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Artificial Cranial Deformation: Potential Implications for Affected Brain Function

Tyler G O'Brien[1]*, Lauren R Peters[1] and Marc E Hines[2]

[1]Department of Sociology, Anthropology and Criminology, University of Northern Iowa, Cedar Falls, USA
[2]Department of Neurology, Covenant Hospital, Waterloo, USA

Abstract

The anthropological study of the ancient cross-cultural practice of artificial cranial deformation (ACD), or intentional head modification, allows for the opportunity to assess the effects of functional interactions of the dynamic altered growth and development processes. Intentionally altering the infant skull is produced through mechanical means by attaching a device to the child's head. Through the application of a deforming apparatus directly to the infant's head, soon after birth and up to as long as four years, the child's head becomes permanently altered. The amount of cranial modification and subsequent deformation is dependent upon the extent of time the molding apparatus is applied to the infant's head. The longer the amount of time applied the greater the resulting stress and subsequent deformation. This paper explores the potential of inhibited cranial development or spatial disorientation and the subsequent effects it may have on adjacent functionally and morphologically related structures, especially as it pertains to brain function. A theoretical analysis is presented because of the practically non-existent data for this ancient practice. However, based onbioarchaeological and neurological analyses of the cranium and brain, it is highly suspected that ACD, in general, would have produced negative results to the lobes and abilities of the individual; such as: influencing vision, object recognition, hearing ability, impairing memory, promoting inattentiveness, inability to concentrate and motor aphasia, contributing to behavior disorders and difficulty in learning new information.

Keywords: Head modification; Neurology; Bioarchaeology

Introduction

The human brain is a complex, fascinating structure of the body which serves many important duties. Brain structure coordinates with brain function and both are interrelated and efficiently rely on one another. Modification of anatomy could result in changes of brain function. Hundreds of years ago, modification was a frequent and common aspect of culture and everyday life in many populations around the world, in particular South America. Skulls have been found which provide evidence that head wrapping techniques were purposefully used to modify the skulls of infants. Research on this topic has produced data and information to discuss artificial cranial deformation (ACD) and increase the awareness on this topic [1,2]. Current research on brain anatomy and function provides knowledge on how the brain works with and throughout the body.

A comparisonof modern knowledge of brain capabilities with ancient head molding techniques that affect the skull and brain breeds interest in questioning the implications of ACD on these people. Unfortunately skulls alone cannot give the answers to ACD effects on brain function and anatomy. However, a thorough investigation on the evidence of both modern brain anatomy, evidence from ACD skulls, and consideration of modern conditions such as plagiocephaly attempt to give a well-rounded discussion of the possible implications that ACD had on brain function. Overall, this research works to understand the possible abilities the brain has to recover and overcome cranial deformation, and the pressures these conditions exude on different areas of the brain. Theoretically, ACD could have had the potential to inhibit, sustain, or improve brain function and capability of these individuals. To determine whether these practices were beneficial or disadvantageous to the individuals an overview of ACD, along with a closer examination of the brain anatomy and an understanding of the brain functions are presented.

Artificial cranial deformation

Artificial cranial deformation is a human cultural phenomenon defined as the product of dynamic distortion of the normal vectors of infantile neurocranial growth through the agency of externally applied forces [3]. Deformed crania give us the opportunity to assess the effects of functional interactions of the dynamic processes of altered growth and development [4-10]. There is "little doubt that inhibited development or spatial disorientation of one cranial structure has profound effects on the final disposition of adjacent functionally and morphologically related structures" [10].

The practice of ACD must begin early in life since the morphogenetic pattern of the head is established by the third month of post-natal life, or perhaps earlier, and once attained does not change [7]. Therefore, the molding process is initiated soon after birth, when the skull is most malleable, and may last until the second year of life [11-13] or as long as four years [14]. The amount of cranial modification and subsequent deformation is dependent upon the extent of time the molding apparatus is applied to the infant's head. The longer the amount of time applied the greater the resulting stress and subsequent deformation.

ACD has been shown to affect different parts of the craniofacial complex [15,6,7,16,17]. Mention of side effects or the pain suffered by the child is limited in the ethnographic accounts of ACD but firsthand accounts mention observing bulging eyes and skin irritations on the infant. Extended modification of the cranial plates may cause injury

***Corresponding author:** Tyler G. O'Brien, Department of Sociology, Anthropology and Criminology, University of Northern Iowa, USA, Tel: 319-273-2789; Fax: 319-273-7104; E-mail: tyler.obrien@uni.edu

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or death to the infant. A method that produced negative effects was probably quickly discontinued [18, 14]. A hypothesis, presented by both Moss [3] and Dingwall [19], implies the practice was well defined within the limits of deformation. Museum collections around the world contain thousands of skulls displaying ACD. A majority of them are of adult individuals which suggests that the process did not influence life expectancy, despite the fact that severely deformed infant crania have been discovered. It is likely that many of those infants with extremely flattened crania died as a result of too tight or too long a binding process.

The human cranium can be deformed in one of two ways: unintentionally or altered with intent [2]. In the past, unintentional deformity occurs when a child's head and body were bound tightly to a cradleboard or flat, hard structure to prevent the infant from moving during the parent's daily activities [12,20,14,9]. Likely modern day examples include positional molding, intra-uterine compression and craniosynostosis [21]. These processes often result in plagiocephalic defects. Physiological and mental defects associated with genetically inherited craniosynostotic syndromes will not be discussed here but can be found elsewhere [21,1].

In contrast, intentional modification was performed in one of two basic styles: annular and tabular [12,13,22]. The annular style involves bands, belts or cords that are wrapped around the infant's head: running transversely across the upper forehead, down along the sides and terminating on the back near the hairline. This technique results in a conical shaped cranium with the posterior parietals extending superiorly and posteriorly, and the bones of the frontal, occipital and cranial base being lengthened [15,7]. The tabular form utilizes boards or hard flat surfaces bound across the child's forehead and tied laterally to a board placed across the back of the head. This method produces a box-like vault shape (i.e., high and short) or bilobular cranium with the postero-lateral parietals (i.e., the bosses) being widened laterally. This style is also characterized by a vertical posterior cranial aspect with a lengthened and widened frontal bone [6].These intentional modifications may have been practiced for a variety of reasons, one being the belief that obtaining this head shape produced higher intelligence in the individual.

Dingwall [19] notes that in an 1833 study at a French insane asylum over 50% of the 431 residents possessed deformed crania and it was "among the more intractable and difficult patients that the extreme forms of deformation were encountered." Further speculations of long-term negative consequences from ACD are documented in historical accounts from ancient Peru. There, Inca rulers dictated that heads be deformed in order to make those who submitted weak and without energy [23] hence the artificially deformed head supposedly made one "silly and easily ruled" [24]. Curiously, Dingwall [19] maintains that there is no solid evidence accounting for the diminishing of the mental faculties of one who is deformed, even though others have raised the issue [25]. Additionally, Gerszten's [14] study of over 400 pre-Columbian skulls shows no apparent difference between the cranial capacities of the deformed and the unreformed skulls, lending support to the view that the brain is unharmed and that normal brain function is unaffected by the deforming procedure. It is noteworthy that ACD is an alteration in direction of growth not magnitude of dimensions [26]. Furthermore, contemporary studies [9] suggest that there is most likely neither injury to the brain nor impairment of the mind in the case of ACD. It is worthwhile to consider that the artificially deformed individual's mental functions or abilities might actually be augmented in some manner.

Brain anatomy and function

All activities and functions of the body are controlled, regulated, and monitored by the brain. Being a highly organized and symmetrical structure, divided into various components that serve specific and important functions, the brain is able to translate and interpret signals. For example, the right side of the brain is dominant for spatial abilities, face recognition, visual imagery, and music. The left side of the brain corresponds to calculations, math, and logical abilities [27]. The major components, the cerebral cortex (cerebrum), cerebellum and the brain stem, work together to produce efficient responses and actions. The functions of the first two are described below. Without any one of its structural components it could not work with the same high quality and efficient speed. Both the brain anatomy and brain function are interrelated and rely on each other.

The cerebral cortex is the largest structure of the brain and rests above most of the other structures [28]. Different lobes are involved in different cognitive and behavior functions [29,30].

The frontal lobe is concerned with intellect, behavior, memory, and movement. This area controls decision making, problem solving, and will [30]. The prefrontal area is associated with the ability to concentrate and elaborate thought. Personality and emotional traits are also controlled here. Damage to the frontal lobe can result in impairment of memory, inattentiveness, inability to concentrate, behavior disorders, difficulty in learning new information, and motor aphasia [29].

The parietal lobe is associated with intelligence, language, reading, and sensation. This area of the brain receives and translates sensory information. The parietal simultaneously interprets signals received from other areas of the brain, such as vision, hearing, motor, sensory and memory. New sensory information received will be translated into memory and given meaning in the parietal lobe. Damage to this area in the brain could result in inability to discriminate between sensory stimuli, inability to recognize parts of the body, inability to recognize self, or inability to write [29].

The occipital lobe is the primary visual reception area and allows for visual interpretation [29]. This lobe is located toward the back of the brain. This area of the brain influences how humans process colors and shapes. Damage to the occipital lobe can cause loss of vision and loss of ability to recognize an object.

The temporal lobe is also involved in visual memory and enables the recognition of objects and other people. The left temporal lobe is involved in verbal memory and understanding language as well as interpreting other's emotions and reactions [31]. Another main function of this lobe is that it is the area for auditory reception, information retrieval, and expressed behavior. Damage to this lobe can result in hearing deficits, agitation, and irritability [29].

The cerebellum is a two-lobed structure located at the back tucked behind and under the two cerebral hemispheres [29]. The main function of the cerebellum is to coordinate balance and movement, such as allowing the body to assume postures and maintain muscle coordination [32]. As a result of the interaction between body parts and the brain through nerve signals the brain can easily regulate and maintain homeostasis. If problems and conditions do arise the brain can be notified and can quickly act to protect and readjust the body [28].

Cranial growth and development

Vault growth predominates during the pre-natal period and the first years of infancy and childhood due to its expanding cerebral contents

[33]. Once the brain reaches its final size, there is very minimal cranial expansion, yet the human cranial system is continuously dynamic throughout life [34] and may be limited or influenced by hereditary or environmental factors (climate, diet, environment, mechanical etc.) [35-38]. During early growth and development the delicate nature of the brain makes it susceptible to many types of damage. The effects of damage to the brain have been seen and experienced by many people. Accidents have caused people to lose abilities and functions they possessed before when their bodies could fully function. These affects have been seen by fast, hard impact to the brain and skull. However, slow and continuous compression and/or tension to the brain and skull can have significantly different results. Slow impact over time on the skull gives the brain the ability to adapt to the stress and strain [30]. For example, studies by Buchman et al. [39] and Brennan and Antonyshyn [37] confirm that local skull morphology and craniofacial growth are affected and can be directed by graded application of pressure. The compressive forces of an externally applied deforming apparatus to an infant's head can be extreme. Additionally, resultant tensile forces combine to produce an abnormal head shape consistent with the applied mechanical stresses. Normal rates and patterns of growth no longer apply when trying to interpret either of them from human skulls that have been artificially deformed.

Plagiocephaly is a condition characterized by the asymmetrical distortion (flattening of one side) of the skull. This deformation can be the result of craniosynostosis (premature fusion of the sutures), infant positioning, or other means of pressure [40]. Deformational or positional plagiocephaly can be initiated prenatally, but more commonly occurs post-natally, like when an infant repeatedly sleeps in the same position, lying on the same side of their head every night. Miller and Clarren [41] conducted a study on long term development outcomes in children diagnosed with deformational plagiocephaly. The study was conducted to determine if there existed an increased rate of later developmental delay in school-aged children, who as infants had presented deformational plagiocephaly without obvious signs at the time of initial diagnosis. They reviewed these patients when they were in school to see how many (or if any) had to receive special educational assistance, physical therapy, or speech therapy. Siblings of these children were used as a control group and were under the same review. The results showed that there was a significant amount of the children (40%) who had developmental difficulties, which resulted in having problems with school work. There was also a clear difference in children with plagiocephaly compared to their siblings in regard to special needs in school.

Results from this study can be applied to the effects that ACD would have had on the children who experienced it. Both plagiocephaly and ACD are very similar in the way that slow, continuous pressure is applied to the skull. In the case of plagiocephaly, the infant is continuously sleeping on the same side of their skull every night or the infant is resting its head on the same side of their caretaker's chest when they are fed by bottle. Pressure is continuously being put on the same side of the malleable skull, while the other side is not. In contrast, most cases of ACD have pressure being placed on both sides of the skull. Both cases result in the abnormal shape of the skull which could affect the brain. This study shows that pressure on the skull and the changing of the skull shape affected the development and cognitive skills of the brain negatively. Cases of ACD, which experience the same traumatic conditions of daily, low pressure as plagiocephaly, could have also been affected negatively by the pressure of the skull.

Neurological effects of ACD

The brain continues to grow up until adulthood. Any modification or alteration of this process may produce changes in normal cerebral function. Brain volume quadruples between birth and adulthood. The growth is not generally from the addition or enlargement of neurons. Most of the generation of neurons has occurred by the seventh month of gestation [42]. Migration of neurons also takes place almost entirely within the period of prenatal development in the human [43]. Neuronal migration continues into the post-natal period. The elaboration of myelination and inter-neuronal interfaces in the form of dendritic connections accounts for the majority of brain enlargement. ACD is performed as a post-natal phenomenon. The frontal cortex obtains connections from a broad array of areas within the brain [44]. The orbitofrontal cortex is on the under and medial surface of the frontal lobes. The orbito-frontalarea in particular hashigh level integration functions. The consolidation of information from multiple brain areas is required to procure resolutions for emotional (and social) context specific decisions. Such abilities are central to even the seemingly simplest human. The most extreme example of such impairment would be pre-frontal lobotomy. Such an extensive damage to connective fibers leads to extremes of sometimes contradictory phenomena of passivity and bizarrely inappropriate social interactions. The third author's (MEH) first-hand experience of such behavior was a patient's social introduction by urinating on the author's shoes. Apparently, according to staff who had known him for years, this was his usual introduction.

The dorsolateral prefrontal areas are specialized (with other association areas) in sequential and logical abilitie [45]; the orbitofrontal cortex (as well as the limbic system and other association areas) specializes in the emotional and social context. The frontal cortex generally can be stated to have connections to and from primary sensory as well as association areas. The key point is the connectivity of one area to another. The frontal areas have extensive connections with the limbic system and they are collectively responsible both for reactive emotive responses as well as templates of emotional set points. The limbic system is itself a group of widespread structures. Some parts are deep to and include parts of the temporal lobe. Limbic networks are responsible for emotional set points, responses and interactions between the processing of pre-conscious information from our autonomic and internal regulation, such as blood pressure, pulse, skin temperature, aspects of attention and arousal. The parietal cortex is also a networking center for regional primary and association areas (from the primary sensory areas and from the temporal lobe auditory cortex as well as sensory integration from the occipital cortex which includes visual information) [46-48]. The information arriving in the frontal areas is preprocessed for relevance to attention, need for concentration and arousal, validity determined by comparative processing of both hemispheres and multiple sensory modalities, as well as comparison to prior experience and memory [49].

Frontal function is often characterized as being heavily utilized to prevent a variety of reflexive and impulsive responses, particularly characteristic of highly practiced skills [50]. For example, a stick (possibly resembling a snake) seen in peripheral vision, may set off an arousal mechanism in the occipital/parietal area that uses one pathway (to the limbic system and arousal areas) to heighten attention, increase heart rate and initiate avoidance due to a predetermined reflex secondary to the confusion of this barely visualized snake-like appearance of the stick. As the reflex begins action, a slower and longer pathway to frontal function arouses to the recognition that this is only a stick. By this time the eyes have been reflexively moved to this area of visual concern and the fovea (central-high acuity) focus on the stick and the frontal cortex which is informed of the now more clear recognition (re-cognition) of the object, and inhibits the reflex to leap away (thus avoiding both an inefficient and potentially injurious action).

Deformation of the frontal cortex (an almost unavoidable consequence of any of the forms of ACD) could give rise to a wide array of problems but those which seem most likely (if there is a negative consequence) would impair higher processing favoring more impulsive and slightly more immature responses. There is speculation that the frontal cortex does not complete maturation until somewhere between late adolescence and the early adulthood [51]. In a large number of patients suffering from traumatic brain injury it is thought that frontal injury, and particularly orbitofrontal injury, comprises one of the most characteristic deficits known as impairment in executive function [52]. Such injury even if moderate or mild, often effects the frontal area of the brain due to roughed cranium beneath and the fact that kinetic forces converge on the heavy overhang of frontal regions beyond the better tethered and protected brainstem (though significant brainstem injury is often swiftly fatal) [53].

As the name implies, the key deficit is impairment in decision making ability. Characteristically, patients who suffer these difficulties are impulsive in decisions that are made, and have difficulty controlling explosive emotional responses while yet passively less able to initiate a sequence of appropriate spontaneous responses. A mixture of several types of injury are attributed to these and other difficulties, but increasingly, injury to connections between the frontal poles of the brain and even with one gyrus with a neighbor, can be found on the most sophisticated image interpretations [54]. It is therefore natural to conclude that the effects of head and brain injury in such situations, with the most noticeable changes being changes in motivation and impulse control, could be similar in ACD. ACD would be expected to have effects most prominently on the frontal areas and on cell migration and synaptic connectivity. If widespread, ACD would have potentially dramatic effects on society. In view of the delay in frontal maturation, a younger population might be said to further exacerbate a trend to impulsivity and explosiveness. A search for any evidence either for or against such trends in the societies which practice ACD could be construed as evidence for or against this line of reasoning.

Physiological considerations of increased intracranial pressure along with extended distances for cell migration and pathway connectivity also might have potential negative effects of frontal compression. Frontal elongation of structures internal to the cranium including arterial and venous circulation and unusual angulation of the ventricular system, which produces the cerebral spinal fluid (CSF) bathing all surface areas of the brain, may be responsible for changes in extracellular tissue pressures and ion concentrations in CSF as well as in extracellular tissues. Non-fatal deformity does argue in favor of non-critical (or potentially even sub-clinical) abnormalities but does not exclude the possibility of pathologies which could expose the effected individuals to a lessened functional ability compared to the pre-deformation potential. In this culture distant in both time and numerous other factors, it is possible that such deformations were of little significance to the society as a whole. However, it can generally be reasonably argued that most afterbirth deformational techniques do not improve functional outcomes of the tissues themselves. In a particular cultural or exposure setting however, one could argue that some advantage could be construed or obtained.

In the occipital area the effects of ACD may be similar but research allows the comprehension of the individual properties of vision even more clearly. The individual attributes of point, line, angle direction of movement and color variation are known to be attributable to individual neuronal populations. Columns of neurons in cortex in primary visual areas connect to secondary columns which process combined attributes which in turn transmit information to complex columns of neurons and in turn to hyper-complex columns which can formulate shapes and seem to "recognize" objects and this columnar organization of cortex forms networks which place context, generalize more abstractly after recognition of novel or prior experience. These latter steps involve association cortex regions in the occipito-parietal cortex. Injury in children suggests that the areas of cortex supporting these functions can be resilient in that recruitment of neighboring areas for assimilation of lost brain areas is common. As with other areas of brain function, addition of subcortical connections along with associated cortical regions is more devastating. Total volume of injury, whether multifocal or not, generally correlates with degree of loss of function.

Discussion and Conclusions

Since ACD is no longer popularly practiced only a theory can be generated about what implication these various ACD practices had on the individuals' mental abilities and functions. Based on similar deforming conditions to the brain, like plagiocephaly, the results of these studies can help in understanding the circumstances of the past. Whether the pressures applied to these areas had harmful, beneficial, or insignificant influences can only be theoretically determined. A closer look at where pressure from the bands was applied on the various lobes of the brain, along with a fair, impartial consideration of the possible implications that occurred, sheds light on some interesting possibilities that ACD practices could have had on the individuals.

The practice of annular ACD would affect the frontal and occipital areas of the skull. Pressure to these areas would potentially affect the functions correlated with these areas in the lobes of the brain. Using information and experiences from the modern world that simulate similar states helps to form a hypothesis of whether the implications were harmful or beneficial. Damage to the frontal lobe that has been documented by doctors shows that impairment to memory, inattentiveness, inability to concentrate, behavior disorders, difficulty in learning new information, and motor aphasia take place. Documentation also shows that the pressure from the bands, from both tabular and/or annular ACD, in the occipital region may have influenced vision and ability to recognize objects. Pressure to temporal areas may induce damage that results in changes of worsening hearing ability, agitation, and irritability [29].

In the cultures that practiced ACD, distant in both time and numerous other factors, it is possible that such deformations were of little significance to the society as a whole. However, it can generally be reasonably argued that most afterbirth deformational techniques do not improve functional outcomes of the tissues themselves. In a particular cultural or exposure setting however, one could argue that some advantage could be construed or obtained. The extent of these implications could have ranged from individuals due to duration and intensity of the pressure. An understanding of the full extent of the implications of ACD may never be met, but perhaps in the future more knowledge will be available through more research in the areas of craniosynostosis and plagiocephaly that could aid in this uncertainty.

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References

1. O'Brien TG, Sensor KP (2004) On the classification of abnormal head shape: interpreting artificial cranial deformation and craniosynostosis. Journal of Paleopathology16: 27-51.
2. O'Brien TG, Sensor ILA (2008) On the effect of cranial deformation in determining age from ectocranial suture closure. Growth Development and Aging 71: 17-27.
3. Moss ML (1958) The pathogenesis of artificial cranial deformation. American Journal of Physical Anthropology16: 269-286.
4. Berry AC, Berry RJ (1967) Epigenetic Variation in the Human Cranium. Journal of Anatomy101: 361-379.
5. Kohn LAP, Cheverud JM (1991) The effects of cradleboarding on the cranial base and face. American Journal of Physical Anthropology, Supplement 12: 106.
6. Cheverud JM, Kohn LAP, Konigsberg LW, Leigh SR (1992) Effects of fronto-occipital artificial cranial vault modification on the cranial base and face. American Journal of Physical Anthropology 88: 323-345.
7. Kohn LAP, Leigh SR, Jacobs SC, Cheverud JM (1993) Effects of annular cranial vault modification on cranial base and face. American Journal of Physical Anthropology 90: 147-168.
8. Konigsberg LW, Kohn LAP, Cheverud JM (1993) Cranial Deformation and Nonmetric Trait Variation. American Journal of Physical Anthropology 90: 35-48.
9. Gerszten PC, Gerszten E (1995) Intentional cranial deformation: a disappearing form of self-mutilation. Neurosurgery 37: 374-381.
10. Rhode MP, Arriaza BT (2006) Influence of cranial deformation on facial morphology among prehistoric south central Andean populations. American Journal of Physical Anthropology130: 462-470.
11. McNeill RW, Newton GN (1965) Cranial base morphology in association with intentional cranial vault deformation. American Journal of Physical Anthropology 23: 241-254.
12. Imbelloni J (1938) Formas, esencia y metodica de las deformaciones cefalicas intencionales. Universidad Nacional de Tucuman. Instituto de Antropologia. Revista Instituto de Antropologia 1: 5-37.
13. Imbelloni J (1963) Cephalic deformations of the indians of Argentina. In: Steward JH, editor. Handbook of South American Indians. Smithsonian Institution Bureau of American Ethnology Bulletin 143: 53-55.
14. Gerszten PC (1993) An investigation into the practice of cranial deformation among the pre-Columbian peoples of Northern Chile. International Journal of Osteoarchaeology 3: 87-98.
15. Anton SC (1989) Intentional cranial vault deformation and induced changes of the cranial base and face. American Journal of Physical Anthropology 79: 253-267.
16. Dean VL (1995a) Sinus and meningeal vessel pattern changes induced by artificial cranial deformation: a pilot study. International Journal of Osteoarchaeology 5: 1-14.
17. Dean VL (1995b) Effects of cultural cranial deformation and craniosynostosis on cranial venous sinus and meningeal vessel pattern expression. PhDthesis, Indiana University.
18. Björk A, Björk L (1964) Artificial deformation and craniofacial asymmetry in ancient Peruvians. Journal of Dental Research 43: 353-362.
19. Dingwall EJ (1931) Artificial Cranial Deformation–A Contribution to the Study of Ethnic Mutilation. John Bale, Sons and Danielsson: London.
20. Allison MJ, Gerszten E, Munizaga J, Santoro C, Focacci G (1981) La practica de la deformación craneana entre los pueblos andinos precolombinos. Chungara 7: 238-260.
21. Cohen MM (1986) Craniosynostosis: Diagnosis, Evaluation and Management. Raven Press: New York, NY.
22. Dembo A, Imbelloni J (1938) Deformaciones Intencionales del Cuerpo Humano de Carácter Etnico. Humanior, Biblioteca del Americanista Moderno. Seccion A, Tomo III. Buenos Aires, Argentina.
23. McGibbon W (1912) Artificially deformed skulls with special reference to the temporal bone and its tympanic portion. Laryngoscope 22: 1165-1184.
24. Rogers SL (1975) Artificial deformation of the head – New World examples of ethnic mutilations and notes on its consequences. San Diego Museum Papers No. 8. San Diego: San Diego Museum of Man.
25. Shapiro HL (1928) A correction for artificial deformation of skulls. Anthropological Papers of the American Museum of Natural History 30: 1.
26. Moss ML, Young RW (1960) A functional approach to craniology. American Journal of Physical Anthropology 18: 281-292.
27. Carruthers P (2006) The Architecture of the Mind. Clarendon Press: Gloucestershire, UK.
28. Beckstead RM (1996) A survey of medical neuroscience. Springer: New York, NY.
29. Turkington C (1996) The Brain Encyclopedia. Facts on File, Inc: New York, NY.
30. Steen GR (2007) The Evolving Brain. Prometheus Books: Amherst, NY.
31. Howard PJ (1994) The Owner's Manual for the Brain. Leornian Press: Austin, TX.
32. Ornstein R, Thompson RF (1984) The Amazing Brain. Houghton Mifflin Company: Boston, MA.
33. Moore WJ, Lavelle CLB (1974) Growth of the Facial Skeleton in the Hominoidea. Academic Press: New York, NY.
34. Herring SW (1972) Sutures – a tool in functional cranial analysis. Acta Anatomica 83: 222-247.
35. Thorogood P (1993) Differentiation and morphogenesis of cranial skeletal tissues. In: The Skull, Hanken J and Hall BK (eds.). Chicago University Press: Chicago, IL 112-152.
36. Enlow DH, Hans MG (1996) Essentials of facial growth. WB Saunders: Philadelphia, PA.
37. Brennan M, Antonyshyn O (1996) The effects of temporalis muscle manipulation on skull growth: an experimental study. Plastic Reconstructive Surgery 97: 13-24.
38. Stini WA (1998) Skeletal development. In: The Cambridge Encyclopedia of Human Growth and Development, Ulijaszek SJ, Johnston FE and PreeceMA (eds.). Cambridge University Press: Cambridge, UK 196-197.
39. Buchman SR, Bartlett SP, Wornorn IL, Whitaker LA (1994) The role of pressure on regulation of craniofacial bone growth. Journal of Craniofacial Surgery 5: 2-10.
40. Lekovic GP, Baker BJ, Lekovic JM, Preul MC (2007) New World cranial deformation practices: historical implications for pathophysiology of cognitive impairment in deformational plagiocephaly. Neurosurgery 60: 1137-1147.
41. Miller RI, Clarren SK (2000) Long-Term Developmental Outcomes in Patients with Deformational Plagiocephaly. Pediatrics 105: e26.
42. Rakic P (1995) Corticogenesis in human and nonhuman primates. In: The cognitive neurosciences, GazzaningaMS (ed.). MIT Press:Cambridge, MA 127-145.
43. Gage NM, Johnson MH (2007) Development. In: Cognition, Brain, and Consciousness: Introduction to Cognitive Neural Science, Baars BJ and Gage NM (eds.). Elsevier: Boston, MA 411-453.
44. Saper CB, Iversen S, Frackowiak R (2000) Integration of the sensory and motor function: the association areas of the cerebral cortex and the cognitive capabilities of the brain. In: Principles of Neural Science, 4th Edition. McGraw-Hill: Columbus, OH 349-411.
45. Goldberg E (2001) The executive brain: frontal lobes and the civilized mind. Oxford University Press: New York, NY.
46. Goldman-Rakic PS (1988) Topography of cognition: Parallel distributed networks in primate association cortex. Annual Review of Neuroscience 11: 137-156.
47. Cavada C (2000) The anatomical connections of the macaque monkey orbitofrontal cortex. A review. Cerebral Cortex 10: 220-242.
48. Price JL (2006) Connections of the Orbital Cortex. In: The Orbitofrontal Cortex, ZaldDH and Rauch SL (eds.). Oxford University Press: New York, NY 39-55.
49. Duncan J, Owen AM (2000) Common regions of the human frontal lobe recruited by diverse cognitive demands. Trends in Neuroscience 23: 475-483.

50. Schneider W, Chein JM (2003) Controlled and automatic processing: behavior, theory, and biological mechanisms. Cognitive Science 27: 525-559.

51. Huttenlocher PR, De Courten C, Garey LJ, Van der Loos H (1982) Synaptic development in human cerebral cortex. International Journal of Neurology16: 144-154.

52. Varney N (1999) Post-Traumatic Anosmia and Frontal Injury. In: The Evaluation and Treatment of Mild Traumatic Brain Injury,Varney NR and Roberts RJ (eds.). Lawrence Erlbaum Associates, London 115-131.

53. Gennarelli TA, Thibault LE (1982) Biomechanics of acute subdural hematoma. Journal of Trauma 22: 680-686.

54. Benson RR (2007) Global White Matter Analysis of Diffusion Tensor Images Is Predictive of Injury Severity in Traumatic Brain Injury. Journal of Neurotrauma 24: 446-459.

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