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Craniofacial development in obese adolescents

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SUMMARY The purpose of this study was to investigate craniofacial morphology in obese adolescents and to compare the morphological data with those of normal adolescents.

The study was based on measurements of lateral cephalometric roentgenograms of adolescents who had been diagnosed as obese. Linear and angular measurements were obtained from cephalometric tracings of 27 females (mean age 15.6 ± 0.83 years) and 23 males (mean age 13.9 ± 0.98 years). The data were compared with corresponding measurements of gender and age matched controls.

The results showed that both males and females in the obesity group exhibited significantly larger mandibular and maxillary dimensions than the controls. On average, mandibular length (Cd–Pgn) was 10 mm greater in males and 8 mm greater in females. Maxillary length (Pm–A) was 3.5 mm greater in males and 3 mm greater in females. When considering vertical dimensions, lower anterior (Ans–Gn) and posterior (S–Go) face height were 4 and 5 mm greater in the obese males, respectively, while in the obese females both these distances were 4 mm greater compared with the controls. Both maxillary (SNA) and mandibular (SNB, SNPg) prognathism were more pronounced in the obesity group than in the control group. This also influenced the average soft tissue profile, which was less convex in the obesity groups. The mandibular plane angle (ML/SN) was smaller in the obesity group than in the control group.

Craniofacial morphology differs between obese and normal adolescents. In general, obesity was associated with bimaxillary prognathism and relatively greater facial measurements.

Introduction

Obesity constitutes a significant public health problem in affluent countries. It is usually defined as an excess of body fat, and may considerably impair the individual's quality of life (Kiess *et al*., 2004). Furthermore, studies have linked obesity to an increased risk of type-2 diabetes mellitus, hypertension, cardiovascular disease, dyslipidaemia, and certain types of cancer (Pi-Sunyer, 1993). Recent surveys have found that 54.9 per cent of United States adults and 20 per cent of Swedish adolescents are overweight or obese (Kuczmarski *et al.*, 1997; Marcus *et al.*, 2004). The prevalence of obesity in children is emphasized by the results of an epidemiological study which shows a clear upward trend in body weight that is equivalent to a 0.2 kg increase in body weight/year at any given age (Freedman *et al*., 1997).

In recent years, investigators have begun to use the body mass index (BMI) to measure whether a subject is overweight. This is calculated as the weight in kilograms divided by the square of height in metres: weight (kg)/[height (m^2)]. Overweight is defined as a BMI of 25.0 to 29.9, while obesity is a BMI of 30 or greater (World Health Organization, 1997; National Heart, Lung, and Blood Institute, 1998).

Human body weight and the level of fat accumulation is influenced by multiple interrelated factors, including health status, basal metabolism, diet, physical exercise, hormonal balance, race, and heredity (Simopoulos, 1987).

As regards heredity, studies have suggested that body weight is subject to a substantial genetic control, which accounts for approximately one-third of the variation in BMI (Bouchard, 1997). Genetic influences appear to contribute to differences among individuals in resting metabolic rate (Rice *et al*., 1996), body fat distribution (Bouchard *et al*., 1998), and weight gain in response to overfeeding (Bouchard *et al.*, 1990).

Growth hormone (GH) secretion is significantly reduced in obese patients (Veldhuis *et al*., 1991; Strobl and Thomas, 1994; Thissen *et al*., 1994; Nam *et al*., 1997). A variety of factors appear to be associated with this phenomenon, although the exact reason for the reduction is not known. Nevertheless, despite their low GH secretion and the potential abnormalities in the peripheral GH–insulin-like growth factor (IGF) system (Nam and Marcus, 2000; Kamel *et al.,* 2004) prepubertal obese children generally exhibit normal or increased height (De Simone *et al*., 1995). Furthermore, it has been suggested that hyperinsulinaemia (Zannolli *et al*., 1993), a low level of IGF-binding protein-1 (IGFBP-1) (Conover *et al*., 1992; Nam *et al*., 1997), and a high level of free IGF-1 (fIGF-1) (Frystyk *et al*., 1995; Nam *et al*., 1997) might also be of importance in this context.

The mechanisms which regulate craniofacial growth and development are complex and include interactions between genes, hormones, nutrients, and epigenetic factors that will give the craniofacial bones their final morphology. Disturbances in any of these mechanisms may result in an

aberrant growth pattern (Van Limborgh, 1982; Thilander, 1995). Cephalometric studies have demonstrated that patients with GH deficiency exhibit a small anterior and posterior cranial base size, a small posterior face height and a small posterior mandibular height (Spiegel *et al*., 1971; Poole *et al*., 1982; Pirinen *et al*., 1994).

In a recent study, increased craniofacial growth was found in obese adolescents (Öhrn *et al*., 2002). However, only a few articles have been published on this subject and further knowledge in the field is required. The purpose of this study was to investigate craniofacial morphology in obese adolescents and to compare the morphological data with those of normal adolescents.

Materials and methods

The study was based on measurements taken from lateral cephalometric roentgenograms of obese adolescents who had been consecutively referred from the National Childhood Obesity Centre, Karolinska University Hospital in Huddinge, to the Department of Paediatric Dentistry, Karolinska Institute for a dental check-up. Fifty subjects, 27 females (mean age 15.6 ± 0.83 years) and 23 males (mean age 13.8 ± 0.98 years), were included in the study.

Lateral head films were obtained from the paediatric files of these subjects. The craniofacial variables evaluated were based on the cephalometric reference points and lines shown in Figures 1 and 2. The data were compared with the corresponding measurements recorded in 16-year-old normal controls (Bhatia and Leighton, 1993).

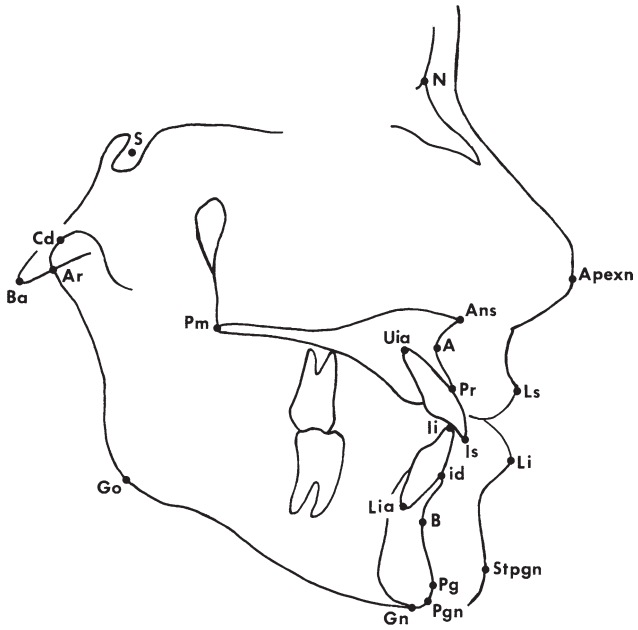

Figure 1 Cephalometric reference points employed in the analysis. A: subnasale, Ans: anterior nasal spine, Apexn: apex nasale, Ar: articulare, B: supramentale, Ba: basion, Cd: condylion, Gn: gnathion, Go: gonion, id: infradentale, Ii: lower incisor edge, Is: upper incisor edge, Lia: lower incisor apex, Li: laberale inferius, Ls: laberale superius, N: nasion, Pg: pogonion, Pgn: prognathion, Pm: posterior nasal spine, Pr: prosthion, S: sella, Stpgn: soft tissue pogonion, Uia: upper incisor apex.

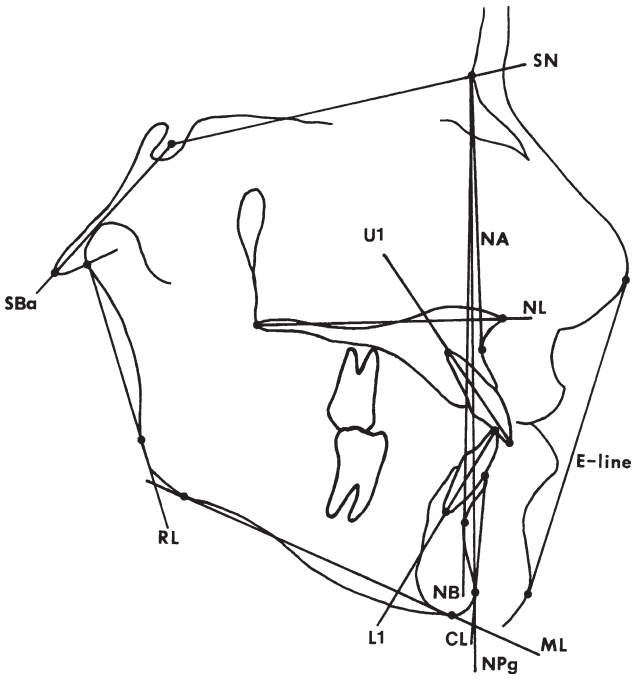

Figure 2 Cephalometric reference lines employed in the analysis. CL: chin line, E-line: aesthetic line, L1: long axis of lower incisors, ML: mandibular line, NA: nasion–subnasale line, NB: nasion–supramentale line, NL: nasal line, NPg: nasion–pogonion line, SBa: sella–basion line, SN: sella–nasion line, RL: ramus line, U1: long axis of upper incisors.

All the measurements were made with an electronic digitizer on-line with a microcomputer. The resolution of the instrument was 0.1 mm and 0.1 degree. The linear and angular variables listed in Tables 1 and 2 were measured. The linear measurements were corrected for the radiographic magnification.

Ethical approval

The investigation was approved by the ethical committee of Karolinska Institutet and Huddinge University Hospital (192/99).

Statistics

In order to determine the reliability of the method, 15 randomly selected radiograms were traced and measured twice with a two-week interval. The error of the method (S_i) was calculated using the formula: $S_i = \sqrt{(\sum d^2/2n)}$, where d is the difference between the first and second measurments and n is the number of double determinations.

The greatest error was found to be 0.85 degrees (L1/ML) for angular and 0.52 mm (S–Pm) for linear measurements.

Mean values and standard deviations were computed for all variables. The level of statistical significance of differences between the mean values obtained from the

experimental and control groups were evaluated with the Student's *t*-test for independent samples.

Results

Both males and females in the obesity group exhibited significantly greater mandibular and maxillary dimensions than the controls (Tables 1 and 2).

In the obesity group anterior cranial base length (SN) was significantly larger in both males ($P < 0.05$) and females ($P < 0.01$) compared with the controls. The average mandibular length (Cd–Pgn) was 9.7 mm greater in males ($P < 0.001$) and 7.5 mm greater in females ($P < 0.001$). Corpus length (Go–Pg) was more than 3 mm greater in the obese children than in the controls ($P < 0.001$ in males, $P < 0.01$ in females). Maxillary length (Pm–A) was 3.5 mm greater in obese males and 3.0 mm greater in obese females ($P < 0.001$). The comparatively greater jaw dimensions recorded in the obese groups were also reflected in significantly ($P < 0.001$) larger values of jaw prognathism (SNA, SNB, SNPg).

As regards vertical dimensions, lower anterior (Ans–Gn) and posterior (S–Go) face height was 4.0 mm ($P < 0.01$) and 4.9 mm ($P < 0.001$) greater in males, and 3.8 mm ($P < 0.01$) and 4.0 mm ($P < 0.001$) greater in females compared with the controls. The mandibular plane angle (ML/SN) was smaller in both the male ($P < 0.01$) and the female ($P < 0.05$) obese groups. However, the maxillary plane angle (NL/SN) only exhibited a reduced value ($P < 0.01$) in the female obese group. Increased proclination of the upper (U1/NL, $P < 0.01$) and lower (L1/ML, $P < 0.001$) incisors was found in the female obese group, who also exhibited a marked mandibular alveolar prognathism (ML/CL, $P < 0.001$). The maxillary dentoalveolar height was also increased in both obese groups (ANS–Pr, $P < 0.05$).

As regards the facial soft tissue, the individuals in the obese groups were characterized by comparatively straight profiles (Convex, $P < 0.001$).

Discussion

Although obese children in general have a decreased level of GH (Veldhuis *et al*., 1991; Strobl and Thomas, 1994; Thissen *et al*., 1994; Nam *et al*., 1997), the present findings show comparatively greater craniofacial dimensions in obese adolescents. This is in agreement with a previous study (Öhrn *et al*., 2002), but in contrast to the findings and the belief that a decrease in GH level will negatively affect

Table 1 Linear (mm) and angular (degrees) craniofacial measurements in 23 obese males and 51 controls.

	Obesity ($n = 23$)		Control ($n = 51$)		Difference
	Mean	SD	Mean	SD	
Age	13.9	0.98	14.0	†	
Angular (degrees)					
SNA	85.0	2.6	80.7	4.1	+4.3***
SNB	82.4	3.1	77.6	3.9	+4.8***
SNPg	83.0	3.1	79.0	4.1	+4.0***
SNBa	131.1	5.5	130.1	5.3	+1.0
NL/SN	6.0	2.9	6.9	3.0	–0.9
ML/SN	28.8	4.7	32.8	5.9	–4.0**
ML/NL	22.8	4.7	25.9	5.8	–3.1*
RL/ML	125.8	5.4	125.0	5.0	+0.8
U1/NL	110.7	5.8	109.4	7.7	+1.3
L1/ML	91.3	8.5	91.8	7.4	–0.5
ML/CL	70.9	7.3	68.6	7.5	+2.3
CONVEX	172.1	7.2	159.9	6.0	12.2***
Linear (mm)					
S–N	70.3	3.1	68.6	2.9	+1.7*
Pm–A	50.2	2.6	46.7	2.7	+3.5***
S–Pm	47.0	3.4	47.3	2.1	–0.3
Cd–Pgn	120.4	6.2	110.7	5.1	+9.7***
Go–Pg	75.9	3.6	72.3	3.6	+3.6***
N–Gn	114.8	6.8	113.6	6.4	+1.2
N–Ans	50.2	3.2	51.5	2.5	–1.3
Ans–Gn	67.3	5.6	63.3	5.1	+4.0**
S–Go	77.2	4.4	72.3	4.7	+4.9***
Ans–Pr	16.7	2.4	15.1	2.3	+1.6*
Ls–E-line	–3.7	2.2	–3.1	1.8	–0.6
Li–E-line	–3.4	3.2	–2.4	1.7	–1.0

SD, standard deviation.
***$P < 0.001$, **$P < 0.01$, *$P < 0.05$.
†Not available. However, considering the method of registration that has been used the SD should be very small.

Table 2 Linear (mm) and angular (degrees) craniofacial measurements in 27 obese females and 53 controls.

	Obesity (n = 27)		Control (n = 53)		Difference
	Mean	SD	Mean	SD	
Age	15.6	0.83	16.00	†	
Angular (degrees)					
SNA	84.4	3.4	80.0	3.9	+4.4***
SNB	82.4	3.6	78.2	3.9	+4.2***
SNPg	83.0	3.5	79.7	4.3	+3.3***
SNBa	133.1	5.0	131.6	4.7	+1.5
NL/SN	6.0	2.4	8.0	4.0	–2.0**
ML/SN	28.5	6.1	31.8	6.6	–3.3*
ML/NL	22.5	5.4	23.8	6.1	–1.3
RL/ML	124.1	6.7	123.8	5.2	+0.3
U1/NL	114.5	7.1	109.4	6.9	+5.1**
L1/ML	95.4	7.1	89.4	6.9	+6.0***
ML/CL	74.3	5.9	67.6	6.6	+6.7***
CONVEX	173.0	5.9	163.0	6.0	+10.0***
Linear (mm)					
S–N	68.4	2.8	66.4	2.3	+2.0**
Pm–A	48.2	2.4	45.2	1.9	+3.0***
S–Pm	45.7	3.0	45.1	2.6	+0.6
Cd–Pgn	116.9	6.2	109.4	4.0	+7.5***
Go–Pg	74.0	4.9	70.9	3.8	+3.1**
N–Gn	111.9	6.2	110.8	6.0	+1.1
N–Ans	48.7	2.6	50.0	2.6	–1.3*
Ans–Gn	65.6	5.4	61.8	5.3	+3.8**
S–Go	75.4	5.0	71.4	4.6	+4.0***
Ans–Pr	16.5	2.8	14.9	2.8	+1.6*
Ls–E-line	–4.1	3.0	–4.9	2.5	+0.8
Li–E-line	–2.6	3.3	–3.4	2.1	+0.8

SD, standard deviation.
***$P < 0.001$, **$P < 0.01$, *$P < 0.05$.
†Not available. However, considering the method of registration that has been used the SD should be very small.

growth of the craniofacial components (Spiegel *et al*., 1971; Pirinen *et al*., 1994).

In the study by Öhrn *et al*. (2002), the obesity group was smaller and the control group consisted of patients with malocclusions of varying severity, who had been referred to an orthodontic department. Furthermore, some children in that control group had already been treated orthodontically. Consequently, that group could not be regarded as a true representation of the population. Finding an appropriate control group is a dilemma in many orthodontic cephalometric projects. Ethical considerations prohibit radiographic exposure of non-patients. For this reason the existence of longitudinal cephalometric growth data from normal populations, which can serve as control material, is limited.

In the present investigation, the control data originated from an extensive longitudinal cephalometric growth study of normal subjects (Bhatia and Leighton, 1993). The data were derived from the records of subjects who had participated in a growth study at King's College School of Medicine and Dentistry, London. The children were recorded at birth, six months of age, and then annually thereafter. None exhibited any gross or congenital abnormalities of development. The radiographic registrations had been carried out between 1952 and 1993. This means that the control and patient material used in the present study were collected at different periods of time. Furthermore, since the control and study materials originate from different population bases, there is a possibility that the recorded dimensional and angular differences between the groups could be due to morphological differences that have been shown to exist between Caucasian samples (Kerr and Ford, 1986). Such differences are small, however, and there is no reason to believe they affected the present results to any significant degree.

The general impression of the present data is that the obese adolescents demonstrated a more advanced dentofacial development than control subjects of a corresponding age. It can be concluded, therefore, that obese individuals have increased growth activity in comparison with age matched individuals of normal constitution. The growth stimulation seemed to have affected all of the separate components of the dentofacial complex to some extent. Although the dentofacial dimensions were comparatively greater in the obese subjects, the facial proportions did not show any pronounced deviation from normality.

The differences recorded between the obese and control group were greater numerically in the male than in the

female group. However, the direction of the deviations from normal values and the significance levels were very similar, with only three exceptions. The obese females exhibited smaller maxillary inclination, greater incisor inclination and a larger lower alveolar prognathism than the controls. These differences from normal values did not exist in the obese male group.

A clear facial prognathism was found in both the male and female obese groups. Some studies indicate that facial prognathism of the jaws is associated with a smaller than average cranial base angle (Björk, 1955; Kerr and Adams, 1988; Enlow and Hans, 1996). In this study, however, the cranial base angle did not differ from the normal values in the obese group. The marked facial prognathism in the obese group, therefore, is most likely due to the comparatively large maxillary (Pm–A) and mandibular (Cd–Pgn) lengths which were recorded in the obese individuals.

In the vertical plane it was interesting to note that posterior face height (S–Go) in the obese adolescents was significantly greater than the corresponding dimension in the control group, whereas anterior face height did not differ between the groups. These results indicate that the increased growth activity which characterizes the obese individuals expresses itself to a greater extent in the posterior than in the anterior facial skeletal components. Furthermore, upper posterior face height (S–Pm) did not differ significantly between the obese and control group. It seems, therefore, that it is mainly growth events in the lower posterior face which are responsible for the increased S–Go dimension in the obese groups. Important areas of growth in this respect are the mandibular condyles and the alveolar processes.

The mean values of the inclination of the maxilla and mandible in the female obese group were smaller than the corresponding values in the controls. As the deviation from normal values was of similar dimension and in the same direction, the resulting effect on the vertical relationship (ML/NL) was minimal. In the obese males, on the other hand, only the mandible exhibited an anterior rotation whereas the inclination of the maxilla was normal. Consequently, in comparison with the controls the vertical relationship in the obese males was relatively deep.

With respect to incisor inclination (U1/NL, L1/ML) and lower alveolar prognathism (ML/CL), there was a distinct difference between the genders. In the male subjects none of these variables differed between the obese and control groups. In the female groups, however, the mean values recorded in the obese individuals were considerably greater than in the controls. Incisor proclination often appears in cases of dental crowding or tongue habits, for example, but the existence of such factors was not recorded in this study. It cannot be determined, therefore, whether or not factors of this kind influenced the dentition of the obese females to a greater extent than the dentitions of the other subjects.

The soft tissue profile was straighter in the two obese groups. This is a consequence of the relatively large mandibular length and the more anterior position of pogonion. In spite of the pronounced chin in the obese groups, the relationship of the lips to the E-line was normal. The normal relationship of the lips to the E-line found in the obese group can be explained in the female group by the more proclined upper and lower incisors.

In a previous study, the relationship between BMI and craniofacial characteristics (Paoli *et al*., 2001) was examined in patients with obstructive sleep apnoea (OSA). Eighty-five men with OSA were divided into two groups according to their BMI (less than 30 or greater than 30). Patients with a BMI below 30 had a shorter anterior cranial base, a smaller mandible and retroposition of the mandible compared with severely obese patients. These results are in agreement with the present findings.

Cephalometric studies have demonstrated that patients with GH deficiency exhibit a smaller anterior and posterior cranial base, small posterior facial height, and small posterior mandibular height (Spiegel *et al*., 1971; Poole *et al*., 1982; Pirinen *et al*., 1994). These findings indicate that factors other than the level of GH may be the cause of the increased craniofacial dimension found in obese adolescents. Hyperinsulinaemia (Zannolli *et al*., 1993) and the high level of fIGF-1 (Frystyk *et al*., 1995; Nam *et al*., 1997) may be such growth stimulating factors. The theory that craniofacial growth may be more dependent on free circulating IGF-1 rather than on the level of GH seems attractive in this context. In a recent study of rats, GH receptors were detected in various components of the temporomandibular joint (TMJ), but not in the fibrous articular surfaces or in the cartilage layers of the condyle. IGF-1 receptors, on the other hand, were found in the fibrous articular surface and particularly in the superior and posterior regions of the condylar cartilage. It seems, therefore, that the expression of GH and IGF-1 receptors is area specific in the TMJ of these animals (Visnapuu *et al*., 2001). Providing the same conditions prevail in humans, the greater mandibular dimensions recorded in the present obese adolescents could be explained by the relatively high level of IGF-1 found in these subjects.

In the field of orthodontics the differences between obese and normal adolescents may be of interest from a clinical point of view. Cephalometric standards, which have been obtained from samples of normal children, are frequently used in the process of orthodontic diagnosis and treatment planning. Taking the present results into consideration, however, it may be necessary to apply somewhat modified treatment goals in obese orthodontic patients.

Conclusion

Craniofacial morphology differs between obese and normal adolescents. In general obesity was associated with bimaxillary prognathism and relatively larger facial dimensions.

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