of age is adequately controlled. A total of 28 factors are computed from the correlation matrix; six of these possess a value greater than 1.0 (Table 10). Utilizing SAS's PROC PRIN (Principal Components Analysis), these six factors were orthogonally transformed to identify which suture sites provide the greatest contribution to matrix values. Orthogonal transformation and rotation (obtained from SAS's varimax peocedure) of the factor pattern indicate where maximum amounts of continuity exist. This establishes some idea of how sutures interact with each other to form biomechanical units (Table 11).

In Factor One, the highest contributing elements

Table 10.

Factors Produced from the Correlation Matrix

Factor	Eigenvalue	Difference	Proportion	Cumulative
1	11.7729	8.6394	0.4205	0.4205
2	3.1335	0.5473	0.1119	0.5324
3	2.5862	1.1399	0.0924	0.6247
4	1.4463	0.1843	0.0517	0.6764
5	1.2620	0.1311	0.0451	0.7215
6	1.1310	0.2607	0.0404	0.7619
7	0.8702	0.2130	0.0311	0.7929
8	0.6573	0.0409	0.0235	0.8164
9	0.6163	0.0311	0.0220	0.8364
10	0.5031	0.0435	0.0180	0.8564
11	0.4596	0.0149	0.0164	0.8728
12	0.4447	0.0373	0.0159	0.8887
13	0.4074	0.2068	0.0145	0.9032
14	0.3805	0.0204	0.0136	0.9138
15	0.3601	0.0650	0.0129	0.9297
16	0.2951	0.0479	0.0105	0.9402
17	0.2473	0.0311	0.0088	0.9491
18	0.2161	0.0121	0.0077	0.9568
19	0.2041	0.0223	0.0073	0.9641
20	0.1818	0.0200	0.0065	0.9706
21	0.1618	0.0216	0.0058	0.9763
22	0.1402	0.0128	0.0050	0.9813
23	0.1275	0.0130	0.0046	0.9859
24	0.1144	0.0083	0.0041	0.9900
25	0.1061	0.0289	0.0038	0.9938
26	0.0772	0.0126	0.0028	0.9965
27	0.0646	0.0321	0.0023	0.9988
28	0.0328		0.0012	1.0000

Table 11.

Factor Patterns and Explained Variance

Suture Site	Factor 1	2	3	4	5	6
ICBRR	0.151	0.833*	0.181	0.167	0.290	0.047
ICBRL	0.153	0.835*	0.154	0.180	0.281	0.566
ICCOR	0.135	0.869*	0.128	0.262	0.109	0.010
ICCOL	0.140	0.858*	0.101	0.257	0.106	0.008
ISBR	0.195	0.674*	0.185	0.095	0.407*	0.100
ISVE	0.106	0.359	0.128	0.039	0.666*	0.917
ISOB	0.178	0.253	0.051	0.011	0.626*	0.048
ISLA	0.089	0.293	0.158	0.170	0.707*	0.085
ILLAR	0.063	0.040	0.246	0.594*	0.590*	0.009
ILLAL	0.094	0.034	0.259	0.621*	0.576*	0.007
ILINR	0.031	0.268	0.217	0.805*	0.166	0.159
ILINL	0.071	0.279	0.202	0.789*	0.154	0.165
ILASR	0.132	0.200	0.176	0.799*	0.267	0.212
ILASL	0.158	0.200	0.110	0.804*	-0.039	0.219
OCBRR	0.846*	0.149	0.233	0.056	0.055	0.069
OCBRL	0.839	0.162	0.260	0.135	0.030	0.055
OCCOR	0.807*	0.109	0.144	0.060	0.206	0.191
OCCOL	0.809*	0.120	0.142	0.064	0.126	0.240
OSBR	0.696*	0.141	0.384	0.139	0.128	0.044
OSVE	0.501*	0.124	0.673*	0.109	0.111	0.071
OSOB	0.187	0.257	0.702*	0.102	0.077	-0.078
OSLA	0.373	0.217	0.747*	0.131	0.105	0.030
OLLAR	0.171	0.091	0.736*	0.298	0.204	0.285
OLLAL	0.160	0.979	0.771*	0.256	0.217	0.238
OLINR	0.296	0.115	0.559*	0.283	0.119	0.474*
OLINL	0.283	0.059	0.614*	0.277	0.078	0.442*
OLASR	0.183	0.020	0.167	0.205	0.059	0.841
OLASL	0.167	0.059	0.130	0.258	0.092	0.824
Variance Explained by each Factor						
	4.161	**4.116**	**4.112**	**4.041**	2.655	2.244

* - Indicates High Contributions

are from the ectocranial surface's coronal and anterior sagittal sutures. These values suggest that these sites operate together as a single unit. Factor Two identifies high contributions from the endocranial's sagittal bregma and coronal regions. All other values are considerably smaller, suggesting that Factor 2 expresses a uniquely anterior module. It is important to recognize that while Factors One and Two identify close to the same sites, each factor identifies a unique aspect of the cranial surface. Within each factor high contributions come solely from a single cranial surface. This is evidence that two distinct regions of the cranial capsule are independently responding to a single stress.

A complex of medial, posterior ectocranial sutures dominate the values from Factor Three and Factor Four identifies a similar complement of endocranial sites, concentrating exclusively on the lambdoid sutures. As with the first two fields, Factors Three and Four also identify the same approximate regions of the skull, but are separated from each other by the aspect examined. There is a considerable drop in the amount of variance explained in the last two factors, suggesting that these relationships are minor. Factor Five defines an endocranial unit consisting of all three lambda sites, vertica, and obelion. A small, but notable

contribution is also provided by the sagittal bregma. This field overlaps the previous one in the expression of lambdoid-lambda observations; the presence of the lamdoid sites in the endocranial skull indicate that this region is contributing to the alleviation of affects from two distinct forms of pressure. Finally, Factor Six renders extremely high contributions from ectocranial asterionic sites and a lower, but very visible value from the ectocranial intermedia sutures.

From data gathered from a combined cranial sample, there is considerable evidence that the suture sites observed follow the same patterns observed in human skeletal populations. With increases in age, there is a greater tendancy for sutures to transform their tissue complement. Further exploration identifies that the pattern is not necessarily a relationship with age, but with other factors. The results provide reasons to argue that suture closure is not necessarily a feature isolated to changes in bone.

CHAPTER X.

CONCLUSIONS

What causes sutures to fuse? Does the transformation from soft fibrous connective tissue to the hardened conglomeration of bone result from biological drives exclusive to bone or is ossification the end product of a unified tissue response to environmental demands? This question does not appear to have received much attention from Anthropologists in the past. This thesis has introduced of a model explaining the general mechanical qualities that govern tissue transformation in the suture margin. By examining modern human crania, this investigation has sought evidence supportive of the model's existance and documented what can be determined about the forces governing closure.

1. The Capsular Model and Prior Approaches

Traditional approaches to post-adolescent suture morphology have inferred an age relationship and concentrated on refining methods of age prediction.

Very little evidence is available suggesting any extensive understanding of suture dynamics and only a few investigations have looked at the interaction with other capsular tissues. The drive behind normal adult suture fusion has never been explored thoroughly enough to infer any causal agent other than bone. Variations in the commencement and termination of fusion have been considered simply as part of the bone's physiological aging process. While not necessarily stated, most research implies that suture closure is thought to begin on the endocranial surface and ossify outward. If the impetus behind change in structural composition of the suture is a singular relationship with age, then the only positive correlation accompanying suture morphology would have been with age.

Values from the correlation matrix, however, do not correspond with this perception. What emerges are consistant regional similarities and differences, whose origin can be traced beyond a simple age relationship. While a complex of highly inter-correlated anterior endocranial sutures is evidence for unified transformation behavior, this complex's independence fropm the ectocranial surface and lack of a cohesive ectocranial structure characterize activity expected within the cranial capsule model. The correlation found in the medial right lambdoid sutures or among the

posterior sagittal sutures add more evidence that something besides age is stimulating these sutures to transform in unique manners.

If age acts as a unified, independent entity to transform the whole sutural tissue complement, its control is anticipated to produce roughly equal suture-to-suture correlations and express no distinct geographic pattern. The results of both correlation and factor analysis provide evidence, that this is not the case. As expressed earlier, the lack of high correlation coefficients is as indicative of independence, as high Rho values. The presence of low correlations between surfaces suggest that environmental influences acting on one site are not present on another.

2. The Capsular Model and Goodness of Fit

It is recognized that while these observations are almost unreconcilable under a unified age-morphology model, the invalidation of one model does not support another. What must be presented is a superior goodness of fit with the data available. The capsular model rejects the assumption that fusion is simply a function of age, rather it implies that the dynamics of tissue transformation are a continuation of physiological

processes in the cranial capsule. When observing the cranial vault as a whole, multiple sites of relatively independent sutural networks would be anticipated - these represent distinct regions localizing and responding to environmental pressures. Unless environmental stimuli were uniform between two sites, such as in bilateral suture correlations, there is little reason to expect the sutural structure to behave in similar manners. The lack of cohesiveness in suture relationships are viewed as extensions of the cranial capsule's adaptive function.

No theoretical constructs have been found in the literature that are capable of adequately modeling post-adolescent suture activity in humans. Asking questions about the contributions various stimuli impose upon the mature form make it necessary to view the suture from several fundamentally different aspects. Sutures are not simply skeletal features, they must be perceived as part of a plexus of interactive tissues defined as the cranial capsule. Their morphology is a direct result of satisfying demands placed on the cranial capsule's environment. Each tissue acts in conjunction with others to isolate pressures and balance demands placed on the whole structure. If a shift in the environment occurs, there is a change in the tissue complex's morphology. If the

demands are too great for the connective tissues to respond by themselves, this transformation can be identified in bone as a shift towards increased osteoblastic cellular activity.

A synthesis of what is known about suture closure suggests that the drives governing suture transformation are not isolated to the bone, and that understanding this involves thinking of the mature suture in terms of biomechanical units functioning in the tissue complex. Most research has divided the suture into landmarks established during the cranial capsule's developmental period. If disproportionate demands are placed on the mature cranial capsule, portions of the bone will react that may not follow the original morphological pattern. The cranial capsule should be partitioned according to adult functional demands, not developmental similarity.

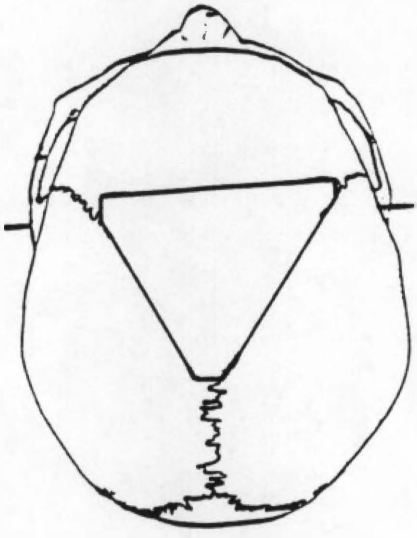
a. Localization of Biomechanical Demands

The identification of biomechanical units serves a two fold purpose. Not only does it provide a biologically valid way of partitioning the capsular structure, but it suggests where the stimuli behind suture change may be originating.

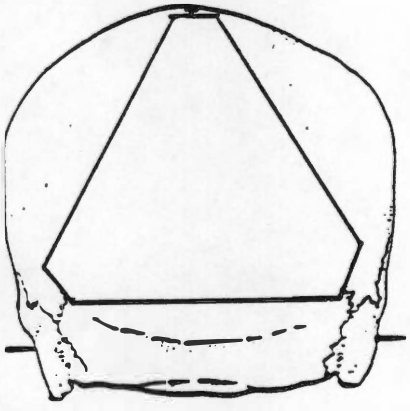
Factor analysis identifies several distinct localized areas of sutural activity operating as

integrated networks responding to simular stimuli. Results of the factor analysis indicate that at least five centers on the vault are undergoing unified sutural tissue transformations (Figures 15 and 16). Factor contributions also tend to congregate in clusters corresponding to adjacent regions of the skull. Each of these centers, particularly those on opposing cranial surfaces and planes are independent entities.

Where are these forces generated? Recognizing that a considerable overlap is present between endocranial and ectocranial sites, the simplest explanation is that more than one force acts on the mature cranial capsule; one of these centers is on the ventral vault and the other focuses on dorsal regions. Force from two drives is believed to affect both aspects. While the ectocranial complexes segregate into two discrete groups, the endocranial compliment contains a medial complex which partially overlaps with the lambdoid group. The strength of the ectocranial dichotomy may suggest where the pressure originates. Structurally, the endocranial surface is at least partially responsive to ectocranial stimuli. Recalling that the pars interna acts as a hinge to deflect external pressure away from the neural mass, it would be reasonable to detect some level of outside influence.



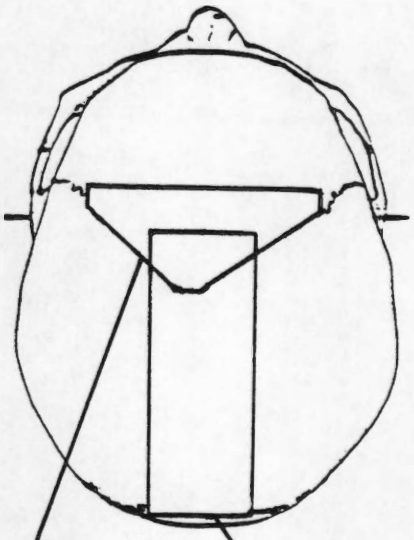
Module 1:
OCBRR
OCBRL
OCCOR
OCCOL
OSBR
OSVE



MODULE 2:
OSVE
OSOB
OSLA
OLLAR
OLLAL
OLINR
OLINL

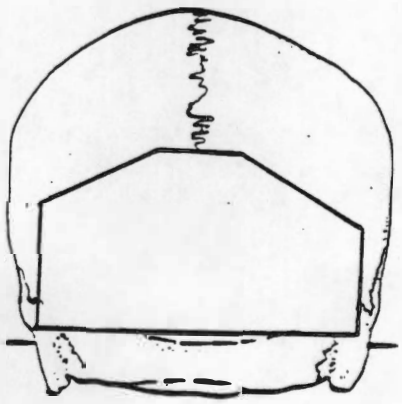
Figure 15.

Biomechanical Complexes on the Ectocranial Surface of the Cranial Capsule



MODULE 3:
ICBRR
ICBRL
ICCOR
ICCOL
ISBR

MODULE 4:
ISBR
ISVE
ISOB
ISLA
ILLAR
ILLAL



MODULE 5:
ILLAR
ILLAL
ILINR
ILINL
ILASR
ILASL

Figure 16.

Biomechanical Complexes on the Endocranial Surface of the Cranial Capsule

It is suggested that the ventral-dorsal complexes probably result from pressures outside the cranial vault.

The presence of distinct ectocranial lambdoid and medio-dorsal complexes, however indicate that these forces cannot be entirely accounted for by ectocranial influence. Neither can the presence of an ectocranial asterionic complex be defined on the basis of anterior-posterior stimuli. It is currently unclear whether the asterionic and intermedia measurements reflect bilateral symmetry or represent a lateral-dorsal complex. The separation of these two sites from other ectocranial sutures and marked reduction in value contributions, are indicative that the complex may be related to different biomechanical activities at the occipital-temporal juncture. It is suggested that the endocranial surface has at least one additional drive center on the ectocranial lateral surface. It is important to recognize that when using the capsular model, location is the dominant feature that defines morphology. `M' may be defined as large (capsular) or small (microscopic), depending on the needs of the research question, but exact identification of the suture's locale clarifies what unique regional affects are present.

This investigation identified that the ectocranial

and endocranial environments have distinctly unique effects on morphology; it must be recognized that contributions from the diploe are likely to be as influential. Defining what stimulates the complexes forming drives is beyond the scope of the data gathered for this thesis. Causal agents can only be hypothesized. The ectocranial anterior complex may reflect changes in the masticatory and supporting tissues and the dorsal unit could stem from either diploic expansion or changes in cranial support structures. Endocranial complexes may be partially affected by vascular changes associated with increased arborization of the menengeal vessels. Further research into these and other anatomical complexes is needed to clarify exactly what causes differential suture activity.

b. Age and the Capsular Model

At a given location, a suture will not contain the same tissue structure throughout life. Neither young or adult sutures remain stable; they do not possess the same form of connective tissue throughout life. Changes in the structural components are based on shifts in the cranial capsule's needs. With age, there is a change in the biomechanical forces shaping the

tissue. Any explanation of the suture's form must recognize the existence of these influences and demonstrate how they relate to the tissue state observed.

Within the capsular model, sutures are not viewed as changing regardless of age, rather they appear to most sensitive to age's influence on the surrounding environment. The ultimate cause for change is still age; what is stressed is where the pressure for ossification orginates. Ossification of the sutural tissues has traditionally been viewed as simply a result of the aging process; the rejection of this explanation does not account for age's relationship with morphology.

How can age change be explained using the capsular model? It is reasonable to suggest that the age effects seen in the suture are not unique osseous drives, but primarily result from the cranial capsule's adaptation to the effects of age on the surrounding environment. The endocranial aspect of the suture can be related to changes in the neural environment; with the subsidence of active neural expansion associated with adulthood, subsequent changes in structure would follow these actions. Likewise, the scalp musculature has been noted to change form with increased age. The sensitivity of ectocranial tissues to meeting these

element's structural needs is believed to be the source of this sutural affect. The capsular environment is also responsive to age related change associated with the diploic space. In effect what sutures record are variances between at least three distinct sets of aging processes - one neural, one muscular, and one diploic. How all three of these forces interact with age, and with each other, determines what tissues will be present at the sutural margin.

c. The Capsular Model and The Suture Throughout Life

In the proposed model, M=(IA)+(OA)+(DA)+E, the morphology at a given suture site is viewed as an expression of three prime forces:

1). Expansive forces from the neural mass pushing the cranial capsule apart.

2). Muscles attached to the skull apply tensile force pulling the bone away from the brain's surface.

3). Expansive forces, principally in the diploe, act to remodel cortical bone in order to increase hematopoietic and storage capability inside the cranial capsule.

Suture activity shifts with age as a result of

these forces. Any model of suture dynamics should reasonably be able to explain much of the morphological change in young as well as elderly crania. Using the capsular model, sutures can be seen to pass through three dramatic phases of biomechanical activity, each reflecting a different combination of the suture's biomechancial regulators.

PHASE 1 - THE YOUNG SUTURE:

The Young Suture is dominated by the cranial capsule's need to provide a tight protective shield around the brain that is capable of rapid expansion. The major biomechanical force present is expansion. This pressure propels the capsule outward. The young suture responds to the functional demands with a unique tissue repertoire. Between the margins, undifferentiated mesenchymal tissue permits rapid connective tissue formation. Accelerated growth in bone is characterized by pronounced periosteal activity on all sides of the bone. The cambial layer is very thick. No diploic space is present.

Bone rims are typically far apart. All proto-suture margins display a smooth, flat junction and there is no interdigitation. These edges are very plastic. Their exact location on the developing skull is not predisposed, rather the proto-suture margin

conforms to the needs of the tissue state. Margin growth is flexible; it responds to pressures brought on by shifts in cerebral growth orientation.

The young suture emerges with the development of the fusiform blastema. The young phase is present throughout the developmental period. In humans, it lasts until approximately two to three years of age. Morphologically, this phase transforms into the adolescent suture with the loss of the ectomennix, commencement of margin interdigitation, and development of the diploic cavity.

PHASE 2 - THE ADOLESCENT SUTURE:

The Adolescent Suture reflects a morphology needed by the cranial capsule to fulfill the demands of the brain enlargement, the musculature to pull away from the skull, and those of the diploe to develop new regions for stem cell production. Expansion of the brain is more leisurely, ossification of the cranial capsule continues until the fusiform blastema is met by the bone margin. The margin looses its smooth profile in response to the pressure present and forms either flat or bevelled junctions. Interdigitation develops as a compromise between needs for a strong bond against ectocranial tension and a breach in the hard tissue for cerebral expansion.

The tissues present during adolescence also reflect biomechanical force compromises. Active connective tissue growth of the ectomennix is exchanged for a more resistant sutural ligament. Sutures loose much of their plastic nature during adolescence and do not respond to modification as readily. This is attributed to a decline in the number of cells present and an increase in collagen fibers. The cambial layer thins and the diploic space develops as tension pulls the ectocranial surfaces away. Hematopoiesis is initiated in the newly opened area.

The adolescent phase is a transitional period between major influences. Its morphology reflects a considerable amount of mediation. In humans, adolescence starts at about two years and terminates in the early twenties, with the first signs of suture closure. There are no sharp lines defining its commencement and termination.

PHASE 3 - THE MATURE SUTURE:

The Mature Suture reflects a decline in endocranial influences on the entire suture area. During this phase brain growth slows to a point where internal apposition and remodeling are capable of balancing expansion with the capsule's protection. This reduces the forces acting on the suture to those

of the musculature and the diploic cavity. This shift in force induces a change in the structures present.

There is a gradual change in most of the mature suture's tissue. The sutural ligament is lost and replaced with either cartilage, bone, or diploe. Ossification and loss of the suture margins allow expansion of the diploe. Closure of the suture isolates the cranial capsule into elements reflecting internal and external environments (Young 1959). Suture fusion also serves to merge the diploic element of each cranial bone into a unified organ. The loss of the internal structure of the suture indicates that the need for a fulcrum is replaced by needs for a rigid structure and more diploic tissue.

The reduction in tissue density during the adolescent state is combined with a decline in the amount of cellular activity. Changes in morphology are more gradual than seen in the earlier phases. Continued growth at the suture margin reduces the ligament, eventually replacing it with different tissue. It is unclear whether this transformation is related to changes in one of the controls governing suture morphology more than any other.

Can it be demonstrated that the capsular model of suture transformation is a more reasonable reflection of reality than any prior construct? The model's

design encapsulates a host of different perspectives and addresses all major features of suture margin dynamics. It can be seen to model the suture throughout life. This aspect recognizes that the same generalized sources for pressure found within the developing suture are still present in the mature form; all that has changed are the responses needed to maintain a biological balance. With the introduction and testing of hypotheses drawn from the model, a superior goodness of fit results.

What has emerged from the data is evidence that while age is an important aspect governing suture morphology, it is not a direct influence. This relationship places any change in the osseous aspect, attributeable simply and directly to age, at a much more minor level than previously anticipated. It is important to remember that the suture is not an isolated tissue network, but rather is one aspect. What is driving changes in morphology is not limited to application on the suture margin, but is part of the cranial capsule as a whole.

The discovery of independent drives in the cranial capsule serves to generate more questions than it answers. There is a surprising lack of understanding in post-adolescent fusion. One major step will be the identification of the agents behind the sutural

complexes. The relationship of diploe to the cranial capsule could not be adequately approached in this investigation, yet has been instigated time and time again as a major influence on the transformation process. It is also unclear what agents are driving discrete suture complexes and how they relate to the aging process.

By developing a thorough understanding of the `whys' and `hows' behind suture closure, its abbarent, erratic behavior will appear to be more reasonable physiological responses. It is hoped that this research will serve as the foundation to continue investigations and serve as a base to look at the cranial capsule as a whole unit changing to meet the needs of the ageing human body.

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Appendix:

Study Sample

Study Sample

ID Number	Sex	Age
Indian and Civil Wars Collection (AFIP):		
0644	M	37
0748	M	40
1140	M	29
1359	M	29
1484	M	37
1493	M	40
1940	M	34
1943	M	26
1948	M	29
1949	M	30
1959	M	28
2164	M	25
2462	M	24
2466	M	66
2476	M	44
2480	M	18
2481	M	27
2486	M	22
2489	M	28
2494	M	25
2509	M	19
3086	M	28
3114	M	34
3117	M	24
3121	M	32
3123	M	24
3126	M	30
3142	M	28
3146	M	18
3184	M	24

Study Sample (continued)

Id Number	Sex	Age
R.J. Terry Collection (NMNH)		
0014R	M	38
0034R	M	44
0109	M	46
0113R	M	31
0126R	M	50
0313R	M	39
0136R	M	59
0137R	M	65
0156	M	68
0167	M	77
0168R	M	68
0181	M	38
0184R	M	42
0187R	M	29
0192	M	73
0194	M	63
0195	M	49
0196	M	27
0198R	M	40
0201	M	55
0207	M	45
0212	M	62
0216	M	45
0219	M	72
0224	M	64
0229	M	41
0230	M	38
0233	M	37
0245	M	65
0246	M	60
0250	M	52
0258	M	64
0259	M	52
0263	M	49
0264	M	67
0266	M	70
0267	M	42

Study Sample (continued)

ID Number	Sex	Age
0268	M	62
0273	M	50
0274	M	45
0277	M	50
0279	M	40
0281	M	79
0282	M	52
0284	M	70
0297	M	50
0301R	M	36
0303R	M	30
0307	M	72
0311R	M	27
0315	M	48
0316	M	57
0317	M	84
0318	M	45
0320	M	43
0325	M	65
0326	M	66
0332	M	50
0342	M	40
0343	M	32
0347	M	69
0352R	M	76
0354R	M	78
0366	M	57
0367	M	57
0369	M	76
0370	M	62
0372	M	71
0380	M	44
0392	M	68
0396	M	75
0406	M	73
0411	M	49
0412	M	62
0413	M	55
0416	M	77
0420	M	64

Study Sample (continued)

Id Number	Sex	Age
0428	M	84
0431	M	42
0434	M	41
0435	M	64
0448	M	47
0453	M	42
0459	M	60
0481	M	68
0493	M	75
0496	M	54
0498	M	78
0499	M	76
0501	M	77
0502	M	82
0506	M	48
0517	M	46
0521	M	67
0527	M	70
0533	M	67
0545	M	44
0547	M	66
0552	M	72
0555	M	56
0557	M	46
0569	M	63
0573	M	59
0588	M	69
0588R	M	60
0591	M	28
0598	M	61
0602	M	49
0605R	M	52
0622R	M	42
0623R	M	80
0624R	M	79
0630	M	72
0633	M	76
0634	M	74
0636	M	51
0645	M	20

Study Sample (continued0

Id Number	Sex	Age
0651	M	64
0672	M	52
0674	M	58
0681	M	53
0691	M	82
0693	M	57
0696	M	32
0697	M	72
0743	M	53
0746	M	71
0747	M	45
0750	M	80
0751	M	41
0752	M	71
0755	M	36
0756	M	47
0762	M	59
0763	M	46
0768	M	54
0783R	M	65
0784	M	68
0795	M	51
0802	M	36
0805	M	87
0806	M	78
0810	M	54
0812	M	56
0814	M	55
0827	M	74
0832	M	84
0835	M	61
0838	M	67
0839	M	41
0842	M	73
0846	M	48
0849	M	47
0852	M	66
0858	M	66
0865	M	69
0867	M	38

Study Sample (continued)

ID Number	Sex	Age
0871	M	56
0874	M	75
0892	M	59
0897	M	43
0912	M	63
0918	M	40
0924	M	43
0931	M	67
0956	M	63
0974	M	77
0979	M	85
0982	M	70
0989	M	30
0991R	M	59
1023	M	20
1024	M	65
1028	M	67
1037	M	52
1043	M	46
1051	M	74
1053	M	58
1061	M	68
1062	M	81
1072	M	56
1073	M	79
1079	M	47
1084	M	47
1088	M	58
1089	M	43
1097	M	54
1102R	M	75
1107	M	78
1111	M	81
1111R	M	38
1112	M	77
1116	M	80
1118	M	74
1121	M	51
1167	M	53
1170	M	41

Study Sample (continued)

Id Number	Sex	Age
1175	M	55
1179	M	75
1194	M	48
1204R	M	44
1216	M	69
1217	M	52
1219	M	79
1220	M	73
1226	M	48
1228	M	79
1229	M	73
1230	M	53
1234	M	69
1242	M	48
1248	M	62
1250	M	53
1255	M	39
1271	M	58
1295	M	70
1301	M	57
1310	M	85
1318	M	43
1321R	M	85
1324	M	49
1327	M	61
1371	M	65
1424	M	51
1428R	M	71
1436	M	56
1437	M	61
1442	M	63
1450	M	50
1458	M	37
1471	M	71
1475R	M	72
1481	M	54
1484	M	60
1495	M	82
1498	M	75
1520	M	52

Study Sample (continued)

Id Number	Sex	Age
1522	M	66
1527	M	74
1532	M	76
1534	M	44
1540	M	58
1543	M	70
1545	M	82
1564	M	30
1569	M	30
1588	M	82
1598	M	33
1607	M	32
006RR	F	69
016RR	F	91
0019	F	45
0022RR	F	63
0041R	F	41
0064R	F	57
0069	F	75
0069R	F	52
0071R	F	75
0072R	F	78
0076R	F	62
0091R	F	65
0096R	F	83
0108	F	70
0112R	F	59
0133R	F	73
0134R	F	66
0135R	F	30
0140R	F	72
0142R	F	82
0161R	F	50
0162R	F	40
0175R	F	84
0206R	F	88
0217R	F	77
0236R	F	60

Study Sample (continued)

Id Number	Sex	Age
0248R	F	70
0249R	F	81
0252R	F	81
0275	F	68
0289R	F	47
0312R	F	64
0319R	F	86
0321	F	61
0321R	F	89
0344RR	F	73
0346RR	F	51
0349R	F	70
0373	F	55
0383RR	F	76
0393RR	F	58
0405R	F	34
0437R	F	44
0451	F	72
0451R	F	48
0456R	F	72
0563	F	41
0580	F	61
0686	F	78
0689R	F	87
0722RR	F	67
0736	F	57
0745R	F	34
0794R	F	79
0834R	F	87
0837R	F	86
0847	F	39
0880	F	27
0899R	F	65
0928R	F	49
0983	F	30
0985RR	F	66
0992R	F	71
1016R	F	54
1054	F	81
1071R	F	75

Study Sample (continued)

Id Number	Sex	Age
1075RR	F	67
1080	F	70
1080RR	F	42
1085R	F	71
1103	F	74
1103R	F	88
1120	F	24
1120RR	F	76
1174	F	53
1186	F	44
1189R	F	68
1199R	F	45
1202R	F	56
1243R	F	50
1270R	F	59
1370	F	80
1405	F	67
1456	F	60
1473	F	74
1476	F	51
1480	F	64
1482R	F	35
1512	F	37
1523	F	36
1562	F	46
1563	F	29
1565	F	85
1566	F	33
1567	F	69
1570	F	62
1572	F	45
1579	F	49
1582	F	46
1587	F	76
1592	F	54
1599	F	41
1601	F	61
1608	F	43
1610	F	61
1612	F	41

Study Sample (continued)

Id Number	Sex	Age
1614	F	38
1616	F	53
1617	F	35
1622	F	58
1623	F	74
1630	F	79
1631	F	83
1633	F	80
1634	F	63
28R	F	70
589R	F	84
617	F	90
80RR	F	55
939RR	F	72

VITA

Hugh Bryson Matternes was born in 1960 in Houston, Texas. He is the son of James R. and Gwendolynn P. Matternes. He grew up in Winston-Salem, North Carolina and received his Bachelor of Arts Degree in 1987 from the University of North Carolina - Greensboro, majoring in Anthropology. He, his wife Jennifer, and their spastic little dog, Calamity Jane, currently reside in North Knoxville.

Mr. Matternes' interests in Anthropology focus on data obtainable from human skeletal remains. He has had considerable experience with the archaeological recovery of human remains from sites all over the Mississippi River Drainage System. His publications emphasize an intense interest in osteology, cemetery social patterning, modern and prehistoric mortuary behavior. Mr. Matternes currently serves as the osteological consultant for the Wickliffe Mounds Research Center. He plans to pursue his education through the PhD level.