

CRANIAL ASYMMETRY IN NEWFOUNDLAND  
MARITIME ARCHAIC AND COLONIAL-ERA  
EUROPEAN SKELETAL POPULATIONS:  
AN EXAMINATION OF DEVELOPMENTAL STABILITY  
AND THE IMPACT OF MUSCULAR ACTIVITY ON  
CRANIAL MORPHOLOGICAL VARIATION

EMILY WEBB







**Cranial Asymmetry in Newfoundland Maritime Archaic and Colonial-Era  
European Skeletal Populations: An examination of developmental stability and the  
impact of muscular activity on cranial morphological variation.**

by

Emily Webb

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in partial fulfillment  
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Department of Anthropology  
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## **Abstract**

This research centres on an investigation of cranial asymmetry in terms of within-individual and intra-sample frequency and etiology, in an effort to elucidate and compare the health, environment and cultural behaviour of Newfoundland Maritime Archaic and Colonial-era European skeletal samples. The samples consisted of thirty-seven Maritime Archaic and forty-one European crania, and were drawn from past Newfoundland and Labrador populations. This research entailed a quantitative examination of morphological variation, focusing on developmental stability, pathology and the influence of muscular activity, and was analyzed in the context of a functional cranial model.

At the individual level, the use of standardized cumulative asymmetry values to characterize each functional unit allowed both empirical description and discussion of asymmetry in functional and developmental contexts. At the sample level, both groups manifested asymmetric variation in regions likely to be reflective of postnatal muscle use, and some differences in physical activity between groups can be defined. On the whole, the Maritime Archaic demonstrated higher levels of asymmetry than the European sample, and this is reflected through an examination of developmental stability and fluctuating asymmetry.

The methodology allowed group-specific explanations of asymmetric variation to be constructed at the sample level, and informative analysis of functional and developmental variation at the individual level. All studied crania were determined to be asymmetric to some extent, and it was possible to gain an understanding of the nature and magnitude of that asymmetry using the functional cranial model and the asymmetry measurements presented in this thesis.

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## Chapter 1 Introduction

*“Nature has furnished the organic world with  
a certain measure of asymmetry.”*

*(Fritsch 1968)*

Some form of asymmetry exists in nearly every aspect of the natural world, from the molecular to the gross anatomical level. Many soft tissue structures in the human body are asymmetric; for example, the lungs, the heart and the brain. The skeleton, however, is overtly symmetric. So why, then, is it reasonable to study skeletal asymmetries? Although there is a general symmetry of form, the skeleton is subjected to an asymmetry of function at the cellular level during development and at the macroscopic level through postnatal activity, which can, over time, lead to a subsequent and corresponding asymmetry of form. An understanding of that asymmetry, particularly the forces directing its manifestation, can provide new information, enriching studies of morphology, growth and development. By taking a broader approach, one that incorporates multiple lines of evidence from genetic, morphological and osteometric studies of skeletal remains and that considers groups or samples rather than single cases, a more complete picture can be created, recognizing differences between diverse groups or between different strata within one group.

The study of cranial morphology has undergone numerous methodological and contextual shifts since its establishment in the eighteenth century, from primarily racial studies to the study of growth and development. Studies of cranial morphology incorporating concepts of biomechanics and growth operate under the functional

paradigm that form is the ultimate outcome of a mechanical interaction between two or more structures, or functional units. Although the skull does not articulate or experience muscular activity the same way that, for example, the appendicular skeleton does, various functional components respond to forces exerted by soft tissue action. That being said, because of the more subtle effects of muscular action, the study of cranial asymmetry may also provide a unique opportunity to examine the influences of developmental stability on skeletal structure.

Although they will be further expanded upon in section 1.3, the primary objectives of this research can be summarized in brief. The most crucial objective is the development of a new set of measurements and interpretive model that can provide information about cranial asymmetry for population samples as well as individual isolated crania. The frequency and cause of observable variation of both normal and pathological origin will ideally elucidate the effects of cultural behaviour and activity, health, and environment on cranial morphological variation in this context. Finally, the possibility of defining a normal versus asymmetric threshold value will be explored.

## 1.1 General Introduction

Asymmetry may be loosely defined as the lack of proportion between parallel parts of an object or thing. In terms of the cranium, it can be a convexity of one part of the neurocranium or simply the uneven distribution of the bony structure over the midline. It is understood that some degree of cranial asymmetry is normal, but the question of how much and what type is considered “abnormal” still remains unanswered. Zivanovic (1982), in comparing size differences between right and left sides of the skull, suggested that a difference greater than two millimeters indicates asymmetry. Using this

criterion, 98% of human skulls in a given population would be considered asymmetric. Thus this differentiation, while interesting, is not very sensitive, rendering it relatively uninformative. The problem, then, of establishing a set of criteria for analysis of asymmetry still remains unresolved. Normal asymmetry is influenced by both internal and external factors; for example, mild developmental irregularities like fluctuating asymmetry or perturbations in the uterine environment. That being said, these same factors, taken to the extreme, can induce detectable cranial asymmetry (Skinner *et al.* 1989; Zivanovic 1982).

Classic cranial asymmetric variation can be visualized most easily as a twisting of the face or neurocranium, but a great deal of subtle disproportionality exists in the entire skull, as well as the rest of the human body. There are many potential causes of this cranial variation, which can be generally categorized as due to post-depositional warping, artificial/culture-induced deformation, and biological processes acting to distort the skull. Post-depositional warping is a pseudopathology caused by the pressure of the burial substrate (i.e., heavy or damp soils) on the delicate arrangement of the bones of the skull. Over time, the calcium matrix of the bone degrades, weakening the overall integrity of the skull. The resultant cranial deformation can, at first glance, be confused with pathological or artificial deformation (Henschen 1966). Purposeful cranial deformation has been extensively researched globally (e.g., after Ortner and Putschar (1985), aboriginal North America, Egypt and Peru), but it has not been shown to be practiced by any of the populations used for this study (Jelsma 2000; Kennedy 1981). The biological causes of cranial asymmetry are numerous and vary widely in terms of etiology. They

can be grouped generally under the categories of developmental defects, congenital anomalies and activity-induced changes in form.

The terms developmental and congenital are often used interchangeably, but they can be used to correctly define two slightly different etiologies. According to Barnes (1994), developmental defects are the result of mistakes or interruptions during morphogenesis, generally occurring quite early during foetal development. The process is mediated by both intrinsic (genetic) factors and extrinsic (biochemical or environmental) factors. Developmental defects influencing cranial form include craniosynostosis and cranial/caudal border shifts, which affect the cranial base. Congenital conditions, while still due to genetic factors to some extent, include some sort of insult or trauma before or at parturition as their more immediate cause. For example, position *in utero* during growth can influence the ultimate shape and/or functioning of involved structures (i.e., congenital hip dysplasia and congenital muscular torticollis).

Even in the absence of developmental defects, perturbations in the developmental environment are still occurring. These perturbations, also known as “random noise”, are detectable by observing fluctuating asymmetry, specifically by measuring deviations from perfect symmetry in bilateral structures. It occurs when an organism is unable to undergo a stable developmental process due to intrinsic and extrinsic factors, and it can thus provide information about these factors in a culture-specific context. The relationship between fluctuating asymmetry and its causative factors is not, however, as straightforward as this implies. Developmental mechanisms, including canalization and stabilization, attempt to counteract the random noise inhibiting stable development at both the population-genetics level and the individual-environmental level. Developmental

stability, as well as any variability ultimately present in the cranium, is therefore a window into the genetic character and the environmental conditions of group members, causing bilateral variation that is detectable, and potentially biologically important, but not specifically pathological.

Although it is more commonly recognized as an influencing factor on intracranial asymmetry, muscular activity may also cause similar bilateral variation in the cranium. Activity-induced asymmetry can be viewed as a divergence from what is accepted as a normal, or symmetrical, form, and, although this is entirely expected, it is important to highlight the fact that muscular activity can cause a deviation from perfect left-right symmetry of the skull. Although a fairly extensive body of work exists concerning bilateral asymmetry of the appendicular and most of the axial skeleton, (e.g., Mays 1999; Mays *et al.* 1999; Steele and Mays 1995; Stokes 1997), cranial asymmetry induced by normal physical activity outside of the masticatory apparatus is not often considered (e.g., Daegling 2004; Wood and Lieberman 2001). The cranium is, however, a dynamic structure influenced, like all other skeletal structures, by the surrounding soft tissue, and thus some indications of activity may be discernable.

## 1.2 Background

Instances of cranial asymmetry have appeared in many distinct populations globally, and, although usually noted when present in archaeological skeletal material, there has been little objective investigation performed and literature reporting is sporadic. There are several reasons for this. First and foremost is the ambiguity in characterizing asymmetry. There is no defined threshold with respect to single elements of the cranium

or of the composite whole, thus making it difficult to determine if the observed asymmetry is truly a significant deviation from “normal”. As well, there is not, at this time, a single set of measurements that capture cranial asymmetry in a meaningful way, so that the nature and magnitude of the anomaly is reflected. It is therefore evident that both the analytical method and the causative factors influencing the manifestation of asymmetry require some investigation.

Over the last two decades, a few researchers in diverse fields have applied metric analysis to the study of cranial asymmetry, considering either the entire cranium (i.e., Douglas 1988) or some smaller aspect (i.e. Anderson 1983). Although craniometry has been successfully and legitimately applied to the study of morphological variation in the context of growth and development since the early twentieth century, its application to left-right asymmetry, an important aspect of human development, is still problematic. The necessity of being able to capture asymmetric variation was highlighted by Woo (1931), who, as an early proponent of morphometric studies, undertook an investigation of asymmetry of homologous bone pairs. He concluded that human crania are inherently asymmetric.

Subsequent research centering on cranial asymmetry in a metric context has been sporadic (Kidd 1954), and often focused on activity-induced asymmetries (i.e., Anderson 1983; Lemay 1977). It was not until the late nineteen-eighties that a base in anthropological literature began to develop (Douglas 1988; Skinner *et al.* 1989; Smrcka *et al.* 1986). Even then, only Douglas attempted to develop an osteometric method for capturing asymmetry. Quantification of any pathology, including cranial asymmetry, is rarely undertaken. It is equally rare to examine asymmetry in an entire sample, regardless

of pathology, in order to gain an understanding of normal variation. Due to the ever-increasing scope of palaeopathology, it has, however, become obvious that standardization of observations and broadened research questions concerning growth and development are absolutely essential for the continued viability of the discipline. Thus, the development of a means of quantifying such a basic concept as bilateral cranial morphological variation has the potential to be of use to the larger discipline of physical anthropology.

### 1.3 Purpose & Objectives

This research centers on an examination of cranial asymmetry in terms of within-individual and sample frequency and etiology, in an effort to elucidate and compare the health, environment and behaviour of the Newfoundland Maritime Archaic, Basque whalers and Colonial Europeans. To do this, several aspects of human osteology will be addressed; specifically, the definition of normal versus asymmetric crania and the development of a quantitative method of ascertaining this. Should the application of this method to available skeletal material demonstrate significant patterns of asymmetry, discussion of potential causes will provide information regarding the cultural behaviour and health of both individuals and population samples.

Discrete skeletal samples from Maritime Archaic, Basque and Colonial European populations have been incorporated into this research, and asymmetry of all individuals, whether a specific asymmetry-inducing defect is evident or not, will be examined. Although linked through geography, that is, a common habitation of Newfoundland and Labrador, each population is distinct both temporally and genetically; thus those

influencing factors, as well as activity and social infrastructure are variables to consider. Of the over 200 individuals represented in the Newfoundland skeletal remains collection, only thirty-seven Maritime Archaic, twenty-four eighteenth and nineteenth century European and seventeen sixteenth century Basque crania, partial crania and/or mandibles were deemed appropriate for this study.

The primary research objective is to develop a methodology and an interpretive model that is both broadly applicable to any cultural group, in addition to being able to provide useful insights into individual and sample-wide patterns of asymmetry. The method and model will then be applied to the Maritime Archaic and European samples in order to elucidate the frequency and etiology of observable variation of both normal origins (incorporating developmental stability and muscular activity) and asymmetry resulting from other causes. The analysis of the asymmetry measurements in the context of the interpretive model, outlined in Chapter 5, will provide information about the effects of cultural behaviour and activity, health, and environment on cranial morphology, and it will determine to what extent inferences can be reasonably made about these factors based on population samples or individual crania. Finally, the feasibility of creating a threshold that distinguishes between normal and asymmetric crania can be discussed in the context of the interpretive model and the observations made during the course of this thesis.

## Chapter 2 The Normal Cranial Form

### 2.1 Normal Asymmetry

What constitutes a “normal” skull shape? The term normal can be defined in many ways, but, in its broadest sense, it means to conform to what is typical or standard. Since it is widely accepted that no skull is perfectly symmetric, normal cranial shape must therefore be defined in such a way that it encompasses a range of slightly asymmetric variations in form. Putting aside for the moment the more dramatic morphological variations caused by, for example, a developmental anomaly, the point of consideration now becomes the establishment of a normal-abnormal threshold.

Due to the lack of a single normal form, this threshold-determination is surprisingly problematic, from both a cultural and a metric perspective. Although the cranium of any healthy individual could be defined as normal, there is no one invariably reliable criterion for deciding what is normal. This is because cranial and facial asymmetries are closely linked to group-specific cultural constructions of beauty and attractiveness, and a normal cranial shape for a given cultural group is one that is cosmetically acceptable. Although Simmons *et al.* (2004) suggest that near-perfect cranio-facial symmetry is an important factor in determining beauty in most modern cultures, there are numerous examples where purposeful cranial deformation has been used to create shapes that might be considered repellant outside of their unique cultural context, for example, in Peru or Egypt (Ortner and Putschar 1985). Creating a broad mathematical threshold value provides a limited amount of information; however, a preliminary study (Webb 2005) suggests that isolating parts of the skull into functional

units may allow more meaning to be attached to similar metric-threshold determinations, for individual crania and for larger samples.

## 2.2 Normal Cranial Growth and Development

There are numerous factors influencing normal cranial growth, and an understanding of these factors reveals some of the underlying causes of the range of normal cranial forms. Normal cranial growth is an immensely complex process. Foetal development and ossification of bony tissue are dealt with more thoroughly in Chapter 3; however, at parturition, all bones are present and beginning to ossify, even though growth and fusion are not yet complete. Dense connective tissue attaches adjacent plates of bone, leaving soft, unprotected areas, known as fontanelles, open to allow compression during birth and rapid postnatal brain growth. During the first nine months of life, the skull may grow anywhere from a quarter to half of its adult volume, reaching full adult proportions by eight or nine years of age. This rapid intracranial growth coupled with the expansion of respiratory passages, as well as tooth development, cause corresponding active growth of bony tissue, eventually allowing contact to occur among cranial and facial bones and thus the formation of sutures (Marieb 1995; Ortner and Putshcar 1985).

Cranial sutures are a type of synarthrotic fibrous joint, allowing limited movement until complete bony fusion occurs, normally in mid- to late-adulthood. Initially, neurocranial and facial bones appear as distinct ossification centres, expanding rapidly and eventually articulating with each other via interdigitating or overlapping bone splices (White 2000). The formation process, including both timing and location, is in part regulated by the growth movements of the bones; that is, the displacement of the bones

relative to each other that occurs during growth (Persson 1989). The development of sutures is thus, to some extent, responsive to external separative forces acting on the bone, such as the volumetric demands of expanding intracranial tissue and spaces (Babler 1989; Persson 1989).

Despite the apparently straightforward nature of cranial growth and suture formation, there is, in fact, a great deal of variation in the procedure and outcome of these processes. Cranial bone growth is mediated by brain growth, which is unevenly distributed between left and right cerebral hemispheres (Steele 2000). According to Steele, this differential growth is somewhat detectable in dry bone by measuring frontal and occipital lengths and widths. Additionally, the malleability provided by the fontanelles in early childhood could lead to incidental (non-purposeful) cranial deformation, such as occipital flattening or mild plagiocephaly (Myslobodsky *et al.* 1987). Since sutures are a response to bone growth, they are in turn influenced by the relative positioning of bone, and therefore both shape and location can be affected by uneven growth. This fact is of particular importance during osteometric studies, which use suture intersection points as landmarks. As well, sutures delimit the bones, such that the ultimate size of homologous skeletal elements or functional units may be perceptibly affected.

The cranial forms created by these factors, uninfluenced by any pathological disturbance, can range from near-perfect symmetry to distinct asymmetry. While visually qualifiable and metrically quantifiable, with variable success, the interpretation of these often subtle morphological variations can be difficult, due to the unique interactions of the factors involved. That being said, a common theme throughout is the importance of

growth and movement, and cranial morphological variation can be interpreted in that context through the functional matrix hypothesis.

## 2.3 Functional Craniology

### 2.3.1 Background

In general, the mammalian head skeleton can be considered to have three distinct functional roles: food gathering and processing, maintenance of respiratory flow and support and protection of associated soft tissues. Typically, investigations of skull mechanics focus on the jaw apparatus, but all structures of the cranium are fully integrated and are thus subject to and respond to forces exerted by soft tissue action, resulting from both mechanical and developmental processes (Russell and Thomason 1993). Thompson (1942) highlights this inter-relationship of form, function and growth, stating that form is the outcome of a mechanical interaction between two or more structures, which can act to limit or stimulate growth. Form, in this context, can thus be understood as a product of the mechanical loads placed on anatomical components and related structures throughout growth and development, manifesting both temporal and causal associations with function. This premise, known as Wolff's law, has been investigated and adapted to reflect current research by Pearson and Lieberman (2004). The development and acceptance of this functional paradigm has had a direct impact on the study of cranial morphological variation of both normal and pathological derivation, by essentially allowing the skull to be viewed as a dynamic framework, rather than a static structure.

Although no unitary theory has been constructed integrating cranial development, biomechanics and evolution, models adapted from the field of mechanical engineering

are often applied to mandibular/masticatory apparatus and facial form interpretation with some success. That being said, models describing post-orbital structures (i.e., the neurocranium and basicranium) are less common and generally less precise. Greave's (1985) model, though over-simplified, is one of the more useful paradigms for understanding cranial morphological variation, and it is particularly helpful in visualizing cranial dynamics. He envisions the skull as an idealized cylinder, with the rostral portion, consisting of the basisphenoid and vomer, acting as a torsion-resisting strut along the midline. Asymmetric forces applied through unilateral (differential) loading will therefore twist the brain case, resulting in variations in neuro- and basicranial form. This model is useful as an heuristic device for understanding the overall effect of mechanical stress on the skull, but it cannot provide an in-depth understanding of the multiple factors that work to shape the skull. A more rigorous approach is outlined in Moss and Young's (1960) and Moss's (1997a, b, c, d) publications concerning functional craniology, which attempt to explain the variation of the form of the skull by incorporating developmental/genetic factors as well as mechanical loading.

### 2.3.2 Moss's Functional Cranial Matrix Hypothesis

The initial concept of a functional cranial model can be traced to van der Klaauw's work during the nineteen forties and nineteen fifties. He proposed that: "the skull may be regarded as a complex of relatively separate functional components, sometimes detached, sometimes united in a morphological whole, but even in this case to a certain extent independent in size, relative position and grouping" (van der Klaauw 1948-1952, as cited in Moss and Young 1960). Moss and various colleagues have

expanded on van der Klaauw's ideas, initially introducing the concept of a functional approach to craniology during the nineteen-sixties. It is now generally accepted by physical anthropologists and researchers in other fields, such as orthodontics, as a plausible theoretical framework for cranial morphological studies.

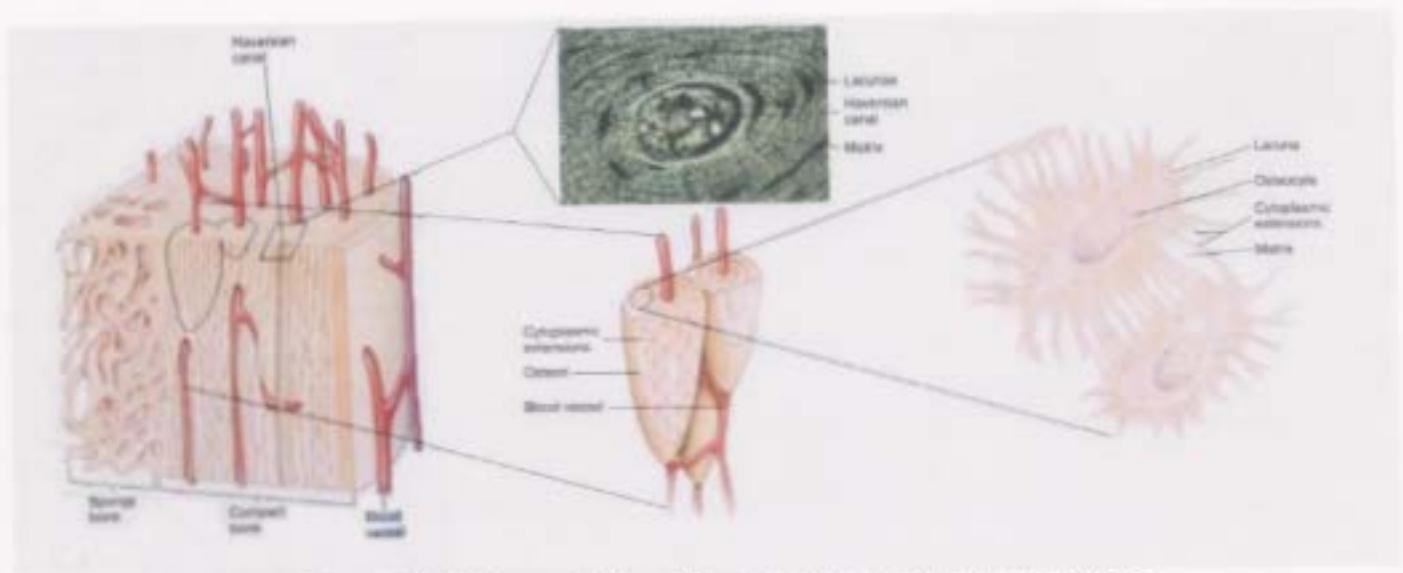
Moss recognized the lack of a solid theoretical foundation upon which to base interpretive studies of cranial morphological variation, stating that while the body of collected data was extensive, research regarding the biological import of the material was comparatively rare. He determined that the application of a functional anatomical analysis would be of benefit to the progress of the discipline of craniology, stimulating new analysis and interpretation. From Moss and Young's (1960) seminal paper, two concepts of particular relevance to this discussion emerge. First, that both normal and abnormal neurocranial development is a result of the interaction of the components of a functional matrix, which consist of the cerebral capsule, the skull base and the meninges. Cranial form is thus a manifestation of the magnitude of neural tissue growth, limited and directed by the growth of the skull base and meninges. Equally important is the recognition that a functional unit is not constrained to follow the boundaries of bone elements; that is, a single bone may be part of two separate functional units along with all or portions of any number of other bones. Subsequent research in the fields of complexity and complex biological systems has allowed the construction of a theoretical framework, and advances in the study of skeletal genetics and biomechanics have allowed some idea of the mechanisms and reasons behind this hypothesis to be constructed.

Complex systems theory provides a framework for understanding the behaviour of complex biological systems, which are made up of several interacting elements. The

integrated nature of these elements, as in the case of a cranium composed of functional skeletal units, allows emergent, self-organizing events to influence morphological variability. Moreover, the high degree of integration among the organism, its genetic parameters and the external environment dictates the nature of the developmental responses, thus ultimately guiding cranial morphology. Many previous theories of development postulated serial, or linear, processing of genetic information and were thus deterministic, considering growth and development to be primarily under genomic (genetic) control. In his examination of development through the lens of complex systems theory, Moss rejects this, suggesting instead that epigenetic factors are more likely to mediate growth and development. The term epigenetic encompasses all extrinsic influences like mechanical loading and intrinsic factors, including all biomechanical, biochemical and bioelectric aspects of the intraorganismal microenvironment. These can act locally to affect a single structure or developmental event, or systemically, influencing large portions or even the entirety of the developing organism. Moss presents an eloquent argument citing principles of hierarchy, emergence and causation, describing growth and development instead as non-linear and resulting in a not-entirely predictable outcome based on its initial (genetic) conditions. Ultimately, he states that the genome acts at the molecular level to regulate the construction of the cellular constituents, but that the immediate causes of morphological variability are epigenetic factors. Only the interaction of both genetic and epigenetic factors provides sufficient and reasonable cause for morphogenesis and therefore morphological variability.

In an effort to apply the tenets of complex systems theory to the functional cranial matrix hypothesis, Moss (1997a, b, c and d) cites several mechano-cellular mechanisms

through which extrinsic factors can impact bone growth and adaptation, specifically mechanotransduction through an osseous connected cellular network (CCN). Mechanotransduction is, succinctly, the way in which cells are believed to sense external stimuli and transform that information into an appropriate cell response (Pearson and Lieberman 2004). Osteocytes serve as the primary mediating cells, connected to each other, the periosteum and the endosteum via canaliculi (see Figure 2.1). This CCN then transmits mechanical loading information to the living osteoblasts, which direct bone activity (i.e., quiescence, modeling, resorption or Haversian remodeling). Moss (1997a) goes on to outline several cellular mechanisms through which bone can be impacted at the cellular level by external environmental stimuli. In this fashion, through the application of complex systems theory and through new understandings of skeletal biology and cell biology, it becomes clear that a relationship exists among developmental factors and post-natal growth, as well as physical activity.



**Figure 2.1: Microscopic Bone Structure (Long Bone Cross-Section)**  
(adapted from Marieb 1995)

In the context of normal cranial growth and development, this discussion pinpoints an additional reason beyond developmental instability for the observed variability in morphology, and that is muscular activity. Variations in the functionality of muscles and muscle groups, even over synarthrotic sutures, must be compensated for in some way, specifically through bony structure modification. Even though there are only a few highly movable joints associated with the skull (i.e., temporo-mandibular joints and the occipito-cervical junction), interaction between the bony elements and soft tissues comprising the functional units dictate morphological variation in a patterned and describable fashion.

#### 2.4 Activity-Induced Asymmetry

Activity-induced asymmetry essentially refers to any dissimilarity in the morphology of bilateral structures caused by differential, that is, left-right, postnatal muscle use. Although the application of this sort of influence is a normal and expected occurrence (Ortner and Putschar 1985), it is possible to define it as low-intensity trauma, albeit of exceptionally long duration. It is generally accepted that the appendicular skeleton adapts dynamically to mechanical loading, and the information gleaned from an analysis of the pattern detectable on skeletal remains is a useful indicator of handedness and behaviour. It is, however, comparatively rare that activity-induced asymmetry is considered for the axial skeleton. Variation following an expected pattern, predicted based on limb use, has been demonstrated to exist in the clavicle, thoracic and lumbar vertebrae and the sacrum (Mays *et al.* 1999; Plochocki 1999; Stokes 1997), but cranial manifestations of non-pathological differential muscle use remain largely unexplored. It

is reasonable to postulate that activity would influence cranial form, particularly when considered in the context of the current trend in evolutionary biology towards integrated skeletal interpretation; that is, different parts of the skeleton cannot be examined in isolation from each other and the rest of the body (Pearson and Lieberman 2004).

The influence of handedness on the cranium is almost exclusively thought to manifest itself through asymmetric intracranial growth. The idea that brain-shape variations influence the shape of the skull is not new. Franz Joseph Gall recognized this in the early nineteenth century, and, while his ideas regarding phrenology were based upon erroneous assumptions and have since been disproved, the basic premise is sound (Lemay 1977). Due to the way in which cranial growth occurs, asymmetries of the soft tissue should be detectable in the skull. Recent research has indicated that several neocortical structures, particularly the planum temporale, planum parietale, the Sylvian fissure and the central sulcus, demonstrate left-right asymmetry reflective of hand preference. While outwardly promising, Steele (2000) reports that the correlation to cranial skeletal structure is inconsistent or uncertain at best, due to the small scale of the deviations involved. That being said, observations of the occipital region have shown potential for some populations (Myslobodsky *et al.* 1987; Steele 2000), but whether this is due to cerebellar asymmetry or muscular activity, or even a combination of the two, has yet to be determined.

Research focusing explicitly on the effects of muscular activity on the cranium is infrequently undertaken, outside of the context of mastication (e.g., Weishampel 1993). Anderson's (1983) examination of the basicranial region to determine the influence of differential limb use on the relative positioning of the occipital condyles is suggestive,

but unsupported by any subsequent research. Anderson postulated that subjecting the muscles in the suboccipital and neck region to persistent tension during growth could potentially result in the angulation of basicranial structures. For example, continual or frequent rotation of the cervical spine in one direction could transmit muscular tension in the neck to the base of the skull. Additionally, Myslobodsky *et al.* (1987), in their investigation of infant cranial asymmetry, suggested that bilateral differences in the morphology of the petrous temporal region, particularly the mastoid processes, might be due to differential muscle use. As well, they hypothesized that lack of muscle use in infancy might promote positional deformities or even preserve changes in cranial shape caused by intrauterine molding beyond early childhood, to the extent that some vestige of that asymmetry may be maintained into adulthood.

It is plausible that areas of the cranium acting as anchors to major muscle groups, or that underlie major muscles, may be apt to demonstrate asymmetry resulting from muscular activity due to the way in which bone behaves when subjected to biomechanical stresses. Bone reacts to mechanical loads at the cellular level, causing observable changes in bone density and geometry through four responses: quiescence, modeling, resorption or Haversian remodeling (Pearson and Lieberman 2004; Ruff 2000). The best understood bone response to mechanical loading is remodeling, which induces bone deposition and thus increases the cross-sectional area of the bone. This reduces the impact of stress by spreading it over a larger area, as well as increasing resistance to twisting and bending. The other major response to increased bone loading is Haversian remodeling. Osteogenic cells are activated and work to prevent or repair fatigue damage and to reinforce bone by arranging collagen fibres along lines of tension. Although not entirely understood, this

process is observed most often in older bone tissue. Quiescence (no response) and resorption are both results of consistently low or unusual decreases in mechanical loading. Resorption can occur when changes in the musculoskeletal system reduce stress in a localized area, for instance, through immobilization of a limb. The causative factors for both quiescence and resorption are unclear, but they may occur as a result of the removal of the epigenetic stimulation of the bone tissue (Ortner and Putschar 1985; Pearson and Lieberman 2004).

Based on the preceding information, and bearing in mind Moss's assertions concerning functional craniology, it is reasonable to hypothesize that bone tissue and structures may be adapting to differential loading, albeit on a smaller scale than the more mobile and weight-bearing parts of the skeleton, such as the pelvic girdle. Consequently, for an accurate understanding of cranial asymmetric variation, the influence of activity is an important consideration.

## **Chapter 3 Human Growth and Development**

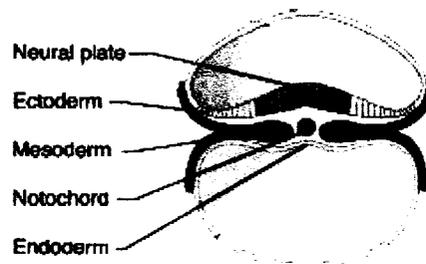
### **3.1 Introduction**

Simply put, human development involves the transformation from egg to phenotype, or the physical form, under genetic and epigenetic control. The development of the human skeleton from fertilization through birth is a delicate, complex process, and normal morphogenesis depends heavily on cellular activity and the timing of events. Any unusual morphology, whether minor variability or a genuine developmental defect, can occur if there is a delay in the occurrence of a “threshold event”. The expression of a defect in a particular structure reflects a disturbance in a specific developmental field, or a portion of developing tissue specific to that structure, at a specific time during morphogenesis.

Of the approximately nine months required for complete growth and development of a foetus, the first nine weeks are particularly relevant to skeletal morphology (Johnson 1988). The early stages of development essentially involve the establishment of the embryo and basic cellular organization, wherein cells form layers that proliferate and interact, eventually becoming distinct tissues and organ systems (Heggeness 1995).

Cells are an important part of the developmental mechanism that interfaces genotype and phenotype. This is due to what Chandebois and Faber (1983) termed “cell sociology”, a concept that describes the interactions among similar and dissimilar cells at several levels of organization and for the accomplishment of an array of tasks and functions. In early embryogenesis, interaction occurs among individual cells; as the complexity of the developing embryo increases, interactions begin to occur among

groups of like cells in the same germ layers, and groups of dissimilar cells in adjacent germ layers (see Figure 3.1). This process is known as embryonic induction, and it involves the differentiation of cells whose growth and development is dependent on the progress of adjacent structures in a sort of domino-like effect. As tissue differentiation and morphogenesis proceeds, dissimilar cell condensations interact to initiate and maintain these increasingly complex developmental processes, which eventually lead to the production and integration of tissues and organs into a functioning embryo (Hall 2003). Development then proceeds through the remainder of the embryonic and foetal stages until parturition.



**Figure 3.1: Germinal Layers in the Early Embryonic Development**  
(adapted from Marieb 1995)

The essential information to be drawn from this discussion is that, first, there is a strong reliance on cellular activity throughout development, and second, that groups of like cells can interact with each other to expand the sphere of cellular functionality. Cell condensations have both identity and coherence that is temporally and spatially specific which, coupled with the ability to interact through processes like migration, signaling and induction, allows the identification of morphogenetic units influenced by epigenetic processes. Because of this, cell condensation behaviours also provide an opportunity for the introduction of variability and defects. In the context of skeletal development, if a

condensation for an individual skeletal element is too small for whatever genetic or epigenetic reason, the resulting element will be small or aplastic. As well, any additional condensations that are developmentally linked through induction may be similarly affected. Alterations in the temporal or spatial behaviour of condensations can lead to any number of abnormal morphologies, including mild irregularities, developmental defects and congenital anomalies (Hall 2003; Willmore *et al.* 2005).

### 3.2 Normal Developmental Asymmetries

A general understanding of the developmental processes leading to the creation of the foetal skeleton is of key importance in gaining the ability to correctly interpret anomalous forms. The effects of mis-timed developmental steps range from dramatic and fatal to mild and asymptomatic, but the overall impact, or likelihood, of these occurrences is mitigated by the stability of the genome and the developmental environment. Even in the absence of recognizable developmental anomalies, variability within each individual population member is present to some extent. While perhaps not of phenotypic significance, this fluctuating asymmetry is detectable and has the potential to be a useful indicator of overall developmental stability in both the individual and the population.

#### 3.2.1 Fluctuating Asymmetry

Examining developmental stability can provide valuable information about the genetic character and the environmental conditions to which a given population is subjected. A means of gauging this ability to resist random errors during growth and development could, as Moller and Swaddle (1996) suggest, provide a general health

certificate for an individual and perhaps even the population. Fluctuating asymmetry provides a quantifiable option for examining this, through the analysis of random deviations from perfect symmetry in bilaterally paired traits. It is thought to occur when an organism is unable to undergo a stable developmental process due to some array of environmental stressors and genetic factors, and the perturbations occurring at that time visibly impact on the morphology of the adult human skeleton (Benderlioglu and Nelson 2004; Simmons *et al.* 2004). It is also possible that asymmetry is created at the molecular level, possibly through random variations in the rates of cellular processes affecting communication and growth, or through inherent molecular asymmetry translating into structural asymmetry via developmental pathways (e.g. Brown *et al.* 1991). Although it is generally agreed that fluctuating asymmetry is present in most plant and animal species, its causes and its applicability to the study of developmental stability are controversial.

For the most part, fluctuating asymmetry, when examined under uncontrolled conditions, appears to be a non-specific indicator of environmental stress impacting a developmentally susceptible individual or group. In the context of environmental stress, fluctuating asymmetry is essentially a measure of available energy. Reductions in energy due to adverse conditions negatively affect maintenance, growth, survival and reproduction, increasing the frequency of asymmetric phenotypes (Moller and Swaddle 1996). Potential stressors, that act to lower the level of available energy, are widely varied and numerous. They can work singly or in concert, influencing both pre- and post-natal life, and include restricted food (quality and quantity), lack of water, adverse climate, illness and disease, pollution (natural or human-created) and population density (Benderlioglu and Nelson 2004; Lens *et al.* 2002; Moller and Swaddle 1996; Mooney *et*

*al.* 1985). The importance of these factors can be influenced heavily by seasonal considerations as well, suggested by studies like Benderlioglu and Nelson's, who noted that late winter and spring births have been shown to have higher levels of fluctuating asymmetry in less industrialized groups.

Moller and Swaddle's meta-analysis of the research discussing developmental stability suggests that there is a small but significant genetic component to fluctuating asymmetry across several species. While they list several possible genetic causes, the most relevant to the discussion of human fluctuating asymmetry is the loss of genetic variation within a single group. Essentially, this loss increases the overall homogeneity of the population, both phenotypically and genetically. Samples drawn from areas of low population density are generally considered to be less able to resist changes in their environment, due to a more limited range of adaptive responses. Such groups would therefore be expected to have higher levels of asymmetry or lower developmental stability (Benderlioglu and Nelson 2004; Moller and Swaddle 1996). That being said, a small number of studies indicate that, after a period of intense stress resulting in selective mortality, developmental stability increases, due to selection against some genotypes (Moller and Swaddle 1996). Thus, in the absence of other sources of information, the degree of fluctuating asymmetry can only be somewhat suggestive of the genetic character of the population.

### 3.2.2 Fluctuating Asymmetry, Directional Asymmetry and Antisymmetry

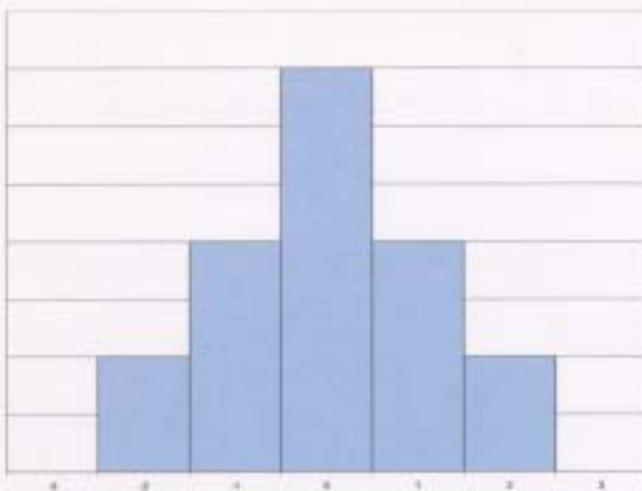
In naturally occurring populations, that is, those not examined under controlled or manipulated conditions, it is often impossible to isolate which type of stress is causing

the observed asymmetry. To further complicate interpretation, fluctuating asymmetry is not the only sort of asymmetry that can be present in a population. Directional asymmetry is a normally occurring variation, unrelated to activity, in which there is a propensity for one side of the trait always to be more highly developed. Although this type of asymmetry is present in soft tissue structures like the liver and lungs, it is not consistently present in the skeleton. The non-random nature of this asymmetry makes it reasonable to think that it is not indicative of developmental instability, but it has been suggested that the magnitude of the variation could be affected by instability. It is important to distinguish directional asymmetry from asymmetry caused by differential limb use. Directional asymmetry is a genetically mediated variation that is somewhat predictable, while the asymmetry caused by activity develops over time through the effects of muscular use and bone remodeling processes. Antisymmetry is similar to directional asymmetry, since it, too, is the exaggeration of one side of a bilateral trait, but it differs in the degree of randomness; the affected side cannot be predicted. It is important to note that neither directional nor antisymmetry have been consistently demonstrated to be present in the human skeleton, in either skeletal or soft tissue structures (Lens *et al.* 2002; Moller and Swaddle 1996).

### 3.2.3 Fluctuating Asymmetry and the Normal Distribution Curve

Fluctuating asymmetry and its appearance in a population sample can be most easily envisioned through the use of a normal distribution curve (Figure 3.2). In a normally distributed population examined for a given measured trait, that is, a population without any unusual left-right variation, individuals should essentially fall within the area

delimited by a normal curve. Those individuals demonstrating some degree of developmental instability for that trait are located further from zero, or perfect symmetry. If the apex of the distribution curve is greater or less than zero, the most common form may not be symmetrical. Similarly, if there are significant tails (skewness), there may be an important number of individuals who deviate from symmetry, but not a high enough number to significantly alter the definition of the most common form (Figure 3.3). In this way, very simple characteristics of a trait distribution among members of a sample can provide information about fluctuating asymmetry, and through this, about developmental stability and variations in the internal (maternal) and external environments (Lens *et al.* 2002; Moller and Swaddle 1996; Simmons *et al.* 2004).



**Figure 3.2: Normal Distribution Curve**



**Figure 3.3: Normal Distribution with Significant Positive Tail**

Fluctuating asymmetry is infrequently investigated in the context of human cranial morphology. The subtle nature of the changes observable in the skull make it difficult to achieve a clear understanding of the trends present in left-right morphological variation in nonpathological cases of asymmetry, but certainly not impossible.

Biologically significant information can be gleaned from a population sample level of analysis concerning developmental stability and environmental/genetic stress, even if the exact nature of the stressor must be determined from other sources, such as analysis of infracranial skeletal material or historical records (Moller and Swaddle 1996).

In addition to the subtle variation caused by fluctuating asymmetry, there are many developmental and congenital conditions that can induce cranial asymmetry of a more obvious kind. The skull is uniquely plastic in terms of how much its morphology can be affected by disease processes, soft tissue modifications and external forces. Permanent bony changes can be indicative of changes in function of some part of the body, whether the cause is genetic, muscular or environmental.

### 3.3 Pathological Asymmetries: Developmental and Congenital Defects

Pathological asymmetries are essentially morphogenetic defects, caused by a variety of factors, and include both developmental and congenital anomalies. In the context of cranial asymmetry, defects impacting the skull and upper cervical vertebrae, such as border shifts, irregular occipital condyle morphology and congenital muscular torticollis, are of primary relevance.

The skeleton, notably the skull and upper cervical vertebrae, is one of the first systems to appear in the developing embryo and complete *in utero* formation, usually by the end of the fourth month. Bone tissue forms through two mechanisms of ossification and through three phases of development. The blastemal phase is essentially an organizational period, during which cell and tissue precursors develop and move to the appropriate location prior to either chondrification or ossification. Bone can then be

produced through membranous or cartilaginous ossification, and both forms contribute to the growth and development of various parts of the skull. Cartilaginous (endochondral) ossification requires the development of a cartilage model before bone can form. In addition to the vertebral bodies and the base of the skull, all long bones and the pelvic girdle develop in this manner. Conversely, membranous ossification proceeds through only two phases, moving directly from the blastemal precursor to bone formation, and this type of ossification is responsible for the development of the flat bones of the skull and some of the bones of the facial skeleton (Barnes 1994).

Developmental defects are the result of mistakes or interruptions during morphogenesis. They range from very minor disturbances to major, potentially fatal, abnormalities; fortunately, most of these changes are minimal and can remain asymptomatic for most of one's life. Etiologically, developmental defects are multifactorial, mediated by intrinsic (genetic) and extrinsic (environmental) factors in an epigenetic interaction. Essentially, this means that certain individuals will be genetically sensitive to a particular defect, and that actual expression of that defect can be influenced by previously described environmental factors. A defect occurs when a specific stage of morphogenesis is reached, usually a time of rapid cellular change (i.e., differentiation, proliferation). The most common disturbance is a delay in the timing of a "threshold event", resulting in hypoplasia (underdevelopment), aplasia (non-development) or, in rare cases, hyperplasia (over development). The expression of a particular defect in a specific region of the body reflects a disturbance in a particular developmental field, or portion of developing tissue specific to a structure, during morphogenesis. Variability in expression is determined by the timing of the interruption of the threshold event (Barnes 1994).

There are numerous developmental fields in the axial skeleton alone, but only those responsible for the cranial base, neurocranium and upper cervical vertebrae are of relevance to this discussion. These are the paraxial mesoderm, prechordal cranial base and blastemal desmocranium. Although the facial skeleton and mandible are also important, the developmental defects in this area have less impact on overall cranial morphology and were not observed to be present to any great extent in the Maritime Archaic or European skeletal samples.

### 3.3.1 The Cervical Vertebrae and Paraxial Mesoderm Defects

The atlas and axis, as well as the exoccipitals and supraoccipitals, develop from columns of developmentally active mesenchymal tissue oriented parallel to the notochord, the vertebral column precursor. These columns subdivide into paired somites, the four most cranial of which go on to form, among other structures, the first and second cervical sclerotomes. The cranial half of the first cervical sclerotome (bone-forming unit) forms part of the exoccipitals and the tip of the dens, while the caudal half is responsible for the anterior and posterior arches, the lateral masses of the atlas and the body of the dens. The remaining structures of the axis are derived from the second cervical sclerotome (Johnson 1988; Tyrrell and Benedix 2004). Of the numerous defects associated with the paraxial mesoderm field, cranial-caudal border shifting and basilar impression have the greatest potential to cause or exacerbate cranial asymmetry in this context.

The human vertebral column normally consists of seven cervical, twelve thoracic, five lumbar and five fused sacral vertebrae (Marieb 1995). Variations in both the number

and regional distribution can be due to border shifting, a developmental defect wherein the affected vertebra is morphologically similar to the vertebrae in an adjacent region (i.e., cranially or caudally), depending on the direction of the shift (Barnes 1994).

The occipitocervical border is a very vulnerable region during development, susceptible to both cranial and caudal shifting of sclerotomes. A possible cause is suggested by Barnes (1994), who states that border shifting in this region may be due to delays in formation of the vertebral developmental unit, affecting neural arches and the occipital vertebral body. The comparatively high degree of susceptibility may also be due to the complex and irregular nature of the development of the first cervical vertebra. The atlas (C1) develops from three chondrification centres, one for each of the lateral masses, which later fuse to form the posterior arch, and one for the anterior arch (Tyrrell and Benedix 2004). Additionally, the second cervical vertebra, the axis, is also forming in an equally irregular fashion. Thus, this ontogenetically restless zone can demonstrate both cranial and caudal border shifting of variable nature and severity (Taitz 2000).

Cranial shifting, the least common border shift, can still have a noticeable impact on this region. While occasionally involving the formation of an occipital vertebra, that is, an “extra” cervical vertebra, manifestation ranges from small protuberances, like precondylar tubercles, located anteriorly to larger bony processes that distort the rim of the foramen magnum. Caudal shifting, the more common border shift in this region, essentially involves the incorporation of the atlas into the occipital bone. This can involve a partial or entire vertebra, and can be symmetrical or asymmetrical. Additionally, paracondylar processes may also develop, either alone or in conjunction with occipitalization of the atlas. These cone-shaped masses of bone appear immediately

lateral to the occipital condyles. For the most part, the morphological variations caused by border shifting in this region are fairly localized and thus affect overall cranial asymmetry in a limited fashion (Barnes 1994). That being said, the effects on the growth of the entire cranium can be quite dramatic for more severe manifestations, as in, for example, the case of an occipitalized atlas.

The final paraxial mesoderm defect that can potentially cause cranial asymmetry is basilar impression, a specific form of caudal shifting (Barnes 1994). Basilar impression can be due either to this caudal shifting or to any other condition that softens bone, such as rickets or osteoporosis. Clinically, there are visible deformities of the face, head and neck, as well as neurological symptoms, such as weakness and paresthesia of the limbs, caused by bony interference in the spinal cord and lower brain (Hughes and Sundaresan 1998).

Skeletal involvement associated with basilar impression varies in severity. The mildest form is an elevation of part of the bony rim of the foramen magnum into the cranial cavity (Bland 1994; Douglas 1988). A slightly more severe manifestation occurs when the occipital region associated with the atlas is depressed, the foramen magnum looks small with upturned edges and the petrous portion of the temporal bone is pushed upwards (Barnes 1994). Finally, the odontoid process of the axis can be displaced posteriorly, causing progressively worse neurological symptoms by impinging on the spinal cord, often leading to death of the patient (Barnes 1994; Douglas 1988). As well, basilar impression can frequently involve occipitalization of the atlas (Hughes and Sundaresan 1998).

### 3.3.2 Cranial Base and Neurocranium

Although the skull is formed by both membranous and endochondral-derived bone, the prechordal cranial base primarily undergoes endochondral ossification. This region includes the basioccipital and anterior portion of the occipital condyles, the petrous temporal bones, parts of the sphenoid and ethmoid and the supraoccipital. This part of the skull will begin to ossify in the third foetal month (Barnes 1994; Johnson 1988). Most of the neurocranium develops from the blastemal desmocranium, which ossifies directly from the blastemal stage without forming a cartilage precursor. The frontal, parietal, medial occipital and squamous temporal bones are identifiable by the end of week four, and ossify during weeks seven to nine (Barnes 1994).

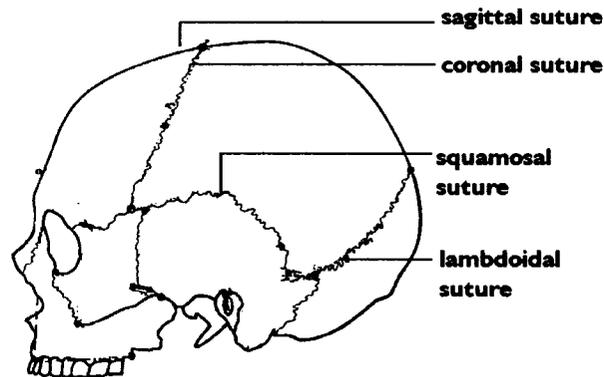
Unlike the majority of cranial structures, the prechordal cranial base develops from a cartilage model, rather than ossifying directly from mesenchymal condensations. The basioccipital and the anterior portion of the occipital condyles ossify from several cartilaginous aggregates, and a delay in development of any part can lead to the hypoplasia or aplasia of any or all parts of the structures formed. Defects in the parachordal cartilages are likely related to achondroplasia, but can also occur as an isolated field defect and can significantly influence the shape of the foramen magnum. For example, a lop-sided foramen magnum can be due to unilateral asymmetric aplasia of basioccipital structures (Barnes 1994). Interestingly, in Snow's analysis of a prehistoric Hawaiian population, a high proportion of individuals demonstrated an unusually asymmetric foramen magnum, suggesting a possible genetic component to defects in this field (Barnes 1994; supported by Douglas 1988).

The blastemal desmocranial field includes the frontal, parietals, superior occipital and squamous temporal bones, as well as the greater wings and pterygoid processes of the sphenoid (Barnes 1994). Due to the way in which normal cranial growth occurs, sutural agenesis is the defect most likely to influence the degree of cranial asymmetry significantly.

Sutural agenesis, or craniosynostosis, results in a failure to differentiate opposing cranial bone precursors, and this can lead to a partial or complete coalescence of cranial bones at any time after birth. It is believed that both genetic (autosomal linkage) and external factors, such as intrauterine infection, birth trauma, or metabolic disorders (Barnes 1994) mediate the manifestation of craniosynostosis. Essentially, the premature fusion of one or more calvarial sutures (shown in Figure 3.4) either in isolation or as part of a polytopic syndrome, results in deformation of the cranium, especially the vault and facial complex (O'Loughlin 1996). Skeletal involvement is mediated by which sutures are fused and at what age this occurs (Ortner and Putschar 1985). Endocranial structures continue to grow normally in spite of the constraint of early fusion, forcing compensatory overgrowth of bony tissue at unfused sutures (Douglas 1988; O'Loughlin 1996).

The skeletal changes resulting from craniosynostosis fall into three categories: scaphocephaly, brachycephaly and plagiocephaly. Scaphocephaly, the most common form, particularly among males, results in a long, narrow vault and is due to sagittal suture fusion, whereas brachycephaly, occurring predominantly in females, is due to premature fusion of the coronal suture. Brachycephaly results in a rounded vault with a high forehead. Plagiocephaly is due to the fusion of any one or more sutures, and creates an asymmetric or parallelogram-shaped cranium. In addition to the direct evidence of

premature fusion, bone ridging along suture lines may also occur as an indicator of this pathology (Douglas 1988).



**Figure 3.4: Sutures Commonly Affected by Sutural Agnesis**

Essentially, developmental defects are caused by disturbances in morphogenesis. They are multifactorial in etiology, and their expression is mediated by the timing of the interruption of threshold events. Pertinent to the study of cranial asymmetry are the developmental defects affecting the paraxial mesoderm, prechordal cranial base and the blastemal desmocranium. Each of these conditions has distinctive features that facilitate diagnosis from skeletal remains.

### 3.3.3 Congenital Causes of Asymmetry

Unlike developmental defects, congenital conditions have more variable etiologies, and may not be due to interruptions in morphogenesis. Due to both genetic and environmental factors, congenital conditions are present at or before parturition, and, as a group, they represent a major cause of mortality. Many congenital conditions are non-

lethal, varying in severity from mild, asymptomatic skeletal variation to more debilitating malformations (Turler 1989). The sole congenital causative factor of cranial asymmetry that will be discussed here is congenital muscular torticollis, a condition of ambiguous etiology that has a marked deformative effect on the cranium.

The primary congenital cause of cranial asymmetry most often cited in archaeological literature is congenital muscular torticollis. It is commonly believed to be caused by trauma associated with parturition, by some genetic factor or by a combination of both. The basic process involves the inducement of lesion formation in the sternocleidomastoid muscle during pregnancy due to foetal position or at birth. The lesion manifests as a dense, palpable mass ten to thirty days after birth. It eventually becomes a fibrous mass of scar tissue, inhibiting growth of the muscle while skeletal growth proceeds uninterrupted. This initially causes a pulling sensation in the neck, followed by an enforced tilt of the head to the affected side (Douglas 1988). Within six weeks, deformation of the bony structures of the face begins, and this progresses into the asymmetry described below. Torticollis is a symptom of many different conditions or injuries, such as blindness in one eye or a lax atlantl ligament, but each of these pathologies have other distinctive characteristics, allowing differentiation from congenital muscular torticollis (Skinner *et al.* 1989). There are several potential causes of this condition, but the most likely involve some characteristic of the uterine environment or birth trauma. Skinner *et al.* (1989) present a literature analysis discussing this, and suggest that the lesion may be caused by complications at birth, such as breech positioning, Caesarian section or forceps-assisted delivery, or by malposition *in utero* (Tien *et al.* 2001).

In congenital muscular torticollis, the cranium is modified almost exclusively by the action of the constricted sternocleidomastoid muscle. According to Douglas (1988), the parietal bone on the affected side is flattened, the occipital bone bulges contralaterally, the occipital condyles may be uneven and rotated, the affected eye is lower and the facial complex is shortened vertically and is broader on the affected side. There is no bias with respect to side affected (Tien *et al.* 2001). Congenital muscular torticollis is a favoured diagnosis for many of the reported cases of cranial asymmetry in archaeological literature (Kidd 1954; Douglas 1988; Skinner *et al.* 1989; Smrcka *et al.* 1986).

The developmental process is extremely complex, requiring accurate timing of key events and specific, stable environmental conditions. Mistakes in development can lead to the formation of a plethora of defects, ranging from mild to inevitably fatal; the defects discussed in this chapter are only a few of those found in the cranium and cervical spine. Congenital conditions, caused by an as yet undetermined combination of genetic susceptibility, environmental conditions and trauma, are equally important, and, like developmental defects vary markedly in severity. Although emphasis has been placed on defects and conditions that cause noticeable pathology, it is important to remember that they are usually quite rare in a typical population.

## **Chapter 4    Methodology and Materials**

In an effort to appropriately situate this thesis within the body of previously conducted research concerning cranial asymmetry, a brief review of both historical and contemporary studies is required. The skeletal samples used in this work have been, and continue to be, the focus of a diverse array of research projects. An introduction to these unique cultural groups is therefore needed to properly contextualize the results and discussion. Finally, the data collection methods and procedures used for both standard and asymmetry measurements will be described in this chapter, and the functional cranial model, expanded upon in Chapter 5, will be briefly introduced.

### **4.1    Past Research and Methodological Background**

Research devoted to cranial asymmetry has been undertaken at various times, with varying degrees of success. One of the first investigations attempted to define a normal “type” skull for a particular population, eventually determining that asymmetry and a high degree of variability in form was the norm for that population (Woo 1931). Subsequent research (e.g., Elderton and Woo 1932; Pearson and Woo 1935; Woo 1937) used complex statistical analysis and impressively large samples to explore morphological variation between genetically distinct groups. However, while acknowledging the applicability of their method to the study of growth and development, these anthropologists often avoided interpreting their results or still attempted to work within an ethnometric context. After World War II, research concerning cranial asymmetry appeared sporadically in the anthropological literature, but it was not until the

late 1970's and 1980's that it was truly the focus of study (e.g., Anderson 1983, Douglas 1988, Lemay 1977).

#### 4.1.2 Early Research

The most thoroughly documented early work related to cranial asymmetry was undertaken during the nineteen-twenties and thirties by a group of statisticians in London, United Kingdom (Elderton and Woo 1932; Pearson and Davin 1924; Pearson and Woo 1935; Woo 1931). The eleven-year study involved approximately 1500 crania, predominantly of Egyptian derivation, and, of interest here, discussed cranial growth and development, using statistical analysis as the major interpretive tool. Possibly the most important aspect of this research is the almost complete absence of an oppressive race-oriented theoretical model directing the outcome. For the most part, ideas beyond growth and statistical analysis were left out of the published reports, allowing greater interpretive freedom for subsequent researchers.

The approach taken by Pearson, Woo and their colleagues involved the examination and measurement of each skeletal element separately, consciously attempting to avoid using what they termed "anthropometric" measurements, that is, those that covered large areas of the cranium or that measured across major cranial units. Over the course of several years, several interesting hypotheses concerning cranial growth and morphology were proposed. Pearson and Davin (1924) suggested that growth is the sole organic controlling factor regarding morphology. They went on to suggest that correlation between different skeletal elements would occur in homologous pairs or if a common "covering" factor exists; for example, the palatal index would correlate strongly

with the upper facial index. Pearson and Woo (1935) also determined that the absolute variation of a trait is not proportional to the absolute size of the element, and that rates of growth vary among individuals and, in terms of direction, within a single element.

Woo (1931) began an investigation into cranial asymmetry, applying sixty-three measurements, fifty of which were bilateral, to approximately eight hundred crania. He concluded that the “normal” type for the human cranium is, similar to many internal organs, asymmetric. He interpreted his data to suggest that there is a consistent right-side dominance in the cranium due to differential growth, or possibly mediated by some genetic control (he suggested race). Following from this research, Elderton and Woo (1932) investigated the distribution of individual measurements to determine if they are normally distributed in the sample; that is, was the population from which the sample was drawn normal for a given trait. They determined that the sample Woo used in his asymmetry research was not drawn from a normal population and that the relative skewness and kurtosis of a given trait becomes increasingly apparent as the sample size increases. Elderton and Woo noted that a normal distribution for a given trait was evident in medium-sized samples, but not in very small or very large samples.

Although this research was exceptional for its time in terms of the questions examined and the interpretations put forth, there are a few aspects of the research that require further consideration. One of the most important determinations involved the decision to focus on the measurement of separate skeletal elements out of context with the rest of the cranium. Pearson and Woo (1935) correctly noted that most ethnometric cranial measurements were taken between homologous bone pairs or across major units of the skull, such as the maximum cranial breadth, and therefore decided to approach

each bone separately. They constructed indices where appropriate and used statistics, particularly correlations, to compare between different groups of bones or different bones; for example, the relationship between adjacent bones, contralateral bone pairs or homologous bone pairs. The type of correlative analysis used did provide information concerning some of their research questions, particularly the examination of the mediating factors in cranial growth, but it did not make the regional, functional relationships within the skull explicit enough that Pearson and his colleagues could notice them. This is due to the lack of a theoretical framework from which to work, but, since Pearson and Woo were essentially breaking new ground, this should not reflect badly on the research outcomes. That being said, a reexamination of their work, particularly Woo's work on asymmetry, from a functional craniological perspective reveals new possible interpretations of their data.

Based on his measurements and analysis of correlations between individual skeletal elements, Woo (1931) determined that the right side of the cranium was, on the whole, larger than the left. He did, however, note that some left-side measurements were significantly larger than the right side, specifically the zygomatic ("malar") region, petrous temporal, maxilla and part of the sphenoid and lower occipital bone (Figure 4.1). The most significant right-side measurements were those in the vault and squamous temporal region, with some dominance in the face and superior-posterior occipital bone. Descriptively, this is reminiscent of the sort of muscle-mediated changes associated with congenital muscular torticollis, but Woo dismissed the pattern as being unimportant in the overall understanding of cranial morphology.

The theoretical background needed to fully understand the results of Woo's research did not become available until the nineteen-forties (Thompson 1942). In any case, the best research is the sort that stimulates more questions than it answers, and the work undertaken by Pearson, Woo and their colleagues was ground-breaking in both anthropology and biostatistics.

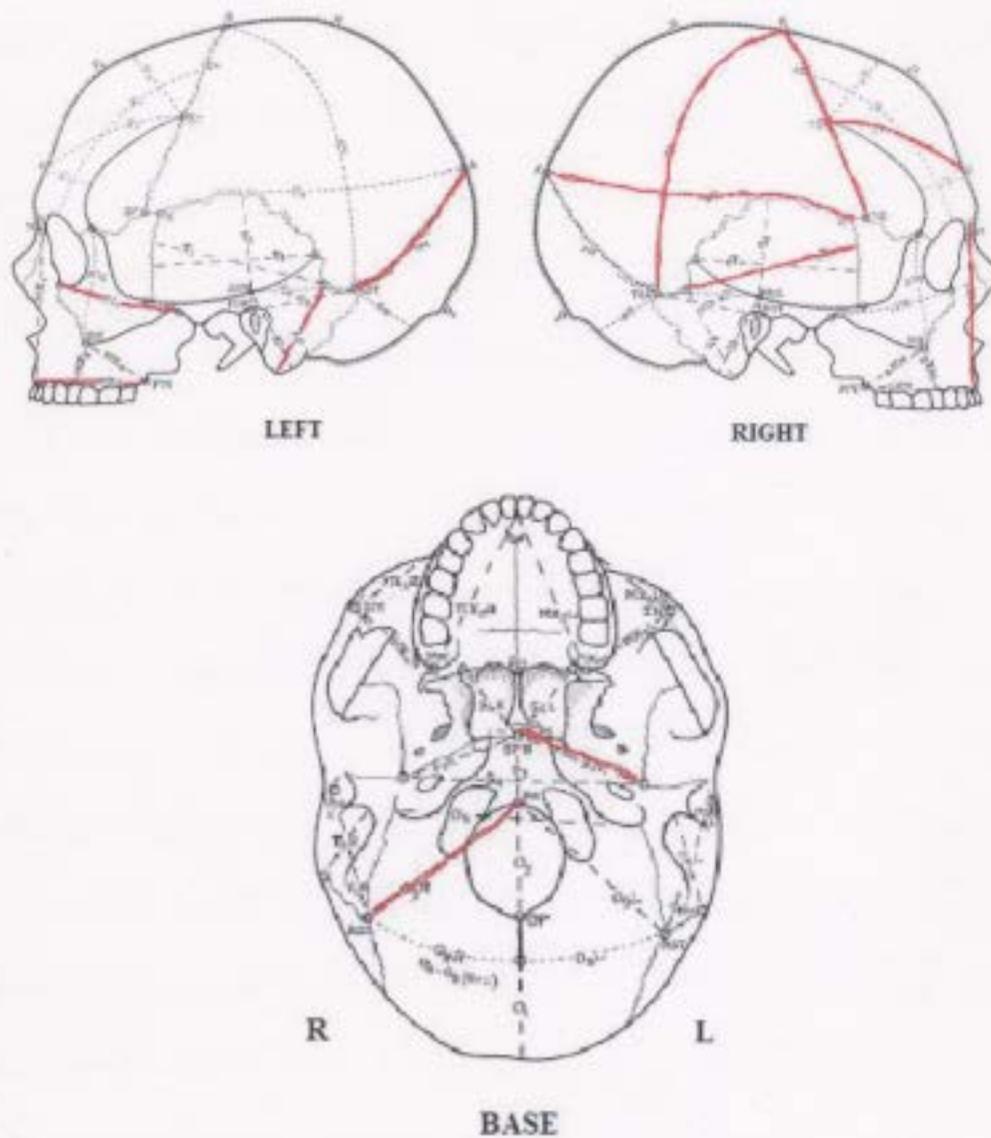


Figure 4.1 – Woo's Significantly Larger Measurements for the Vault and Cranial Base

#### 4.1.2 Recent Studies of Cranial Asymmetry

In the last sixty years, cranial asymmetry has rarely been mentioned explicitly in anthropological literature, and even less frequently analyzed by some quantifiable means. Reporting tends to take one of three routes: written description, measurements generated through imaging or via metric recording. One of the earliest, and certainly most exact, descriptions of cranial asymmetry involves a middle-aged male cranium from a Late Woodland site in Ontario, reported by Kidd (1954). He accurately describes the cranial form associated with torticollis. This descriptive approach continues to be a legitimate way of noting pathological cranial asymmetry in human skeletal populations (e.g., Smrcka *et al.* 1989).

In contrast, more metric approaches to quantification and interpretation have been applied to pathological instances of asymmetry. Computerized tomography and radiology have also been used with some success. Studies of this nature tend to involve only a few measurements, such as angles or widths, based on radio-opaque or some other pre-determined landmarks (e.g., Lemay 1977; Skinner *et al.* 1989). Direct analysis of dry bone, similar to Pearson and Woo's work, has also been undertaken, directed towards the investigation of specific research questions (e.g., handedness – see Anderson 1983). Of note is Douglas' (1988) work on an Hawaiian skeletal sample demonstrating a high frequency of pathological cranial asymmetry. She compiled a set of measurements designed to capture asymmetry and mathematically describe the observed pathology, but was ultimately dissatisfied with the metric aspects of the analysis.

The most appropriate method of analysis is highly dependent on the context of the remains, such as the number of pathological specimens within a sample or the availability

of a coherent population sample to examine. Even so, Pearson and Woo (1935) have demonstrated that analyzing an entire population sample, regardless of the presence of pathology, can maximize the information gathered and has the potential to suggest new avenues of research. Questions concerning base-line levels of asymmetry in different groups, the causes for this normal variation and the meaning of unusual frequencies of asymmetry remain open for analysis, and have the potential to provide insight into the factors influencing cranial growth and development in different populations.

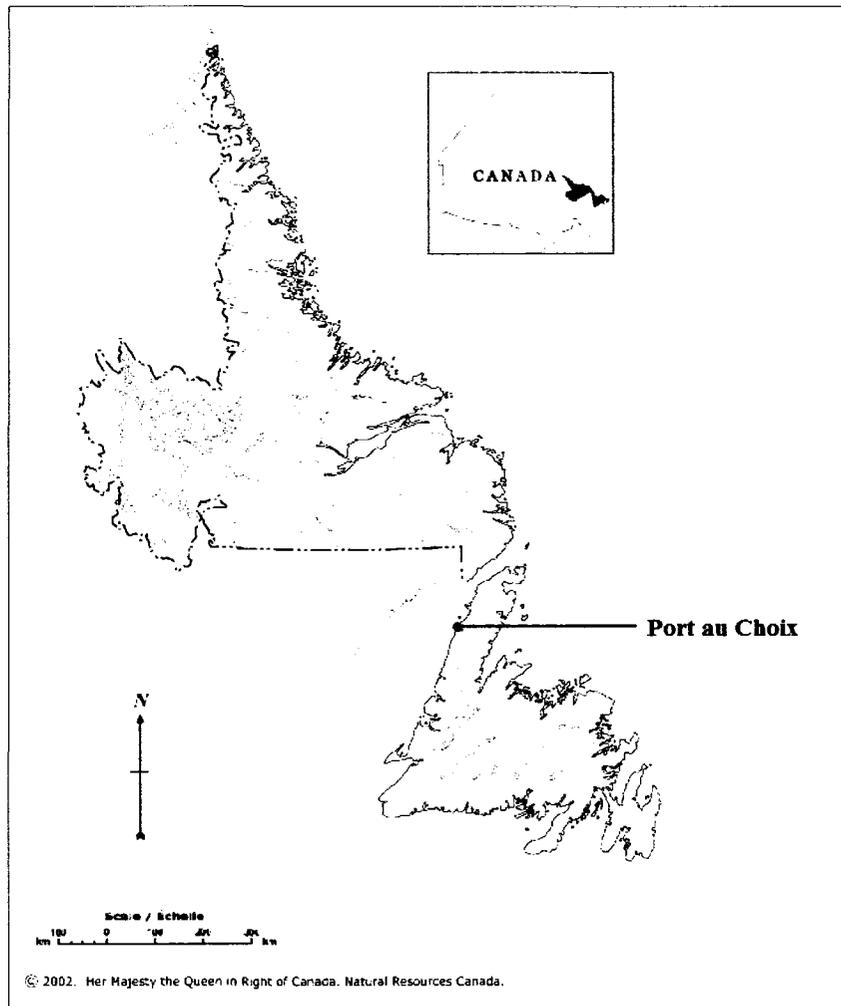
#### 4.2 Skeletal Samples: Excavation and Group History

The standard and asymmetry measurements that will be outlined in Section 4.3 were applied to complete crania, partial crania and mandibles drawn from the Newfoundland Provincial Museum human remains collection. In order for a partial cranium to be incorporated into this study, at least one functional unit had to be intact and not severely (i.e., visibly) distorted by post-depositional warping. From this collection, which is curated at Queen's College on the Memorial University campus, seventy-eight adult individuals were measured from Maritime Archaic and European groups, including both Basque and Colonial-era European samples. Although these two groups were combined in an effort to equalize sample size, each group has a distinct cultural and archaeological history that assists in setting the context for this research. To that end, the excavation and cultural histories of the Maritime Archaic, Basque and Colonial-Era European samples will be reviewed separately.

#### 4.2.1 The Maritime Archaic Sample

Located in Port au Choix (Figure 4.2), the Maritime Archaic cemetery was excavated primarily by Dr. J. A. Tuck and Memorial University from 1968 to 1970. Prior to this time, human remains were recovered or obtained by Elmer Harp throughout the nineteen-forties and nineteen-fifties, and it was Harp who initially identified the site as Port au Choix-3 in the early nineteen-sixties (Tuck 1976). The site consists of four separate loci, three of which (I, II and IV) are Maritime Archaic in origin. The unusually good bone preservation, due to an alkaline burial matrix, allowed over one hundred individuals to be identified and recovered (Jelsma 2000). The site, which would have been located on an island at the time of use, was radiocarbon dated to within a range of 4900 – 4400BP (Jelsma 2000; Tuck 1976).

Evidence for the Maritime Archaic tradition has been found on the northeastern coast of North America, in Nova Scotia, New Brunswick, Quebec, Newfoundland and Labrador, Maine, New Hampshire and Vermont (Jelsma 2000). In Newfoundland and Labrador, the Maritime Archaic developed as an adaptation to post-glacial era conditions approximately 8000 years ago, and disappeared as a distinct culture from the archaeological record around 3500BP (Tuck 1976). Although technically a hunter-gatherer group, their reliance on the sea as a resource results in a few important divergences from the classic model, outlined by Pálsson (1988). She suggests that, unlike the traditional small, highly mobile groups, maritime hunter-gatherer groups tend to have more permanent settlements and larger, more complex societies.



**Figure 4.2: Port au Choix, Newfoundland and Labrador**

In terms of resource availability, the Port au Choix people fit maritime hunter-gatherer pattern, preferring sandy beaches, protected coves and proximity to a river (Jelsma 2000; Pálsson 1988). Both stable isotope analysis and an analysis of the faunal remains found in the cemetery suggest that marine-based foods constituted a high proportion of the typical Port au Choix diet, as well as terrestrial mammals (e.g., bear, fox and caribou) and birds (e.g., auk and goose), presumably supplemented by available

plants and fruits. It is important to note that, according to pollen analysis, the vegetation and climate around Port au Choix has changed little over the millennia (Jelsma 2000). Of additional interest is Skinner and Newell's (2000) determination through an investigation of hypoplasia of the primary canine that maternal diet was, on the whole, uniquely sufficient compared to other ancient human populations. Thus, the overall resource availability for the Maritime Archaic of Port au Choix was adequate, and this is reflected in the general good health of the population sample.

Merbs' (1983) study of degenerative joint disease as an indicator of activity patterns in a Canadian Inuit group has provided an analytic framework for similar analyses in other hunter-gatherer populations, including Marshall's 1990 examination of the Newfoundland Maritime Archaic. The list of proposed activities, adapted from Merbs, includes general human characteristics like bipedalism, erect posture and side dominance, as well as culture-specific behaviour like harpoon/spear-throwing, hammering, lifting, paddling, cutting and sewing. This array of activities is further supported by the types of tools associated with the burials, which include tool kits for both terrestrial and marine hunting, wood, bone, antler and stone-working and sewing equipment (Tuck 1976). Even so, Marshall (1990) did not detect any significant side-to-side variation in the upper limb for males or females, and only a slight right-side dominance for males in the lower limb. This may be due to lack of activity-specialization seen in more complex societies, causing each individual to engage in several different types of activity, thus equalizing stresses on limb bones and, by extension, the manifestation of osteoarthritic lesions which would minimize skeletal evidence for unilateral loading. From both the archaeological record and osteological analysis, it is

apparent that the Maritime Archaic were involved in a range of behaviours and led generally active lifestyles, necessitated by the nature of their subsistence patterns.

#### 4.2.2 The European Sample

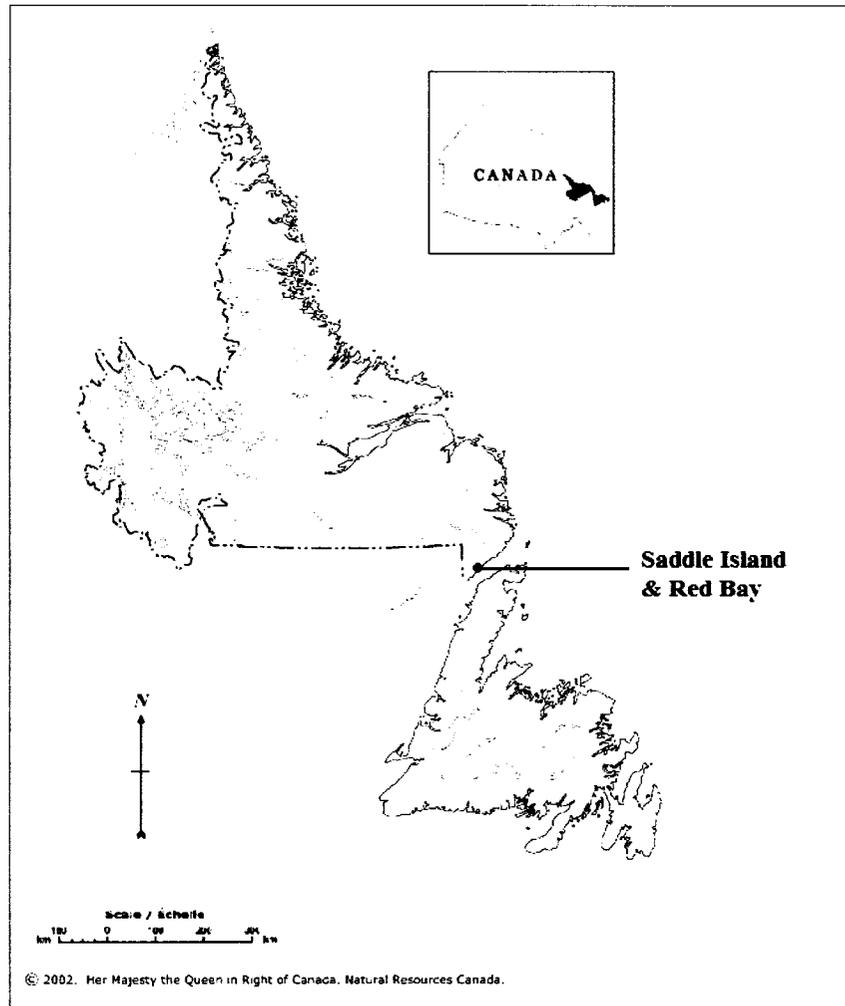
The European sample, consisting of forty-one individuals, was drawn from several sources, including the sixteenth century Basque cemetery on Saddle Island, Red Bay, and a series of eighteenth and nineteenth century burials distributed across Newfoundland. Due to their unique cultural history, this sample has been subdivided to facilitate description of the excavation, the recovered remains and any activity or behaviour specific to either the Basque whalers or the more heterogeneous Colonial European sample.

##### 4.2.2.1 The Basque Whalers

The Basque sample was excavated from a single cemetery on Saddle Island, Red Bay, Labrador by Dr. J. A. Tuck and Memorial University in the early nineteen-eighties (Figure 4.3) (Tuck and Grenier 1989). A Basque site in this location was initially proposed by Selma Barkham as a result of archival research, and then later verified by surveying the area (Proulx 1993). The Red Bay site is fairly extensive, consisting of shipwrecks, work buildings and living sites as well as a cemetery. Sixty-two graves were excavated, containing approximately 140 individuals, of which all but two are adult male Caucasians. Preservation of remains is extremely variable, due to both variations in depth of interment and differences in burial substrate, and some skeletons were almost completely degraded (Tuck and Grenier 1989). As a result, only about one-third of the

individuals recovered are curated, while the other two-thirds were reburied immediately following the initial excavation. The cemetery is thought to have been in use from approximately AD1540 to the late fifteen-hundreds, although some sources suggest a Basque presence in Red Bay as early as the end of the fifteenth century (for cod fishing) or the fifteen-twenties (Vasconcellos and Heyman 2002; Waddell 1988).

The Basques' traditional home is in the Pyrenees in southwestern France and northeastern Spain, and they are both culturally and, to some extent, biologically distinct from surrounding groups. They were among the earliest Europeans to begin whaling, possibly as early as the eleventh century, and are generally considered to have been technologically advanced in terms of both ship construction and whaling technique (Tuck and Grenier 1989; Waddell 1988). By the sixteenth century, the Basques had numerous shore stations bordering the Strait of Belle Isle, notably along the eastern shore of Red Bay and the southeastern side of Saddle Island (Tuck and Grenier 1989). The excavated site is significantly larger than most other stations that were in the vicinity, hosting more than fifteen ships at a time during peak usage, and it was occupied at least four months of every year (Rowe 1980; Tuck and Grenier 1989). During the early seventeenth century, Basque whaling activity gradually diminished, eventually ceasing altogether, due to a combination of dwindling bow and right whale stocks, war and power shifts in Europe, and new, more easily accessible whaling grounds around Spitsbergen, Norway (Proulx 1993; Tuck and Grenier 1989).



**Figure 4.3: Location of Basque Cemetery, Red Bay, Newfoundland and Labrador**

By virtue of their profession, the Basque whalers were an extremely active group, subject to a wide variety of activities and stresses. In addition to basic activities like bipedalism, the Basque whalers were involved in sailing-type activities (e.g., climbing and lifting), settlement activities (e.g., construction and woodworking) and whaling-specific activities (e.g., rowing, harpooning, rendering and butchering) (Proulx 1993; Tuck and Grenier 1989). Similar to the hunting and gathering Maritime Archaic, there is

a distinct lack of permanent trade specialization, since it is likely that a single individual might have many jobs during the four to six months of the year spent as a whaler, and another, potentially different, array of activities during the off-season. Although the excavated remains were characterized as robust and healthy, with no explicit evidence for cause of death (Tuck and Grenier 1989), it is likely that harsh living conditions, inconsistent nutrition, exposure to the weather and to pollutants released during the rendering process, in conjunction with an extremely high activity level may have increased physical stress levels, weakening the immune system or lessening long-term health in a way not manifest on the skeleton. In the context of osteological evidence, the Red Bay Basque skeletal sample can, however, be characterized as representing a healthy, active segment of the Basque population.

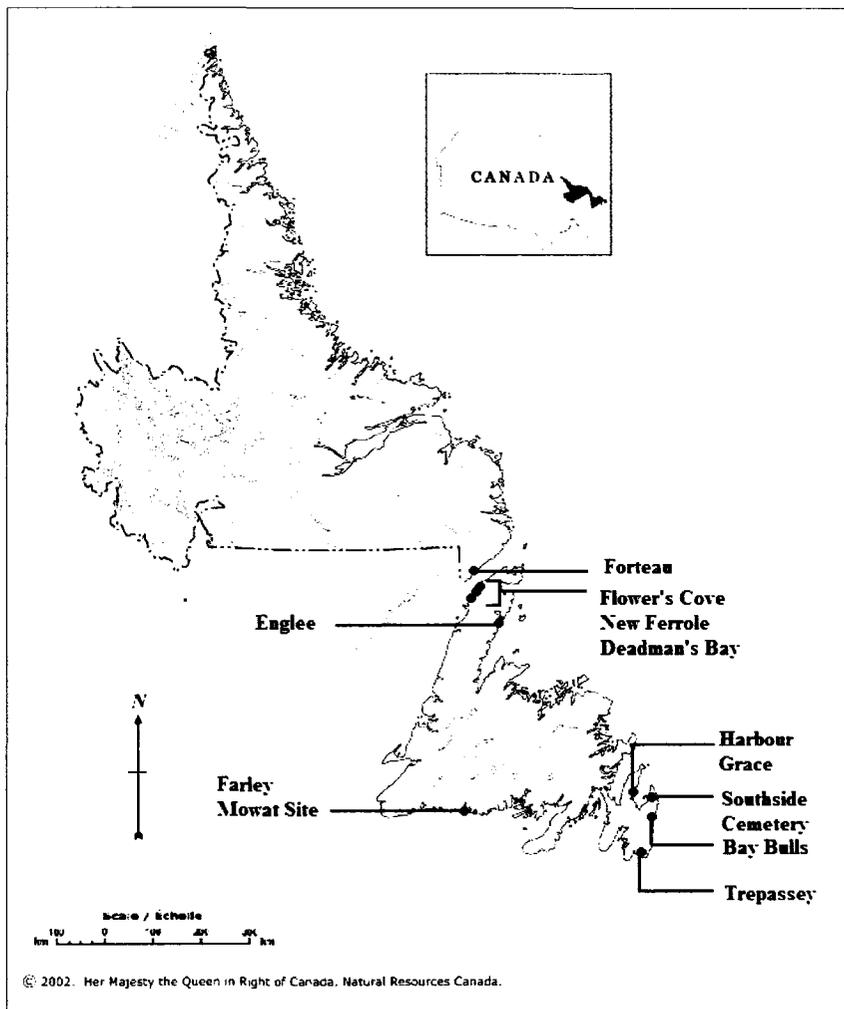
#### 4.2.2.2 The Colonial Europeans

Unlike the Maritime Archaic and Basque population samples, the colonial European sample is not from a single excavation site, nor was it excavated by a specific group of archaeologists. Preservation and overall completeness of the remains is therefore extremely variable. All remains do date from the eighteenth to early twentieth centuries, and approximately two-thirds of the twenty-four individuals chosen for this study were recovered from the Avalon peninsula (Figure 4.4). Of this number, eleven individuals were disinterred from the Southside Naval cemetery in St. John's, which is known to date to the mid- to late 1700's and to be of mixed English-Irish descent (Von Hunnius 1998).

This cultural mix is typical for most settlements in Newfoundland in the past two hundred years. According to Rowe (1980), there has been a continuous English presence

in Newfoundland from AD1610 until the present time. By 1650, there were approximately 2000 people living in fifteen settlements scattered from Cape Race to Cape Bonavista, along the English Shore (Prowse 1895). Although permanent settlers were not explicitly encouraged by the British government at this time, the overall growth and development of the colony was certainly impacted by British politics. The war with France (1689-1713) and the fluctuating nature of the fisheries production had particularly strong influence on the colony, with emigration to America and returns to England common responses to poor conditions in Newfoundland (Rowe 1980).

During the seventeenth century, there was a strong French presence in Newfoundland, concentrated on the area defined as the French Shore, which stretched initially from Cape Bonavista to Pointe Riche, shifting later to Cape St. John through Cape Ray. Prior to this time, French settlers were located from Cape Race to Placentia, as well as the coast of the Northern Peninsula down to Bonavista and the west coast (French Basques). The cessation of French colonization was due to the political situation in Europe and elsewhere abroad, causing France to officially cede Newfoundland to England at the end of the seventeenth century. The French presence was thus primarily restricted to offshore fishing by the end of the seventeenth century, decreasing dramatically throughout the eighteenth and nineteenth centuries and withdrawing entirely by 1904 (Janzen 1998).



**Figure 4.4: Locations of Recovered Colonial European Remains, Newfoundland and Labrador**

By 1730, the English-Irish settlements spread from Notre Dame Bay to the Burin Peninsula. This population growth was assisted by a large-scale immigration from Ireland, as well as economic crowding in the St. John's area, which encouraged migration north along the eastern coast in the late 1700's and early 1800's. The population of Newfoundland remained small and unstable for some time, but, supported by the expansion of the English fishery, American trade and the impediments to the migratory

fishery caused by various wars and political tension, it eventually began to grow in earnest, with populations reaching 20 000 during the 1790's and doubling by 1815 (Prowse 1895; Rowe 1980).

Immigration to Newfoundland occurred primarily from southwestern England and southeastern Ireland, resulting in a mixed English-Irish society by the end of the eighteenth century, and this formed the base for subsequent colonization and population growth (Newfoundland and Labrador Heritage 1997; Rowe 1980). The Avalon Peninsula was settled predominately by the Irish, for example, by 1753 all major communities were established and populated by Irish immigrants, and thus it is likely that most if not all skeletal remains in the Colonial European sample are of Irish-English descent. With the exception of one individual with developmental defects, the sample has a generally healthy appearance, with no obvious cranial or dental pathology or trauma.

#### 4.3 Data Collection Methods and Materials

Skeletal samples from the Maritime Archaic, Basque and Colonial European populations were examined and data were collected over a period of six weeks in May and June 2005 at Queen's College, Memorial University of Newfoundland. Each of seventy-eight individuals was described qualitatively as well as quantitatively, a process taking between thirty and forty minutes per skull depending on the integrity and completeness of the remains. Under ideal conditions, each skull was subject to twenty-four standard cranial measurements and thirty-five asymmetry measurements, each taken for both left and right sides. As well, six photographs were taken and a visual assessment of morphology was performed.

#### 4.3.1 Qualitative Description

Each skull and/or mandible was assessed in terms of general robusticity and morphology, post-mortem damage and visually discernable asymmetric variation. Overall size, shape of the frontal bone, rugosity in the nuchal and mastoid regions and the shape of the mental region were examined and recorded, both to verify previous sex assessments (see Jelsma 2000), as well as to give a general picture of muscle impact on the morphology of the skull. Post-mortem damage, whether due to crushing or disarticulation, post-depositional warping or poor reconstruction, was also recorded, in order to provide information on possible sources of error at the individual level and to provide a means of excluding overly-damaged remains. For the visual assessment of asymmetry, the base, face, vault and mandible were each examined separately, and any twisting, uneven mass distribution or flattening was noted. An overall subjective assessment of general cranial asymmetry for the more complete crania was also determined. An example of the data collection sheets used is included as Appendix A.

In addition to the written description of the remains, six photographs were taken of each skull, including the mandible, using a Panasonic Lumix® DMC-LC50 digital camera (3.2 megapixels). Left and right lateral and base views were taken, as well as a frontal view with the cranium held in the modified craniometer (see Section 4.3.2) when possible. Anterior and posterior views of the mandible, placed on a level surface, were also taken when appropriate. All photographs were taken using the modified craniometer as a sizing guide and from a distance of approximately fifty-two centimeters from the base to the camera.

### 4.3.2 Quantitative Description

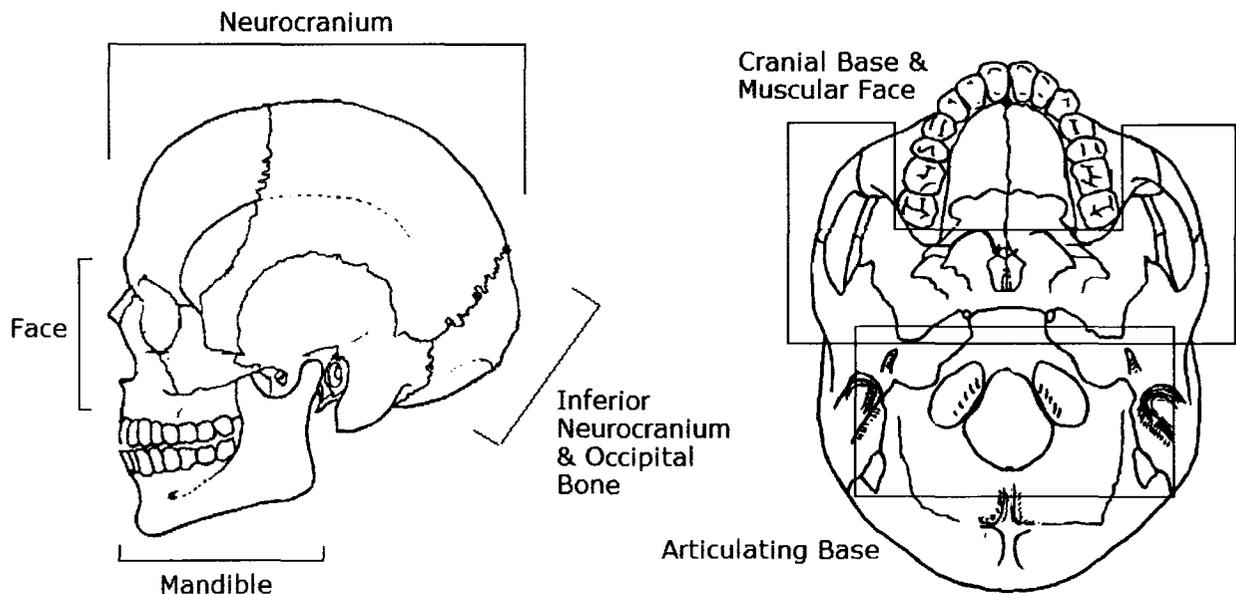
As a starting point, a set of twenty-four standard cranial measurements was taken for each cranium and mandible (Appendix B). Brothwell (1972) and Bass (1971) were used as references for these measurements, which are described in Table 4.1. The measurements chosen are those most commonly taken, and can be used to create descriptive indices, useful in characterizing the population samples, as well as providing base-line information for broader comparative purposes. The equipment required for each measurement is listed in Table 4.1, but for the most part, spreading calipers, sliding calipers and a mandibulometer are all that is required for the standard cranial measurements selected for this study. The asymmetry measurements make additional use of a coordinate caliper for fraction and subtense measurement and a tape measure for arcs (see Table 4.2). The data sheet used for the recording the standard and asymmetry measurements is included in Appendix A.

The development of the new set of asymmetry measurements for this thesis took into consideration previous metric analyses, particularly those conducted by Pearson, Woo (Elderton and Woo 1932; Pearson and Davin 1924; Pearson and Woo 1935; Woo 1931) and Douglas (1988), and used Moss's functional craniology (Moss and Young 1960) as a theoretical framework. Based on both Moss's work and subsequent studies of primate skulls (e.g., Daegling 2004; Wood and Lieberman 2001), as well as experimental studies of cranial component interaction (e.g., Hotye 1989; Persing *et al.* 1991), the cranium was divided into six functional units (Figure 4.5), the face, neurocranium, mandible, inferior neurocranium/occipital bone, cranial base/muscular face and the articulating base. Measurements were designed in this fashion to examine variation within a unit, as well as

the variation detectable by comparing functional units through statistical analysis. Ideally, this will elucidate the variation both in different regions of the skull, but more importantly, it will reveal the interaction of different parts of the skull with each other and with the infracranial skeleton.

**Table 4.1 Standard Cranial Measurements**  
(After Bass 1971; Brothwell 1972)

<b>Name</b>	<b>Code</b>	<b>Description</b>	<b>Equipment</b>
Max. Cranial Breadth	MCB	max. biparietal breadth	Spreading Calipers
Max. Cranial Length	MCL	max. glabella – opisthocranium	Spreading Calipers
Basion-Bregma Height	BBH	basion – bregma	Spreading Calipers
Bistephanic Breadth	BSB	stephanion – stephanion	Spreading Calipers
Bizygomatic Breadth	BZB	zygion – zygion	Spreading Calipers
Upper Facial Height	UFH	chord nasion – alveolare	Sliding Calipers
Total Facial Height	TFH	chord nasion – gnathion	Sliding Calipers
Palatal Length	PAL	chord staphylion – orale	Sliding Calipers
Palatal Breadth	PAB	chord bi-endomolaric	Sliding Calipers
Maxillary Breadth	MAB	chord bi-ectomolaric	Sliding Calipers
Maxillary Length	MAL	chord alveolare – staphylion	Sliding Calipers
Basion – Nasion	BAN	chord basion – nasion	Sliding Calipers
Basion – Alveolare	BAV	chord basion – alveolare	Sliding Calipers
Min. Frontal Breadth	MFB	min. breadth temporal crests	Sliding Calipers
Nasal Breadth	NAB	max. aperture breadth	Sliding Calipers
Nasal Height	NAH	chord nasion – nasospinale	Sliding Calipers
Bicondylar Breadth	BCB	chord mandibular condyles	Sliding Calipers
Bigonial Breadth	BGB	chord gonion – gonion	Sliding Calipers
Symphysis Height	SYH	chord gnathion – infradentale	Sliding Calipers
Max. Projective Length	MPL	posterior condyle – mentale	Mandibulometer
Foramen Magnum Breadth	FMB	max. internal breadth	Sliding Calipers
Foramen Magnum Length	FML	chord basion – opisthion	Sliding Calipers
Bimastoidale Breadth	BMB	chord mastoidale – mastoidale	Sliding Calipers
Biasterionic Breadth	BAB	chord asterion – asterion	Sliding Calipers



**Figure 4.5 – Functional Units Defined for the Asymmetry Measurement Package Development**

To that end, the newly compiled set of thirty-two asymmetry measurements has been tested and applied to the cranial samples used in this research. A selection of previously defined asymmetry measurements, specifically those that suited the theoretical premise of this study and that used standard cranial landmarks, were drawn from Woo (1931) and Douglas (1988). Note that acronyms used throughout this study were adapted from these sources, as well as Bass (1971) and Brothwell (1972), but have been modified as required to avoid overlap or confusion. Standard measurements that lent themselves to bilateral application were taken from Brothwell (1973). Additional measurements were created, based on standard landmarks, for the cranium and particularly the mandible, which had not been thoroughly investigated before. The asymmetry measurements were tested and refined through preliminary analysis (Webb 2005) and further modified during

the data collection process in response to problems or emerging research questions. Table 4.2 describes each measurement taken, including the landmarks used, functional units involved and the source. It is accompanied by Figure 4.6, which aids in defining each measurement and understanding the layout of the package. Appendix C provides a more accurate diagram of cranial landmarks.

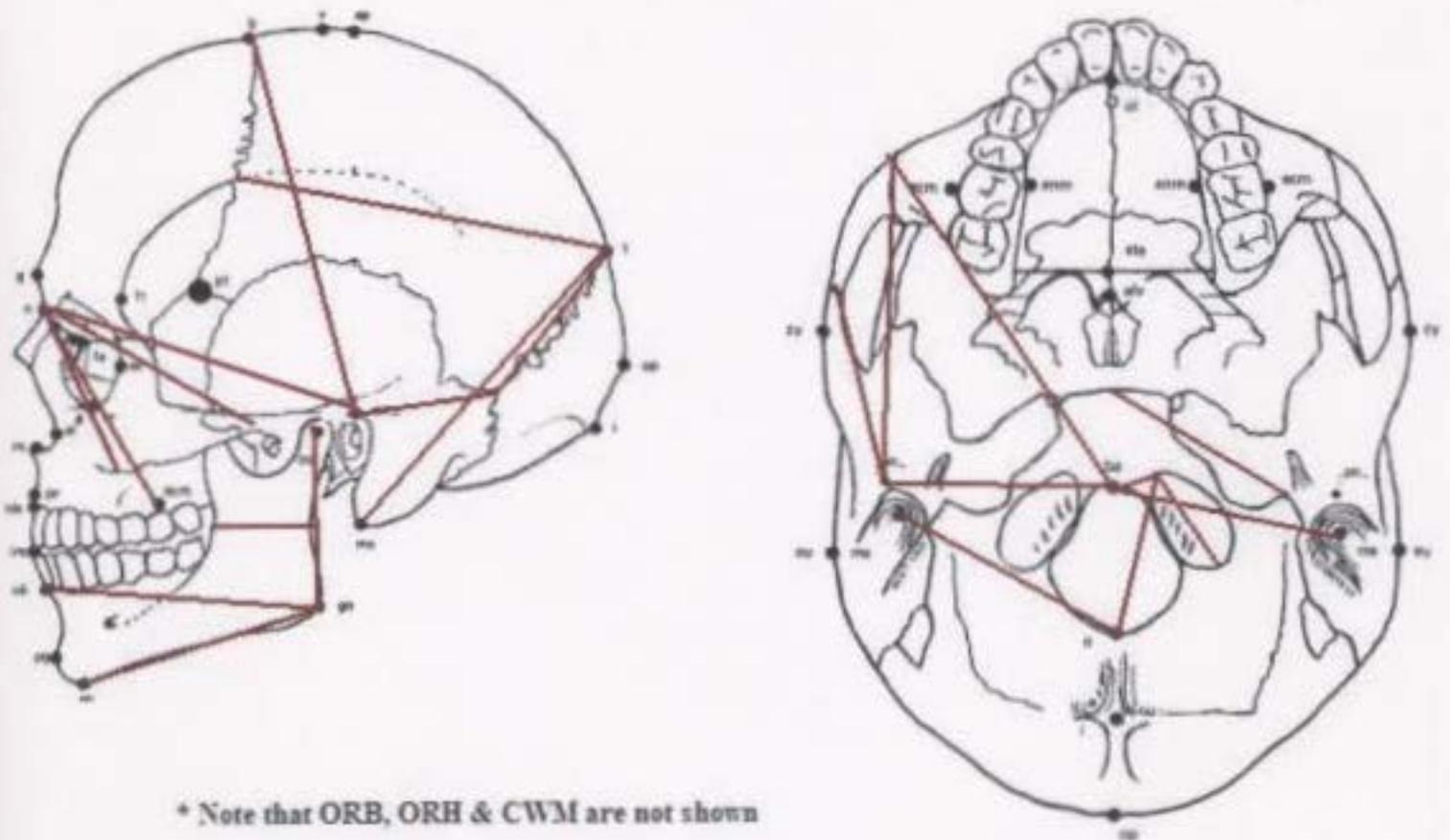


Figure 4.6: Asymmetry Measurement Package

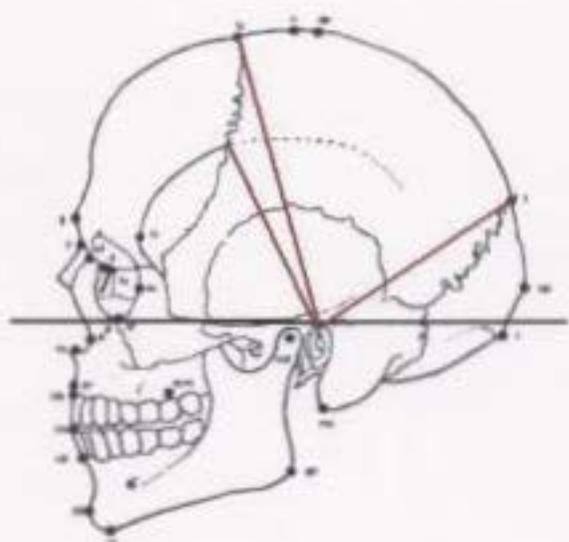
**Table 4.2: Asymmetry Measurements**

<b>Functional Unit</b>	<b>Code</b>	<b>Description</b>	<b>Source</b>
Neurocranium	TCH	Porion – Bregma	Douglas
	PBA	Arc	Webb
	TST	Subtense	Webb
	PBF	Fraction	Webb
Neurocranium	LTC	Lambda – Temporal	Webb
	LTA	Line/Coronal Suture	Webb
	LTF	Arc	Webb
	LTS	Fraction Subtense	Webb
Face	NZM	Nasion – Zygomaxillary Suture	Douglas
Face	ORB	Orbital Breadth	Brothwell
Face	ORH	Orbital Height	Brothwell
Face	NEM	Nasion - Ectomolare	Webb
Face	NAZ	Nasion - Zygion	Webb
Face	NAP	Nasion - Porion	Douglas
Cranial Base & Muscular Face	BZM	Basion – Zygomaxillary Suture	Douglas
	PZT	Porion – Zygomatoco- temporal suture	Webb
Cranial Base & Muscular Face	PZM	Porion – Zygomaxillary Suture	Webb
Cranial Base & Muscular Face	BAP	Basion – Porion	Douglas
Cranial Base & Muscular Face	SP1	Posterior Sphenoid – Sphenobasion	Woo
Mandible	RBR	Minimum Ramus Breadth	Brothwell
Mandible	MBL	Mandibular Body Length	Brothwell
Mandible	RHT	Ramus Height	Brothwell
Mandible	CWM	Maximum Condylar Width (mandible)	Webb
Mandible	GID	Gonion - Infradentale	Webb
Articulating Base	BAM	Basion - Mastoidale	Webb
Articulating Base	OCC	Maximum Articular Length – Occipital Condyles	Douglas
Articulating Base	BSC	Basion – Anterior Occipital Condyle	Webb
Articulating Base	OPC	Opisthion – Anterior Occipital Condyle	Webb
Articulating Base	MAO	Mastoidale - Opisthion	Webb
Inferior Neurocranium & Occipital Bone	AMO	Asterion - Lambda	Woo
Inferior Neurocranium & Occipital Bone	ASA	Asterion – Porion*	Woo (*modified from auriculare)
Inferior Neurocranium & Occipital Bone	LMD	Lambda - Mastoidale	Webb

In addition, a modified version of a craniometer was designed by the author and constructed by Memorial University technical staff. Its purpose was to investigate cranial suture asymmetry and to facilitate accurate photography that reflects asymmetric variation. Based on the Frankfort Horizontal plane (White 2000) and existing equipment for establishing this plane of reference for crania, it creates an independent plane in three-dimensional space. This provides a reference point that is a function of the skull but not part of the skull itself. Three measurements (see Table 4.3 and Figure 4.7) were used to examine the left-right variation of three suture-based landmarks integral to the measurement portion of this study. The measurements were performed by measuring arcs, using a tape measure, from the plane to the specific landmark point. Douglas (1988) noted the difficulty in accurately photographing asymmetric crania, in terms of the difficulties in both positioning the skull and in capturing three-dimensional asymmetry in a two-dimensional photograph. Elevating the skull, and causing its position in space to be influenced only by the position of the external auditory meatuses, allows more informative photographs and eliminates issues in balancing the skull in on a flat surface.

**Table 4.3: Craniometer Measurements**

<b>Functional Unit</b>	<b>Code</b>	<b>Description</b>	<b>Source</b>
Neurocranium	MBR	Arc: Plane - Bregma	Webb
Neurocranium	MSP	Arc: Plane – Stephanion	Webb
Neurocranium	MLD	Arc: Plane - Lambda	Webb



**Figure 4.7 – Craniometer Measurements & Cranium in Craniometer**  
(Note: measurements taken were arcs, i.e., following the surface of the bone, not chords)

## **Chapter 5    Development of the Functional Cranial Model**

The human cranium can be envisioned as a self-organizing, integrated construct of interacting functional units, the creation of which is directed and mediated by several mechanisms, including genetic control, developmental mechanisms, muscular action and bone response to mechanical stress and strain. The patterns and relationships acting in this system are discernable at several levels of organization, specifically the molecular and cellular level, the morphological level for both functional units and crania, the small-sample level and the larger population level. The model briefly outlined in Chapter 4 is based on this perception of the skull as a dynamic, adaptive structure made up of several independent but interacting units. Accepting Moss's functional matrix hypothesis, the skull can be viewed through the framework of complex systems theory, allowing a better understanding of both the processes and outcome of cranial growth and development at all levels of organization.

### **5.1    Background Information for Constructing the Functional Cranial Model**

In the context of the functional cranial model as it applies to cranial asymmetry, there are two types of mechanisms for self-regulation of the cranial form; stabilization acting at the individual level, and canalization acting at the population level. These mechanisms may be applied inconsistently across an individual, to the extent that some characters, or units, are more heavily regulated than others. Fluctuating asymmetry is a reflection of the activity of stabilization and canalization, and simultaneously provides a means of quantitatively observing these mechanisms. Complex systems theory provides a

framework for understanding how small and random perturbations can stabilize into patterns discernable at both individual and population levels.

### 5.1.1 Self-Organization and Complex Systems

Self-organization is a defining aspect of a complex system, examining as it does the way in which intrinsic properties and interactions among elements of the system act to create order (Green 2000). In this sense, each object is both a constituent of some larger system as well as a discrete system in its own right. There are several mechanisms or pathways through which a complex system can be created, including computation-like iteration, emergence, evolution and non-linear/non-equilibrium dynamic processes (Green 2000). Of greatest relevance to the discussion of cranial morphology are non-linear/non-equilibrium dynamic complex systems, as applied by Klingenberg (2003), Starke *et al.* (2003) and Willmore *et al.* (2005). Rooted in chaos theory, this type of system does not allow predictive statements to be made from the properties or behaviour of an individual constituent of the system, but predictions can be made about the system as a whole. For example, while each cranium may be unique in its growth and development and thus cannot be predicted to exist in the same sense that a “type” skull is postulated, a sample of crania can be used to construct an idea of the dynamic behaviour of the population as a whole system. Similarly, a specific cranium can be predicted in terms of morphology, but its constituent functional units cannot. This raises the question of how a chaotic, unstable system can lead to self-organization and the emergence of recognizable, meaningful patterns of interaction. In an open system, that is, one that is impacted by external forces, entropy, or disorder, must decrease. Because any system

must strive towards order, minor irregularities are reinforced by the system, or some aspect of it, in an effort to self-stabilize. It is in this fashion that these minor irregularities evolve into a large-scale pattern (Prigogine 1980). Therefore, while at close inspection, or in examining its individual constituents, a system might appear random or lacking in organization, there is always some sort of underlying pattern of self-directed or emergent organization present.

In order for a living system to want to verge on chaos, there must be some benefit accrued by the organism. In this case, a certain amount of developmental instability (or chaos) is, as Darwin suggested, the cost of the population variability necessary to preserve the potential for evolutionary change (Barnes 1994). It is possible that existing at the edge of chaos allows a species to adapt better to change and therefore provides a selective advantage to slightly more variable forms (Kauffman 1992). Green (2000), however, proposes a more dynamic process, wherein a given object flips back and forth between chaotic and stable forms, stimulated by external forces. There would exist an integrated phase with minimal variation, which would be impacted by some external stimulus, such as a teratogenic agent or other alteration in the environment, and pushed into an unstable or chaotic phase. This highly variable form would then gradually adapt itself, or evolve, and reintegrate into a new stable form. It is, therefore, evident that it is beneficial from an evolutionary and adaptive standpoint for living organisms to exist in a non-equilibrium dynamic state since it increases the potential array of stable, adapted forms.

## 5.1.2 Mechanisms Influencing Growth and Development

Although the ultimate goal of this research is a better understanding of the final physical form of the cranium, there are several underlying systems and processes at work throughout an individual's life that impact on cranial morphology. In the context of the proposed functional cranial model, these can be loosely grouped as the genetic system and developmental mechanisms, and bone responses to mechanical loading through muscular activity.

### 5.1.2.1 The Genetic System and Developmental Mechanisms

The growth and development of an organism begins with its genetic system. This system is composed of all the DNA-encoded information that provides guidance for protein formation and molecular manufacturing. The genetic system expands in a linear fashion, through the iteration of similar functions and components. In addition to its definition as a distinct system, it can also be considered as a constant set of initial conditions, or a starting point, for the expansion of the decidedly non-linear developmental system (Moller and Swaddle 1996). The ultimate manifestation of the genetic system is the genotype, the established pattern through which further growth and development is mediated.

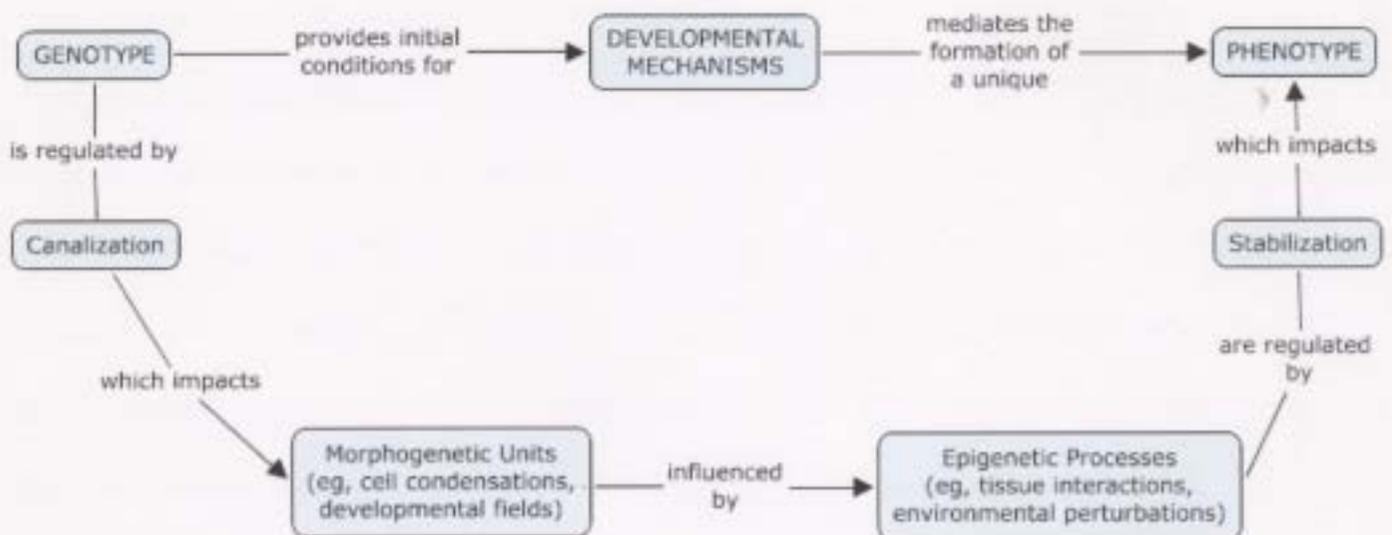
Even though the genotype is fully established and fixed early in the life of an organism, the resultant phenotype, that is, the actual physical form, is usually more variable than a given genome would suggest. Therefore, in addition to genetic differences between individuals or populations, some other source or reason for phenotypic variability must exist. Moller and Swaddle (1996) suggest that variability in mediation

between genotype and phenotype may be due in part to variability in cell formation and quality. This is caused by perturbations in the developmental environment, such as poor maternal health or pollution (e.g., THC poisoning *in utero*), and is regulated through the impact of developmental mechanisms.

Developmental mechanisms can be described through the examination of two mechanisms, canalization and stabilization (Clarke 1998b; Willmore *et al.* 2005). While both have essentially the same goal, that is, the creation of a healthy organism, the level at which these mechanisms act, as well as the direct outcome, differs somewhat. Canalization reduces variation among individuals of the same group by buffering against phenotypic variation caused by genetic and/or environmental perturbations and irregularities. The outcome of this process is the gradual elimination of developmentally or genetically sensitive genotypes, or aspects thereof, from subsequent generations. Another developmental mechanism is stabilization, which acts at the individual level to correct for phenotypic variability caused by developmental noise or accidents *in utero*. In this way, stabilizing processes mitigate the severity or presence of developmental and congenital defects, and in this way have a direct impact on individual morphology.

Cell quality and the related ability to perform necessary tasks and functions adequately impact the efficiency of both canalization and stabilization. During the development process, aggregates of similarly functioning cells, known as condensations, act as actual physical mechanisms, performing many divergent tasks as constituents of developmental fields. As discussed in Chapter 3, the timing and location of the set of tasks performed by cell condensations are important. As a consequence of this, cell condensations also provide a means for introducing variability into the developmental

process. Any inadequacy or failure of stabilization or canalization can detrimentally impact the function of cell condensation units. Different degrees of variation would then be introduced through, for example, poor communication between cell condensations mediating between genotype and phenotype, or between developing left and right sides of a bilateral organism (Hall 2003; Moller and Swaddle 1996). Therefore, the overall relationship can be conceptualized as:

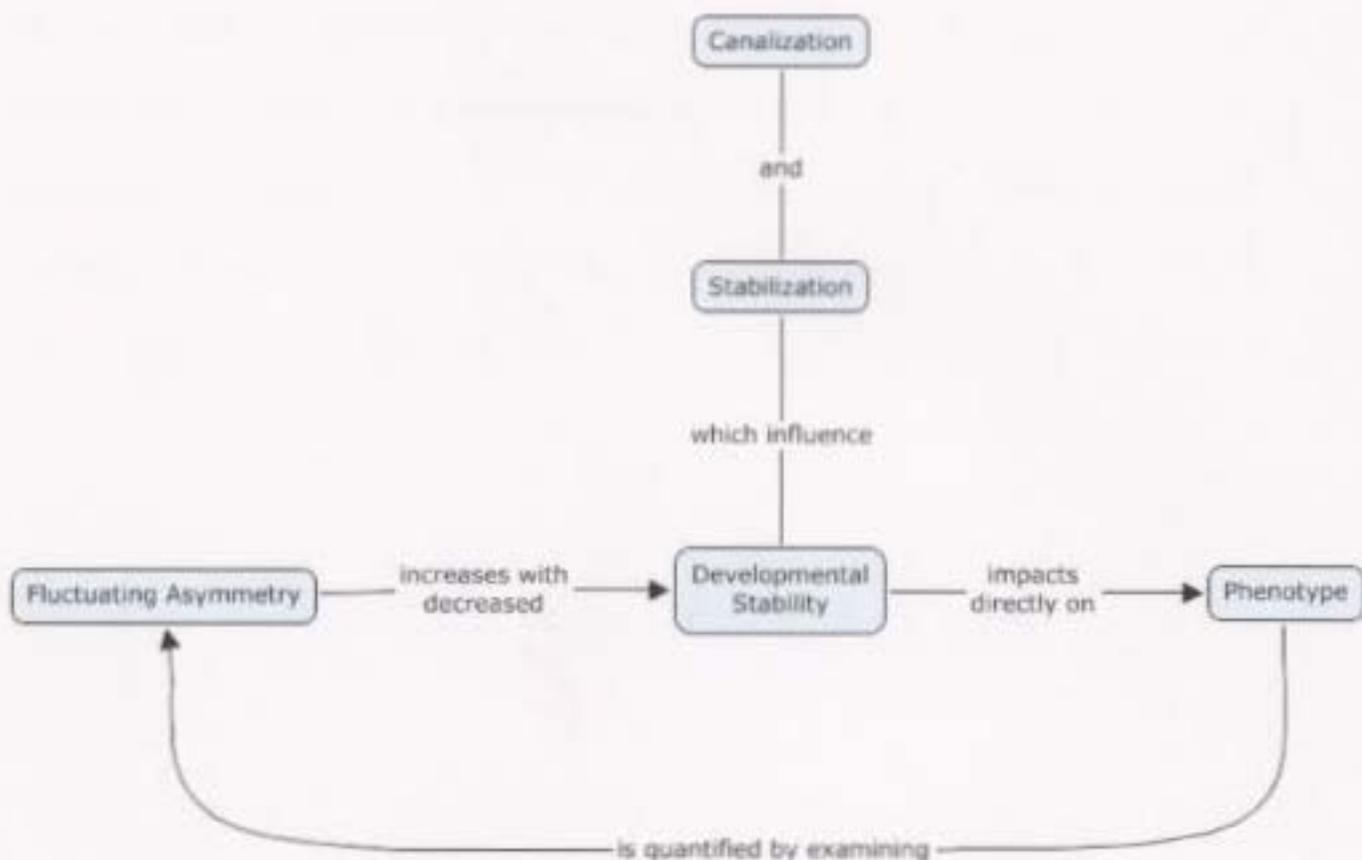


A key consideration when applying the concept of developmental mechanisms to cranial asymmetry is the extent to which these variation-reducing mechanisms impact the morphology of a given character. Clarke (1998b) proposes that the degree of impact is reflective of the functional or reproductive importance of the structure, to the extent that these crucial characters will be less likely to demonstrate significant amounts of variability among individuals of a given population sample. This is supported by the presence of some consistency within and among individuals and populations concerning which characters are more stable or less stable than others (Clarke 1998b).

There is also a significant relationship between fluctuating asymmetry, the major means of quantifying developmental instability, and developmental mechanisms.

Fluctuating asymmetry could be an indicator of the action of either canalization or stabilization, or perhaps of both acting concurrently. Despite this interpretive ambiguity, it effectively serves as a tool for observing developmental mechanisms at work. Clarke (1998b) states that fluctuating asymmetry increases as stabilization decreases, and that phenotypic variation increases as canalization decreases. It is possible that higher or lower levels of fluctuating asymmetry present in the developing and growing organism would be able to dictate further developmental responses, such as increased stabilization efforts; this notion is expanded upon in Figure 5.1. The integrated nature of fluctuating asymmetry with developmental mechanisms, and its correct definition as a complex system, is supported by Klingenberg's (2003) hypothesis. He suggests that fluctuating asymmetry is a non-linear dynamic system, in which small perturbations, such as developmental noise or accidents, result in large-scale responses, such as entire genotypes within a population sensitive to environmental variation.

Of key importance to the construction of a functional cranial model is the understanding that there are at least two types of mechanisms by which an organism self-corrects for variation in cell growth and performance, specifically canalization and stabilization. Further, these mechanisms are applied differentially across an individual, to the extent that some characters may be more rigorously stabilized than others. Fluctuating asymmetry is a simple but relatively informative reflection of these mechanisms acting at both individual and population-sample levels. Finally, an understanding of complex systems, in the context of the developmental of the ultimate physical form, demonstrates how very small perturbations on a cellular scale can eventually emerge into patterns of growth and development discernable among individuals and population samples.

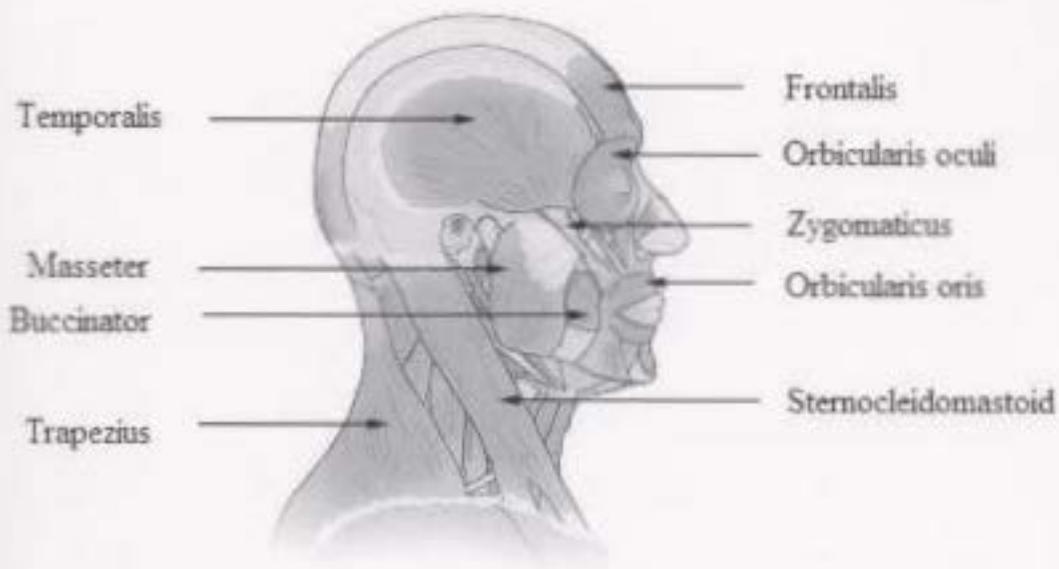


**Figure 5.1: Integration of Fluctuating Asymmetry as an Observational Tool into the Developmental System**

### 5.1.2.2 Bone Responses and the Impact of Muscular Activity

It is generally accepted that bone is able to respond dynamically to mechanical loading caused by muscular activity (e.g., Mays 1999; Plochocki 1999, Steele and Mays 1995). Section 2.4 introduces this notion, and proceeds to explain in detail the four responses of bone at the cellular level to applied forces. For the most part, any type of muscular influence on the skull will be less dramatic than what might be observed in the infracranial skeleton. This is due largely to the domed shape of the human cranium, which distributes force over a larger area to minimize stress on a given region (Pearson and Lieberman 2004). There is, however, muscle tissue overlying the entire cranium, and

parts of the cranium serve to attach major neck and shoulder muscles (Figure 5.2). Some of these muscles and regions of attachment could be more active in shaping morphology than others, such as the temporalis and sternocleidomastoid muscles, and there are several theories concerning the reasons behind bone responding as it does.



**Figure 5.2: Major Muscles of the Head, Neck and Shoulders**  
(adapted from Marieb 1995)

Skeletal muscles are attached to and cover the bony skeleton, and are responsible for mobility, joint stabilization and maintenance of posture. Although there are many forms and arrangements of skeletal muscle in the human skeleton, it is usual for a muscle to originate and insert between at least two locations on bones in order to direct the movement or function of that aspect of the bony system. Attachment between bone and muscle can occur directly or indirectly via tendons or sheet-like aponeuroses (Marieb 1995). Although many of the muscles associated with the cranium act to stabilize or to produce fine muscle movement required for facial expressions, there are certain areas impacted by more active muscles, particularly those spanning between the mandible and

the cranial base/muscular face units, the mandible and soft tissue structures of the mouth and throat, and the occipital bone/mastoid processes and the neck and shoulders. Figure 5.2 highlights a few of the more substantial muscles attached to the base of the cranium and the mandible.

Bone response to increased or decreased mechanical loading through Haversian remodeling, resorption, quiescence and modeling is described in detail in section 2.4, but it is of interest to note that several authors, particularly Carter and Beaupre (2001) and Rubin and Lanyon (1984), suggest that bone will also modify its response to differential loading differently in juveniles and adults, and based on the region affected. There are several theories concerning this dynamic response, including Carter and Beaupre (2001), Lieberman and Crompton (1998) and Ruff *et al.* (1994). All three agree that bone modeling increases proportionally to strain, but Lieberman and Crompton note that this activity varies with location. Carter and Beaupre suggest that bone adapts itself to optimize strain levels, acting to maintain a viable and functional system during growth. They expand on this idea by proposing that growth is a function of biological and mechanobiological components. The biological component consists of intrinsic properties like hormones and genetic control, and this component decreases in impact as age increases and as major linear growth decreases into adulthood, thus rendering this component sensitive to age and region. The mechanobiological component induces modeling to optimize strain with regard to direct or indirect mechanical loading, and is therefore responsive to activity. Support for this can be found in studies of bone mineral density, which demonstrate that bone responsiveness to loading decreases with age, but

does have an increased influence on bone morphology during adolescent growth spurts (Pearson and Lieberman 2004).

Based on this information, it is reasonable to infer that significant muscle attachments, particularly to the cranial base and mandible, are involved in physical activity to the extent that some evidence of this could be discernable on dry bone. This idea is most elegantly supported by the consideration of a cranium with congenital muscular torticollis, in which the shape and function of the cranium is dramatically and somewhat predictably impacted by muscular activity. Carter and Beaupre's (2001) theory concerning the interaction of biological and mechanobiological components to mediate bone response highlights a common theme of regional differences within individuals, and is also suggestive of the sort of interactivity at the postnatal cellular and gross anatomical level required by complex systems theory.

An understanding of the interactive relationships among the genetic system, developmental mechanisms, bone response to loading and the impact of muscular activity allows the reconciliation of local instability with the appearance of recognizable patterns on a larger scale. At any of level of analysis (e.g., individuals or samples), information about group or unit stability can emerge, as can trends concerning the distribution of instability of a given character or sample. This discussion therefore provides important background information for understanding the results described in Chapter 6 and for creating an appropriate functional cranial model.

## 5.2 Development of the Model

Although it will be greatly expanded upon, succinctly, the proposed model suggests that the medial cranial base structures and the face are subjected to fairly rigorous control through developmental mechanisms and are thus less likely to exhibit variation due to developmental noise or genetic instability. The remainder of the cranium, the form of which is still initially regulated by developmental mechanisms to some extent, is more apt to be affected by muscular activity throughout life and would thus demonstrate some asymmetry.

The cranial base, neurocranium and face are derived from embryologically distinct regions, but they grow in a morphologically integrated fashion through the developmental and functional interactions previously described. The basic premise of the functional cranial model is that some functional units will be more stable, that is, less subject to left-right asymmetry, than others, due to the influence of both developmental regulating mechanisms and muscular activity. The cranial base, which extends forward to the ethmoid and incorporates medial basal structures posteriorly to the nuchal region, acts as a structural foundation for the face and neurocranium (Barnes 1994). The metric analysis of the influence of the basicranium on overall cranial shape performed by Lieberman *et al.* (2000) highlights the importance of a stable base in reducing variation in the form of the entire cranium.

The cranial base, derived predominantly from the prechordal cranial base developmental field, undergoes rapid postnatal growth to reach full adult size more quickly than the rest of the cranium (Lieberman *et al.* 2000). This region grows through elongation, flexing at the several synchondroses, and by widening laterally at sutures,

such as the occipito-mastoid suture. Lieberman *et al.* determined that, while the region demonstrates minimal variability, when there is some abnormality, it is most often due to variations in base width and ultimately has a profound impact on neurocranial shape. This is supported by the experimental work of Persing *et al.* (1991) on abnormal suture growth using an animal model. They determined that changing suture formation in the lateral aspect of the sphenoid resulted in statistically significant changes in neurocranial morphology, likely by impacting vault sutural growth. As well, changes in the anterior cranial base resulted in significant changes in neurocranial growth, and it is important to note that comparatively small changes in suture formation result in increasing variability in morphology. Thus, while the medial cranial base is generally stable, any deviation in the lateral portions can significantly impact on the neurocranium. That being said, the neurocranium is also influenced by the growth and expansion of endocranial soft tissue structures, which affect overall shape fairly directly (Steele 2000).

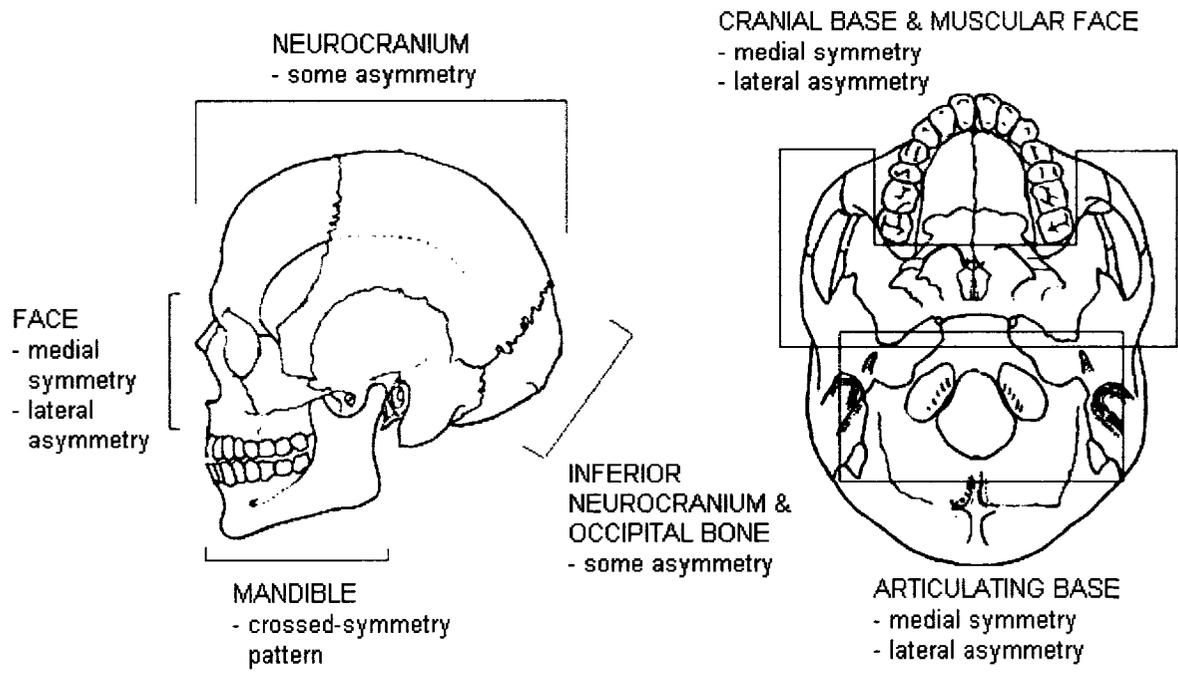
Muscular activity may also exert an influence on both the stability of the cranial base and, through its integration via sutural growth, neurocranial variation. Figure 5.2 presents some of the major muscles attaching to the articulating base and inferior neurocranium/occipital bone functional units, such as the trapezius, which assists in shoulder movement, or the sternocleidomastoid, which assists in head movement (Marieb 1995). It is generally accepted that these regions, particularly the mastoid processes and nuchal area, respond to muscle action (Schwartz 1995). Thus, even though the medial structures of the cranial base are likely subject to increased canalization and stabilization to reduce variation in both the cranial base and neurocranium, lateral and posterior

structures may be more variable than expected due to postnatal muscular activity and growth.

Although the interaction between cranial base and neurocranial structures is reasonably straightforward, stability of the facial skeleton is less clear. Like the neurocranium, most of the bones of the face are formed through intramembranous ossification, dictated by the first branchial arch and frontonasal developmental fields (Barnes 1994). Facial bones are in articulation with each other and with both neurocranial and basicranial skeletal elements, but Lieberman *et al.* (2000) and Persing *et al.* (1991) determined that there was no consistent causative relationship between abnormal cranial base growth and facial morphology. Lieberman *et al.* suggest that perhaps twenty-five percent of all observed facial variability among individuals may be related to general cranial form, such as a narrow neurocranium and base resulting in an equally narrow face. That being said, the high level of connectivity among the face, cranial base and neurocranium makes it reasonable to assume that, like the cranial base and neurocranium, the face would reflect the variability of the rest of the cranium. The mitigating influences of canalization and stabilization could potentially be acting to increase the stability of some facial structures. This is supported by Simmons *et al.* (2004), who, in their study of human preferences and facial symmetry, determined that human perceptions of symmetry in the face are related to traits felt to be most reflective of developmental stability. Their measurements focused on the position of major facial features, such as the position of the eyes, width of the nose, level of the ears and the centrality of the mouth, and symmetry of these features was found to relate strongly to perceived attractiveness between genders. It is therefore possible that in the interests of reproductive success or due to some other as

yet undetermined reason for facial symmetry, medial facial structures and important (e.g., highly visible) features have become more heavily regulated by developmental behaviour, specifically canalization and stabilization.

Lateral and inferior facial structures are likely to be influenced by another factor, specifically muscular action and bone responses. The proposed functional cranial model distinguishes between the face and the cranial base/muscular face, since there are important muscle attachments to the lower face and zygomatic arches related to mandible activity. Due to the bony and soft tissue structures involved in mastication, mechanical strain heavily impacts lower and mid-facial growth and modeling, the result of which may change over time due to mediation through Carter and Beaupre's (2001) proposed theory of bone response. Loading would be high on the mandible and around the zygomatic arches, decreasing further away from teeth and muscle attachments. The mandible itself has been suggested by Weishampel (1993) to exhibit a crossed-symmetry pattern, created as a product of localized bite forces over the mandible, as well as twisting forces along the opposing mandibular body. Thus, while the upper and mid-facial structures, impacted minimally by muscular activity, are likely stabilized by genetic and developmentally dictated factors, potential for variation is introduced due to individual behaviour after development. The general trends expected by the proposed functional cranial model are summarized in Figure 5.3.



**Figure 5.3: Summary of Expected Asymmetry Proposed By Functional Cranial Model**

## Chapter 6 Results

Seventy-eight individuals were evaluated, measured and analyzed to create the main body of data for this research. Of that number, thirty-seven were Maritime Archaic and forty-one were of general European origin. It is important to note that five individuals from each sample (for a total of ten) consisted solely of a mandible. Although both male and female crania were present in each sample, there were significantly fewer female crania. As a result, it was not feasible to consider sex as a variable. As well, due to the occasionally fragmentary nature of some remains, a complete set of measurements could not be taken for each individual. Thus, the sample size for a given individual, or a given measurement, is variable, ranging from  $n=3$  to  $n=30$ . The measurement set was not considered for analysis beyond the individual level if the sample size was ultimately less than  $n=10$  for either sample group. This variability in sample size, coupled with the limited number of remains available and the variations in normality of the data, precluded the use of more powerful parametric statistical tests and tools for the most part. Even so, appropriate descriptive and comparative tests were performed, usually at the 95% ( $p=0.050$ ) significance level unless otherwise stated.

### 6.1 Analytical Framework

Several authors (e.g., Moller and Swaddle 1996, Palmer and Strobeck 1992) discuss and compare various methods of evaluating asymmetry, particularly the more subtle variations used to observe developmental stability, in studies of other species, and, from these meta-analyses, a general idea of the key points and trends to examine can be

determined. Studies of gross human asymmetry in the infracranial skeleton (e.g., Cuk *et al.* 2001) were also reviewed, comparing this methodology to that used for developmental asymmetry and attempting to reconcile the two into a feasible statistical protocol, bearing in mind sample limitations and time constraints. To that end, a statistical-descriptive approach has been used as an overall framework, in an effort to maximize the information gathered and the interpretational ability of the analysis. Standard descriptive statistics, such as mean and standard deviation, and appropriate significance testing have been used to construct a consistent, and therefore comparable, statistical picture at each level of analysis. Although means and sample-based descriptive statistics have been applied in order to examine the magnitude of the asymmetry, the overall distribution of individuals within the sample, including significant outliers and clusters of individuals, is of equal, if not greater importance when considering the nature of that asymmetry. No “type” skull is ultimately created at any stage of analysis. The final synthesis of each level, that is individual, sample and inter-sample, involves the qualitative comparison of these quantitative pictures.

The organization of the analysis is loosely based on Howell’s (1973) three-tier concept, where data are examined at the individual, within-sample and inter-sample levels. The individual level involves a general assessment of asymmetry in each functional unit for each cranium, and the application of a significance test to the entire data set to determine if the differences observed among the six functional units are, on the whole, truly significant. The within-sample analysis entails a detailed description of the shape and character of the distribution, as well as significance testing of the mean and normality. The foramen magnum indices and the subset of measurements designed to

examine suture asymmetry are also subjected to a comparable analysis. The population sample comparison is both qualitative and statistical, using significance testing where appropriate, as well as considering the overall character of asymmetry and sample distribution in each functional unit. The statistical tests and analyses used are summarized in the following table, and are explained in detail as they appear in this chapter.

**Table 6.1 Summary of Data Manipulation**

<b>Level</b>	<b>Statistic</b>	<b>Description</b>	<b>Purpose</b>
<b>I</b>	Right - Left	Signed difference for each asymmetry measurement	Basic determination of side-to-side variation; mitigates influence of structure size
	Standardized Cumulative Asymmetry Value	Sum of absolute differences divided by the number of measurements	Absolute magnitude of asymmetry for each functional unit
	Friedman Test	Non-parametric analysis of variance	Examines differences among units within one sample
	Mann-Whitney Test	Significance of difference between two independent samples	Determine difference between sample functional units (e.g., European mandible unit v. Maritime Archaic mandible unit)
<b>II</b>	Shape of Distribution†	Standard deviation, skewness, kurtosis, Kolmogorov-Smirnov test for normality	Describe the shape of the distribution of the signed asymmetry values within a sample
	Magnitude of Asymmetry‡	Mean, one sample t-test	Determines the degree of right or left-favouring asymmetry for a given measurement within a sample
	BASOPX	Basion-Opisthion Asymmetry Index (analyzed as † and ‡)	Compares difference in shape between left and right indices
	FORIDX	Foramen Magnum Index	Describes distribution of shape of the foramen magnum within the samples
<b>III</b>	Suture Asymmetry	Signed differences (analyzed as † and ‡)	Describes asymmetry of position of suture landmarks
	Mann-Whitney Test for Suture Asymmetry and FORIDX	Significance of differences between two independent samples	Examine differences between Maritime Archaic and European samples for the FORIDX and suture asymmetry measurements

## 6.2 Level I Analysis: Individual Asymmetry

Based on Moller and Swaddle's (1996) suggestion of using unsigned absolute asymmetry values ( $|R-L|$ ) for description at the individual level, a standardized, cumulative asymmetry value was calculated for each functional unit for each individual.

For example:

$$\text{Individual X (Articulating Base)} = (|BAM| + |OCC| + |OPC| + |BSC| + |MAO|) / N_i,$$

where  $N_i$  is the number of measurements for which a real value exists.

Repeating this procedure for all six functional units for each individual provides a fairly sensitive and intuitively sensible gauge of the amount and distribution of asymmetry for a given cranium or partial cranium. Individual asymmetry profiles for each individual are provided in Appendix D. This analysis highlights both the asymmetry within a functional unit, as well as making relationships among units more explicit.

In order to determine if the differences observed among functional units are generally significant, a Friedman's test was performed (Zar 1999). This is a non-parametric equivalent to an analysis of variance, and, because the data are not normally distributed, subsequent testing to identify which functional units differed significantly from each other could not be performed. The Friedman's test did reveal that, on the whole, there are important differences among units for both Maritime Archaic and European individuals at the 95% confidence level ( $p=0.050$ ). As well, an examination of the standard deviations for each functional unit demonstrates that some units are notably less variable than others, particularly the articulating base and the face, while the mandible is the most variable in its manifestation of asymmetry (see Table 6.2). In order to compare Maritime Archaic and European samples at this level, a Mann-Whitney test

was performed, which examines the similarity of a given value between two independent samples. With the exception of the mandible and the inferior neurocranium and occipital bone units (significantly different at 90% confidence,  $p=0.10$ ), the two samples essentially manifest the same individual variations in asymmetry.

**Table 6.2 Level I Analysis - Sample Size, Range and Standard Deviations**

<b>Functional Unit</b>	<b>N</b>	<b>Minimum†</b>	<b>Maximum†</b>	<b>Mean</b>	<b>Standard Deviation</b>
<b>Maritime Archaic</b>					
Neurocranium	21	2.0	9.8	3.9	1.83
Face	22	0	5.0	2.1	1.24
Inferior Neurocranium & Occipital Bone	25	0.7	6.0	2.2	1.50
Cranial Base & Muscular Face	26	0	7.0	2.0	1.43
Mandible	20	1.0	9.5	2.0	2.30
Articulating Base	30	0.4	4.0	1.9	0.75
<b>European</b>					
Neurocranium	26	2.3	7.0	3.9	1.16
Face	25	0.8	5.0	1.2	0.99
Inferior Neurocranium & Occipital Bone	25	0.5	5.5	2.9	1.30
Cranial Base & Muscular Face	24	0.5	4.8	2.2	1.11
Mandible	26	0	8.3	3.1	1.55
Articulating Base	28	0.4	4.2	1.7	0.96

(†note: standardized cumulative asymmetry values were used; thus, for range values, the real range is +/- the given value)

### 6.3 Level II Analysis: Within-Sample Asymmetry

For each of the thirty-two asymmetry measurements taken (see Table 4.2), a signed asymmetry value was calculated by subtracting left from right values; a positive difference meant that the right side was larger, and a negative value the left. A set statistical protocol was applied, including the statistics describing the shape of the distribution (standard deviation, kurtosis, skewness and Kolmogorov-Smirnov test for normality) and those examining the significance of the mean (mean and one-sample t-test), as well as histograms of both raw data and standardized z scores. Microsoft® Office Excel 2003 and SPSS 13.0 were used for data handling and analysis. A normal distribution is perfectly symmetrical, with a mean of zero. Sixty-eight percent of the sample falls within one standard deviation of the mean, and ninety-five percent within two standard deviations. A Kolmogorov-Smirnov test with a Lilliefors significance correction is used to determine if a given sample distribution is normal, in this case examined at the 95% ( $p = 0.050$ ) level of significance. Skewness and kurtosis describe deviations from a perfect normal distribution. Skewness describes asymmetry in the context of significant left (negative) or right (positive) tails, and is deemed to be an important deviation if the skew statistic is approximately twice the standard error. Kurtosis measures the degree and importance of any clustering around a central point of observations within the sample distribution. A positive kurtosis statistic suggests a leptokurtic, or highly clustered dispersion of data points, while a negative statistic indicates a more widely dispersed platykurtic distribution.

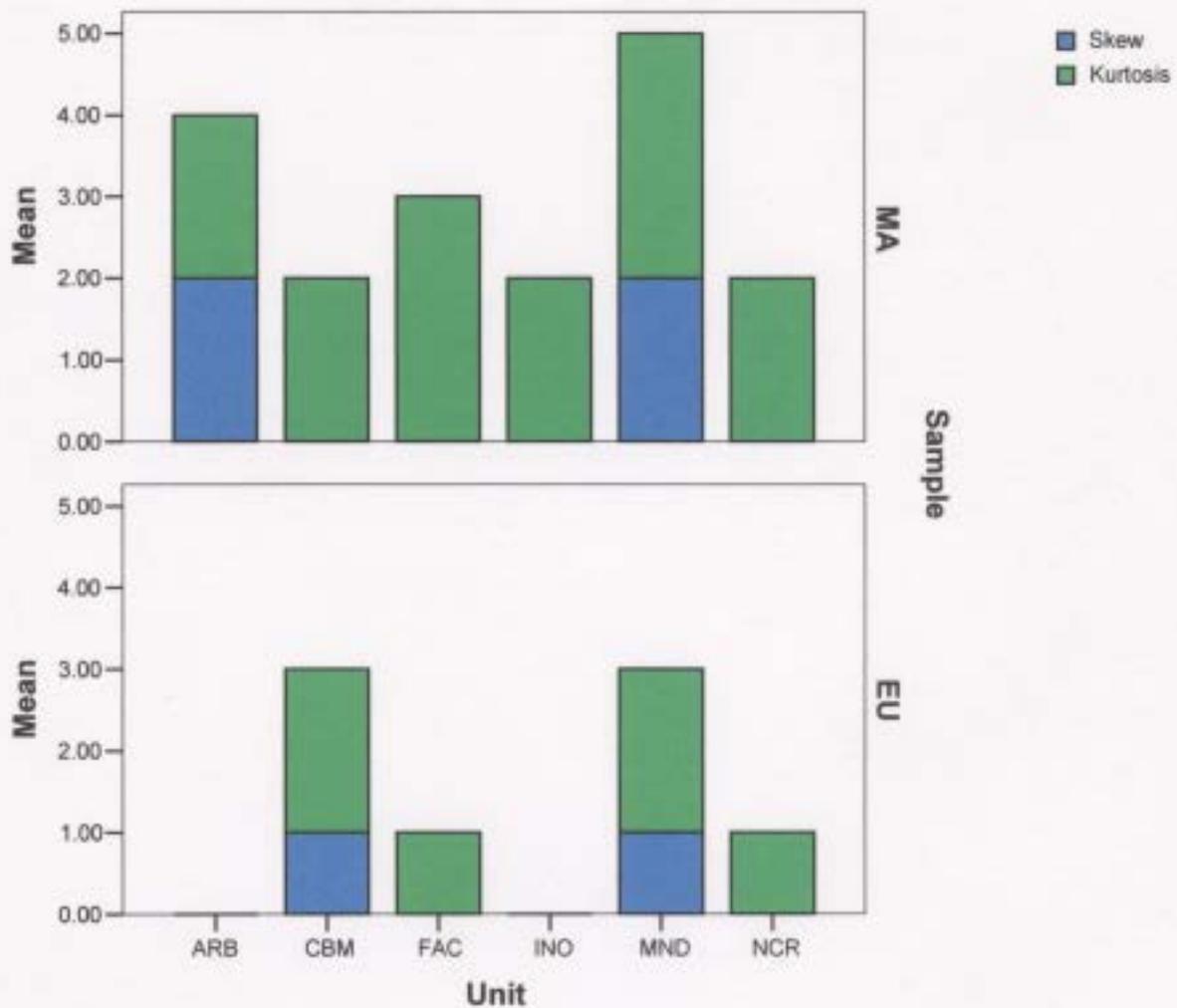
One-sample t-tests were used to determine if the mean is significantly different from zero, at both the 95% ( $p=0.050$ ) and the 90% (0.100) levels of significance. A set of

example calculations is provided in Appendix E. The mean, used in conjunction with skewness, kurtosis and normality statistics, is not defining an “average” form. The shape statistics delimit a field in which most data points for the sample are located, and the mean defines an area within that field where there is an increased probability or likelihood that a given individual will be found for that measurement. The mean, in this context, is a sample tendency for the measurement in question, and the t-test determines if the tendency suggested by the mean is statistically significant.

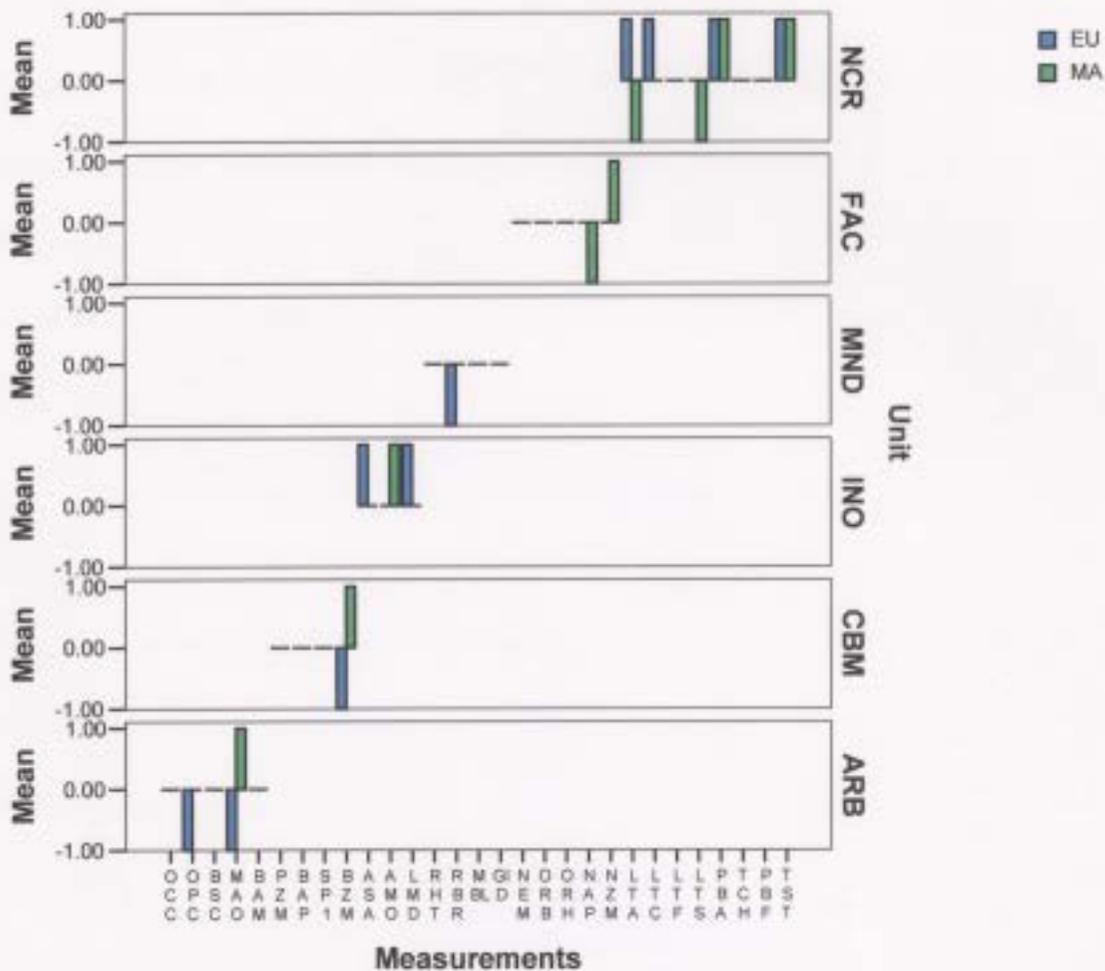
### 6.3.1 Within-Sample Analysis – Maritime Archaic and European Samples

In order to facilitate analysis, measurements are divided into functional units and considered here in the context of trends in each of those functional units. Figures 6.1 and 6.2 present a summary of the results obtained through the previously outlined statistical protocol; detailed outcomes of the analyses are attached in Appendix F. Figure 6.1 presents a summary of the degree of skewness and kurtosis in each functional unit. For the purposes of this analysis, these statistics are essentially measures of variability and the nature of asymmetry within a sample. Additionally, when each measurement is considered independently, variability can be localized to some extent to a specific region within the unit. An ideal, or perfectly symmetrical, set of measurements would be without any skew or kurtosis, and thus any deviation is suggestive of variability within the sample. Similarly, Figure 6.2 presents an overview of units demonstrating any trends towards mean differences significantly above or below zero at either 90% or 95% confidence. In Figure 6.2, an ideal or perfectly symmetric result would be a mean of zero, and thus no indication will appear on the figure. A value of +1 (see left vertical axis)

indicates a sample trend towards right-side dominance, while -1 indicates a trend to left-side dominance. Each functional unit is presented separately (see right axis), and the acronyms are defined in the figure caption.



**Figure 6.1: Number of Measurements per Functional Unit Demonstrating Skewness and Kurtosis for Maritime Archaic (top) and European (bottom) Samples**



**Figure 6.2: Comparison of Significant Directional Tendencies of Means Within Each Functional Unit Demonstrating Magnitude of Asymmetry for Each Sample** (NCR: neurocranium; FAC: face; MND: mandible; INO: inferior neurocranium & occipital bone; CBM: cranial base & muscular face; ARB: articulating base; note that measurement codes are defined in Table 4.2)

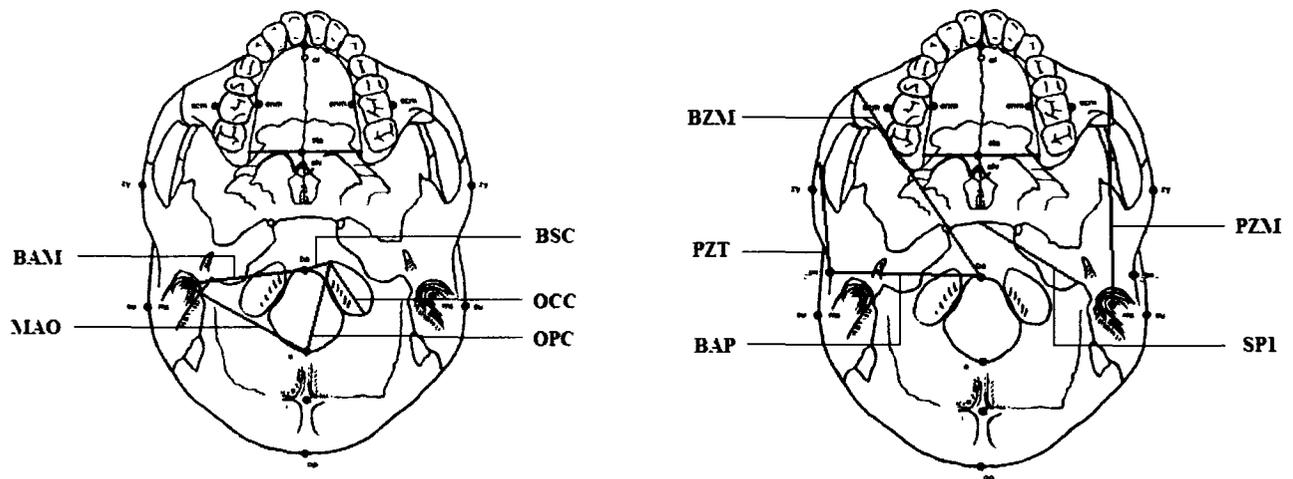
In comparing the Maritime Archaic and European samples at the functional unit level, distinct and important differences in patterns of asymmetry emerge. Succinctly, the Maritime Archaic sample demonstrates a general trend to right-side structural dominance in three functional units, the articulating base, cranial base/muscular face and inferior neurocranium/occipital bone units, and alternating left and right structural dominance for

the mandible, face and neurocranium units. Conversely, the European sample demonstrates trends towards left-side structural dominance for the articulating base and cranial base/muscular face units and right-side structural dominance for the inferior neurocranium/occipital bone and neurocranium units. Like the Maritime Archaic, the mandible unit manifests both left and right-side structural dominance, but the face is essentially symmetric.

The articulating base unit was described through five measurements focusing on the relationship among the foramen magnum, occipital condyles and mastoid processes (Figure 6.3). As demonstrated in Figures 6.1 and 6.2, the Maritime Archaic sample demonstrates important variability in this unit, as well as a slight positive trend; the mean for MAO (mastoidale-opisthion) is significantly located to the right of zero. There is no suggestion of intra-unit regional variability; that is, lateral structures do not manifest variability differently than medial structures. Thus, the Maritime Archaic presents some right-side structural dominance and a comparatively high degree of variability in this unit. The European sample manifests less variability in the articulating base unit, but Figure 6.2 is suggestive of a potentially significant negative (left-favouring) trend. Again, no intra-unit regionalization is discernable, but the overall trend for this sample is towards left-side structural dominance.

The cranial base/muscular face unit consists of five measurements examining the anterior portion of the cranial base and the inferior aspect of the face where major muscles attach (Figure 6.3). The overall assessment for the Maritime Archaic is that a slight right-side structural dominance exists in this functional unit. There is some variability present, demonstrated by Figure 6.1, as well as a slight positive trend (Figure

6.2). There is no intra-unit variation for either the Maritime Archaic or the European sample. The European sample has a slight overall left-side structural dominance, demonstrated through the slightly higher amount of variability and the slight negative trend apparent in Figure 6.2.

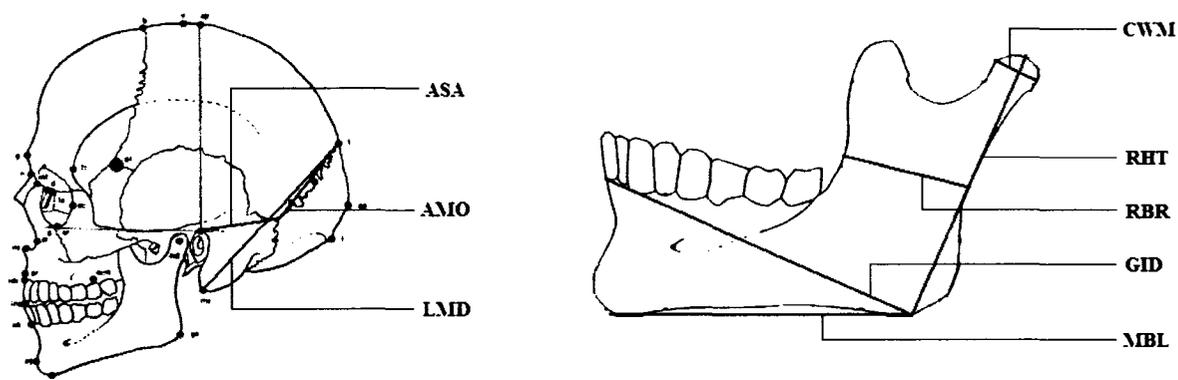


**Figure 6.3: The Articulating Base (left) and the Cranial Base/Muscular Face (right) Functional Units**

Three measurements are used to examine the inferior neurocranium/occipital bone unit, and these are focused on the lateral aspects of the petrous temporal bone and the occipital bone (Figure 6.4), thus presenting no real avenue for intra-unit variation to manifest. Figures 6.1 and 6.2 present some variation and a slight positive trend for the Maritime Archaic sample, and no variability with a positive trend for the European sample. Therefore, the overall trends in this functional unit are for right-side structural dominance, which manifests most strongly in the European sample.

Data collection for the mandibular functional unit entailed the collection of five measurements, of which four have been analyzed, comparing the size and shape of the mandibular body and ramus (Figure 6.4). The Maritime Archaic sample demonstrates a

comparatively high level of variability, but no directional trend related to significance of means. Based on the direction of the skewness statistics, alternate left-right, or crossed, patterns of asymmetry become evident, but are not, of course, significant or distinct enough to be reflected in Figure 6.2. The European sample demonstrates less variation, but Figure 6.2 shows a slight negative trend of the mean for the ramus breadth measurement, and through this and the direction of the variability statistics, a slight crossed-symmetry pattern does begin to become apparent.

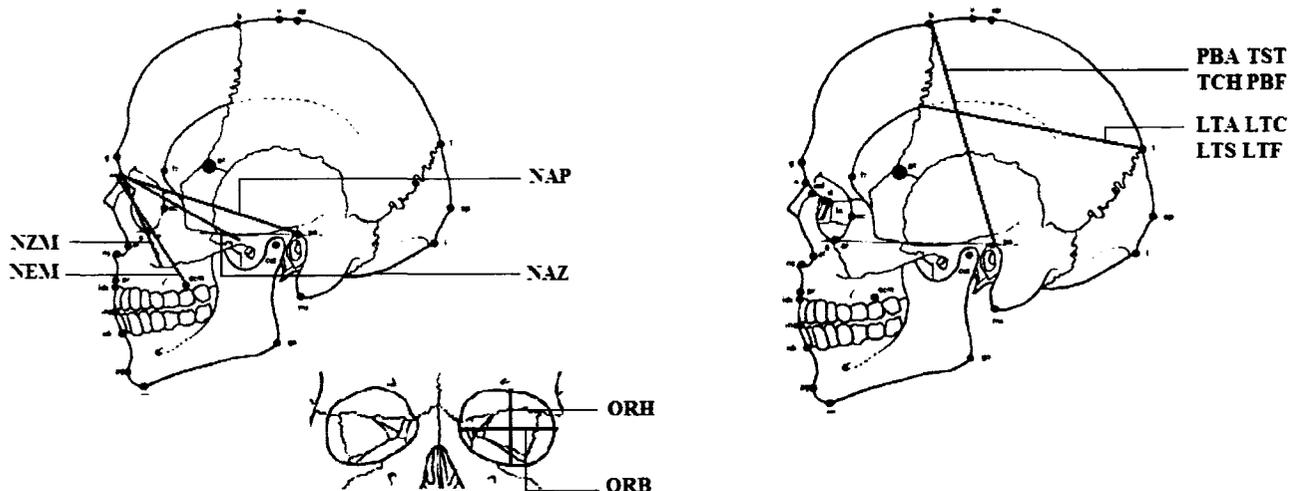


**Figure 6.4: The Inferior Neurocranium & Occipital Bone (left) and Mandible (right) Functional Units**

The six measurements of the face unit examine the shape of the orbits, and compare both the shape of the zygomas and the maxillae, and the location of the nasal aperture (Figure 6.5). In this unit, the Maritime Archaic demonstrates some variability and both positive and negative trends for the means of nasion-porion (NAP) and nasion-zygomaxillary suture (NZM) measurements. There is some suggestion of intra-unit regional variation in asymmetry for the Maritime Archaic sample, but the European sample demonstrates unusually high symmetry and, by extension, no intra-unit differences. Moreover, there is no suggestion of any trends away from symmetry of the mean and only slight variability in orbital breadth measurement. The European sample

therefore presents an exceptionally symmetric pattern for the face unit, in contrast to the more asymmetric pattern for the Maritime Archaic.

The neurocranium unit consists of eight measurements examining the shape of the parietal bones and the location and size of the parietal eminences, as well as the parieto-temporal region and the position of greatest convexity on the lateral aspect of the cranium (Figure 6.5). The Maritime Archaic sample presents some variability, but there are significant trends in the mean (Figure 6.2) suggesting left-side structural dominance for some lambda-stephanion measurements and right-side structural dominance for some bregma-porion measurements. For the European sample, there is minimal variability in the neurocranium in terms of skewness or kurtosis, but there is a significant right structural trend, evident in Figure 6.2. Thus, the Maritime Archaic sample reflects a crossed-symmetry pattern, whereas the European sample presents a significant right-side trend.



**Figure 6.5: The Face (left) and Neurocranium (right) Functional Units**

Although there is less statistically important asymmetry in the European sample, the unusual distribution of that asymmetry may be significant. The sample can be characterized as demonstrating left-side dominance in the articulating base and cranial base/muscular face units, with right-side structures favoured for the inferior neurocranium/occipital bone and neurocranium functional units. The face is exceptionally symmetric, and the mandible has both left and right-side dominance depending on the parts of that functional unit examined. The Maritime Archaic sample asymmetry can be characterized as demonstrating important variation favouring right-side structures in the articulating base, cranial base/muscular face and inferior neurocranium/occipital bone regions of the cranium, as well as both left and right side dominance in the face, mandible and neurocranium. Setting aside left-right asymmetry, there is some sort of variation present in every functional unit, but particularly in the neurocranium, mandible, articulating base and the face.

### 6.3.2 The Foramen Magnum

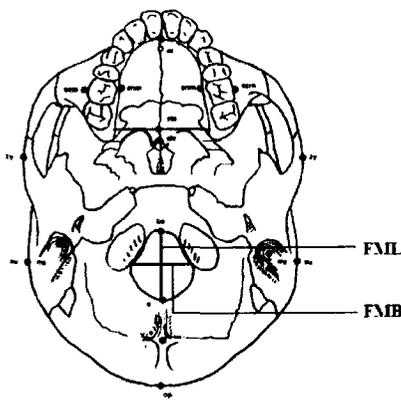
During the data collection stage of this research, it became apparent that both the shape of the foramen magnum and the orientation of the occipital condyles varied, sometimes quite noticeably, among individuals. Since this region is subject to developmental perturbations (see Chapter 3), an index designed to capture the shape of the foramen magnum (FORIDX), and a Basion-Opisthion shape asymmetry index (BASOPX) that determines differences in the relationship of the occipital condyle to the foramen magnum, were developed.

The foramen magnum index creates a bivariate index using the foramen magnum breadth (FMB; see Table 4.1) and the foramen magnum length (FML), such that:

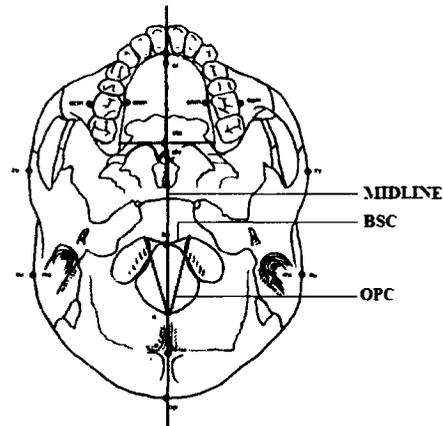
$$\text{FORIDX} = \frac{\text{FMB} \times 100}{\text{FML}}$$

where FORIDX = 100 indicates a perfect circle.

Figure 6.6a denotes the exact location of the measurements taken. Both the Maritime Archaic and the European FORIDX values are normally distributed ( $p=0.050$ ), and a Mann-Whitney test, which determines if the two sampled populations are equivalent for a given trait, did not indicate any significant differences between the two samples. That being said, the range of values in the Maritime Archaic sample is distinctly greater, as is the variance (a measure of variability in the data), than it is for the European sample. The scatterplot in Figure 6.7 make the differences in distribution and variability explicit.



**Figure 6.6a: Measurements Used for the Foramen Magnum Index (FORIDX)**



**Figure 6.6b: Measurements Used for the BASOPX Asymmetry Index**

The position of the occipital condyles in relation to the foramen magnum and a somewhat arbitrarily defined midline between basion and opisthion (see Figure 6.6b) is

also variable. To attempt to capture this, an asymmetry index, involving the measurements BSC and OPC, that subtracts left and right shapes, was designed.

$$\text{BASOPX} = \left[ \frac{\text{BSC-R} \times 100}{\text{OPC-R}} \right] - \left[ \frac{\text{BSC-L} \times 100}{\text{OPC-L}} \right]$$

where a higher difference between right and left sides is indicative of greater asymmetry of form.

Since this essentially creates data in the same form as the main set of asymmetry measurements, the index output can be analyzed similarly. Both the Maritime Archaic and European samples are normally distributed and do not have means significantly different from zero. The Maritime Archaic sample does manifest some unusual characteristics, notably an important right tail and a very high standard deviation, which is a measure of data dispersion, or variability. Thus, though neither sample demonstrates any glaring asymmetry, the Maritime Archaic sample statistics are suggestive of a noticeable amount of variability within the population, while the European sample suggests a less variable population regarding the shape of the foramen magnum and any orientational asymmetries of the occipital condyles.

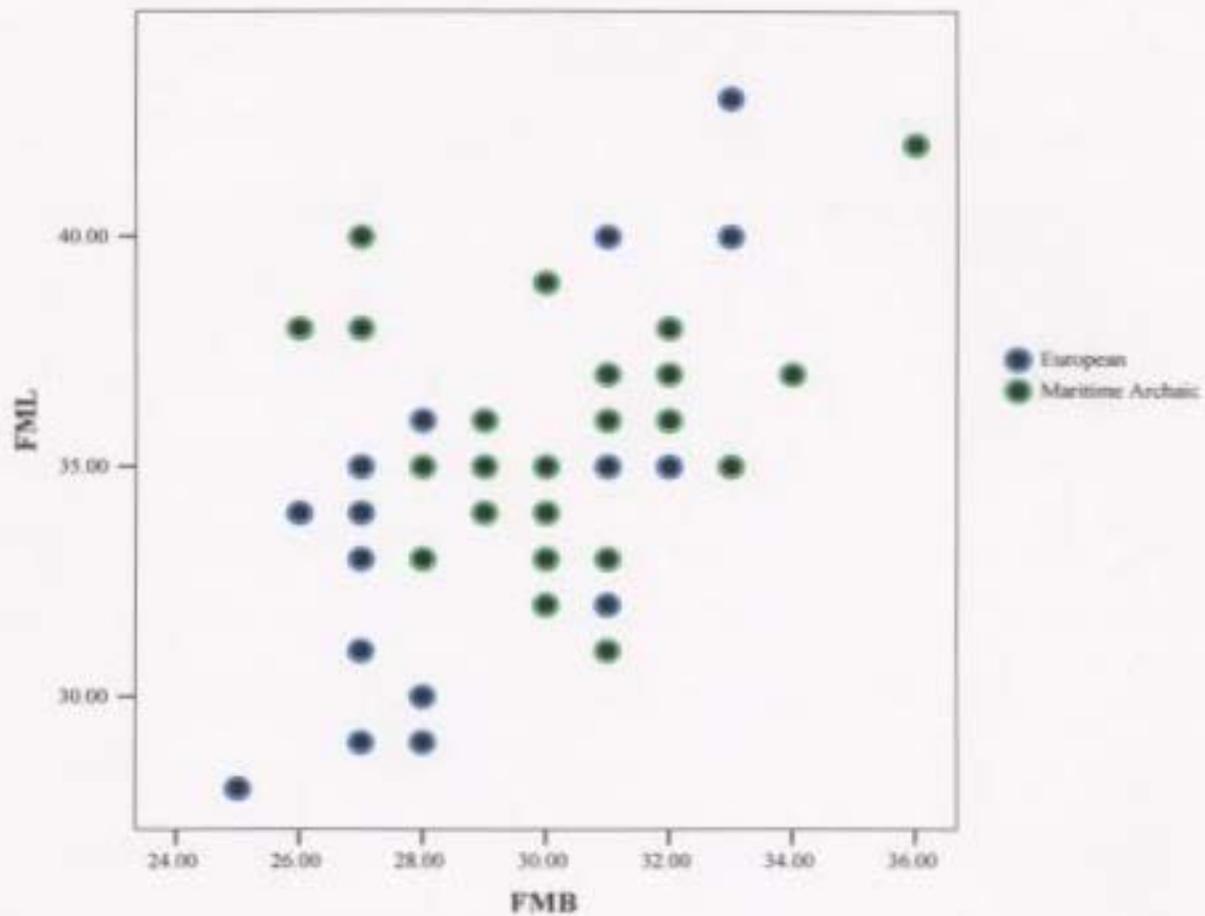


Figure 6.7: Scatterplot of Foramen Magnum Index for European (blue) and Maritime Archaic (green) Samples

### 6.3.3 Suture Asymmetry

The examination of variation in suture position involves collecting a series of left and right measurements, then subtracting them to obtain a signed asymmetry value in the same fashion as the bulk of the asymmetry measurements taken. The measurements were

chosen to observe the position of bregma (MBR), lambda (MLD) and the intersection of the superior temporal line and the coronal suture (MSP; approximately at stephanion). Although both European and Maritime Archaic samples had high standard deviations for all three measurements, all distributions were determined to be normal ( $p=0.050$ ) based on the Kolmogorov-Smirnov test. Distribution shape statistics were applied and the significance of the means was also examined (see Table 6.3).

The Maritime Archaic sample statistics suggest minimal, but crucial, asymmetry, with a significantly negative mean concerning the location of the intersection of the temporal line and the coronal suture (a landmark point of several measurements for the neurocranium unit). As well, MBR is significantly leptokurtic, and unskewed, about a zero mean. For both Maritime Archaic and European samples, MLD is a fairly symmetric measurement, with only mild platykurtosis present for the European sample. Additionally, the European sample has significantly positive means for both MBR and MSP, and an important right tail for MSP.

**Table 6.3 Suture Asymmetry Measurements: Shape of Distribution and Significance of the Mean**

Measurement	Shape of Distribution			Significance of Mean
	Skew	Kurtosis	Normality	
<b>MBR</b>				
Maritime Archaic	ns	1.720	normal	ns
European	ns	ns	normal	Right-favouring, $p= 0.050$
<b>MLD</b>				
Maritime Archaic	ns	ns	normal	ns
European	ns	-1.384	normal	ns
<b>MSP</b>				
Maritime Archaic	ns	ns	normal	Left-favouring, $p= 0.050$
European	1.305	3.163	normal	Right-favouring, $p= 0.050$

In addition to these descriptive and significance statistics, the normality of the complete set of data permits the use of more powerful parametric statistics. An analysis of variance (ANOVA) was performed for the Maritime Archaic and European samples, determining that there is a significant difference among MBR, MLD and MSP for each group. The results of the ANOVA test are included in Appendix F. For the Maritime Archaic and European samples, MSP is subject to a higher level of variation, which suggests that the temporal line/coronal suture intersection is more apt to be asymmetric. For the European sample, the results of the ANOVA suggest that MBR is also subject to a higher level of variation.

It is evident, then, that suture position is variable and, by extension, landmark points like bregma, lambda and the temporal line/coronal suture intersection, vary asymmetrically when measured from a fixed point in space, created in this case by the modified craniometer (see Section 4.3.2).

#### 6.4 Level III Analysis – Inter-Sample Comparison

This stage of analysis is essentially a qualitative comparison of the statistics calculated for the level II analysis (section 6.3). Although there are statistical tests available for comparing two population samples, they were not appropriate for the most part, due to the small and variable sample size among measurements. As well, several data sets were not normally distributed, thus rendering comparative statistical tests inapplicable to the samples. That being said, the data for suture asymmetry was consistently normal, and was therefore subjected to a statistical test for determining the significance of any differences between samples.

The results can be examined for important trends in the context of functional units that display or do not display asymmetry, as well as the direction of that asymmetry within each unit. Of the six functional units examined, only the face does not consistently manifest some form of asymmetry in at least one measurement. As well, certain medial basicranial structures demonstrate symmetry, particularly in the European sample, indicated by the BASOPX index and some individual measurements in the articulating base functional unit. Aside from these exceptions, the presence of some degree of asymmetry is common to both Maritime Archaic and European samples. Of interest in the Maritime Archaic sample is the increased number of significant tails and leptokurtosis, which suggests the presence of both extremes and a high degree of similarity among members of the population. This is, of course, indicative of variability; therefore, in terms of the presence or absence of asymmetry, the results compared between samples suggest that some functional units are less apt to display a high magnitude of asymmetry than others, and the European sample is less variable in nature than the Maritime Archaic.

There are also distinct similarities and differences between patterns of the direction of asymmetry. For both samples, the articulating base and the cranial base/muscular face functional units manifest significant one-sided asymmetry, although the structurally dominant side is reversed between samples. Both the mandible and the inferior neurocranium/occipital base units have similar patterns of asymmetry between samples. The mandible tends to have measurements demonstrating both left and right side structural dominance within that same structure, while the inferior neurocranium/occipital bone tends to be predominantly right side dominant for Maritime

Archaic and European samples. The face is consistently fairly symmetric for both groups medially, but the Maritime Archaic have some left and right lateral asymmetry.

The neurocranium is more difficult to characterize. In terms of the suture position, a Mann-Whitney test determined that there are no significant inter-sample differences for bregma or lambda, but that the intersection of the temporal line and the coronal suture is significantly different ( $p=0.050$ ) between the two samples. The European sample demonstrates consistent significant right side asymmetry, while the Maritime Archaic sample has measurements with significant left and right asymmetry.

## 6.5 Summary of Results

Based on these results, it is apparent that some degree of asymmetry is normally present for the human crania examined in this study. Both the European and Maritime Archaic samples manifested some type of asymmetry, captured through an examination of the sample distribution of left/right side differences. There are, however, significant differences in the pattern and degree of asymmetric variation between samples, as well as among functional units, in terms of both the dominant direction of asymmetry and its prevalence. In addition to the patterns of variation suggested by the asymmetry measurement package, the foramen magnum index (FORIDX) and the basion-opisthion index (BASOPX) proved to be reflective of both the symmetry of the European sample and the asymmetry of the Maritime Archaic sample. The statistics examining suture position also revealed some interesting asymmetry, the impact of which on cranial asymmetry as a whole must certainly be considered. Having established that asymmetry

is a significant and important influencing factor on cranial morphology, it now becomes essential to evaluate potential causes.

## **Chapter 7 Discussion**

Before engaging in an in-depth discussion of this work, it is necessary to recall that the functional cranial model proposed in Chapter 5 suggests that the medial aspects of the cranial base and face units are more rigidly controlled through developmental mechanisms. Thus, these regions are less likely to exhibit variation due to developmental noise or genetic instability. While the form of the remainder of the skull is still regulated by developmental mechanisms, it is more likely to be influenced by muscular activity throughout life and would thus demonstrate some asymmetry.

In an effort to facilitate comparison of the results in the context of the functional cranial model, the discussion is organized in a fashion similar to Chapter 6. The factors influencing cranial asymmetry and the application of the model to individual crania are first generally discussed, then demonstrated through two case studies. Subsequently, at the sample level, the observations made in Chapter 6 are compared to the expectations of the model, and important deviations from that model are discussed. Finally, the explanatory value of the information derived from the model is considered in the appropriate cultural context, and the feasibility of defining a threshold between normal and asymmetric cranial forms is discussed.

### **7.1 Individual Asymmetry for Maritime Archaic and European Samples**

For any individual from either of the sampled populations, it is impossible to predict the distribution of variation for every measurement, or even for every functional unit, with a high degree of accuracy. Each individual is unique, in terms of both genetic background and life history, and this is absolutely reflected by the data presented in

Appendix D. That being said, section 6.2 identifies some important trends concerning the variability within and among functional units.

Clarke (1998a) suggests that an individual that is asymmetric for one trait need not be asymmetric for any other trait or, if a specific character is asymmetric, there may be differences in magnitude. This is borne out by the significant differences among functional units for both the Maritime Archaic and the European samples. As well, some functional units are more variable than others, suggesting either greater variation in morphology among individuals or, since units are described through a set of measurements, that some regions of a unit are more apt to be asymmetric than others. This suggests that the assignment of an individual asymmetry rating, e.g., a single number or value describing the degree of asymmetry present in a given skull, is not possible to undertake, since no regular numerical relationship exists among characters. In this case, this is true whether one considers within unit variation or within individual variation.

In the context of the functional cranial model, the suggested patterns of variation are beginning to emerge at this point. Since analysis is taking place at the individual level, factors influencing morphology uniquely for each sample member must be considered. Thus, while canalization is important for establishing the optimum genotype for the group, the focus here is narrower, examining developmental noise and stabilization at an individual level. The exertion of developmental control seems to be unit-dependent; both Maritime Archaic and European samples demonstrate similar amounts of variation per functional unit. Muscular impact cannot be reasonably discussed at this point, since it is too individual-specific (but see section 7.1.1 for individual case

studies). That being said, developmental noise, the perturbations occurring in the uterine and developmental environment, can be considered.

Developmental noise is random in its impact, in terms of which developmental fields or cell condensations it will influence, as well as at what stage of development it will occur. A defect, whether minor or severe, can occur if a threshold event is interrupted, often inhibiting or slowing the carefully timed cascade of cellular and structural interactions needed for healthy growth and development (Barnes 1994). Differences in the morphology and degree of variability can therefore be at least partially attributed to differences in susceptibility to developmental perturbations of various structures; that is, the window during which a developing field is vulnerable is different for each set of structures. Teratogenic agents and other microenvironmental anomalies, introduced at a specific time, may then only be able to create variation in a given number of susceptible fields (e.g., those vulnerable at that time), thereby making some characters more variable than others in that individual.

Thus, while useful information can be gathered about individuals, the random nature of the variability induced by developmental noise makes it essentially impossible to make predictive assertions concerning a single individual of any sample. That being said, an informative analysis, considering variation of both developmental and muscular etiology, can be undertaken on isolated crania, as demonstrated through the following case studies.

### 7.1.1 Case Studies – NP 56 and NP 163A2

The crania chosen to demonstrate the type of information that can be gathered from application of the asymmetry measurement package at the individual level are both visibly asymmetric, but the end form of that asymmetry is different and it is due to different factors. NP 56 is a Palaeoeskimo cranium not included as a subject in this research, and demonstrates asymmetry attributable to congenital muscular torticollis. Similarly, NP 163A2, a Colonial European cranium with mandible, manifests some asymmetry and a unique morphology, as well as fusion of the first cervical vertebra to the occipital bone, a developmental defect.

It is important to note that NP 56 is Palaeoeskimo and thus not a member of the populations sampled. It is included here because, as an obviously pathological specimen, it provoked the initial question – the quantification of cranial variation – that prompted this thesis research. It also provides an opportunity to apply the techniques used on a visually apparent pathological specimen independent of the samples used to construct the model and test the method. The individual is represented by an adult cranium, with some postmortem damage to the articulating base and medial cranial base/muscular face units, and neither a mandible nor any part of the infracranial skeleton was recovered. As evident in Figures 7.1a and 7.1b, the cranial morphology is highly asymmetric and likely a result of congenital muscular torticollis, a condition that has many potential etiologies, including a genetic predisposition to a postural uterine deformation or birth trauma due to improper parturition techniques. The classic morphology associated with congenital muscular torticollis includes a short and unilaterally broad face and flattening of the mid-

vault on the affected side with contralateral bulging; therefore, in the case of NP 56, the sternocleidomastoid muscle was likely constricted on the left side.



**Figure 7.1a: Posterior View of NP56**



**Figure 7.1b: Frontal View of NP56**

The asymmetry analysis (Table 7.1) highlights the extreme nature of the variation in all measurable functional units. Although the proposed model suggests regulation of the medial facial and cranial base structures through developmental behaviour, it is likely that the significant impact of the altered muscle action occurring after development overwrote the results of any variability-reducing mechanisms. In support of this, Persing *et al.* (1991) stated that changes in the neurocranium can result in cranial base deformation, albeit to a lesser extent than the reverse (that is, when cranial base morphology is altered and impacts the neurocranium). Even so, the face and cranial base/muscular face units are somewhat less variable than the neurocranium and the inferior neurocranium/occipital bone units. Additionally, an examination of the left-right differences for individual measurements reveals a lengthening of the right parietal as well as an exaggerated bulge, captured by the porion-bregma subtense and fraction

measurements. Left-dominant measurements include those involving the lateral muscular face and the posterior portion of the articulating base, as well as the porion-bregma chord (see Appendix G for NP 56 data).

**Table 7.1 Standardized Cumulative Asymmetry Values for Case Study Subjects**

Functional Unit	NP 56	NP 163A2
Neurocranium	9.10	5.40
Face	3.00	1.30
Inferior Neurocranium & Occipital Bone	4.30	1.30
Cranial Base & Muscular Face	3.50	5.50
Mandible	N/P	6.00
Articulating Base	(4.00)*	1.50

\*one measurement only

In this example, the asymmetry measurement package was able to accurately reflect the observed morphology to allow the creation of a comparable picture of the pathology, and it supports the etiological determination by specifying structures that are unilaterally enlarged or changed in some way. It is therefore a useful tool in reinforcing qualitative observations and in understanding among-unit interactions in pathologically malformed crania. It is also interesting to note that, although the degree of variation is dramatically higher than what would normally be expected, the pattern of variation loosely follows the proposed model, with a very high cumulative asymmetry value in the neurocranium and lowest values in the face and cranial base/muscular face units.

Individual NP 163A2 is an adult Colonial European specimen recovered from Southside Road cemetery, a mid-eighteenth century interment. The cranium and mandible have incurred some postmortem erosion damage to the left orbital margin, zygoma, maxilla and mandibular ramus. The fusion of the first cervical vertebra to the

occipital condyles is a gross anatomical developmental defect, caused by a caudal border shift, and a cleft palate is also present (Figure 7.2). Though not of significance for this analysis, it is interesting to note that the branchial arch field, including the palate, develops at the same time as the cervical spine (Taitz 2000). This potential linkage is suggestive of some sort of irregularity in the developmental environment impacting at that time. Barnes (1994) notes that the impact on overall cranial morphology for this type of defect is variable; however, the fused joint normally allows the “nodding” movement, so there would likely be changes in muscle action to compensate for this bony restriction. It is also possible that, due to the fusion of the first cervical vertebra, the dens of the axis could have impinged on the soft tissue in the region of the foramen magnum, thereby causing a basilar impression and the potential for all its attendant neurological symptoms to be present.

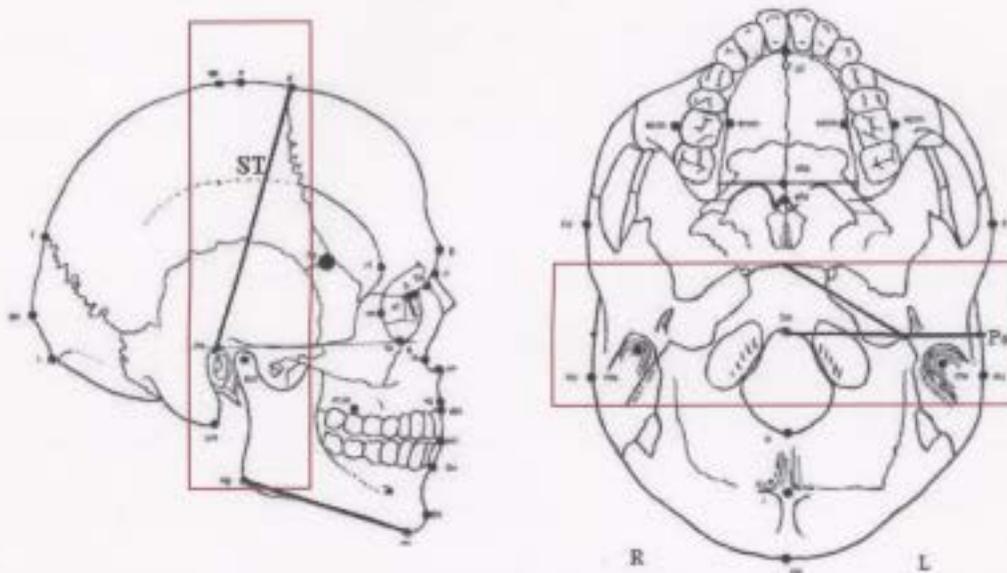


**Figure 7.2: Cranial Base View of NP163A2**

From the asymmetry analysis, further statements concerning specific cranial morphology and left-right differences can be made. Table 7.1 summarizes the standardized cumulative asymmetry values for each functional unit, and it is apparent that the face, inferior neurocranium/occipital bone and articulating base are fairly low in left-right variability. The neurocranium, cranial base/muscular face and mandible are higher in left-right variability, but it is important to note that the overall effect is less than would be expected for Klippel-Feil Syndrome, a type of osseous torticollis that causes some facial asymmetry (Barnes 1994). An examination of each measurement taken reveals that, within the cranial base/muscular face unit, the variability is due to left-side dominance of the lateral sphenoid and the basion-porion measurements, while the neurocranium demonstrates right-side dominance of the porion-bregma arc and subtense, and the mandible has a longer right mandibular body length.

Considering this in light of the proposed functional cranial model, it is important to note that the variation associated with the usually stable cranial base/muscular face unit is located in the lateral portion. As well, other variation in the cranium is localized in a medial section in the frontal plane (Figure 7.3), that is, in the areas surrounding the defect, which suggests that these deviations from symmetry may be compensatory for altered skeletal and muscular action. Should the model prove to be useful upon examination of the population samples, other possible explanations for the observed morphology could be proposed. Developmental stabilization mechanisms could still be acting to maintain medial facial and articulating base symmetry in spite of the presence of a defect that would have been evident fairly early *in utero*. In terms of the muscular impact on the cranium, the inferior neurocranium and occipital bone unit does not

manifest any important asymmetry, suggesting normal muscular activity in this area responsible for anchoring shoulder-moving muscles.



**Figure 7.3: Schematic of NP163A2 Showing Measurements Demonstrating Asymmetry and Regions Impacted by Fused First Cervical Vertebra**

Based on the proposed model, it seems likely that the observed variability is a result of compensatory muscular action changes and defect-induced changes in lateral cranial base/muscular face unit structures exerting influence on neurocranial and mandibular structure and function.

Although it is not reasonable to assign an asymmetry rating to an entire cranium, valuable descriptive information, useful on an individual basis, can be obtained. As well, some general information about the degree of variability within functional units can be inferred if an entire sample is examined at this level of analysis. Even though the relationships among structures and functional units are non-linear in terms of growth and development, thus precluding the use of an asymmetry rating, the standardized cumulative asymmetry values are real numbers, indicating a true difference in structure

size between right and left sides. The key factors influencing cranial morphology in this context are the interaction between developmental noise and developmental stabilization, and the impact of postnatal muscular activity on the cranium as it grows. The individual crania discussed are both pathological, albeit of different etiologies, and serve to illustrate the heuristic value of the asymmetry measurement package applied to individual crania.

## 7.2 Within-Sample Asymmetry and a Comparison of Maritime Archaic and European Samples

The results of the within sample analyses for both samples indicate that the cranial form is by its nature variable to some extent. Significant trends and patterns concerning the location and degree of variability for the Maritime Archaic and European samples emerge at this level of analysis. This is to be expected, since, as a complex system, it is only in the examination of an entire sample of unique individuals that predictive or descriptive statements can be made about the behaviour of the group. Thus, while it is of course impossible to predict the ultimate form of a specific cranium, minor irregularities and variations can begin to crystallize into a better understanding of the cranial morphology of the group.

Although both the Maritime Archaic and the European samples did demonstrate patterns of variation, it is in the examination of the degree of that variation and in the directional aspect of the asymmetry that they differ most markedly. The Maritime Archaic sample manifested some variation in all functional units. In terms of asymmetry, the sample demonstrated right-side structural dominance in the articulating base, cranial base/muscular face and inferior neurocranium/occipital bone units, as well as both left

and right asymmetry in the face, mandible and neurocranium units. The European sample was less variable than the Maritime Archaic and, in contrast, bilateral asymmetry favoured the left side in articulating base and cranial base/muscular face units and the right side in the inferior neurocranium/occipital bone and neurocranium units. As well, there was an absence of asymmetry in the face and a crossed-symmetry pattern evident in the mandible unit. Therefore, since both samples do demonstrate variation in the form of cranial asymmetry, the point for consideration now becomes the congruence of the observed variation with that expected by the proposed functional cranial model, which is outlined in Table 7.2.

**Table 7.2 Comparison of Observed Variation Against the Expected Variation**

<b>Functional Unit</b>	<b>Expected Variation (Model)*</b>	<b>Observed Maritime Archaic Variation</b>	<b>Observed European Variation</b>
Neurocranium	YES	YES	YES
Articulating Base			
- medial	NO	<i>YES</i>	<i>YES</i>
- lateral	YES	YES	YES
Inferior Neurocranium & Occipital Bone	YES	YES	YES
Mandible	YES†	<i>SOME</i> †	YES†
Cranial Base & Muscular Face			
- medial	NO	<i>YES</i>	<i>YES</i>
- lateral	YES	YES	YES
Face			
- medial	NO	NO	NO
- lateral	YES	YES	<i>NO</i>
-			
Suture Position			
- MBR	NO	NO	<i>YES</i>
- MSP	SOME	<i>YES</i>	<i>YES</i>
- MLD	NO	NO	NO
FORIDX	NO	<i>SOME</i>	NO
BASOPX	NO	<i>YES</i>	NO

\*YES = asymmetry; SOME = some asymmetry/symmetry present; NO = symmetry; † = crossed-symmetry pattern

### 7.2.1 Deviations from the Proposed Functional Cranial Model

In nearly all points, the observed variation matches that suggested by the model, but the examination of the few notable exceptions provides a better understanding of the sampled populations than complete congruence might. Two of the points of divergence from the model are shared by both Maritime Archaic and European samples, specifically the presence of asymmetry in the medial cranial base and the degree of asymmetry of suture positioning. Additionally, the Maritime Archaic demonstrates less asymmetry than expected in the mandible and the European sample manifests more symmetry in the lateral face than the model proposes.

#### 7.2.1.1 Variability in the Medial Cranial Base

While both the Maritime Archaic and European samples present trends indicative of some degree of variation in the cranial base, the Maritime Archaic sample manifests comparatively more variability in this region. The model suggests that there ought to be less variation in this region, which incorporates the medial cranial base/muscular face and the medial articulating base units. This hypothesis is based on the importance of a stable cranial base for normal growth and function of the cranium and surrounding soft tissue. The cranial base provides important bony support for numerous foramina allowing the passage of nerves and blood vessels into the surrounding tissues (Lieberman *et al.* 2000), as well as articulation with the cervical spine and providing the foundation for growth and development. Due to its structural and functional importance, it follows that Clarke's (1998a) assertion that this importance dictates the degree of regulation by developmental control mechanisms would apply.

The examination of the Maritime Archaic remains did not reveal any glaring developmental defects in this region, making the explanation of one or two highly pathological individuals causing the observed sample variation untenable in this instance. In the context of this research, this variability could be suggestive of either periodic increase in the level of developmental noise or instability in the genetic system, or of muscular activity throughout the growth period impacting the structures in this region. The developmental environment is very vulnerable to external influences at certain points, particularly during threshold events, embryonic induction and general cell growth and differentiation early in the pregnancy. Section 3.2 discusses several potential stressors that act to reduce available energy and thereby create phenotypic (morphological) variation within a sample, including restricted food, adverse climate, infection, metabolic abnormalities and pollution. Moreover, Benderlioglu and Nelson (2004) suggest that there is an important seasonal component to the condition of the developmental environment; namely, that maternal health and nutrition in a hunter-gatherer group may be likely to fluctuate throughout the year, thus rendering the uterine environment more suitable or less suitable for growth and development at different times of the year. The authors noted that late winter and spring births demonstrated more fluctuating asymmetry (e.g., asymmetry caused on an individual level by developmental noise) than other births; note that this type of influence would affect the whole skull.

Asymmetry is also known to have a small but crucial genetic component, such that the loss of genetic variation within a group can increase levels of fluctuating asymmetry. Canalization, the developmental mechanism that acts at the population level to reduce among-individual variability, may still attempt to eliminate sensitive genotypes

from the group and thus increase genetic and developmental stability. If, however, the ability to resist a particular effect of environmental change is not present, it is difficult to introduce it or to have it emerge within a small gene pool. It is known that in genetically homogeneous groups the range of adaptive responses found in the genotype is lower than in more heterogeneous groups, which consequently lowers the ability of that group to respond or resist changes in the environment (Moller and Swaddle 1996).

There is also the possibility that muscular activity may be impacting this region throughout the growth period. Carter and Beaupre's (2001) model of bone response to strain suggests that growth is a function of biological components whose influence changes with age and as major growth ceases, and of mechanobiological components that cause modeling to occur in response to applied direct or indirect loads in an effort to optimize bone shape. The late ossification of the basisphenoid synchondrosis in this region may lengthen the opportunity for strain to have an obvious impact on the medial structures; Anderson (1983) suggested that handedness would have an observable influence on the basisphenoid and occipital condyles in particular. While perhaps an optimistic expectation based on the gross anatomical methods used here, it might still be reasonable to expect some sort of an exaggeration of right-side structures, since it is usual for around eighty percent of any population to be right-handed (Ortner and Putschar 1985). If this is the case, a similar pattern of asymmetry should be apparent in the other regions of the cranium subject to muscular activity.

The Maritime Archaic, the more variable of the two samples in this region, does demonstrate an overall trend to right-side dominance in the units likely to be subject to muscular activity, as well as important right tails for both the basion-anterior occipital

condyle chord and the BASOPX index. Even so, it is unlikely to be the exclusive cause of the variation in the medial cranial base. Marshall's (1990) examination of side-specific osteoarthritic changes to limbs and joints did not reveal any particular pattern of preferential limb usage in this group. Thus, while it is likely that an overall high activity level may have contributed to the asymmetry observed, previous research indicates that it is unlikely to account for all the variation present. The analysis of some of the measurements does not suggest a specific side, merely that there is left-right variability in the sample in that region or functional unit.

The potential influence of environmental or genetic stress on the variability in the medial cranial base is likely to be somewhat more important in this group. Although Pálsson (1988) suggests that a maritime hunter-gatherer lifestyle was likely more stable in terms of subsistence and movement, it is entirely possible that fluctuations in the quality and quantity of available resources would be common on a seasonal basis. This would, coupled with the potential for infection, impact the developmental environment negatively. Genetic homogeneity would also play an important role since, although Kennedy (1981) does conclude that the Maritime Archaic were exogamous (that is marrying outside of their immediate group), the overall population of the entire geographic region would be limited, due to the environment, resource availability and the lack of technology or even impetus to travel very great distances. It is possible that this is, as alluded to Chapter 5, a large-scale manifestation of small, individual-based perturbations, both resulting in and caused by sensitivity in the genotype to changing environmental conditions. It is most likely that, while muscular activity does play an important role in shaping cranial base morphology, the environmental conditions and

genetic system have a greater effect on the ultimate form of this ontogenetically sensitive region for the Maritime Archaic sample.

The European sample manifests less asymmetry in this region, with only the opisthion-anterior occipital condyle chord (OPC) and the lateral sphenoid measurement (SP1) demonstrating any tendency to bilateral variation. The two indices concerning the foramen magnum were essentially symmetric and of low variation. There is very little tendency towards asymmetry in the entire sample; that being said, lateral structures in the cranial base/muscular face and articulating base units are somewhat asymmetric, similarly favouring the left side. Unlike the Maritime Archaic, the mandible also presents a more emphatic pattern of crossed-symmetry. Coupled with the absence of any data concerning infracranial asymmetry or patterns of osteoarthritic wear to provide supporting or conflicting evidence, this suggests that the medial articulating base unit might be prone to react to muscular activity in this fashion.

Due to the nature of the sample, it is impossible to presume any genetic homogeneity, since part of the sample is sixteenth-century Basque and the other a broad sample of eighteenth and nineteenth-century Colonial European. While certainly one individual, NP163A2, manifests a major developmental defect, there is, according to BASOPX and FORIDX, very little variation in this region which further supports the hypothesis that the observed asymmetry is muscular in this instance. The foramen magnum and occipital condyles are fairly sensitive to developmental upsets, which may range from minor protuberances to third condyles or the more severely asymmetric foramina caused by conditions like basilar impression. There are, however, no major manifestations of this sensitivity for the European sample. As a result of the broader

cultural background of the European sample, developmental regulation can be attributed to mechanisms like canalization, which would be better able to suppress environmentally sensitive genotypes at the population level. Bear in mind that, in this instance, the population referred to spans a four hundred-year period and much of Western Europe. The interaction between developmental noise and developmental stabilization would still of course be acting to create and mitigate the effects of unstable environmental conditions on an individual level, resulting in the observed variation. It therefore seems most appropriate to attribute the asymmetry and general variability observed in the European sample to the impact of muscular activity, manifesting in a broader sampling of individuals, and fluctuating asymmetry, as an outcome of developmental noise occurring at the individual level.

#### 7.2.1.2 Variability in Suture Position

The location of suture growth is largely a passive, secondary outcome of cranial bone growth and endocranial soft tissue expansion. Sutures are synarthrotic fibrous joints and allow limited flexibility and movement, which decreases over time as they eventually fuse or obliterate completely in middle adulthood. Although the proposed functional cranial model suggests that, as a part of the neurocranium unit, bregma (MBR), lambda (MLD) and the intersection of the coronal suture and the temporal line (MSP) should also demonstrate some asymmetry, it is implicitly assumed in most osteometric methods, including this one, that these landmark points will be located medially or in the same position bilaterally. This was, however, not the case in all individuals; MSP was found to

be asymmetric for both European and Maritime Archaic samples, and MBR was found to be asymmetric for the European sample only.

Most likely the result of both muscle action and uneven rates of growth between left and right neurocranial bones, the midline landmarks of lambda and bregma are, for the most part, not subject to significant levels of asymmetry. The European sample was, however, somewhat right dominant for the location of bregma. It is important to note that the porion-bregma arc (PBA) measures the same region of the parietal and temporal bones, and PBA (European) does demonstrate similar right dominant asymmetry. This suggests that the variation in MBR may in fact be due to asymmetry of the temporal/parietal region and not asymmetry of bregma itself. It is equally important to note that PBA (Maritime Archaic) is similarly asymmetric, and MBR for that sample demonstrates no significant asymmetry; thus, it is difficult to make any real inferences in this instance. The neurocranium units for both groups do demonstrate different patterns of bilateral asymmetry; the Maritime Archaic has both left and right-favouring measurements, whilst the European is entirely right-favouring. It is possible that the right-favouring asymmetry of bregma is due to the same muscular and/or developmental factors at work on all neurocranial structures in the European sample; in any case, this raises an interesting point for future consideration.

There is also significant asymmetry in the location of the intersection of the temporal line and coronal suture for both samples. Although it is similar to stephanion, this is not a commonly used landmark point; it was used in this study as part of the lambda-temporal line measurement examining neurocranial asymmetry. The temporal line is a region of muscle attachment for the temporalis, which inserts on the coronoid

process of the mandible and is involved in closing and maintaining the at-rest position of the mandible. The asymmetry observed in this region suggests that the variation in the lambda-temporal line chord and arc may be due to lateral and anterior asymmetry, rather than in the more stable lambda region. The cause of this is almost certainly due to differential muscle activity and the mildly asymmetrical development of the cranial base; thus, it is unsurprising that variability should be found in both the Maritime Archaic and the European samples. It is also reasonable to postulate some link to mandible function, perhaps reflected in that unit's crossed-symmetry pattern, but it is impossible to infer a relationship from this analysis.

#### 7.2.1.3 Other Deviations from the Proposed Functional Cranial Model

In addition to the deviations from the model common to both samples, the Maritime Archaic also manifested a less definite crossed-symmetry pattern in the mandible and the European sample had a higher degree of symmetry than expected in the lateral face. In the Maritime Archaic sample, the mandible manifests some general variability with a few significant tails in the distribution, favouring both left and right sides. Although not a focus of this work, the unusually healthy dentition (e.g., low attrition) that was observed suggest that the maritime subsistence diet may have been less difficult to masticate than a Basque or Colonial European diet (Ortner and Putschar 1985). Moreover, Merbs (1983) suggests that hunter-gatherer group members may have used their mouths as an “extra hand”; perhaps in this case, that use occurred bilaterally or not at all. It is also possible that the measurements chosen are not adequate to the task of

capturing the variation present in the mandible; a re-evaluation and perhaps an expansion of the mandible unit measurements is most likely required.

There is a high degree of symmetry present in the lateral face for the European sample; with the exception of some minor variability in the nasion-ectomolare chord, all measurements demonstrate symmetry. It is possible that the balanced nature of the asymmetry demonstrated by the mandible is influencing the shape of the lateral face or that canalization was able to act to the extent that the entire face is more apt to be symmetrical, in lieu of just the median. It seems, however, more plausible that the problem lies rather in the data itself. The European sample suffered more heavily than the Maritime Archaic in terms of postdepositional damage, particularly to the lateral face and zygomatic arches, to the extent that the nasion-zygion chord measurement could not be taken on a sufficient number of individuals. Moreover, there are fewer European faces to measure, even without the additional consideration of structural damage. It is therefore contended that if a larger or more intact sample could be examined, lateral facial asymmetry might be detectable.

### 7.3 Explanatory Value of the Functional Cranial Model

The utility of this approach can be gauged based on the information about the sampled populations that can be gleaned from application of the model, specifically in terms of health, behaviour and activity, and environment. The two samples are temporally distinct, but are also composed differently; the Maritime Archaic sample is drawn from contiguous cemeteries, and the European sample is composed of a set of disparate colonial interments and a series of Basque burials. To some extent, the output of

the functional cranial model must be evaluated comparatively; that is, by asking what unique factors make the two samples different in some aspects but not in others. Even so, the interpretation of the remains can be placed in the appropriate context of previously performed, relevant archaeological and historical research.

Activity and behaviour encompass a wide range of culture-influenced factors, including postnatal physical activity, parturition practices and marital behaviour. Both groups demonstrate asymmetric variation in regions likely to be impacted by post-developmental muscular use (e.g., lateral portions of the articulating base unit) and, while the European sample is relatively balanced in terms of structural dominance of one side over the other, the Maritime Archaic sample manifests an emerging tendency to right side structural dominance for many units. Having provisionally rejected handedness as a direct cause for this variation, it is possible to advance the hypothesis that the emerging trend is due to a general similarity of activity among most groups members; that is, less variation in behaviour among group members.

Neither the Maritime Archaic nor the European sample shows evidence of the detrimental parturition practices, such as assisted births or twisting during birth, that induce congenital conditions like muscular torticollis (e.g., Skinner *et al.* 1989). It is possible that at least some of the asymmetric flattening/bulging of the neurocranium is due to the positional deformity following birth or intrauterine molding immediately preceding birth suggested by Myslobodsky *et al.* (1987) that can carry over into adulthood. Another aspect of cultural behaviour is the way in which family groups and, by extension, genetic character, are created. The genetic character of the sampled populations suggested by this research supports the notion that, while both groups are

predominantly exogamous, low population density seems to increase genetic homogeneity. Thus, the European sample exhibits more heterogeneity while the Maritime Archaic sample appears to be more homogeneous; this is, of course, exacerbated by the sample composition. This is reflected not only in the differing degree of developmental stability, notably of the medial cranial base, but also in the dispersal of measurements. The European sample is less variable amongst individuals on the whole, but there are individuals with discernable developmental defects of varying severity. In contrast, the Maritime Archaic sample does not demonstrate evidence of developmental defects of the cranium, but it is more variable and there are often extreme (outlying) subjects for a given measurement. Thus, while the European sample has low developmental noise (that is, as can be inferred from fluctuating asymmetry) and a few defects, the Maritime Archaic has a comparatively higher level of developmental noise, but no glaring defects in the developmental fields examined.

Developmental stability, including developmental noise and fluctuating asymmetry, is also mediated by extrinsic environmental factors. It has been established that there is an important difference in stability between the Maritime Archaic and the European samples, and there are two likely causes for this in the context of environmental impact. It could indicate differential ability of the genetic system to resist perturbations in the environment, that is, to buffer against unexpected occurrences like maternal illness or pollutants. It is also possible that the observed differences are due instead to constant extreme environmental stress or a high degree of fluctuation in environmental conditions while individuals are *in utero* developing. Real developmental noise is most likely a sort of equilibrium relationship between buffering ability and

degree of stress, and both factors highlight the importance of place of birth and the maternal environment in understanding factors influencing cranial morphology in this context.

While most of the Maritime Archaic sample was likely born and interred in essentially the same environment, albeit with the potential for unpredictable environmental fluctuations during development, the same cannot reasonably be said for the European sample, drawn as it is from several locations and time periods characterized by rapid socio-economic change. Additionally, the place of birth cannot be reasonably assumed to be constant for all members of the sample and could potentially range over most of Western Europe and North America. An assessment of environment can thus only be very general; in this case, a low-stress environment is indicated for the most part with few fluctuations, although there is one individual with a major developmental defect. Even though Newfoundland in the eighteenth and nineteenth centuries was a maritime-based economy, infrastructure existed to adequately sustain the population through seasonal changes in food production and fishing. Similarly, the Basques' primary residence was in Spain and it is reasonable to assume both that many whalers were likely born there and that provisions to feed whalers were often transported from their homeports. In this fashion, the European sample was buffered externally against changing resource availability. Instability caused by poor maternal health would still be a possibility, but some form of medical infrastructure would most likely have been available, particularly in the latter eighteenth and nineteenth centuries.

The Maritime Archaic were likely subject to fewer nutritional stresses than many hunter-gatherer groups, due to the exploitation of maritime resources (Pálsson 1988).

Even so, some variability in the environment or resource availability may have existed, probably in the form of seasonal fluctuations and changing living conditions, which would manifest as low level developmental noise with few extremes. Therefore, the Maritime Archaic lived in a comparatively high stress environment, and this stress was likely caused by a high degree of change in environmental conditions throughout the year. Conversely, the Europeans likely lived in a more constant environment, stabilized by external sources and infrastructure, though of indeterminate quality in terms of nutrition and health. Thus, through the functional cranial model, inferences can be made concerning the type of environment in which development occurred (both intrinsic and extrinsic), as well as activity and cultural behaviour patterns.

#### 7.4 Distinguishing Normal and Asymmetric Crania

The question of how to make a distinction between normal and asymmetric crania is an important theoretical point, and a surprisingly complex problem. It is impossible for a static “threshold” value, beyond which any cranium is always asymmetric, to be defined for a single skull or even a single functional unit. Certain trends can be expected based on functional unit, e.g., some regions being consistently more likely to exhibit variation. These consistencies can be used to determine if a cranium is exhibiting unusual morphology or if it falls generally within expected limits for a particular functional unit. Ultimately, however, a definition of “normal” must be sample specific to be strictly accurate. That being said, Lieberman *et al.*'s (2000) understanding of the impact of the basicranium was not population-specific, so “normal” for a given sample can be defined most accurately through a functional cranial model adapted to that sample. On an

individual basis (e.g., for isolated crania), the model can still both describe defect-induced changes and assist in the understanding of the effect of those changes in a functional manner; recall that the model distinguished between two different asymmetric shapes fairly clearly through standardized cumulative asymmetry values.

Perhaps a better approach is to define asymmetry, rather than to attempt to describe the normal cranial form, since, as Woo (1931) stated, some degree of asymmetry is entirely to be expected. The methodology used for this research captures and describes asymmetry on two levels, for individuals and samples, and an asymmetric skull will, as previously alluded to, deviate from the sample-adapted functional cranial model or will demonstrate unusually high standardized cumulative asymmetry values. Even so, a really clear distinction between normal and asymmetric still cannot be made in the absence of a pathological condition.

The problem is essentially the relative importance of magnitude of asymmetry and nature of asymmetry. Is the asymmetry of one functional unit more important than that of a different unit? It has been established that asymmetry of those functional units encompassing the basicranial region has important developmental and functional repercussions, but, numerically, this asymmetry will reflect more strongly in the neurocranium through the “ripple effect” mechanism previously described. In this sense, the nature and region of the asymmetry is more important than magnitude, particularly when variation is found in regions that ought to be most symmetric. Another potential approach is to simply count the number of units demonstrating asymmetry, but once again, magnitude versus nature must be considered. From an interpretive standpoint, minor variation in all functional units can mean something completely different (e.g.,

developmental instability) than extreme variation in only a few units (e.g., developmental defect). Therefore, based on the functional cranial model and methodology developed in this thesis, a blanket numerical threshold for distinguishing asymmetric and normal skulls cannot exist for all crania within a sample. Even so, the standardized cumulative asymmetry values can be used in conjunction with the functional cranial model to empirically analyze asymmetry at the individual level and to define asymmetry on a case-by-case basis. At the sample level, such determinations of the degree and cause of asymmetry are less precise, but going through the process of applying the model to a population sample can provide some idea of the general trends and patterns present. It can also assist in fine-tuning the model and defining individual skulls drawn from that sample more certainly as normal or asymmetric.

## 7.5 Summary of Asymmetry Analysis and Interpretation

One of the most important outcomes of this discussion is the degree of continuity between the individual variation and the emerging trends and patterns evident upon examination of larger samples. As well, it is now possible to empirically examine cranial asymmetry at the individual level from a functional perspective. The degree of congruence between the proposed functional cranial model and the Maritime Archaic and European samples is satisfactory, particularly in light of the deviations from the model and the information obtainable about these groups through devising possible explanations for those deviations. The heuristic value of the model, when it is taken in conjunction with the theoretical and background information provided in Chapter 5, allows a framework for the construction of reasonable, group-specific explanations of variation

and asymmetry, and a better understanding of the interaction of functional units. The examination of suture positions is helpful in isolating the location of asymmetry noted in the neurocranium, while simultaneously raising an important point about assumptions made in the osteometric method. The minor departures from the model of the Maritime Archaic mandible sample and the European face sample, while possible to explain to some degree, are more likely due to measurement package construction and insufficient data. Finally, although it is not possible, based on this work, to devise a static numeric definition of cranial asymmetry, asymmetric forms can be defined for individuals, both singly and within a studied sample.

## Chapter 8 Conclusion

*“The human skull is definitely and markedly asymmetrical.*

*It is not a question of the bones of individual crania differing from a symmetrical type,  
but the type cranium is itself asymmetrical.”*

*(Woo 1931)*

### 8.1 Research Objectives Revisited

The primary undertaking of this research and the aspect upon which the bulk of effort has been focused is the development of a set of measurements and interpretive model that can provide information about cranial asymmetry for entire population samples as well as individual isolated crania. The data collection portion entailed the application of an exploratory set of asymmetry measurements to the selected crania from the Maritime Archaic and European samples to determine the efficacy of the new array of measurements and to provide quantitative data examining the frequency and nature of any variation in morphology. The interpretation of these data was based on the proposed functional cranial model, a variation on and an expansion of Moss's functional matrix hypothesis, and the resulting information was placed in an appropriate archaeological and historic context for the two cultural groups. Through this, the relationships among health, cultural behaviour and activity, and environment, and their impact on cranial morphology, were elucidated and discussed. Moreover, it has been demonstrated that inferences can be made about these factors based on an examination of patterns of cranial morphological variation at the individual and sample levels. The final objective of this research was to ascertain if a determination can be made between normal and asymmetric

crania, and, in doing so, to attempt to determine when a cranium can be said to be normal or asymmetric.

The theoretical background and foundation for the interpretation of the metric data was developed with Moss's functional matrix hypothesis as an initial concept, and was further expanded through the application of complex systems theory. Defining the cranium through complex systems theory allowed it to be conceptualized as a dynamic, integrated construct, that can be examined either singly as a system of functional units or as a constituent of a system composed of individual crania. The body of the functional cranial model was gleaned from a review of current growth and development literature examining developmental stability, bilateral asymmetry and skeletal biology, as well as the way in which these factors interact with each other and react to the external environment. In this way, an adaptive but informative model was developed to explain the variation in cranial form across groups and within individuals.

The major difficulty encountered in the development and analysis of the asymmetry measurement package was in maximizing sensitivity to small variations in bilateral structures. Differences of only a few millimetres between left and right sides are biologically significant, particularly when one considers the "ripple effect" nature of minor asymmetry in the cranial base translating into more visible asymmetry in the neurocranium. However, because these differences are so small, it is difficult to equate statistical significance to biological significance. The best way to compensate for this lack of congruency is to design the analysis to address functional units, resulting in pooled or cumulative differences at the individual level (e.g., standardized cumulative asymmetry values), and to use simple, sample-based statistics to create a multi-focus

picture. In this way, patterns can be simultaneously examined at the functional unit or group level in the context of the sample, for both the Maritime Archaic and European samples without creating a type skull. Furthermore, the complex systems aspect of the theoretical model validates choosing this approach since predictions are not made specifically; only general trends and probabilities based on either functional units or sample patterns are considered.

The most critical research objective was to develop a means to document cranial asymmetry through which the magnitude and nature of any observed variation could be recorded. The set of measurements compiled for this study was able to adequately document asymmetry for the chosen samples. Based on the concept of fluctuating asymmetry, left-right differences can be equated with variation or a lack of stability. Through application of the functional cranial model, it has been determined that the degree and incidence of variability changed within and among individuals for both samples in a fashion broadly predicted by the model; as well, the model allows some inferences about cause to be made, at both the individual and population sample level. Pathological etiologies are only attributable at the individual level, but normal causes of cranial asymmetry include both developmental stability and muscular activity, and can be discussed for both individuals and samples.

Although health, cultural behaviour and activity, and environment each have a definite impact on cranial morphological variability, the type of inferences that can be made changes depending on the organizational level of observation. At the individual level, the functional cranial model and asymmetry measurements can be used to gather information about all three factors. Any unusual asymmetry present can be examined

from both a functional perspective (e.g., the impact on function), and a morphological perspective by comparing the shape of different functional units, as well as through the lens of fluctuating asymmetry. Fluctuating asymmetry is indicative of instability in the developmental environment which, at this level, is mediated directly by developmental stabilization and influenced by maternal health during growth and development *in utero*. It is therefore possible to create a functional picture of a given cranium, such that inferences can be made about developmental stability and fluctuating asymmetry, muscular activity, and maternal health and environment, depending on the type of cranial morphological variability present.

Expanding the asymmetry analysis to the population-sample level allows inferences and hypotheses to be made about the entire group. Some acuity is by necessity lost in shifting to this level of organization, or analytical perspective; for example, casuses of specific defects or changes in form due to specific muscle uses cannot be directly addressed. Even so, some types of activity, genetic character and the impact of external environmental factors can all be examined in the sample context. In terms of activity, very rough patterns of muscle use can be discerned through consistent exaggeration of one side over the other in regions affected by attached muscles. Attribution of this asymmetry to a specific cause is difficult, but, depending on the strength of the pattern, inferences can be made, such as general similarity of activities among group members.

Genetic character, such as the degree of homogeneity and the ability to adapt to changing conditions, can also be discussed. Fluctuating asymmetry, in this case in the context of developmental stability and canalization, is an important indicator of genetic

character, as well as intra-group variability; that is, the dispersion of individuals within the sample. Genetic character and environmental impact are intertwined in terms of their influence on fluctuating asymmetry. The ability to maintain developmental stability is affected by both genetic character and environmental stress. Changing levels of stress, either among individuals or over time for the whole group, can result in developmental noise, which impacts on the individual level to become a recognizable pattern of instability for the sample.

Information that is new, and that further supports prior research concerning the Maritime Archaic and Colonial-era Europeans, has emerged from this study of cranial asymmetry. The patterns of variation and stability described for the Maritime Archaic are indicative of a fairly genetically homogeneous group, which, coupled with known exogamous behaviour, suggests a low-density, partially sedentary population. Despite the slight side-dominance in regions susceptible to muscular activity, it follows from Marshall (1990) that group members may have been involved in a variety of activities (i.e., minimal trade or task specialization), the array of which would be fairly similar for each individual. There is no evidence of major developmental defects in the cranium, which suggests a low incidence of severe intrauterine environmental fluctuations. That being said, there is a high level of fluctuating asymmetry, and this is likely due to a low ability to buffer against random noise, coupled with an increased susceptibility to seasonal fluctuations in environmental conditions and varying resource availability impacting maternal health.

In contrast, the European sample represents a more varied group, drawn from a wider geographic area and a larger population. There is also some evidence of asymmetry

in regions of the cranium expected to reflect muscular activity, which may be an emerging pattern describing handedness, a specific behaviour or task specialization. Participation in a narrower range of activities (e.g., specialization) may result in a different, less balanced, distribution of muscular forces over the skeleton, allowing handedness to become discernable. As well, even though there were major developmental defects present, there is less fluctuating asymmetry in the European sample than in the Maritime Archaic sample, likely due to a similar combination of genetic and environmental factors. It is possible that the environment in which the individuals comprising the European sample lived was more constant in terms of resource availability and that, as a genetically broader group, they were better able to buffer against random fluctuations during development.

## 8.2 Future Research Considerations

Both the new set of asymmetry measurements and the functional cranial model would benefit from application to a larger and different population sample, for both verification and refinement of the model and measurements on another “normal” population sample. As well, the set of measurements designed for the mandible requires reevaluation. This is a unique and difficult structure to analyze, since it is likely to manifest both left and right asymmetry in different regions, as well as being subjected to torsion forces. A new approach to this region may be needed, and a better understanding might be gained perhaps through examining angles, such as the gonial angle, or through the creation of an index, such as those designed for the foramen magnum, to examine left/right shape differences. Creating an artificial midline from the symphysis directed

posteriorly, and then measuring the midline to condyle and midline to gonion chords could accomplish this fairly easily. However it might be analyzed, the mandible certainly bears further examination than is currently incorporated in the model. Additionally, asymmetry of dental tissue could be incorporated, whether through measurement of morphology or an examination of attrition and patterns of wear, into the data collected for this study, or to further studies of cranial asymmetry.

One of the interesting outcomes of this research is the ability to create a functional unit-based numerical picture of pathological asymmetry. It would be particularly interesting to apply this methodology to a series of pathological crania to compare the impact of, for example, a defect or category of defects in order to determine if the influence on morphology and functionality is consistent. As well, it would be of benefit to test the ability of the measurements and model to create comparable pictures of similar and different pathologies within and among population samples. There are also other regions of the skeleton that have been examined from a functional perspective, such as the os coxae-sacral complex (e.g., Herneth *et al.* 2004; Ruff 2005) or the foot (e.g., Le Minor and Wolff 2004), that might benefit from a similarly designed functional model.

### 8.3 Summation

The most important objective of this research was to develop both a means of quantifying cranial asymmetry and an explanatory model founded on Moss's functional matrix hypothesis. The functional cranial model and the asymmetry measurements are most powerful, in terms of ease of use and maximization of information, at the individual level. Through the use of standardized cumulative asymmetry values, an intuitive and

empirically testable picture of the cranium in this context emerges. Although some sensitivity is lost at the sample level, trends and patterns emerge, and these can be discussed in their own right or used to refine the functional cranial model for the interpretation of individual case studies. In this way, by reflecting on a single skull as a constituent of a larger population, a better understanding of cranial asymmetry can be developed.

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**Appendix A**  
Data Sheets

**Subject #**  
**Date:**

**Visual Assessment**

Gender:

Size:

Mastoid:

Brow:

Frontal:

Nuchal:

Mental:

Damage:

Asymmetry:

1. Base:

2. Vault:

3. Face:

4. Mandible:

*Standard Cranial Measurements*

<i>Code</i>	<b>Measurement</b>	<b>Code</b>	<b>Measurement</b>
MCB		BAV	
MCL		MFB	
BBH		NAB	
BSB		NAH	
BZB		BCB	
UFH		BGB	
TFH		SYH	
PAL		MPL	
PAB		FMB	
MAB		FML	
MAL		BMB	
BAN		BAB	

**Subject #**

**Date:**

---

**Asymmetry Measurements**

<b>Code</b>	<b>Left</b>	<b>Right</b>	<b>Code</b>	<b>Left</b>	<b>Right</b>
ASA			ORH		
LTC			BZM		
LTA			AMO		
LTF			SP1		
LTS			BAP		
PBA			OCC		
TCH			BSC		
TST			OPC		
PBF			BAM		
NAP			MAO		
NAZ			LMD		
PZT			RHT		
NEM			MBL		
NZM			RBR		
PZM			CWM		
ORB			GID		

---

**Pictures**

View: Lateral      Photo No:  
View: Frontal      Photo No:  
View: Base      Photo No:  
View: Mandible      Photo No:

---

**Machine Measurements**

MBR:  
MSP:  
MLD:

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**Notes:**

## Appendix B

### Standard Cranial Measurements – Explanatory Note

MCB	-	maximum cranial breadth
MCL	-	maximum cranial length
BBH	-	basion-bregma height
BSB	-	bistephanic breadth
BZB	-	bizygomatic breadth
UFH	-	upper facial height
TFH	-	total facial height
PAL	-	palatal length
PAB	-	palatal breadth
MAB	-	maxillary breadth
MAL	-	maxillary length
BAN	-	basion-nasion chord
BAV	-	basion-alveolare chord
MFB	-	minimum frontal breadth
NAB	-	nasal aperture breadth
NAH	-	nasal height
BCB	-	bicondylar breadth
BGB	-	bigonial breadth
SYH	-	symphyseal height
MPL	-	maximum projective length
FMB	-	foramen magnum breadth
FML	-	foramen magnum length
BMB	-	bimastoidale breadth
BAB	-	biasterionic breadth

Standard Cranial Measurements – Maritime Archaic  
(adapted from Bass 1971 and Brothwell 1972)

Subject	MCB	MCL	BBH	BSB	BZB	UFH	TFH	PAL	PAB
NP-01B	125.00	174.00	128.00	112.00	.	63.00	.	.	43.00
NP-06	136.00	186.00	131.00	114.00	135.00	66.00	112.00	.	43.00
NP-08	.	.	.	.	.	.	.	.	.
NP-09	140.00	178.00	122.00	126.00	135.00	64.00	.	37.00	40.00
NP-10A	135.00	178.00	130.00	101.00	.	65.00	98.00	34.00	43.00
NP-12A	144.00	191.00	132.00	111.00	.	69.00	.	41.00	41.00
NP-16A	133.00	180.00	131.00	100.00	.	75.00	118.00	43.00	37.00
NP-15	.	.	.	.	.	.	.	.	.
NP-16B	.	.	.	.	.	.	.	.	.
NP-18A	.	.	.	.	.	.	.	.	.
NP-18B	140.00	177.00	126.00	111.00	121.00	61.00	.	42.00	35.00
NP-21	138.00	185.00	.	118.00	.	.	.	.	.
NP-22D	150.00	187.00	134.00	116.00	146.00	69.00	.	40.00	40.00
NP-25	.	.	.	.	.	.	.	.	.
NP-27A	145.00	188.00	132.00	115.00	139.00	72.00	122.00	41.00	39.00
NP-28A	137.00	180.00	128.00	113.00	133.00	68.00	109.00	43.00	43.00
NP-29	144.00	179.00	137.00	118.00	145.00	67.00	113.00	40.00	44.00
NP-32	137.00	175.00	135.00	103.00	.	65.00	111.00	.	38.00
NP-34	140.00	191.00	134.00	110.00	.	.	.	.	35.00
NP-35A	139.00	184.00	135.00	114.00	143.00	68.00	119.00	43.00	39.00
NP-37B	142.00	174.00	130.00	114.00	.	66.00	112.00	42.00	39.00
NP-37C	.	.	.	110.00	.	.	.	.	.
NP-40A	.	.	.	.	.	.	.	.	42.00
NP-44A	134.00	179.00	122.00	108.00	135.00	64.00	107.00	44.00	41.00
NP-44B	136.00	180.00	127.00	.	.	64.00	.	38.00	42.00
NP-47A	132.00	186.00	133.00	112.00	.	71.00	118.00	43.00	36.00
NP-49A	139.00	179.00	129.00	103.00	.	69.00	115.00	39.00	36.00
NP-50A	.	.	.	.	.	.	.	.	.
NP-50B	.	.	.	.	.	.	.	37.00	40.00
NP-52	136.00	171.00	123.00	93.00	135.00	66.00	113.00	38.00	38.00
NP-60A	143.00	178.00	129.00	99.00	143.00	64.00	115.00	.	45.00
NP-60B	145.00	172.00	.	.	132.00	.	.	.	.
NP-60D	.	.	.	.	.	65.00	.	38.00	39.00
NP-61A	138.00	172.00	121.00	97.00	129.00	63.00	.	.	41.00
NP-61B	136.00	183.00	119.00	.	126.00	66.00	107.00	41.00	37.00
NP-63	148.00	179.00	.	106.00	.	.	.	.	.
NP-67	146.00	189.00	131.00	122.00	.	.	.	.	.

### Standard Cranial Measurements – Maritime Archaic

Subject	MAB	MAL	BAN	BAV	MFB	NAB	NAH	BCB	BGB
NP-01B	67.00	.	106.00	102.00	.	19.00	52.00	.	.
NP-06	66.00	.	96.00	91.00	105.00	24.00	53.00	120.00	108.00
NP-08	.	.	.	.	.	.	.	121.00	109.00
NP-09	66.00	53.00	104.00	101.00	106.00	28.00	51.00	.	.
NP-10A	66.00	47.00	102.00	95.00	98.00	21.00	48.00	.	94.00
NP-12A	69.00	54.00	106.00	105.00	115.00	21.00	54.00	.	.
NP-16A	63.00	52.00	111.00	108.00	.	25.00	58.00	.	88.00
NP-15	.	.	.	.	.	.	.	136.00	111.00
NP-16B	.	.	.	.	.	.	.	.	.
NP-18A	.	.	.	.	.	.	.	121.00	111.00
NP-18B	59.00	51.00	95.00	96.00	98.00	21.00	49.00	.	.
NP-21	.	.	.	.	102.00	.	.	.	100.00
NP-22D	67.00	49.00	99.00	96.00	109.00	23.00	54.00	.	.
NP-25	.	.	.	.	.	.	.	127.00	118.00
NP-27A	66.00	49.00	99.00	95.00	102.00	25.00	55.00	119.00	113.00
NP-28A	65.00	53.00	106.00	105.00	108.00	24.00	52.00	121.00	97.00
NP-29	70.00	54.00	103.00	100.00	106.00	22.00	49.00	132.00	120.00
NP-32	65.00	51.00	109.00	104.00	105.00	23.00	49.00	121.00	105.00
NP-34	65.00	53.00	108.00	.	180.00	26.00	61.00	.	109.00
NP-35A	67.00	54.00	100.00	102.00	107.00	26.00	51.00	126.00	103.00
NP-37B	67.00	46.00	100.00	97.00	.	23.00	54.00	127.00	111.00
NP-37C	.	.	.	.	.	.	.	.	.
NP-40A	69.00	52.00	.	.	.	27.00	.	132.00	119.00
NP-44A	65.00	52.00	99.00	96.00	103.00	24.00	55.00	122.00	104.00
NP-44B	65.00	51.00	101.00	105.00	.	26.00	53.00	.	.
NP-47A	65.00	52.00	109.00	108.00	100.00	24.00	56.00	126.00	111.00
NP-49A	60.00	46.00	98.00	95.00	101.00	23.00	49.00	112.00	96.00
NP-50A	.	.	.	.	.	.	.	.	109.00
NP-50B	65.00	51.00	.	.	.	.	.	127.00	117.00
NP-52	57.00	43.00	95.00	91.00	93.00	24.00	52.00	121.00	102.00
NP-60A	69.00	54.00	102.00	99.00	95.00	24.00	49.00	125.00	115.00
NP-60B	.	.	97.00	.	.	.	.	116.00	97.00
NP-60D	66.00	47.00	.	.	.	24.00	46.00	117.00	99.00
NP-61A	65.00	47.00	95.00	98.00	.	.	.	.	.
NP-61B	61.00	53.00	95.00	102.00	.	23.00	51.00	105.00	92.00
NP-63	.	.	.	.	.	.	.	.	.
NP-67	.	.	.	.	.	.	.	.	.

### Standard Cranial Measurements – Maritime Archaic

Subject	SYH	MPL	FMB	FML	BMB	BAB
NP-01B	.	.	34.00	37.00	105.00	94.00
NP-06	29.00	79.00	28.00	35.00	98.00	105.00
NP-08	31.00	97.00	.	.	.	.
NP-09	.	.	30.00	34.00	108.00	111.00
NP-10A	23.00	76.00	29.00	36.00	108.00	106.00
NP-12A	.	.	31.00	36.00	114.00	123.00
NP-16A	32.00	80.00	29.00	34.00	106.00	103.00
NP-15	31.00	86.00	.	.	.	.
NP-16B	.	.	.	.	.	116.00
NP-18A	30.00	86.00	.	.	.	.
NP-18B	.	.	29.00	35.00	99.00	109.00
NP-21	32.00	79.00	23.00	.	102.00	111.00
NP-22D	.	.	32.00	38.00	.	126.00
NP-25	30.00	85.00	.	.	.	.
NP-27A	32.00	85.00	27.00	38.00	103.00	118.00
NP-28A	32.00	82.00	30.00	39.00	101.00	110.00
NP-29	25.00	80.00	30.00	35.00	108.00	115.00
NP-32	32.00	89.00	32.00	37.00	98.00	111.00
NP-34	30.00	95.00	.	46.00	.	123.00
NP-35A	35.00	95.00	27.00	40.00	113.00	111.00
NP-37B	31.00	81.00	32.00	36.00	106.00	113.00
NP-37C	.	.	.	.	.	.
NP-40A	28.00	82.00	.	.	.	.
NP-44A	23.00	77.00	30.00	32.00	107.00	128.00
NP-44B	23.00	81.00	28.00	28.00	.	122.00
NP-47A	32.00	94.00	26.00	38.00	102.00	107.00
NP-49A	29.00	80.00	30.00	33.00	106.00	113.00
NP-50A	31.00	84.00	.	.	.	.
NP-50B	31.00	86.00	29.00	34.00	.	.
NP-52	29.00	81.00	31.00	37.00	103.00	123.00
NP-60A	31.00	86.00	36.00	42.00	109.00	114.00
NP-60B	22.00	80.00	33.00	35.00	103.00	110.00
NP-60D	26.00	76.00	31.00	37.00	.	.
NP-61A	.	.	31.00	33.00	100.00	103.00
NP-61B	27.00	82.00	31.00	31.00	106.00	112.00
NP-63	.	.	.	.	.	.
NP-67	.	.	28.00	33.00	102.00	121.00

## Standard Cranial Measurements – European

Subject	MCB	MCL	BBH	BSB	BZB	UFH	TFH	PAL	PAB
NP-53	144.00	.	126.00	131.00	.	.	.	.	.
NP-54A	144.00	.	.	123.00	.	.	.	.	.
NP-54B	146.00	193.00	133.00	98.00	.	.	.	.	.
NP-71	.	170.00	128.00	104.00	.	55.00	94.00	34.00	38.00
NP-154	.	.	.	.	.	66.00	.	35.00	33.00
NP-155	.	.	.	.	.	.	.	.	.
NP-163A1	137.00	185.00	129.00	113.00	.	59.00	.	31.00	34.00
NP-163A2	145.00	186.00	132.00	113.00	.	61.00	111.00	29.00	31.00
NP-163A3	132.00	188.00	143.00	90.00	.	69.00	117.00	46.00	36.00
NP-163A4	139.00	188.00	134.00	107.00	.	70.00	.	39.00	33.00
NP-163A5	.	162.00	127.00	.	.	65.00	.	29.00	34.00
NP-163A6	.	.	.	.	.	.	.	.	.
NP-163A8	.	.	.	.	.	.	.	.	.
NP-163A1	.	.	.	.	.	65.00	.	36.00	33.00
NP-163B1	.	.	.	.	.	.	.	.	.
NP-163B3	.	.	.	.	.	.	.	.	.
NP-163B9	.	.	.	.	.	.	.	.	.
NP-165	134.00	172.00	136.00	101.00	.	52.00	108.00	40.00	35.00
NP-178	.	.	.	.	.	.	.	.	.
NP-179A	.	186.00	128.00	.	.	.	.	.	.
NP-181A	150.00	174.00	131.00	110.00	.	61.00	.	30.00	35.00
NP-181B	144.00	181.00	131.00	118.00	.	65.00	105.00	35.00	35.00
NP-181C	.	.	.	.	.	.	.	.	.
NP-181E	.	170.00	128.00	.	.	69.00	99.00	37.00	.
NP-181F	137.00	173.00	.	110.00	.	.	.	35.00	30.00
NP-182	140.00	.	.	.	.	.	.	.	.
NP-183A	.	.	.	.	.	66.00	.	35.00	38.00
NP-183B	.	.	.	.	.	.	.	.	.
NP-184A	.	182.00	123.00	.	.	74.00	117.00	38.00	28.00
NP-184B	.	.	132.00	.	.	.	.	.	.
NP-184C	.	.	.	.	.	.	.	.	.
NP-184D	.	180.00	120.00	.	.	66.00	.	.	.
NP-186A	133.00	166.00	137.00	105.00	.	62.00	95.00	31.00	34.00
NP-186B	.	.	.	114.00	.	.	.	.	.
NP-212	.	.	.	.	.	.	.	.	.
NP-243	132.00	166.00	121.00	96.00	120.00	66.00	.	35.00	35.00
NP-267	137.00	173.00	131.00	106.00	.	66.00	.	38.00	37.00
NP-271	155.00	184.00	128.00	120.00	136.00	71.00	112.00	36.00	41.00
NP-272	147.00	.	.	124.00	.	.	.	.	.
NP-273	146.00	178.00	131.00	125.00	133.00	62.00	.	35.00	34.00
NP-274	141.00	183.00	137.00	116.00	135.00	68.00	118.00	37.00	37.00

## Standard Cranial Measurements – European

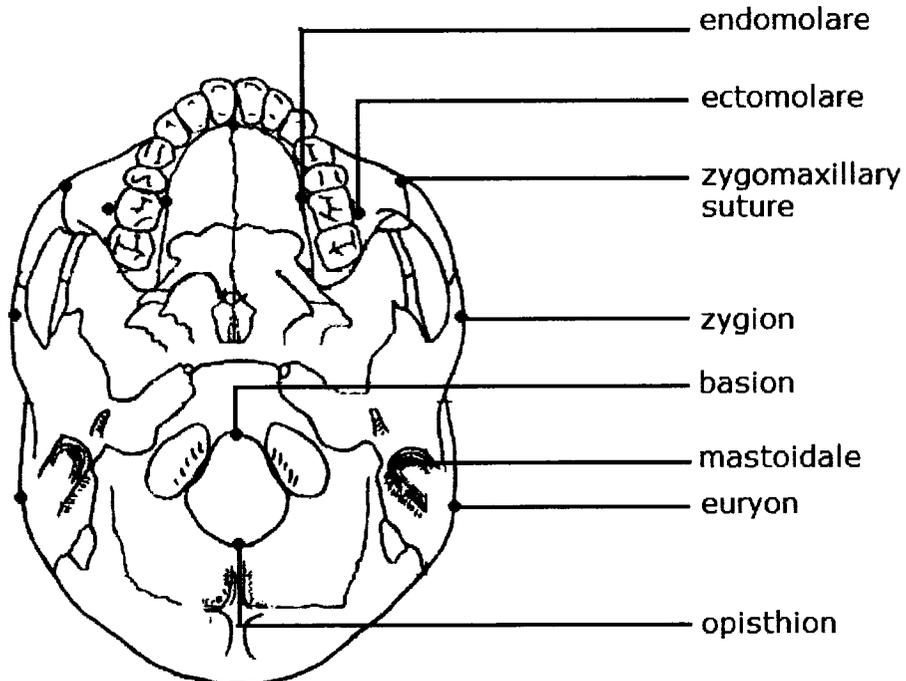
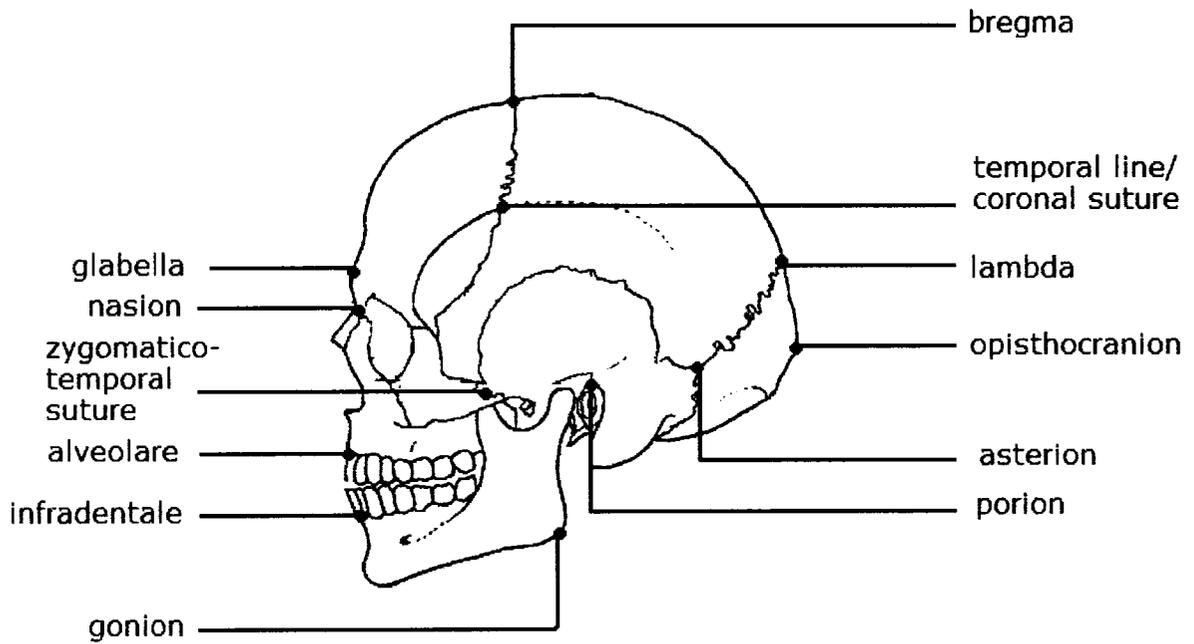
Subject	MAB	MAL	BAN	BAV	MFB	NAB	NAH	BCB
NP-53	.	.	.	.	.	.	.	.
NP-54A	.	.	.	.	.	.	.	.
NP-54B	.	.	102.00	.	.	.	.	.
NP-71	62.00	44.00	91.00	87.00	99.00	25.00	43.00	112.00
NP-154	56.00	50.00	.	.	92.00	21.00	51.00	.
NP-155	.	.	.	.	.	.	.	.
NP-163A1	53.00	42.00	96.00	83.00	89.00	24.00	47.00	.
NP-163A2	58.00	43.00	100.00	82.00	84.00	20.00	49.00	.
NP-163A3	68.00	56.00	111.00	105.00	87.00	23.00	52.00	.
NP-163A4	55.00	49.00	104.00	99.00	88.00	24.00	50.00	.
NP-163A5	.	.	81.00	77.00	.	.	47.00	.
NP-163A6	.	.	.	.	.	.	.	119.00
NP-163A8	.	.	.	.	.	.	.	.
NP-163A16	57.00	48.00	.	.	.	23.00	50.00	.
NP-163B1	.	.	.	.	.	.	.	117.00
NP-163B3	.	.	.	.	.	.	.	115.00
NP-163B9	.	.	.	.	.	.	.	.
NP-165	65.00	48.00	98.00	96.00	91.00	29.00	42.00	112.00
NP-178	.	.	.	.	.	.	.	105.00
NP-179A	.	.	98.00	.	.	.	.	.
NP-181A	52.00	39.00	93.00	77.00	90.00	.	48.00	.
NP-181B	58.00	44.00	93.00	83.00	90.00	.	46.00	.
NP-181C	.	.	.	.	.	.	.	.
NP-181E	.	.	98.00	90.00	.	.	50.00	.
NP-181F	.	.	.	.	87.00	.	.	.
NP-182	.	.	.	.	.	.	.	.
NP-183A	50.00	45.00	91.00	85.00	.	23.00	47.00	.
NP-183B	.	.	.	.	.	.	.	.
NP-184A	51.00	50.00	102.00	94.00	.	.	55.00	.
NP-184B	.	.	.	.	.	.	.	.
NP-184C	.	.	.	.	.	.	.	.
NP-184D	.	.	92.00	87.00	.	.	.	.
NP-186A	51.00	44.00	87.00	85.00	88.00	19.00	42.00	100.00
NP-186B	.	.	.	.	.	.	.	.
NP-212	.	.	.	.	.	.	.	.
NP-243	59.00	39.00	89.00	81.00	86.00	20.00	50.00	.
NP-267	62.00	50.00	96.00	92.00	93.00	26.00	49.00	.
NP-271	65.00	48.00	102.00	94.00	97.00	23.00	49.00	117.00
NP-272	.	.	.	.	.	.	.	.
NP-273	46.00	48.00	90.00	85.00	95.00	25.00	53.00	.
NP-274	61.00	52.00	101.00	100.00	96.00	26.00	50.00	118.00

## Standard Cranial Measurements – European

Subject	BGB	SYH	MPL	FMB	FML	BMB	BAB
NP-53	.	.	.	31.00	32.00	.	106.00
NP-54A	.	.	.	.	.	.	.
NP-54B	.	.	.	.	36.00	103.00	118.00
NP-71	101.00	23.00	69.00	.	35.00	103.00	.
NP-154	94.00	25.00	68.00	.	.	.	.
NP-155	.	.	.	28.00	36.00	.	.
NP-163A1	88.00	.	70.00	27.00	31.00	.	112.00
NP-163A2	102.00	26.00	74.00	27.00	34.00	99.00	109.00
NP-163A3	92.00	22.00	75.00	33.00	43.00	94.00	103.00
NP-163A4	.	30.00	.	31.00	40.00	.	110.00
NP-163A5	.	21.00	.	29.00	35.00	.	.
NP-163A6	100.00	23.00	69.00	.	.	.	.
NP-163A8	85.00	24.00	74.00	.	.	.	.
NP-163A16	.	.	.	.	.	.	.
NP-163B1	98.00	18.00	58.00	.	.	.	.
NP-163B3	111.00	30.00	77.00	.	.	.	.
NP-163B9	82.00	26.00	83.00	.	.	.	.
NP-165	96.00	31.00	79.00	26.00	34.00	94.00	103.00
NP-178	105.00	22.00	84.00	27.00	29.00	91.00	111.00
NP-179A	.	29.00	78.00	28.00	30.00	.	.
NP-181A	103.00	25.00	69.00	30.00	32.00	101.00	118.00
NP-181B	93.00	.	79.00	30.00	39.00	100.00	114.00
NP-181C	.	.	.	30.00	34.00	.	111.00
NP-181E	91.00	24.00	74.00	33.00	35.00	95.00	108.00
NP-181F	.	.	.	29.00	.	.	108.00
NP-182	.	.	.	30.00	33.00	100.00	114.00
NP-183A	.	.	72.00	32.00	35.00	93.00	.
NP-183B	.	.	.	28.00	29.00	102.00	106.00
NP-184A	88.00	25.00	79.00	27.00	33.00	103.00	123.00
NP-184B	99.00	.	65.00	33.00	40.00	101.00	112.00
NP-184C	.	.	.	31.00	37.00	106.00	123.00
NP-184D	.	.	.	28.00	35.00	.	.
NP-186A	86.00	24.00	71.00	25.00	28.00	83.00	103.00
NP-186B	.	.	.	.	.	.	.
NP-212	.	.	.	27.00	35.00	100.00	116.00
NP-243	.	.	.	30.00	32.00	98.00	112.00
NP-267	.	.	.	31.00	35.00	120.00	119.00
NP-271	103.00	26.00	79.00	31.00	35.00	102.00	122.00
NP-272	.	.	.	.	.	.	.
NP-273	.	.	.	31.00	35.00	109.00	114.00
NP-274	102.00	25.00	80.00	32.00	37.00	107.00	108.00

## Appendix C

### Standard Cranial Landmarks (adapted from Bass 1971)



## Appendix D

### Individual Asymmetry Profiles - European

Subject	NCR	FAC	INO	CBM	MND	ARB
NP-53	5.30	.	3.00	.	.	1.70
NP-54A	2.80	.	.	.	.	.
NP-54B	9.80	.00	1.70	2.00	.	2.00
NP-71	4.00	2.20	.	7.00	1.00	1.00
NP-154	.	1.80	.	.	9.50	.
NP-155	.	.	.	.	.	2.30
NP-163A1	2.60	2.00	3.00	2.30	4.00	1.30
NP-163A2	5.40	1.30	1.30	5.50	6.00	1.50
NP-163A3	6.30	3.20	3.00	3.30	2.70	2.20
NP-163A4	3.00	.60	6.00	3.30	.	1.70
NP-163A5	.	.	.	.	.	2.00
NP-163A6	.	.	.	.	6.40	.
NP-163A8	.	.	.	.	3.00	.
NP-163A1	.	1.00	.	.	.	.
NP-163B1	.	.	.	.	1.50	.
NP-163B3	.	.	.	.	2.20	.
NP-163B9	.	.	.	.	1.60	.
NP-165	2.40	1.60	1.70	1.50	1.30	1.20
NP-178	.	.	5.00	.00	2.00	2.00
NP-179A	.	.	.	.	.	1.00
NP-181A	2.80	.70	.70	2.00	6.00	.40
NP-181B	3.60	5.00	2.00	1.00	1.50	3.00
NP-181C	.	.	3.00	1.50	.	1.00
NP-181E	.	3.00	5.00	1.00	1.00	1.60
NP-181F	3.10	4.00	2.50	.	.	.
NP-182	.	.	2.00	2.00	.	2.40
NP-183A	.	3.50	.	2.50	.	2.20
NP-183B	.	.	2.30	2.50	.	4.00
NP-184A	.	3.50	1.00	2.00	2.70	.80
NP-184B	.	.	2.00	1.50	5.00	1.60
NP-184C	.	.	5.30	3.50	.	1.80
NP-184D	3.00	2.00	.	1.50	.	1.70
NP-186A	3.10	1.80	1.70	1.30	1.80	.80
NP-186B	2.00	.	.	.	.	.
NP-212	.	.	4.00	1.50	.	1.00
NP-243	3.50	1.00	2.00	1.80	.	1.60
NP-267	3.40	1.00	4.30	1.50	.	1.00
NP-271	4.50	1.80	1.70	1.50	1.40	2.60
NP-272	6.50	.	.	.	.	.
NP-273	3.80	1.30	5.30	2.60	.	1.20
NP-274	2.60	1.30	2.70	1.40	2.00	1.20

Note - these are standardized cumulative asymmetry values; thus range is +/- the given value

Legend: NCR – neurocranium

INO – inferior neurocranium and occipital bone

MND – mandible

FAC – face

CBM – cranial base and muscular face

ARB – articulating base

## Individual Asymmetry Profiles – Maritime Archaic

Subject	NCR	FAC	INO	CBM	MND	ARB
NP-01B	3.80	4.80	1.30	3.00	.	3.60
NP-06	2.80	1.50	1.30	2.00	2.40	1.40
NP-08	.	.	.	.	1.20	.
NP-09	4.50	2.00	2.70	1.00	.	.40
NP-10A	3.80	2.80	1.70	2.30	8.30	2.50
NP-12A	3.80	1.40	1.30	1.50	.	1.20
NP-16A	4.10	2.00	4.30	4.80	2.00	.80
NP-15	.	.	.	.	2.00	.
NP-16B	7.00	.	.	.	.	.
NP-18A	.	.	.	.	2.00	.
NP-18B	5.10	2.40	2.30	1.00	.	3.80
NP-21	4.10	.	2.00	.	1.70	1.00
NP-22D	4.00	1.70	5.50	2.80	.	1.70
NP-25	.	.	.	.	3.00	.
NP-27A	3.60	1.50	5.00	1.60	.80	2.00
NP-28A	4.90	2.00	1.70	2.20	.60	.60
NP-29	4.10	1.70	2.00	1.60	.80	1.60
NP-32	3.40	2.80	.70	1.70	.60	1.80
NP-34	6.00	5.00	3.00	3.00	3.30	1.70
NP-35A	2.40	1.70	2.00	1.00	2.60	3.50
NP-37B	2.30	1.60	2.00	1.00	1.60	2.50
NP-37C	3.50	.	.	.	.	.
NP-40A	.	.	.	.	1.40	1.00
NP-44A	3.10	1.50	1.00	1.20	2.00	1.60
NP-47A	2.60	1.40	2.30	4.80	2.00	4.20
NP-49A	5.10	2.00	1.00	.50	2.00	1.60
NP-50A	.	.	.	.	4.00	.
NP-50B	.	2.50	.	.	1.40	2.30
NP-52	4.50	1.00	1.70	1.80	1.60	1.80
NP-60A	3.60	1.70	2.70	2.20	1.50	2.00
NP-60B	.	2.00	4.00	1.00	1.30	1.20
NP-60D	.	.80	.	.	.00	1.70
NP-61A	2.60	3.00	2.00	2.40	.	1.40
NP-61B	.	1.80	1.00	1.60	2.20	1.00
NP-63	5.30	.	.	.	.	.
NP-67	2.30	.	.50	1.00	.	2.20

Note - these are standardized cumulative asymmetry values; thus range is +/- the given value

Legend: NCR – neurocranium

INO – inferior neurocranium and occipital bone

MND – mandible

FAC – face

CBM – cranial base and muscular face

ARB – articulating base

## Appendix E

Example of Statistical Analysis: NZM (Nasion-Zygomaxillary Suture)  
(generated with SPSS)

### Descriptive Statistics

**Descriptive Statistics**

	N	Mean	Std.	Skewness		Kurtosis	
	Statistic	Statistic	Statistic	Statistic	Std. Error	Statistic	Std. Error
NZMMA	23	1.0435	2.26592	.018	.481	.194	.935
NZMEU	14	.5714	1.82775	.220	.597	-.691	1.154
Valid N (listwise)	14						

(note: there is no significant skew or kurtosis; low standard deviation)

### One-Sample t-Test

**One-Sample Test**

	Test Value = 0					
	t	df	Sig. (2-tailed)	Mean Difference	95% Confidence Interval of the Difference	
					Lower	Upper
NZMMA	2.209	22	.038	1.04348	.0636	2.0233
NZMEU	1.170	13	.263	.57143	-.4839	1.6267

(note: mean statistic for Maritime Archaic only is significantly different from zero)

### Normality Test

**Tests of Normality**

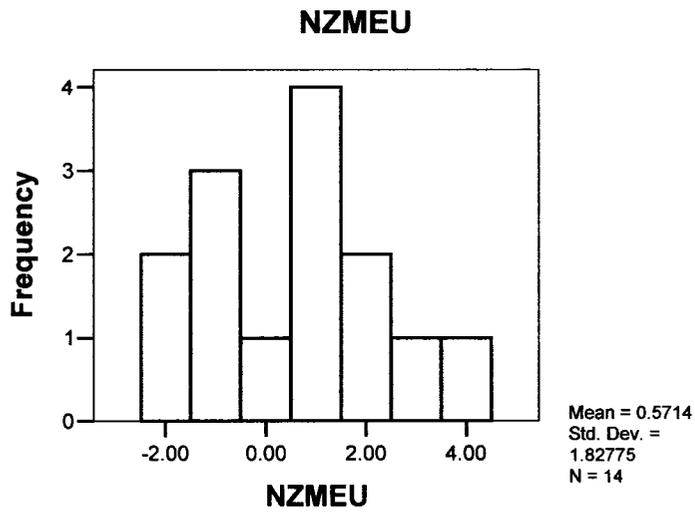
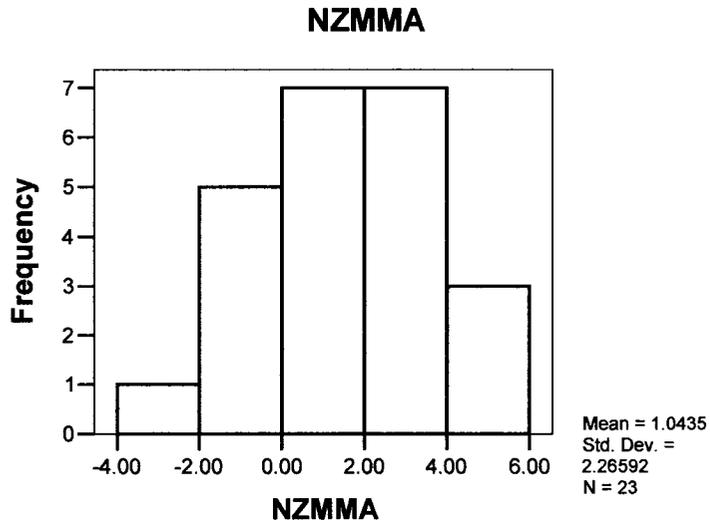
	Kolmogorov-Smirnov <sup>a</sup>			Shapiro-Wilk		
	Statistic	df	Sig.	Statistic	df	Sig.
NZMMA	.157	14	.200*	.963	14	.777
NZMEU	.164	14	.200*	.946	14	.498

\*. This is a lower bound of the true significance.

a. Lilliefors Significance Correction

(note: both Maritime Archaic and European samples are normally distributed)

# Histograms



## Appendix F

### Detailed Results of Asymmetry Analysis Summarized in Figures 6.1 and 6.2 Maritime Archaic Sample

Functional Unit	Shape of Distribution			Significance of the Mean
	Skew	Kurtosis	Normality	
<b>Articulating Base</b>				
OCC	ns	ns	normal	X = 0 p= 0.050
OPC	ns	ns	normal	X = 0 p= 0.050
BSC	1.379	2.784	non-normal	X = 0 p= 0.050
MAO	ns	ns	normal	X > 0 p= 0.050
BAM	1.033	3.022	normal	X = 0 p= 0.050
<b>Cranial Base &amp; Muscular Face</b>				
PZT	ns	ns	normal	X = 0 p= 0.050
PZM	ns	1.676	normal	X = 0 p= 0.050
BAP	ns	1.795	normal	X = 0 p= 0.050
SPI	ns	ns	non-normal	X = 0 p= 0.050
BZM	ns	ns	normal	X > 0 p= 0.050
<b>Inferior Neurocranium &amp; Occipital Bone</b>				
ASA	ns	4.179	non-normal	X = 0 p= 0.050
AMO	ns	1.036	normal	X > 0 p= 0.050
LMD	ns	ns	normal	X = 0 p= 0.050
<b>Mandible</b>				
RHT	ns	ns	non-normal	X = 0 p= 0.050
RBR	1.043	3.628	non-normal	X = 0 p= 0.050
MBL	ns	ns	normal	X = 0 p= 0.050
CWM	ns	1.639	non-normal	X = 0 p= 0.050
GID	-1.738	5.526	normal	X = 0 p= 0.050
<b>Face</b>				
NEM	ns	ns	normal	X = 0 p= 0.050
ORB	ns	ns	normal	X = 0 p= 0.050
ORH	ns	-1.076	normal	X = 0 p= 0.050
NAP	ns	1.308	non-normal	X < 0 p= 0.100
NAZ	ns	-1.009	normal	X = 0 p= 0.050
NZM	ns	ns	normal	X > 0 p= 0.050
<b>Neurocranium</b>				
LTA	ns	ns	normal	X < 0 p= 0.100
LTC	ns	ns	normal	X = 0 p= 0.050
LTF	ns	ns	normal	X = 0 p= 0.050
LTS	ns	2.374	normal	X < 0 p= 0.100
PBA	ns	ns	normal	X > 0 p= 0.100
TCH	ns	-1.316	normal	X = 0 p= 0.050
PBF	ns	ns	normal	X = 0 p= 0.050
TST	ns	ns	normal	X > 0 p= 0.100

Detailed Results of Asymmetry Analysis Summarized in Figures 6.1 and 6.2  
European Sample

Functional Unit	Shape of Distribution			Significance of the Mean
	Skew	Kurtosis	Normality	
<b>Articulating Base</b>				
OCC	ns	ns	non-normal	X = 0 p= 0.050
OPC	ns	ns	non-normal	X < 0 p= 0.050
BSC	ns	ns	normal	X = 0 p= 0.050
MAO	ns	ns	normal	X < 0 p= 0.100
BAM	ns	ns	normal	X = 0 p=0.050
<b>Cranial Base &amp; Muscular Face</b>				
PZM	ns	ns	normal	X = 0 p= 0.050
BAP	ns	ns	normal	X = 0 p= 0.050
SP1	-1.386	1.287	non-normal	X = 0 p= 0.050
BZM	ns	1.314	normal	X < 0 p= 0.100
<b>Inferior Neurocranium &amp; Occipital Bone</b>				
ASA	ns	ns	normal	X > 0 p= 0.100
AMO	ns	ns	normal	X = 0 p= 0.050
LMD	ns	ns	normal	X > 0 p= 0.100
<b>Mandible</b>				
RHT	2.728	8.417	non-normal	X = 0 p= 0.050
RBR	ns	ns	normal	X < 0 p= 0.100
MBL	ns	ns	non-normal	X = 0 p= 0.050
GID	ns	2.705	normal	X = 0 p= 0.050
<b>Face</b>				
NEM	ns	ns	non-normal	X = 0 p= 0.050
ORB	ns	-1.233	normal	X = 0 p= 0.050
ORH	ns	ns	normal	X = 0 p= 0.050
NAP	ns	ns	normal	X = 0 p= 0.050
NZM	ns	ns	normal	X = 0 p= 0.050
<b>Neurocranium</b>				
LTA	ns	ns	normal	X > 0 p= 0.050
LTC	ns	ns	normal	X > 0 p= 0.050
LTF	ns	ns	normal	X = 0 p= 0.050
LTS	ns	ns	normal	X = 0 p= 0.050
PBA	ns	ns	normal	X > 0 p=0.050
TCH	ns	ns	normal	X = 0 p= 0.050
PBF	ns	ns	normal	X = 0 p= 0.050
TST	ns	-1.040	normal	X > 0 p= 0.050

## Suture Asymmetry Measurements – Analysis of Variance Output

### Maritime Archaic Sample

#### ANOVA

MA

	Sum of Squares	df	Mean Square	F	Sig.
Between Groups	204.986	2	102.493	6.421	.003
Within Groups	941.788	59	15.963		
Total	1146.774	61			

(generated with SPSS)

### European Sample

#### ANOVA

EU

	Sum of Squares	df	Mean Square	F	Sig.
Between Groups	177.333	2	88.667	4.441	.018
Within Groups	778.571	39	19.963		
Total	955.905	41			

(generated with SPSS)

## Appendix G

### Raw Data – NP-56 Palaeoeskimo Cranium

<b>Measurement</b>	<b>Left</b>	<b>Right</b>	<b>(R-L)</b>
ASA	49	52	3
LTC	131	142	11
LTA	164	178	14
LTF	59	80	21
LTS	34	38	4
PBA	177	177	0
TCH	148	140	-8
TST	37	45	8
PBF	81	88	7
NAP	119	112	-7
NAZ	98	95	-3
PZT	54	53	-1
NZM	62	65	3
PZM	89	83	-6
ORB	44	43	-1
ORH	34	33	-1
AMO	92	86	-6
MAO	65	61	-4

### Standard Cranial Data

<b>Measurement</b>	<b>Value</b>
MCB	131
MCL	192
BSB	110
BZB	142
UFH	72
PAL	43
PAB	48
MAB	71
MAL	48
MFB	93
NAB	22
NAH	60
FMB	29
BMB	113
BAB	111







