Skeletal maturity, dental maturity, and eruption in young patients with Turner syndrome

Marit Midtbø and Agnar Halse
Department of Orthodontics and Facial Orthopedics and Department of Oral Radiology, School of Dentistry, University of Bergen, Bergen, Norway

Midtbø M, Halse A. Skeletal and dental development in patients with Turner syndrome. Acta Odontol Scand 1992;50:303–312. Oslo. ISSN 0001–6357.

A major problem for patients with Turner syndrome is their small body height. The rapid biotechnologic development has now made treatment with growth hormone possible at a larger scale. The aim of this investigation was to evaluate skeletal maturity, dental maturity, and eruption in a group of young patients before hormone therapy. The material comprised 33 patients aged 7–16.7 years. The skeletal maturity, as judged from hand radiographs, was on an average 2.3 years retarded ($p < 0.001$) and showed increasing retardation with increasing age. The dental maturity, assessed from the formation stages of the permanent teeth on panoramic radiographs, was accelerated, with a mean value of 1 year ($p < 0.001$). The timing of clinical eruption did not differ significantly from that of our reference material; the Turner girls were on an average 3.7 months ahead. Several patients had local eruption problems, especially in the maxillary lateral segments. It is suggested that disharmony between tooth size and arch size may contribute to this problem. □ *Age determination by teeth; age determination by skeleton; tooth eruption; X-chromosome*

Marit Midtbø, School of Dentistry, Årstadveien 17, N-5009 Bergen, Norway

In 1938 Turner (1) described seven phenotypic females who had dwarfism, sexual infantilism, webbing of the neck, and cubitus valgus. No obvious cause of the disturbance was observed. The etiology was disclosed by Ford et al. (2), who reported that the karyotype in a 14-year-old phenotypic female with this syndrome was 45X. The partial or total absence of a second sex chromosome (X chromosome monosomy) is at present considered to be characterized by four cardinal features: 1) female phenotype; 2) short stature; 3) sexual infantilism owing to rudimentary gonads; and 4) various somatic abnormalities. Any of these features may be modified by the degree of sex chromosome deficiency. It is therefore useful to consider this syndrome of gonadal dysgenesis and its variants as a continuum of clinical features ranging from those of the typical 45X phenotype to a normal individual.

Among the somatic abnormalities are delayed skeletal maturity and deviations in skeletal morphology, especially prevalent in the hands, arms, legs, and neck but also seen in the jaws and in the basal areas of the skull (3–5). The anterioposterior dimension of the alveolar arch of the mandible is less than in normal girls (3). Laine & Alvesalo (6) have reported that the alveolar arch of the mandible is broader and shorter, both absolutely and especially in relation to the maxilla, where the most predominant finding is a narrow arch (3, 7).

The first extensive dental examination of Turner patients was performed by Filipsson et al. (8), who reported a tendency for early eruption of the teeth. This has later been verified by Lindsten et al. (9) and Ogiuchi et al. (10). Kari & Alvesalo (11) evaluated both the development and eruption of the permanent teeth and found that the 45X girls were clearly earlier than the normal control girls.

The rapid biotechnologic development has now made possible the production of growth hormone on a larger scale and at a more reasonable price. The immunologic aspects of treatment with growth hormone are also less problematic than earlier. The Department of Paediatrics, University of Bergen,

has started hormone treatment of a group of young Turner patients. Since a series of biologic variables are affected in such patients, and treatment may be conceived to influence these in various manners, a thorough supervision of the patients through the treatment period is desirable. The present paper evaluates the skeletal maturity, the state of tooth eruption, and tooth development at the start of the treatment with growth hormone and estrogen.

Materials and methods

This investigation is part of a systematic study of Turner patients whose intention is to evaluate growth and development before, during, and after therapy with growth hormone and estrogen. The karyotyping, the hormone therapy, and the study of general variables are performed by the Department of Paediatrics, University of Bergen.

The present material consisted of 33 patients, aged 7–16.7 years (Table 1) and living in different parts of Norway. Their karyotypes were 45X ($n = 23$), 45X/46XX ($n = 3$), 46X,i(Xq) ($n = 3$), 45X/46X,r(Xq) ($n = 1$), 45X/46X,i(Xq) ($n = 1$), and 45X/46XY ($n = 1$). One patient was not karyotyped. Before the start of hormone treatment the following recordings were made at the Departments of Oral Radiology and Orthodontics, School of Dentistry, University of Bergen: 1) Five intraoral photographs: one frontal view, occlusal views of the mandibular and maxillary arches, and lateral views of the right and left side in habitual occlusion; 2) Plaster models; 3) Radiograph of the left hand; 4) Orthopantomogram; and 5) A full series of intraoral radiographs.

Table 1. Turner patients distributed by age

Age (years)	No. of patients
7.1–9	5
9.1–11	8
11.1–13	8
13.1–15	6
15.1–17	6
Total	33

Skeletal maturity

The skeletal maturity was assessed independently by three examiners. Radiographs of the left hand were compared with the standard of Greulich & Pyle (12), and the majority decision was used. In accordance with the standard both the skeletal age and the chronologic age were given in years and months. Subsequently, the values were converted into years with two decimals. The assessments of the three examiners were identical in 24 patients. In six patients there was agreement between two of the observers. Disagreement never exceeded 1 year. The assessment of three patients was difficult, mainly owing to malformations. In these patients the examiners together decided the skeletal age.

Dental maturity

The maturity registration was done on the orthopantomograms. The seven left mandibular teeth were rated on an eight-stage scale by the method of Demirjian et al. (13). One patient had hypodontia of the second molar on the left side, and the corresponding tooth on the opposite side was classified. One patient had no orthopantomogram, and the periapical pictures were used. Each of the assessed maturity stages was given a certain score. The sum of the scores for each individual was converted into a dental age, expressed in years in accordance with the instructions given by Demirjian & Goldstein (14) and using magnified photographs of the median curves. In the classification 0.1-year steps were used. The scoring was done by three observers, and the majority decision was used. At least two of the examiners agreed on every assessment. Disagreement never exceeded one maturity stage. In 95% of the assessments the ratings were identical. As reference material the French-Canadian sample of normal girls presented by Demirjian & Goldstein (14) was used.

Clinical eruption

A tooth was considered erupted when a

part of it had pierced the oral mucosa. The registration of clinical eruption was done separately on models and on intraoral slides and was performed by two observers. The results for the two methods of registration and by both observers were identical for all the patients. The number of erupted teeth was converted into a dental age expressed in years in accordance with the method of Hägg & Taranger (15), whose material was also used as a reference standard. The dental development of our sample can be expressed as the difference between dental age and chronologic age. The information can also be presented in terms of standard deviation scores, relating the dental development to a frequency distribution characterizing a normal sample. Both these methods were used.

Local eruption disturbances were also recorded.

Statistics

The difference between skeletal age on the basis of hand radiographs and chronologic age was compared statistically by means of Student's paired *t* test.

The difference between dental age on the basis of maturity scores and chronologic age was also tested by Student's paired *t* test. Three patients were not included in the calculation because their chronologic age exceeded the age at which all teeth have normally completed root development. The difference between dental age on the basis of tooth counts and chronologic age was compared statistically by means of Wilcoxon sign rank test. Sixteen patients were not included. The Mann–Whitney test was used to test the differences in skeletal and dental maturity between the 45X karyotypes ($n = 23$) and the nine patients with mosaics and isochromosome karyotypes. One patient with unknown karyotype was not included.

All statistical evaluations were performed by means of a computer program (16).

Results

Skeletal maturity

The skeletal maturity was found to be significantly retarded ($p < 0.001$, paired *t* test) according to the American standard of Greulich & Pyle (12). The mean retardation for the whole group of Turner patients was 2.30 years; the minimum was 0.17 years, and the maximum retardation 4.17 years. The minimum and maximum retardations were found in the youngest and oldest girl, respectively (7 and 16.67 years old). Virtually all the patients had retarded skeletal development. The results are illustrated in Fig. 1. No significant differences were found between the 45X patients ($n = 23$) and the nine patients with mosaics and isochromosome karyotypes.

Dental maturity

The difference between chronologic and dental age are given in Table 2. The mean difference was 1 year ($p < 0.001$, paired *t* test), which means that the dental maturity of the Turner patients was 1 year advanced compared with the French-Canadian reference material of Demirjian & Goldstein (14).

The maximum acceleration was 3.5 years

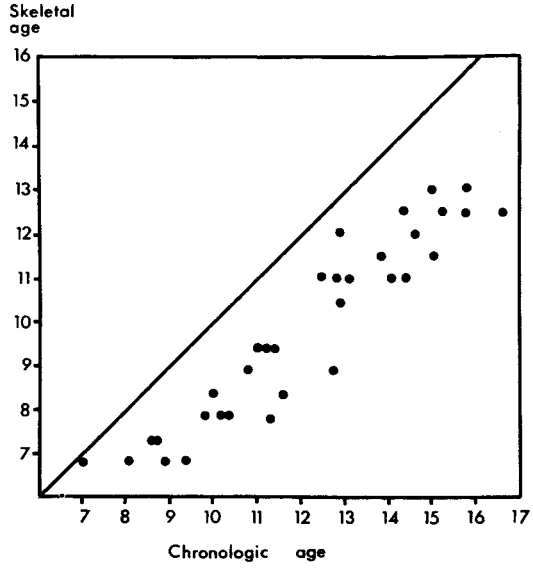

Fig. 1. Skeletal maturity of young patients with Turner syndrome.

Table 2. Comparison of chronologic and dental age on the basis of maturity scores in Turner patients aged 7–16.67 years. The mean difference, 0.99 years, is significant ($p < 0.001$, paired t test)

Patient no.	Chronologic age (years)	Maturity scores	Dental age (years)	Difference
1	7.00	82.1	8.5	1.50
2	8.08	79.9	8.3	0.22
3	8.67	78.4	8.1	−0.57
4	8.75	82.2	8.5	−0.25
5	8.92	97.3	12.1	3.18
6	9.42	90.4	9.8	0.38
7	9.92	96.8	11.8	1.88
8	10.00	92.4	10.2	1.20
9	10.17	90.7	9.9	−0.27
10	10.33	98.0	12.7	2.37
11	10.67	97.7	12.4	1.73
12	10.92	98.4	13.0	2.08
13	11.00	96.8	11.8	0.80
14	11.17	92.1	10.2	−0.97
15	11.33	95.1	11.0	−0.33
16	11.50	99.5	15.0	3.50
17	12.50	97.7	12.4	−0.10
18	12.75	100.0	15.5	2.75
19	12.83	100.0	15.5	2.67
20	12.83	99.5	15.0	2.17
21	12.92	100.0	15.5	2.58
22	13.08	100.0	15.5	2.42
23	13.92	100.0	15.5	1.58
24	14.08	99.5	15.0	0.92
25	14.42	100.0	15.5	1.08
26	14.42	100.0	15.5	1.08
27	14.67	100.0	15.5	0.83
28	15.08	100.0	15.5	0.42
29	15.08	100.0	15.5	0.42
30	15.25	100.0	15.5	0.25
31	15.75	100.0	15.5	
32	15.83	100.0	15.5	
33	16.67	100.0	15.5	

Patients 31–33 were not included in the statistical calculation.

and occurred in an 11.5-year-old patient. Nine patients were more than 2.1 years advanced, and in addition, eight patients were 1–2 years ahead of the reference material. One patient was retarded about 1 year in her dental development, and five other patients showed dental development retarded from 1 to 6 months.

The dental maturity scores for the Turner patients plotted on the dental maturity percentiles of the reference group are shown in Fig. 2. As illustrated, most of the patients had a dental maturity comparable with the 90 and 97 percentiles of the reference material, and only one patient was situated on the 10 percentile. No significant differences were found between the various karyotypes.

Clinical eruption

The number of erupted teeth and the dental age assessed from the state of eruption in the 17 patients aged 7–12.5 years are shown in Table 3. The mean difference between the dental age and the chronologic age was 3.7 months ($p > 0.05$, Wilcoxon test). This means that the clinical eruption of the permanent teeth in this sample was 3.7 months accelerated compared with normal girls in the Swedish reference group.

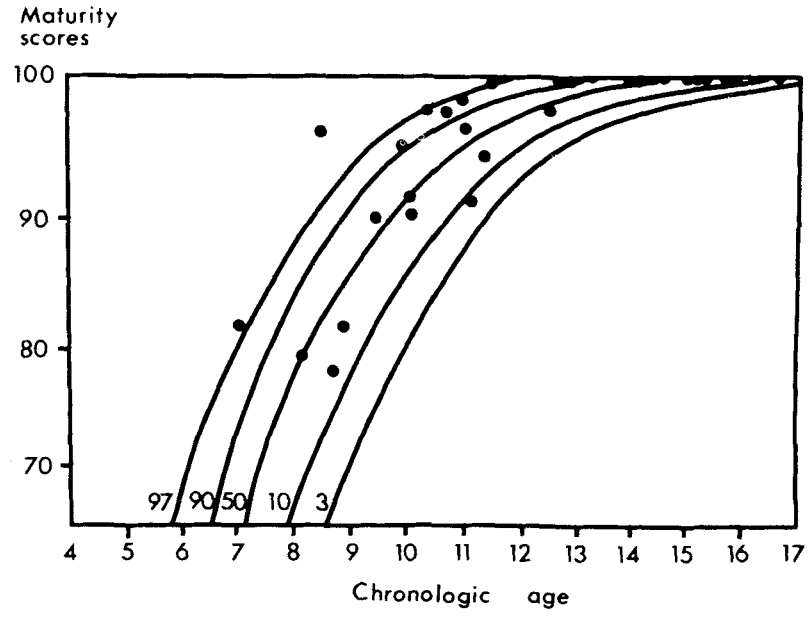

Fig. 2. Dental maturity scores of Turner patients, plotted against dental maturity percentiles of the reference group of Demirjan & Goldstein (14). Patients 15.5 years or more are not included.

The dental development of the patients, expressed as the difference between the dental age and the chronologic age, showed a wide range. The extremes were one girl of 8.92 years who was 1.81 years accelerated and a girl aged 11.33 years showing 1.68 years' retardation compared with the normal girls.

The dental development of the Turner patients expressed in terms of standard deviation scores is shown in Fig. 3. Ten patients were about 1 SD accelerated and five were about −1 SD delayed compared with the reference standard (15). No significant differences were found between the various karyotypes.

Table 3. *Number of erupted teeth and corresponding dental age in Turner patients aged 7–12.5 years*

Patient no.	Chronologic age (years)	Erupted teeth (*n*)	Dental age (years)	Difference
1	7.00	11	7.89	0.89
2	8.08	10	7.50	−0.58
3	8.67	10	7.50	−1.17
4	8.75	12	8.59	−0.61
5	8.92	20	10.73	1.81
6	9.42	17	10.35	0.93
7	9.92	23	11.27	1.35
8	10.00	20	10.73	0.73
9	10.17	13	9.33	−0.84
10	10.33	24	11.54	1.21
11	10.67	26	12.08	1.41
12	10.92	27	12.40	1.48
13	11.00	24	11.54	0.54
14	11.17	15	9.93	−1.24
15	11.33	14	9.65	−1.68
16	11.50	26	12.08	1.41
17	12.50	28	12.50	0.00

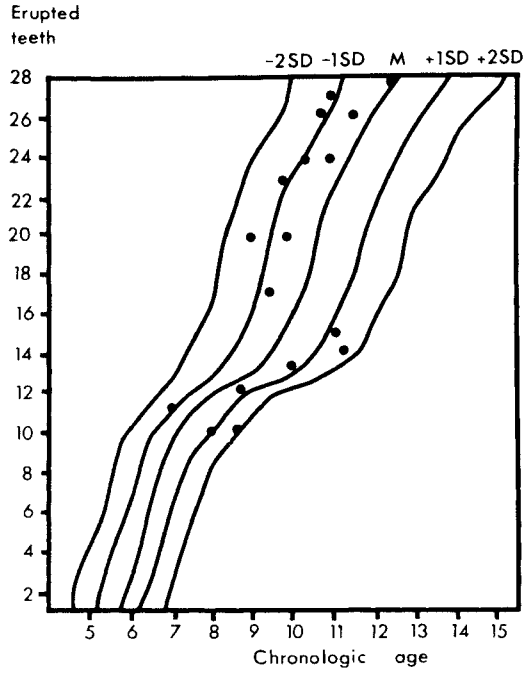

Fig. 3. Number of erupted teeth in Turner patients aged 7–12.5 years compared with the mean and standard deviation scores of the reference group of Hägg & Taranger (15).

Local eruption deviations

Several patients showed local eruption deviations. Since these were assumed to be associated with the syndrome, they will be reported in some detail.

In one patient aged 12.8 years with karyotype 46X,i(X_q) all the permanent teeth were erupted except the maxillary second molars. These showed nearly full root development and were positioned with their crown against the distal root of the first molar and the root extending in a distal direction (Fig. 4A). The third molars were in the tuber region close to the apex of the root of the second molar. As illustrated, the second molar could not erupt without intervention.

In another patient aged 9.9 years with karyotype 45X, 23 teeth were erupted. The maxillary second deciduous molars persisted and showed poor root resorption (Fig. 4B). Both the second premolars were in close contact with the root of the first premolar; the crown was distally positioned and the root mesially. The direction of eruption of these teeth was so oblique that it seemed unlikely that they would erupt without intervention.

In a third patient aged 11 years, karyotype 45X, all the permanent teeth were erupted except the second molars. The lower second molars seemed to erupt without problems. The second maxillary molars appeared to be retained near the cementoenamel junction of the first molar and to resorb its distal surface (Fig. 4C).

In a fourth patient aged 10.3 years with karyotype 45X the first right maxillary molar was partly erupted and the second molar unerupted (Fig. 4D). The root development appeared nearly complete. There was no obvious reason why the first molar did not erupt regularly.

A fifth patient aged 13.1 years with karyotype 45X showed all teeth erupted except the second maxillary premolar on the right and the second molar on the left side. The premolar had the typical appearance of retention due to lack of space, probably caused by early extraction of the deciduous molar. The second molar (Fig. 4E) seemed retained due to lack of space and close relation to the third molar.

In a sixth patient aged 14.1 years with karyotype 45X all permanent teeth were erupted except the right maxillary second molar. This tooth showed a delayed root development and was situated very close to the distal surface of the first molar (Fig. 4F).

In a seventh patient, aged 8.9 years, karyotype 45X, who was severely affected by the syndrome also mentally, the right mandibular first molar was partly erupted. Radiographs showed the tooth mesially tipped with the crown towards the distal surface of the second deciduous molar and only the distal part of the occlusal surface piercing the mucosa.

Discussion

In the presently investigated group of Turner patients we observed delayed skeletal development, advanced dental development,

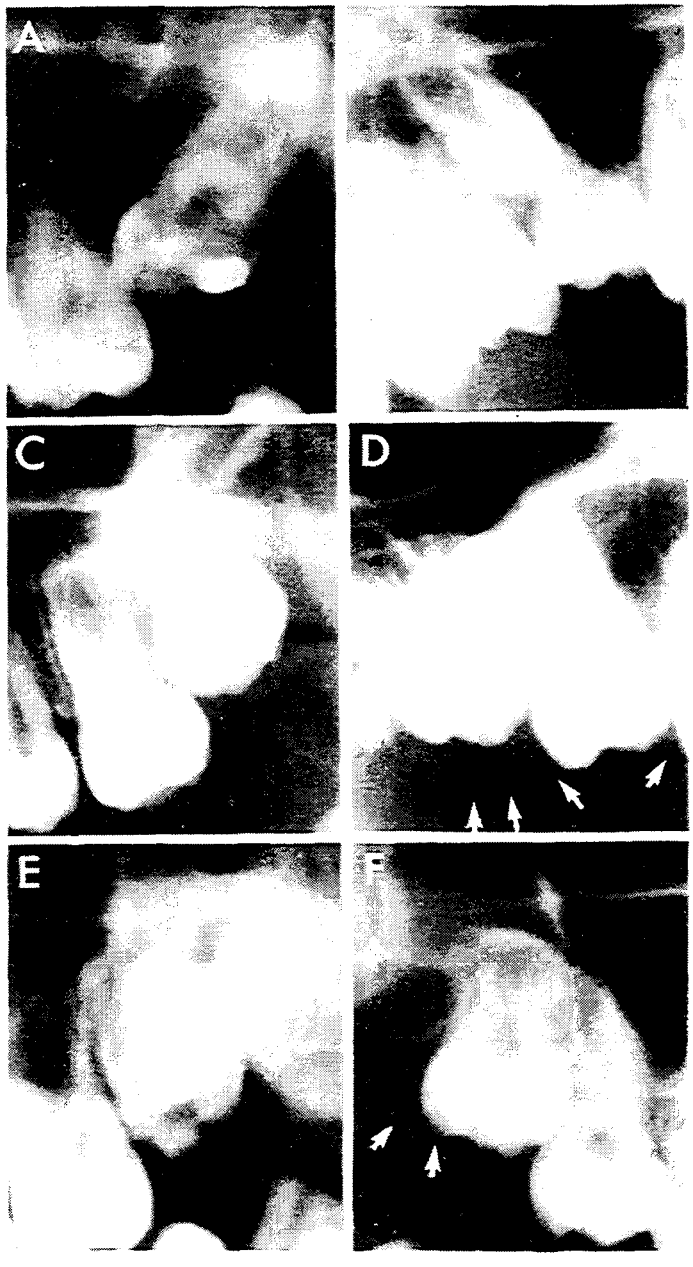

Fig. 4. Local eruption deviations in six Turner patients. In patients with bilateral problems only one side is illustrated.

and a moderately advanced or normal dental eruption.

The maturity stage of an individual can be expressed by means of several variables. Skeletal age expresses the biologic timing of the skeleton, most often visualized on a hand radiograph in which the maturity stages are connected to certain events of skeletal developmental changes. The most commonly used variable for assessing dental maturity is dental eruption. It must, however, be emphasized that tooth formation and eruption are essentially different processes, which can be differently influenced by genetic, environ-

mental, and hormonal factors (17). When studying tooth formation and eruption in Turner patients, the influence of such factors are to some extent unknown. This is one of the reasons we wanted to study dental formation and eruption separately.

Hägg & Mattson (18) evaluated the precision and accuracy of three methods for estimation of chronologic age on the basis of tooth formation. They found that the method of Demirjian et al. (13) was most reliable. A major disadvantage of all methods based on tooth formation is that they are most reliable for younger individuals. With increasing age the number of teeth under development decreases, and the ratings must be based mainly on root formation, which is a process of few 'events' and long duration.

The accuracy and precision of assessing chronologic age by analysis of tooth emergence were studied by Hägg & Hägg (19). They found that this method was better than the methods based on tooth formation. They also maintained that the method was especially useful in cross-sectional investigations.

The interrelation of various factors for skeletal, dental, and sexual maturity has been widely investigated in normal populations. Generally, the correlation between dental maturity, on the one hand, and the skeletal and sexual maturity, on the other, has been shown to be low (20–24). In contrast, correlation between skeletal and sexual maturity is high (22, 25).

This indicates that the variables body height and skeletal and sexual maturity are regulated by the same factors, whereas dental development is regulated differently. In conditions with sex chromosome abberations these regular patterns are frequently disturbed. There are probably several reasons for such deviations. It is, however, possible that the lack of one X chromosome influences the growth and its regulatory mechanisms more directly (5, 26, 27).

Delayed skeletal maturity, in this investigation 2.3 years according to the American standard of Greulich & Pyle (12), has also been reported earlier. Jensen (3) found the skeletal maturity in a group of Danish Turner girls to be 2 years delayed according to the same standard, whereas Webber et al. (4) reported a mean delay of 2.5 years, and Ogiuchi et al. (10) 2.1 years. All investigations show that the skeletal maturity is most retarded in the girls of older age groups. Maximum retardation found in our material was 4.2 years in a 16.7-year-old girl. Jensen (3) found a maximum retardation of 10 years in a 24-year-old woman.

Delayed skeletal maturity is probably caused mainly by hormonal factors and the delay towards puberty seems to be due to lack of estrogen. Park et al. (5) studied the effects of sex chromosome constitution and estrogen treatment on increase in height and on skeletal and sexual maturation. They found the rate of skeletal maturation lower than normal during the 2 years before estrogen treatment and higher than normal during the 2 years after starting treatment.

The findings of Webber et al. (4) are also interesting. They showed that only women with 45X and its variants had significantly retarded skeletal maturity. The women with 47XXX had normal skeletal maturity. This was further discussed by Alvesalo & Tammisalo (28) and Alvesalo et al. (29), who also found that the tooth size was dependent on the number of X chromosomes. The more Xs, the larger the teeth, mainly due to an increase in the enamel layer. It seems that genes on the X chromosome control growth in general and that there is an X-chromosomal influence on stature, tooth size, and enamel thickness.

Dental development is, according to Demirjian et al. (22), unrelated to other developmental systems. It is subject to less variation in relation to chronologic age and appears to be controlled independently. Our findings seem to confirm this also for the Turner patients. The correlation between dental and skeletal maturity was even less than in normal girls. Whereas the skeletal maturity was retarded, the dental maturity was advanced, and the clinical eruption was close to normal. Hormonal factors may contribute to this deviation. As earlier reported, lack of estrogen has been shown to delay the skeletal maturity (5). Our findings indicate that the dental maturity is unaffected or per-

haps even accelerated by the same shortage of estrogen.

In our investigation dental maturity was advanced 1 year. This is in accordance with several authors (8–11). A weakness of our figures may be that we did not have a Nordic control material and therefore used Demirjian's material of French-Canadian girls. A Finnish investigation by Haavikko (30) reports, however, that the rate of formation of the permanent teeth of Finnish children agrees closely with that of white children from the United States. Nyström et al. (31), however, using Demirjian's method for assessing dental maturity, found that the Finnish girls were on an average 3.5 months ahead of the French-Canadian girls at the age of 4–9 years and 9 months ahead in the 10- to 14-year age group.

The clinical eruption was advanced by a mean of 3.7 months compared with the Swedish reference material (15). No correction was made for local eruption problems. Such correlations might have resulted in a somewhat higher value. Acceleration was not significant, and the difference found in timing of the dental maturity and the clinical eruption may give rise to further investigations. The findings are, however, in agreement with those of Ogiuchi et al. (10). In their Japanese material they found that the mean dental age estimated from the maturity stages was 1.22 years advanced, whereas the eruption of the teeth was not affected. According to Ogiuchi et al. (10) this can be explained by the fact that shorter roots occurred in all their patients. This leads to advanced maturity by earlier finished root formation.

Lindsten et al. (9) compared the time of eruption of teeth in normal children and children with Turner syndrome and other sex chromosome abberations and found that both X- and Y-chromosomal factors influenced dental development.

According to a theory of Barlow (32), heterochromatic X chromosomes influence dental development in the sense that excess heterochromatin leads to delayed eruption of teeth by slowing down the cell cycle and diminishing cell division. Early eruption of teeth in Turner patients has been observed by Filipsson et al. (8) and Lindsten et al. (9). Galindo & Baar (33) and Zajaczkowska et al. (34) observed delayed tooth eruption in a case of 49 XXXXY and 49 XXXXX, respectively.

In our material of Turner patients we observed several local eruption disturbances in the lateral segments of the maxilla, most prevalent for 45X patients. The maxillary alveolar arch in these patients has been shown to be significantly narrower but of normal anteroposterior length (3, 7, 10). This will reduce the overall space in the arch. Even though the mesiodistal diameter of the teeth is reduced (28, 35–37), there seem to be a discrepancy between the maxillary alveolar arch size and the sum of the mesiodistal diameters of the teeth. In some patients this may lead to problems especially for the latest erupting teeth. In our investigation eruption problems were found in 6 of 33 patients related to the lateral segments of the maxilla, whereas only 1 patient showed faulty eruption in the mandibula.

Reduced body height is the most predominant finding in Turner patients. Since growth and its regulatory mechanisms seem to be influenced by genes on the X chromosome, and these genes also seem to influence tooth size, it can be anticipated that the same mechanisms also may be responsible for other dimensional changes observed in these patients. We also know that lack of estrogen has a retarding effect on the maturation of the skeleton. It seems likely that all these disturbances in the normal pattern for growth and development may cause disharmony in the relationship between certain structures. This may contribute to the higher frequency of lateral crossbites and perhaps also to the observed disharmony in tooth size in relation to jaw size.

Acknowledgement.—Appreciation is expressed to the Department of Paediatrics, University of Bergen, for their initiative to collaborate with the Departments of Orthodontics and Radiology in this field.

References

1. Turner HH. A syndrome of infantilism, congenital webbed neck, and cubitus valgus. Endocrinology 1938;23:566–74.

2. Ford CE, Jones KW, Polani PE, Almeida JC, Briggs JH. A sex-chromosome anomaly in a case of gonadal dysgenesis (Turner's syndrome). Lancet 1959; 1:711–3.
3. Jensen BL. Kanio-facial morfologi og skeletal modenhet ved Turner's syndrom [thesis]. Copenhagen: University of Copenhagen, 1974.
4. Webber ML, Puck MH, Maresh MM, Goad WB, Robinson A. Skeletal maturation of children with sex chromosome abnormalities [short communication]. Pediatr Res 1982;16:343–6.
5. Park E, Bailey JD, Cowell CA. Growth and maturation of patients with Turner's syndrome. Pediatr Res 1983;17:1–7.
6. Laine T, Alvesalo L. Size of the alveolar arch of the mandible in relation to that of the maxilla in 45, X females. J Dent Res 1986;65:1432–4.
7. Laine T, Alvesalo L, Lammi S. Palatal dimension in 45, X-females. J Craniofac Genet Dev Biol 1985;5:239–46.
8. Filipsson R, Lindsten J, Almquist S. Time of eruption of the permanent teeth, cephalometric and tooth measurements and sulphation factor activity in 45 patients with Turner syndrome with different types of chromosome abberations. Acta Endocrinol 1965;48:91–113.
9. Lindsten J, Filipsson R, Hall K, Leikrans S, Gustavson K-H, Ryman N. Body height and dental development in patients with Turner's syndrome. Helv Paediat Acta 1974;34 Suppl:34–46.
10. Ogiuchi H, Takano K, Tanaka M, et al. Oro-maxillofacial development in patients with Turner's syndrome. Endocrinol Jpn 1985;32:881–90.
11. Kari M, Alvesalo L. Dental maturity in 45X females [abstract]. J Dent Res 1985;64(Spec Iss):Abstract 1510.
12. Greulich WW, Pyle SI. Radiographic atlas of skeletal development of the hand and wrist. Stanford, Calif.: Stanford University Press, 1959.
13. Demirjian A, Goldstein H, Tanner JM. A new system of dental age assessment. Hum Biol 1973; 45:211–27.
14. Demirjian A, Goldstein H. New systems for dental maturity based on seven and four teeth. Ann Hum Biol 1976;3:411–21.
15. Hägg U, Taranger J. Dental development, dental age and tooth count. Angle Orthod 1985;55:93–107.
16. Ryan BF, Joiner BL, Ryan TA. Minitab handbook. 2nd ed. Boston: Duxbury Press, 1985.
17. Demirjian A, Dentition. In: Falkner F, Tanner JM, editors. Human growth. 2nd ed. London: Baillere Tindall, 1978:413–44.
18. Hägg U, Mattson L. Dental maturity as an indicator of chronological age: the accuracy and precision of three methods. Eur J Orthod 1985;7:25–34.
19. Hägg U, Hägg E. The accuracy and precision of assessment of chronological age by analysis of tooth emergence. J Int Ass Dent Child 1986;17:45–52.
20. Anderson D, Thompson GW, Popovich F. Interrelationship of dental maturity, skeletal maturity, height and weight from age 4–14 years. Growth 1975;39:453–62.
21. Filipsson R, Hall K. Correlation between dental maturity, height development and sexual maturation in normal girls. Ann Hum Biol 1976;3:205–10.
22. Demirjian A, Bushang PH, Tanguay R, Kingnorth Patterson D. Interrelationships among measures of somatic, skeletal, dental and sexual maturity. Am J Orthod 1985;88:433–8.
23. Hägg U, Taranger J. Dental emergence stages and the pubertal growth spurt. Acta Odontol Scand 1981;39:295–306.
24. Hägg U, Taranger J. Maturation indicators and the pubertal growth spurt. Am J Orthod 1982;82:299–309.
25. Hägg U, Taranger J. Skeletal stages of the hand and wrist as indicators of the pubertal growth spurt. Acta Odontol Scand 1980;38:187–200.
26. Flipsson R, Hall K, Lindsten J. Dental maturity as a measure of somatic development in children. Adv Metab Disord 1978;9:425–51.
27. Varrela J, Vinkka H, Alvesalo L. The phenotype of 45, X females: an anthropometric quantification. Ann Hum Biol 1984;11:53–66.
28. Alvesalo L, Tammisalo E. Enamel thickness in 45, X females' permanent teeth. Am J Hum Genet 1981;33:464–9.
29. Alvesalo L, Tammisalo E, Therman E. 47, XXX females, sex chromosomes, and tooth crown structure. Hum Genet 1987;77:345–8.
30. Haavikko K. The formation and the alveolar and clinical eruption of the permanent teeth. An ortopantomographic study [thesis]. Proc Finn Dent Soc 1970;66:103–70.
31. Nyström M, Haataja J, Kataja M, Evälahti, Peck L, Kleemola-Kujala E. Dental maturity in Finnish children, estimated from the development of seven permanent mandibular teeth. Acta Odontol Scand 1986;44:193–8.
32. Barlow P. The influence of inactive chromosomes on human development. Hum Genet 1973;17:105–36.
33. Galindo J, Baar HS. The XXXXY sex chromosome abnormality. Arch Dis Childh 1966;41:82–6.
34. Zajaczkowska K, Korniszewski L, Wolff-Pladòwska A. A case of quintuple X syndrome (49 XXXXX). J Ment Def Res 1970;14:305–11.
35. Townsend C, Jensen BL, Alvesalo L. Reduced tooth size in 45X (Turner syndrome) females. Am J Phys Anthropol 1984;65:367–71.
36. Mayhall J, Alvesalo L, Townsend G. Tooth crown size in 46, X, i (XQ) females [abstract]. J Dent Res 1987;66(Spec Iss): Abstract 22.
37. Varrela J, Townsend G, Alvesalo L. Tooth crown size in human females with 45, X/46, XX chromosomes. Arch Oral Biol 1988;33:291–4.

Received for publication 20 March 1992