A ROENTOGRAPHIC CEPHALOMETRIC ANALYSIS OF THE CEREBRAL PALSIED PATIENT

by

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INTRODUCTION

William Little[1] first described cerebral palsy in 1861. The medical profession has since been actively engaged in investigating the condition and in developing methods of treatment.

Only in recent years, however, has the dental profession begun to take an active interest in the dental problems of the cerebral palsied. A few years ago, and to a lesser degree today, cerebral palsy patients were turned away from many dental offices when they sought treatment. However, within the past 10 to 15 years the profession has awakened to its responsibility to administer to the dental needs of the handicapped. This awareness has been accompanied by an increase in the number of investigations into the dental problems of the cerebral palsied. Most reports on dental problems were based on clinical impression, but recently efforts have been made to carefully investigate their dental problems.

A number of studies have investigated the incidence of malocclusions in the cerebral palsy patient. Several investigators[2,3,4,5] reported that cerebral palsied patients have a high incidence of malocclusions. They point to an increase in both the number of class II malocclusions and the number of open bites. This they attribute to the abnormal tongue and lip action often seen in the cerebral palsied patient.

Of the many studies on the dental problems of the cerebral palsied, only one utilized roentographic cephalometry. This cephalometric analysis was done by Album and associates[6] at the Philadelphia Center for Research in Child Growth and the Orthodontic Clinic of the University of Pennsylvania Dental School, as a part of a pilot study evaluating the dental profile of cerebral palsied patients. Their study was limited to a few measurements. They suggested that cerebral palsy patients should be studied with a more detailed roentographic-celphalometric appraisal.

This study will seek to determine, by use of cephalometric analysis, if the dental and oro-facial complex of cerebral palsy patients is significantly different from that of the non-cerebral palsied patient.

REVIEW OF THE LITERATURE

Cerebral palsy is a condition in which the patient has a neuromuscular dysfunction due to a nonprogressive brain lesion or lesions. Crothers[7] wrote, "The disorder of motor control is only one, and often the least, of the difficulties which face these children. Inevitably, many children with impairment of the brain may have mental irregularities and some will have convulsions, others will have defects of hearing or eyesight or other sensory difficulties." The incidence of this condition in the United States is three per 1,000 population.[8]

Classification of Cerebral Palsy

Cerebral palsy is classified into five major groups:[8]

1. Spastic type, characterized by the hyperactive reflex and an exaggerated stretch reflex of the affected parts of the body (50 per cent of the cases).
2. Athetosis, characterized by slow, involuntary, uncontrollable and purposeless movements (25 per cent of the cases).
3. Ataxic type, characterized by a lack of balance and equilibrium; the gait is like that of a drunken person (nine per cent of the cases).
4. Tremor type, characterized by intention and non-intention tremors (one per cent of cases).
5. Rigid type, characterized by hypertonic muscles which stiffen when attempts are made to move them (15 per cent of cases).

When a patient exhibits characteristics of more than one type of cerebral palsy, the condition is described as mixed. A patient might be described as mixed spastic athetoid.

Cerebral palsy is also classified according to the area of involvement:[9]

Monoplegia...........Involves one limb.

Hemiplegia...........Involvement of one side of the body.

Quadriplegia.........Involvement of all four limbs.

Paraplegia...........Involvement of the legs only.

Etiology

Many etiological factors cause cerebral palsy. Prenatal and perinatal factors account for 80 per cent of the cases. Principal prenatal causes are: developmental defects (some are genetic), infection (rubella) and prematurity. Anoxic damage and isoimmunization injuries (kernicterus) are the most important perinatal factors. The most important postnatal cause is infection. Other causes are trauma, tumor, cerebrovascular accidents and surgical complications.[8]

Mothers of cerebral palsied children have histories of a high incidence of abortion, premature birth, stillbirths, and severe illness prior to pregnancy.[8]

Occlusion

The literature reveals controversy regarding an increase in the number of malocclusions in the cerebral palsied patients compared to the normal.

Rosenstein[2] reported 75 per cent more class II malocclusions in cerebral palsied patients than in the normal population.

Jackson,[3] in a study of 84 white cerebral palsied children between ages three and 17, found that 76.2 per cent had a well defined malocclusion, compared to 52.4 per cent malocclusions in the normal group of 380. The following is a breakdown in the different types of malocclusion among the cerebral palsy children:

Class I malocclusion	53.1 per cent
Class II malocclusion	42.2 per cent
Class III malocclusion	4.7 per cent

Both the cerebral palsied patients and the normal group showed practically the same percentage of malocclusion in each of the three classifications. Jackson also reported that the young cerebral palsied patients with the deciduous dentition showed more normal occlusion and less severe malocclusion than did cerebral palsied children with mixed dentition. He felt that abnormal muscle function was an important factor in the development of these malocclusions.

Watson,[4] after examining 119 cerebral palsied children in Australia, reported no higher incidence of malocclusion in the cerebral palsied than in normal children. He concluded that abnormal muscle movement does not seem to cause malocclusion.

Lyons[5] examined 50 cerebral palsied children, ages 10 to 14 years, and found that 36 (72 per cent) had malocclusions.

Rosenbaum[10] studied 124 cerebral palsied children between the ages of six and 12 and a control group of 141 non-cerebral palsied children in the same age group. Both groups showed similar percentages of malocclusion.

There is variation in the reported incidence of open bite in cerebral palsied children. Jackson[3] reported 21 per cent open bites in his group of 84 cerebral palsied children. Lyons[5] reported seven (14 per cent) open bites in 50 cerebral palsied children. Rosenbaum[10] reported 14 (11.3 per cent) open bites in 124 cerebral palsied children and 18 (12.7 per cent) open bites in the control group of 141. In Rosenbaum's study the amount of open bite was more than twice as much in the cerebral palsied group, 2.9 mm. in the cerebral palsied children and 1.2 mm. in the normal group.

There are also varied reports on the incidence of high arched palates. Jackson[3] and Trausch[1] reported a higher incidence of high arched palates. Siegel[11] reported that there was no significant difference in the number of high arched palates between his cerebral palsy group of 65 and his control group of 65.

Bruxism

Many investigators reported a high incidence of bruxism among cerebral palsy patients. Jackson[3] found bruxism in 36 (43 per cent) of the 84 cerebral palsied children and a 21 per cent incidence in the control group of 380. Watson[4] reported that 28

(23.5 per cent) of 119 cerebral palsy children exhibited bruxism. Rosenbaum[10] stated that 54 (44 per cent) of 124 cerebral palsied children exhibited bruxism. In his control group, 29 (21 per cent) of 141 showed bruxism. Siegel[11] examined 65 cerebral palsied children and a control group of 65. He found bruxism in 27 per cent of the cerebral palsied patients and no bruxism in the control group.

Muscle Function and Malocclusion

Those who reported a higher incidence of malocclusion in the cerebral palsied attributed this to the increase in abnormal oral musculature function. These questions arise: (1) Does abnormal muscle function cause malocclusion? (2) If so, is this due to a change in the position of the teeth and shape of the alveolar process or is it the result of the effect of muscle function upon the facial skeleton (the size, shape and relation of the maxilla and mandible to each other and to cranial base)?

The majority of writers today agree that the main factors governing the pattern of skeletal bone are in general inherited.[12,13,14]

The alignment of the teeth and the form of the arches is influenced by the shape and size of the jaws and by muscle forces set up by patterns of movement between the lips, tongue and cheek in swallowing, feeding, in facial expression and possibly speech.[13] The fact that the forces exerted by the tongue and circumoral musculature are not equal indicates that there are factors other than muscle balance which are important in the stability of the dentition.[12] Winders,[15] by use of strain gauges connected to an oscillograph, found that there is more pressure exerted on the dentition during function by the tongue than by the perioral musculature.

Subtelny[16] states: (1) The basic bony component extending from the condyle to the chin cannot be readily affected by the usual muscle forces. (2) Structural areas such as the coronoid process seem to be dependent upon the presence and function of muscle. (3) The dento-alveolar area seems to be most readily affected by muscles and the lips.

Lundstrom[17] did a roentographic cephalometric analysis of 50 monozygotic and 50 dizygotic twins and concluded that genetic factors are twice as important as non-genetic factors in the skeletal pattern of facial profile. Van der Linden[18] states:

> "The permanent interaction between genetic and environmental factors directs the growth of the craniofacial complex (including the development of malocclusion). Genetic factors seem to have the greatest influence, whereas environment factors appear to be of minor importance. Environmental factors, including orthodontic therapy, probably primarily affect the dental alveolar region and the interrelationship of the individual bony elements. The morphology of the individual bones seems to be under rather rigid genetic control."

Abnormal Swallow

Gwynne-Evans[13] stated that Rix, in 1946, was the first to observe that many abnormal dental anomalies seemed to be related to an abnormal swallow. Many conditions recorded in the literature, have been attributed to an abnormal swallow:[19]

1. Narrowing of the maxillary arch.
2. Protrusion of the upper anterior teeth.
3. Drastema between the anterior teeth.
4. Open bite.

Straub[20] suggested that a patient's tongue being interdental during swallow can cause mandibular growth to be altered to cause a steeper mandibular plane.

The swallow pattern of an infant is different from that of a mature adult. In the infant, during swallow the tongue extends between the gum pads to touch the cheeks and lips. The muscles of the lips and cheek contract in order to seal the walls of the oral cavity and resist the force of the tongue during deglutition.[13]

With the eruption of the teeth, the pattern of swallow changes. Instead of the tongue extending between the arches, it is confined by the arches, since the teeth come into occlusion during swallow. As one swallows, the tongue presses upward and forward against the hard palate and inner surface of alveolus, then there is a peristolic contraction of the tongue to squeeze the food back.[13] Since the teeth confine the tongue during swallow, the lips remain passive and there is no perioral contraction. Subtelny[21] stated that Tulley observed that patterns of lip and tongue activity do change with time, growth and maturation.

Some people do not develop a mature swallow after the teeth erupt. In other words, during swallow:[13]

(1) The teeth do not come into occlusion.

(2) The tongue moves anteriorly or laterally between the teeth during swallow to touch the cheeks and lips.

(3) The perioral muscles contract to resist the force of the tongue.

Many times children will retain the so-called "infantile swallow" up to and beyond eight years of age. Weinberg[22] stated that the tongue being interdental during swallow should not be considered abnormal up to eight years.

Abnormal Swallow and Malocclusion

There is considerable disagreement on the relationship between an abnormal swallow (tongue thrusting) and malocclusion.

Atkinson[23] stated that an open bite seems to result from one basic cause, swallowing with the teeth apart.

Kydd[24] used pressure transducers utilizing resistance strain gauges to measure the forces exerted on the anterior dentition by the lingual and perioral musculature during the act of swallow. He selected females, ages 14 to 20 years, who had open bites treated orthodontically. The first group, who were tongue thrusters, had a relapse to the anterior open bite within one year after orthodontic treatment. The second group, normal swallowers, retained normal overbite and overjet one year after treatment. Kydd found that the mean tongue pressure was twice as great in the tongue thrusters.

Kydd[25] cited that Akramine, Kydd, Mendel and Kraus demonstrated by use of strain gauges mounted on the upper anterior teeth, that patients with anterior open bites exert more pressure upon their teeth for a longer period of time during deglutition than patients without an open bite.

Kydd[25] found that the tongue thrusters swallowed at the mean rate of 37.25/hour. The normal swallowers had a deglutition rate of 61.41/hour. Since tongue thrusters swallow at about one-half the rate of normal swallowers the total force is about equal in tongue thrusters and normal swallowers.

Hovell[26] stated that Nord suggested several years ago that soft tissue activity may be secondary to malocclusion. Most orthodontists in England tend to feel that the great majority of atypical swallow result from, and are not the cause of, malocclusion or variation in soft tissue and skeletal morphology.

Rogers[19] stated that in many of the orthodontic cases he treated, improved tooth position has resulted in the elimination of the abnormal swallow.

Subtelny[21] suggested that tongue thrusting is an associated factor rather than a causative factor in many malocclusions. Subtelny[16] said, "Tongue thrusting is not an established cause of defective speech and/or malocclusion."

Jackson,[3] Trausch,[1] and Koster[27] and others reported that abnormal muscle forces seen in cerebral palsied patients result in dento-alveolar deviations. They believed abnormal swallow to be an important cause of open bite. Jackson[3] stated that hyper-tonicity of the buccal and labial tissues acts on the dental arches preventing their anterior and lateral growth. He also stated that this hypertonicity results in maxillary and mandi-bular arches which are narrow, resulting in a crowded condition of the maxillary and mandibulary incisors.

Trausch[1] reported that 20 (23.2 per cent) of 86 cerebral palsy children had an involuntary tongue thrust.

Rosenbaum[10] reported that 44 (35.5 per cent) of 124 cerebral palsy children exhibited tongue thrusting and that 37 (26.2 per cent) of 141 non-cerebral palsied children were tongue thrusters. This is not a statistically significant difference in the incidence of tongue thrusting.

Both Rosenbaum[10] and Trausch[1] reported a higher incidence of tongue thrusting among the athetoid type of cerebral palsy children.

Rosenbaum[10] stated that, on the basis of his study one might theorize that malocclusion and the factors affecting occlusion are not more common in cerebral palsied children.

Hovell,[26] Moyers,[26] and Kydd[24] pointed out that studying the relationship of muscle and occlusion is extremely difficult. Moyers[26] said, "Our interest in the relationship of muscle and malocclusion outruns our knowledge."

<u>Roentographic Cephalometric Study</u>

Album, et al,[6] did a roentographic cephalometric study of 45 cerebral palsy children seven to 10 years of age. The mean age for the boys was 8.6 years and the mean age for the girls was 8.3 years. When facial composites of the cerebral palsied boys and girls were compared with eight-year-old standards, no discernable facial-skeletal deviations could be detected. Cephalometric analysis revealed the only deviation to be a mid-facial protrusiveness, when measured by the angles SNA and SNB. This protrusiveness was statistically significant only in females at the .05 level.

STATEMENT OF THE PROBLEM

This study sought to determine by cephalometric analysis if the dental and oro-facial complex of cerebral palsy patients is significantly different from that of the non-cerebral palsied. Because of the controversy as to the incidence of malocclusion in cerebral palsy patients and since there has been only one reported cephalometric analysis of these patients, it was felt that a more complete cephalometric analysis would add to our knowledge of their dental oro-facial complex.

EXPERIMENTAL PROCEDURE

Twenty-six cerebral palsied patients between the ages of seven and 18 were selected at random from those attending the Indiana University Cerebral Palsy Dental Clinic. A few patients were excluded when it was decided that they had insufficient neck control to safely be placed in a cephalostat.

The patients' medical records were examined and the parents were questioned in order to obtain the necessary information to complete the history portion of the data sheet (Figure 1).

In conducting the clinical examination, the patient was engaged in conversation while clinical observations were being made.

The patient was considered a mouth breather if he habitually kept the lips apart and if he could not hum.

In evaluating swallow, if there was any movement of the lips, it was recorded as excessive perioral activity. If the lips moved during swallow, excessive activity of the orbicularis oris complex was recorded. If there was puckering of the mentalis muscle during swallow, excessive mentalis muscle activity was recorded.

In determining if the teeth were together during swallow, the patient was asked to hold the lower lip with the fingers and swallow. This procedure was repeated several times while the teeth were observed.

The relationship of the maxillary first permanent molars to the mandibular first permanent molars was determined. A vertical line, which originated at the tip of the mesial buccal cusp, was drawn on the buccal surface of the maxillary first molar. With the teeth in occlusion, a measurement was taken of the horizontal distance between the line and the buccal groove of the mandibular first permanent molar. This measurement was taken using a millimeter rule which had been reduced in size. The measurement was recorded to the nearest one-half millimeter and was designated (+) if the groove was anterior to the pencil line and (-) if the groove was posterior to the pencil line.

The relationship of the cuspids was determined. A vertical line originating at the tip of the cusp was drawn on the labial surface of each cuspid. With the teeth in occlusion, the horizontal distance was measured between the vertical lines. If the pencil line on the mandibular cuspid was anterior to the line on the maxillary cuspid the measurement was recorded (+). If the lower cuspid line was posterior to the upper cuspid line the measurement was recorded (-).

When questioning of the parents revealed that the child ground the teeth and if worn facets were detected on the teeth bruxism was recorded. For purposes of the study, the bruxism was considered moderate if the facets were confined to enamel and severe if dentine was visible on the facets.

After approximately 10 patients had been examined, five were re-examined by a speech therapist. The author and the speech therapist were in substantial agreement on the clinical examination.

Lateral cephalometric radiographs were taken. The patients were placed in a B. F. Wehmer cephalometer and the x-ray film was exposed in cassettes with intensifying screens. The exposure time was seven-tenths second, using 90 kilovolts and 15 milliamperes. The x-ray film was Kodak Blue Brand medical x-ray film BBA59.

A wrist plate was also taken using 90 kilowatts and 15 milliamperes with a one-tenth second exposure time (Figure 2). These wrist plates were then evaluated to establish skeletal age by making comparisons with the standards of skeletal development in Greulich's Radiographic Atlas of Skeletal Development of the Hand and Wrist.[28]

A tracing of the lateral headplate was made on acetate tracing paper using a sharp 4-H drawing pencil. When bilateral structures could be seen, both were traced and the mean of the two structures was used in making measurements. The following landmarks (Figure 3) were recorded on each tracing:

1. Nasion (N) - the most anterior point on the fronto-nasal suture.
2. Sella (S) - the center of sella turcica, determined by inspection.

3. Articulare (Ar) - the point of intersection of the posterior border of the mandibular ramus with the temporal bone.

4. Gonion (Go) - a point formed by the intersection of the lines drawn for ramal plane and mandibular plane.

5. Pogonion (Pg) - the most anterior point on the chin button determined by a tangent perpendicular to mandibular plane.

6. Gnathion (Gn) - the most inferior point on the contour of the chin.

7. Anterior Nasal Spine (ANS) - the tip of the anterior nasal spine.

8. Posterior Nasal Spine (PNS) - the point of intersection of nasal floor and the projection of the apex of the pterygomaxillary fissure.

9. Point A (A) - the deepest midline point on the maxilla below ANS.

10. Point B (B) - the deepest midline point on the mandible between pogonion and the alveolar crest.

11. Incision Superius (Is) - the incisal point of the most prominent maxillary central incisor.

12. Incision Inferius (Ii) - the incisal point of the most prominent mandibular central incisor.

These landmarks, a protractor and a millimeter rule were used to make the following measurements to the nearest one-half millimeter or one-half degree:

Cranial Base

1.	N-Ar(FH)	length of cranial base (measured parallel to Frankfort horizontal)
2.	N-S-Ar	cranial flexure angle

Maxilla

3.	PNS-A(FH)	length of maxilla (measured parallel to Frankfort horizontal)

Mandible

4.	Ar-Go	length of ramus
5.	Go-Pg	length of body
6.	Ar-Go-Gn	gonial angle

Facial Height

7.	ANS-N($\perp$FH)	upper facial height (measured perpendicular to Frankfort horizontal)

Dental Height

8.	Is-ANS($\perp$FH)	upper dental height (measured perpendicular to Frankfort horizontal)
9.	Ii-Gn($\perp$FH)	lower dental height (measured perpendicular to Frankfort horizontal)

Position

10.	A-B(OP)	the relationship of the anterior of the maxilla to the anterior of the mandible along occlusal plane.
11.	N-A(FH)	the relationship of the anterior of the maxilla to nasion (measured parallel to Frankfort horizontal)
12.	N-B(FH)	the relationship of the anterior of the mandible to nasion (measured parallel to Frankfort horizontal)

Frankfort horizontal (FH). A line determined by dropping a line 7° below the line S-N.*

Occlusal plane (OP). A line drawn by bisecting the first molar and central incisor occlusion.

Mandibular plane (MP). A line drawn tangent to the inferior border of the mandible.

Ramal plane. A line drawn tangent to the posterior of the ramus of the mandible.

The cephalometric measurements of the cerebral palsied patients were statistically compared with normative data of non-cerebral palsied patients.

The normative data was compiled by the Orthodontic Department of Indiana University from material obtained from the

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*Since it is difficult to accurately locate porion and orbitale to establish Frankfort horizontal, the Orthodontic Department of Indiana University has determined by cephalometric standards that on the average a line 7° below S-N is parallel to Frankfort horizontal.

Child Research Council, Denver, Colorado. The Council and its material have been described by Waldo[29] and McDowell.[30] The normative data was developed from serial records of 14 males and 16 females who had not been treated orthodontically.

DATA

Table IV indicates the number of patients whose swallow exhibited the following characteristics: (1) lips passive (2) lips active (3) orbicularis oris muscle active (4) mentalis muscle active (5) tongue interdental and (6) teeth apart during swallow.

Computations for this project were performed at the Indiana University Medical Center Research Computation Center.*

The cephalometric data of the cerebral palsied patients and the non-cerebral palsied patients were analyzed (Tables V and VI).

Comparison of the cephalometric data of male cerebral palsied patients with male non-cerebral palsied patients (Table V) revealed the following significant differences:

1. The cranial flexure angle (N-S-Ar) was more acute in the cerebral palsied patients ($p < .05$).
2. The length of the maxilla [PNS-A(FH)] was longer in the cerebral palsied patients ($p < .05$).
3. Gonial angle (Ar-Go-Gn) was more obtuse in the cerebral palsied patients ($p < .05$).

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There were no significant differences in the other cephalometric measurements.

Data comparing cephalometric measurements of female cerebral palsied patients with the female non-cerebral palsied patients (Table VI) revealed the following significant differences:

1. The cranial flexure angle (N-S-Ar) was more acute in the cerebral palsied patients ($p < .05$).
2. The length of the body of the mandible (Go-Pg) was greater in the cerebral palsied patients ($p < .01$).
3. Lower dental height [Ii-Gn(⊥FH)] was longer ($p < .01$).

There were no significant differences in the other cephalometric measurements.

Comparing cephalometric measurements of male spastic type cerebral palsied patients with the male non-cerebral palsied patients, (Table VII) data revealed the following significant differences:

1. The cranial flexure angle (N-S-Ar) was more acute in the cerebral palsied patients ($p < .05$).
2. Point A on the maxilla was positioned more anteriorly in relation to nasion [N-A(FH)] in the cerebral palsied patients ($p < .05$).

3. Point B on the mandible was positioned more anteriorly in relation to nasion [N-B(FH)] in the cerebral palsied patients ($p < .05$).

Data comparing cephalometric measurements of female spastic type cerebral palsied patients with the female non-cerebral palsied patients (Table VII) revealed the following significant differences:

1. The cranial flexure angle (N-S-Ar) was more acute in the cerebral palsied patients ($p < .01$).
2. The length of the body of the mandible (Go-Pg) was greater in the cerebral palsied patients ($p < .01$).
3. Lower dental height [Ii-Gn(⊥FH)] was longer ($p < .01$).
4. Point B on the mandible was positioned more anteriorly in relation to nasion [N-B(FH)] in the cerebral palsied patients ($p < .05$).

Data comparing the cephalometric measurements of the seven cerebral palsied patients whose lips were passive during swallow, with the cephalometric measurements of 19 cerebral palsied patients whose lips were active during swallow, (Table VIII) revealed the following significant differences:

1. The length of the ramus of the mandible (Ar-Go) was greater in patients with active lips during swallow ($p < .05$).

For those cephalometric measurements which were affected by sex, chronological age, and/or skeletal age, the comparison was made by the method of analysis of covariance.

Data comparing the cephalometric measurements of cerebral palsied patients with interlabial gap with cerebral palsied patients without interlabial gap (Table IX) revealed the following significant differences:

1. Point A on the maxilla was positioned more posteriorly in relation to nasion [N-A(FH)] in those patients with interlabial gap ($p < .01$).
2. Point B on the mandible was positioned more posteriorly in relation to nasion [N-B(FH)] in those patients with interlabial gap ($p < .05$).

Analysis and comparison of cephalometric measurements of cerebral palsied patients who exhibit bruxism with cerebral palsied patients who did not, revealed no significant differences (Table X).

Analysis and comparison of the molar and cuspid relationship data of the cerebral palsied patients (Tables XI, XII, XIII and XIV) with the molar and cuspid relationship data of Rosenbaum's[10] normal group (Table XV) revealed that the lower right cuspid was positioned more anteriorly in the cerebral palsied patients. There was no significant difference in the right molar, left molar and left cuspid relationships.

The skeletal ages of the cerebral palsied patients, as determined by use of wrist plates, were compared with their chronological ages. Analysis revealed a significant difference in skeletal age. The cerebral palsied patients were significantly retarded in skeletal development ($p \leq .05$) (Table XVI).

TABLES AND FIGURES

Figure 1. Data sheet used to record history and findings of clinical examination.

Name_________________________Birthdate__________Hosp.No._______

Address____________________________Sex__________Date___________

Classification______________________Distribution_________________

HISTORY

Illnesses of long duration

____

____

Illnesses which could affect facio-skeletal development

____

____

Naso-pharyngeal airway problems

Chronic upper respiratory infections

_______Yes

_______No

Type ____________

____

Allergy

_______Yes

_______No

_______No. of yrs. afflicted

_______Mos. per yr. affected

Has it been treated

_______Yes

_______No

Asthma

______Yes

______No

______No. of yrs. afflicted

______Mos. per yr. affected

Snore

______Yes

______No

______Frequency

HABITS

Thumb or finger sucking

______Yes

______No

Lip biting

______Yes

______No

Use pacifier

______Yes

______No

(Figure 1, continued)

CLINICAL EXAM

Rest

______Lips together
______mm. interlabial gap
______Nose breather
______Mouth breather
______mm. freeway space

Bruxism

_____Not present
_____Moderate
_____Severe
_____Day
_____Night

Tongue

______Within dental arch
______Interdental
     _____Anterior
     _____Lateral

Swallow

Lips

______Passive
______Excessive perioral activity
     _____Orbicularis oris complex
     _____Mentalis

Lip trapped under maxillary
anterior teeth
______Yes
______No
     _____Teeth together swallow
     _____Teeth apart swallow

Tongue

______Within dental arch
______Interdental
     _____Anterior
     _____Lateral
     _____Vigorous
     _____Passive

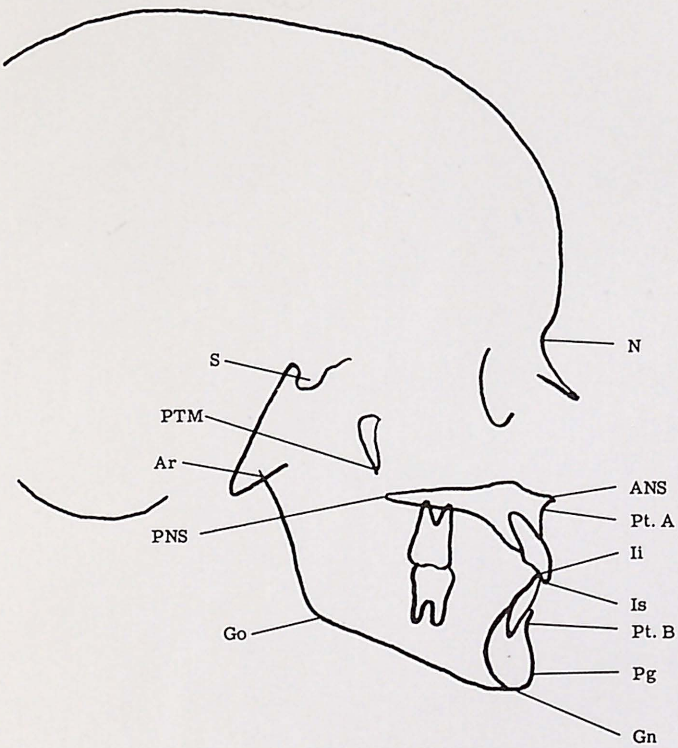
Occlusion

Molar
______mm. Right _____mm. Left

Cuspid
______mm. Right _____mm. Left

Figure 2. This is an example of a wrist plate radiograph used in evaluating skeletal age. The wrist plate is of an 8 year and 9 month old female whose skeletal age is 6 years and 3 months.

Figure 3. This is a diagram showing the cephalometric landmarks used in this study.



N
S
PTM
Ar
ANS
Pt. A
PNS
Ii
Is
Go
Pt. B
Pg
Gn

TABLE I

Distribution of cerebral
palsied patients by sex.

	Number
Males	13
Females	13

TABLE II

Medical classification
of the patients.

Classification	Number
Spastic	21
Athetoid	4
Mixed spastic athetoid	1

TABLE III

Classification of the patients according to topographical involvement.

Topography	Number
Left hemiplegia	9
Quadriplegia	7
Right hemiplegia	5
Paraplegia	2
Diplegia	2
Monoplegia	1

TABLE IV

Number of patients showing the following characteristics during swallow. (Some of the patient's swallow exhibit more than one of these characteristics).

Lips passive	7
Orbicularis oris muscle active	19
Mentalis muscle active	7
Tongue interdental	4
Teeth apart swallow	1

TABLE V

Analysis and comparison of the cephalometric data of male cerebral palsied patients with male non-cerebral palsied.

MALES
(Xn - Xcp)†

Age:	7(2)‡	8(2)	11(1)	13(2)	14(2)	15(1)	16(1)	17(1)	18(1)	Over all ages
Variable										
N-Ar(FH)	-2.85	-5.90	7.10	-7.65	6.55	6.70	-3.00	-0.50	6.90	0.797
N-S-Ar	3.95	2.15	2.90	-1.60	9.50	13.70	2.80	5.40	14.30	5.913*
PNS-A(FH)	-1.70	-3.50	-3.50	-2.85	-4.75	-2.10	-3.40	4.00	3.20	-1.623*
Ar-Go	-0.45	-1.70	4.10	-4.60	-1.90	6.30	-6.70	15.10	6.30	1.831
Go-Pg	0.50	-6.20	-3.20	-1.50	-1.95	5.60	-2.30	3.00	2.40	0.018
Ar-Go-Gn	-1.85	-4.35	1.10	-10.85	-1.30	-14.10	-4.20	-15.20	-10.80	-6.850*
ANS-N(FH)	-1.10	-5.40	5.60	-1.95	1.10	-1.40	-3.90	0.80	6.80	0.056
IsANS(⊥FH)	-1.00	0.05	0.50	-2.25	-0.75	2.40	----	-0.10	4.90	0.643
Ii-Gn(⊥FH)	-0.20	-2.10	0.70	-6.10	-1.60	-5.10	-0.90	1.10	2.30	-1.339
A-B(OP)	-0.65	0.00	-1.60	2.45	0.40	2.90	-2.80	-7.90	-2.80	-1.1055

(Continued)

TABLE V (Cont.)

Age:	7(2)	8(2)	11(1)	13(2)	14(2)	15(1)	16(1)	17(1)	18(1)	Over all ages
Variable										
N-A(FH)	-0.30	-1.10	-3.60	2.65	-4.00	-3.60	-6.20	9.00	-4.00	-1.2476
N-B(FH)	1.75	-0.40	-9.20	5.35	-2.85	-4.00	-7.00	11.90	-11.40	-1.7349

† = mean for normal group minus mean for cerebral palsy group
‡ = number in each age group
* = significant at .05 level

TABLE VI

Analysis and comparison of the cephalometric data of female cerebral palsied patients with female non-cerebral palsied.

FEMALES
(Xn - Xcp)[†]

Age:	6(2)[‡]	7(1)	8(1)	9(1)	11(1)	12(2)	14(1)	15(1)	16(1)	17(2)	Over all ages
Variable											
N-Ar(FH)	-0.25	-1.30	-0.20	-1.40	-3.10	2.20	-7.40	-2.20	0.50	-0.65	-1.277
N-S-Ar	12.05	13.20	6.60	4.10	12.10	8.00	7.30	3.80	1.30	2.70	5.642[*]
PNS-A(FH)	-6.20	-7.10	-7.60	-2.90	-10.10	1.00	1.50	-7.00	1.10	-1.60	-3.893
Ar-Go	-3.25	-6.60	2.80	3.60	-9.90	4.20	-5.10	-3.70	5.00	-2.05	-1.502
Go-Pg	-1.45	-7.90	-3.60	-6.40	-5.70	-2.30	-4.80	-2.20	-6.00	0.80	-3.963[**]
Ar-Go-Gn	-7.30	6.70	-9.80	4.20	3.90	-6.20	-11.70	-0.10	-1.90	-2.55	-2.475
ANS-N(FH)	-12.70	3.90	-3.70	-2.20	-5.30	3.25	1.10	-1.30	-4.10	-3.00	-2.143
Is-Ans(⊥FH)	-1.05	-5.10	-0.60	1.20	0.70	0.25	0.00	-1.20	4.90	-1.35	-0.224
Ii-Gn(⊥FH)	-5.00	-8.20	-4.50	-3.70	-2.60	-1.55	-2.70	-5.20	-1.60	-2.00	-3.707[**]
A-B(OP)	-0.40	-2.60	-5.80	0.20	-5.60	-3.35	-3.10	3.70	-0.40	-2.00	-1.9337

(Continued)

TABLE VI (Cont.)

Age:	6(2)	7(1)	8(1)	9(1)	11(1)	12(2)	14(1)	15(1)	16(1)	17(2)	Over all ages
Variable											
N-A(FH)	-2.75	-2.30	2.20	-1.10	-5.30	-0.50	1.40	-5.70	-0.80	-0.75	-1.5406
N-B(FH)	-4.75	-7.10	4.70	-2.80	-10.40	-3.20	-6.20	-5.10	-2.10	0.45	-3.6469

† = mean for normal group minus mean for cerebral palsy group
‡ = number in each age group
*= significant at .05 level
**= significant at .01 level

TABLE VII

Analysis of the data comparing cephalometric measurements of spastic type cerebral palsied patients with non-cerebral palsied.

(Xn - Xcp)†

Variable	Male (n=9)‡	Female (n=12)
N-Ar(FH)	2.519	-1.027
N-S-Ar	6.024*	6.192**
PNS-A(FH)	-1.498	-4.293
Ar-Go	1.123	-1.602
Go-Pg	0.004	-4.063**
Ar-Go-Gn	-4.157	-2.619
ANS-N(FH)	1.528	-1.838
Is-ANS(⊥FH)	1.674	-0.449
Ii-Gn(⊥FH)	0.522	-3.482**
A-B(OP)	-1.3139	-2.3587
N-A(FH)	-3.0532*	-2.1406
N-B(FH)	-4.5683*	-4.9969*

† = mean for normal group minus mean for cerebral palsied group
‡ = number of patients
* = significant at .05 level
** = significant at .01 level

TABLE VIII

Analysis of data comparing cephalometric measurements of cerebral palsied patients whose lips were active during swallow with those whose lips were passive during swallow.

Variable	Passive lips(n=7)† Mean	Active lips(n=19) Mean	t-test	
N-Ar(FH)	82.286	83.053	n.s.‡	
N-S-Ar	120.929	119.026	n.s.	
PNS-A(FH)	50.500	50.053	n.s.	
Ar-Go	39.973	45.378	p < .05*	§ Adjusted for chronological age and skeletal age
Go-Pg	74.633	74.477	n.s.	§ Adjusted for sex and chronological age
Ar-Go-Gn	132.016	126.257	n.s.	§ Adjusted for chronological age and skeletal age
ANS-N(FH)	49.714	50.868	n.s.	
Is-ANS(⊥FH)	25.637	26.891	n.s.	§ Adjusted for sex and skeletal age
Ii-Gn(⊥FH)	40.053	39.191	n.s.	§ Adjusted for sex and chronological age
A-B(OP)	-0.928	1.184	n.s.	
N-A(FH)	-0.724	-0.180	n.s.	§ Adjusted for chronological and skeletal age
N-B(FH)	-2.714	-6.815	n.s.	

† n = number
‡ = no significance
§ = adjusted by method of analysis of covariance
* = significant at .05 level

TABLE IX

Analysis of data to compare the cephalometric measurements of cerebral palsied with interlabial gap with cerebral palsied patients without interlabial gap.

Variable	Inter-labial gap(n=6)† Mean	No inter-labial gap(n=20) Mean	t-test	
N-Ar(FH)	83.667	82.600	n.s.‡	
N-S-Ar	119.250	119.625	n.s.	
PNS-A(FH)	48.250	50.750	n.s.	
Ar-Go	40.911	44.827	n.s.	§ Adjusted for skeletal age
Go-Pg	74.158	74.627	n.s.	§ Adjusted for sex and chronological age
Ar-Go-Gn	130.000	127.150	n.s.	
ANS-N(FH)	52.083	50.100	n.s.	
Is-ANS(⊥FH)	26.446	26.569	n.s.	§ Adjusted for sex and skeletal age
Ii-Gn(⊥FH)	40.716	39.035	n.s.	§ Adjusted for sex and skeletal age
A-B(OP)	1.583	0.325	n.s.	
N-A(FH)	-4.000	0.775	$p < .01$**	
N-B(FH)	-11.500	-3.975	$p < .05$*	

† n = number
‡ = no significance
§ = analyzed by method of analysis of covariance
* = significant at .05 level
** = significant at .01 level

TABLE X

Comparison of cephalometric measurements of cerebral palsied patients who exhibit bruxism with those who do not.

Variable	Bruxism (n=5)† Mean	No bruxism (n=21) Mean	t-test	
N-Ar(FH)	80.194	83.478	n.s.‡	§Adjusted for chronological age
N-S-Ar	117.100	120.119	n.s.	
PNS-A(FH)	46.900	50.952	n.s.	
Ar-Go	40.639	44.705	n.s.	§Adjusted for skeletal age
Go-Pg	73.111	74.854	n.s.	§Adjusted for sex and chronological age
Ar-Go-Gn	128.100	127.738	n.s.	
ANS-N(FH)	48.300	51.095	n.s.	
Is-ANS(⊥FH)	25.699	26.750	n.s.	§Adjusted for sex and skeletal age
Ii-Gn(⊥FH)	38.175	39.720	n.s.	§Adjusted for chronological age
A-B(OP)	2.400	0.190	n.s.	
N-A(FH)	-2.100	0.095	n.s.	
N-B(FH)	-5.600	-5.738	n.s.	

†n = number

‡ = no significance

§ = analyzed by method of analysis of covariance

TABLE XI

Relationship of the upper right first permanent molar to the lower left first permanent molar.

Millimeters	Number of patients
+2.5	2
+2.0	1
+1.0	3
0.0	8
-1.0	1
-1.5	2
-2.0	2
-2.5	1
-4.0	2
-5.0	1
total	23

TABLE XII

Relationship of the upper left first permanent molar to the lower left first permanent molar.

Millimeters	Number of patients
-3.0	1
-2.5	1
-1.5	2
-1.0	1
0.0	7
-1.0	1
-1.5	4
-2.0	4
-2.5	1
total	22

TABLE XIII

Relationship of the upper right cuspid to the lower right cuspid.

Millimeters	Number of patients
+6.0	1
+5.0	3
+4.5	3
+4.0	2
+3.5	1
+3.0	10
+1.5	1
+1.0	1
0.0	1
-1.5	1
-2.0	1
total	24

TABLE XIV

Relationship of the upper left cuspid to the lower left cuspid.

Millimeters	Number of patients
+6.5	1
+5.0	2
+3.5	3
+3.0	5
+2.5	1
+2.0	4
+1.0	2
0.0	2
-2.0	1
total	21

TABLE XV

Analysis and comparison of the molar and cuspid relationship data of the cerebral palsied patients with the molar and cuspid relationship data of Rosenbaum's normal group.

Tooth Relationship	Normal Group (Rosenbaum)		Cerebral Palsy Group		t-test
	n†	Mean	n	Mean	
Right Molar	122	-0.51	23	-0.59	n.s.‡
Left Molar	119	-0.29	22	-0.36	n.s.
Right Cuspid	110	2.03	25	3.00	$p<.01$**
Left Cuspid	115	2.67	21	2.50	n.s.

† = number of patients

‡ = no significance

** = significant at .01 level

TABLE XVI

Analysis and comparison of the cerebral palsied patients' skeletal ages with their chronological ages.

Analysis of Variance

Source of Variation	Degree of Freedom DF	Mean Square MS	F-test	
Sex	1	432.6923		
Subjects in sex	24	4208.6794		
Ages	1	784.6923	4.942	$p < .05$
Sex x ages	1	99.6923	0.628	n.s.
Subjects x ages in sex	24	158.7758		
total	51			

(Mean ages in months)

	Normal Age	Skeletal Age
Male	153.15	142.61
Female	144.61	139.61
Both	148.88	141.11

Difference between mean chronological age and skeletal age of group is significant at .05 level.

DISCUSSION

In this study an effort was made to determine by use of roentographic cephalometrics if the dental and oro-facial complex of cerebral palsied patients is significantly different from that of the non-cerebral palsied.

The data indicates that there is a significant difference in the cranial flexure angle in both male and female cerebral palsied patients. The cranial flexure angle is more acute. Possibly there is a real difference in the cranial base of cerebral palsied patients.

The gonial angle was significantly more obtuse in the male cerebral palsied group but not in the female cerebral palsied group. The obtuse gonial angle might be related to posture. Many cerebral palsied patients tend to have an "open mouth posture." Some investigators have suggested that posture could affect bone resulting in a more obtuse gonial angle.

Table V shows maxillary length [PNS-A(FH)] to be significantly longer in the male cerebral palsied patient. There is a trend for this measurement to be larger from seven to 17 years. This change in trend at age 17 could be due to random variations of serial data and the fact that the sample is small. Table V shows the male mean difference in maxillary length (-1.623mm.) to be significant at .05 level. Table VI shows that the female mean difference in maxillary length [PNS-A(FH)] is -3.893mm.; however, this difference is not significant because there was greater variation of this measurement in the female than in the male. We need larger samples to confirm if there is a significant difference in maxillary length between cerebral palsied patients and non-cerebral palsied patients.

The length of the body of the mandible (Go-Pg) and the lower dental height [Ii-Gn(⊥FH)] were significantly larger in the female cerebral palsied group but not in the male cerebral palsied group. The fact that the mean difference in these measurements is significant in the female but not in the male suggests that the female cerebral palsied patient might grow faster with respect to these two measurements. However, we need larger samples to confirm these findings.

Of the 26 patients, 21 were the spastic type. Data of the male spastic cerebral palsied patients revealed the anterior of the maxilla, point A and mandible, point B, to be positioned further forward in the cerebral palsied group. In the female cerebral palsied patient, only the anterior of the mandible, Point B, was positioned significantly further forward.

Lower dental height was greater in the female spastic cerebral palsied group. These cephalometric measurements should be investigated further using a larger sample.

Those patients whose lips were active during swallow had a longer ramus than those patients whose lips were passive. The longer ramus would tend to increase the lower facial height, and an increase in lower facial height might explain these patients having active lips during swallow. If the lower facial height were greater, lip length might not be great enough to effect lip seal during swallow unless the lips were active.

Comparison of cerebral palsied patients with interlabial gap with those without interlabial gap revealed points A and B to be positioned more posteriorly in those with interlabial gap. One could speculate that if these patients with interlabial gap attempted to keep the lips together, the force on the teeth might retrude the teeth resulting in points A and B being positioned more posteriorly.

Comparison of molar and cuspid relationship measurements with Rosenbaum's[10] normal group revealed no significant differences except in the right cuspid relationship. The lower right cuspid was positioned further forward in relation to the upper right cuspid in the cerebral palsied group. The author cannot explain this result. Measurements of this type alone can give a false impression of the occlusion. Missing teeth, resulting in drifting of teeth, can greatly influence these measurements.

Comparison of the mean measurements which tend to show the relationship of the maxilla and mandible to each other and to cranial base, [A-B(OP)] , [N-A(FH)] and [N-B(FH)], do not show a class II or class III skeletal tendency. One cannot make any valid conclusions on the incidence of skeletal malocclusions on the basis of means since there is so much variation in the facio-skeletal parts that skeletal malocclusions are determined by how well the parts fit together in the individual.

The skeletal ages, determined by wrist plates, of the cerebral palsied patients was significantly less than their chronological ages. The mean difference between skeletal age and chronological age was 7.77 months. It is felt that those skilled in matching wrist plates with wrist plate standards should be able to do this accurately within six months, therefore the significance which can be attached to this finding is highly questionable.

On close observation of the cerebral palsied patients' characteristic of swallow (Table IV), one might speculate that he could find these same findings among a random group of non-cerebral palsied. The fact that 19 patients showed activity of the orbicularis oris muscle during swallow is not very significant since a patient was considered to have an active orbicularis oris if there was only the slightest movement of the lips.

The cephalometer used to radiograph the cerebral palsied group was different from the one used for the non-cerebral palsied group. The B. F. Wehmer cephalometer used for the cerebral palsied had a focal spot to midsagittal plane distance of 60 inches. The cephalometer used for the non-cerebral palsied patients had a focal spot to film distance of 91 inches. The fact that these cephalometers were different results in a greater enlargement of linear measurements on the cerebral palsied patients than in the non-cerebral palsied group.

This was not a purely random sample of cerebral palsied patients since a few patients were excluded for selection when it was decided that they had insufficient neck control to be safely placed in a cephalostat.

Further studies are needed combining the use of roentographic cephalometrics, electromyography and strain gauges. Strain gauges can be used to measure muscle forces. A more complete cephalometric analysis should be done, including measurements of cranial base, mandibular plane and lower dental height. The selection of a random normal sample who would be radiographed on the same cephalometer and who would be subject to the same muscle function examination would make it possible to do a more meaningful study.

SUMMARY AND CONCLUSIONS

Controversy as to the incidence of malocclusion in the cerebral palsied and the effect of abnormal muscle function on the facial skeleton led to the author's interest in this study.

Twenty-six cerebral palsied patients between the ages of seven and 18 were examined. A history was taken for each patient and a data sheet was completed to record molar and cuspid relationships. The function of the tongue and lips during swallow was also recorded.

Cephalometric radiographs were taken and traced for all patients. The following measurements were made: length of cranial base [N-Ar(FH)] , cranial flexure angle (N-S-Ar), length of maxilla [PNS-A(FH)] , length of mandibular ramus (Ar-Go), mandibular body length (Go-Pg), gonial angle (Ar-Go-Gn), upper facial height [ANS-N(⊥FH)] , upper dental height [Is-ANS(⊥FH)], lower dental height [Ii-Gn(⊥FH)] , relation of anterior of the maxilla to the anterior of the mandible [A-B(OP)], relation of the anterior of the maxilla to nasion [N-A(FH)], and the relationship of the mandible to nasion [N-B(FH)].

The cephalometric measurements of the cerebral palsied patients were statistically compared with normative data of a non-cerebral palsied group. The normative data was compiled by the Orthodontic Department of Indiana University from material obtained from the Child Research Council, Denver, Colorado.

Statistical analysis revealed the following differences:

1. The cranial flexure angle (N-S-Ar) was more acute in the cerebral palsied patients.
2. In the male cerebral palsied group the gonial angle (Ar-Go-Gn) was more obtuse.
3. In the male cerebral palsied group, the length of the maxilla [PNS-A(FH)] was greater.
4. In the female cerebral palsied group, the length of the body of the mandible (Ar´-Go) was greater.
5. In the female cerebral palsied group, the lower dental height [Ii-Gn(⊥FH)] was larger.
6. In the male spastic type cerebral palsied group, point A on the maxilla was positioned more anteriorly in relation to nasion [N-A(FH)].
7. In the male spastic type cerebral palsied group, point B on the mandible was positioned more anteriorly in relation to nasion [N-B(FH)].
8. In the female spastic type cerebral palsied group, point B on the mandible was positioned more anteriorly in relation to nasion [N-B(FH)].
9. Analysis of the data comparing the cephalometric measurements of the cerebral palsied patients whose lips were passive during swallow with the cephalometric measurements of the cerebral

palsied patients whose lips were active during swallow revealed that in the group whose lips were active during swallow, the length of the ramus (Ar-Co) was greater.

10. Analysis of the data comparing the cephalometric measurements of the cerebral palsied group with interlabial gap with the cerebral palsied group without interlabial gap revealed the following differences:
    a. In the group with interlabial gap, point A on the maxilla was positioned more posteriorly in relation to nasion [N-A(FH)].
    b. In the group with interlabial gap, point B on the mandible was positioned more posteriorly in relation to nasion [N-B(FH)].

Radiographs were taken of the hands and wrists of the cerebral palsied patients and evaluated to establish skeletal age by making comparisons with the standards of skeletal development in Gruelich's Radiographic Atlas of Skeletal Development of the Hand and Wrist.[28] The difference in the skeletal ages of the patients and their chronological ages was small enough that it could be attributed to error in matching wrist plates.

Although this study showed a significant difference in some cephalometric measurements, there is no evidence in this study that the facial skeleton of cerebral palsied patients deviates markedly from that of the non-cerebral palsied. Also there is no evidence to support the position that there is a greater incidence of malocclusion among cerebral palsied patients.

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ABSTRACT

A Roentographic Cephalometric Analysis

of the Cerebral Palsied Patient

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This study sought to determine if the dental and oro-facial complex of cerebral palsied patients is different from that of the non-cerebral palsied.

Twenty-six cerebral palsied patients between the ages of seven and 18 were examined. A history was taken and a data sheet was completed to record molar and cuspid relationships. The function of the tongue and lips during swallow was also recorded.

Cephalometric and hand and wrist radiographs were taken of each patient. The cephalometric measurements were statistically compared with the normative data of a non-cerebral palsied group. This analysis revealed the cranial flexure angle to be more acute and the gonial angle to be more obtuse.

Comparison of the hand and wrist plates with normal standards did not show wide enough differences to conclude that cerebral palsied patients' skeletal age deviates from the chronological age.

Although there was a difference in some cephalometric measurements, there is no evidence in this study to show that the incidence of malocclusion and the incidence of factors that contribute to malocclusion are found with greater frequency among cerebral palsied patients.