ORIGINAL ARTICLE

Forecasting the timing of peak mandibular growth in males by using skeletal age

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Introduction: It is generally believed that the orthodontic treatment of a patient with a Class II malocclusion and a small mandible is enhanced by good growth at puberty, so that the timing of peak mandibular growth at puberty becomes of interest. **Methods:** To test the belief that skeletal age, whether early, average, or late, can be used to predict the timing of maximum growth of the mandible, whether early, average, or late, the predictive relationship between skeletal age and peak mandibular growth velocity (PMdV) at puberty was evaluated in 94 boys by using their longitudinal records from 4 to 18 years of age. Skeletal age was determined for each subject at ages 9 through 14 by using the method of Greulich and Pyle. **Results:** At age 9, the Greulich and Pyle measurements predicted that 30 of the 94 subjects would have delayed PMdV equal to or exceeding 1 SD (of the mean age for PMdV), and 10 would have advanced PMdV equal to or exceeding 1 SD. When the actual age of PMdV was determined retrospectively from plots of annual mandibular growth increments, it was found that only 4 of the 30 in the delayed group had actually experienced delays in PMdV, and only 2 of the 10 in the advanced group had experienced accelerated PMdV. **Conclusions:** Skeletal age is not a reliable predictor of the timing of PMdV. (Am J Orthod Dentofacial Orthop 2007;131:327-33)

Many orthodontists believe that the treatment of Class II malocclusion is optimized when it can be timed to take place during the period of greatest mandibular growth. Identifying that period in each patient is complicated because the pubertal growth spurt occurs at various chronological ages. Hence, it would be desirable to have a reliable way of forecasting when the maximum growth of the mandible at puberty will occur in a patient. Skeletal age derived from the maturation stages of the carpals and metacarpals has been used for that purpose for over half a century.

Houston et al[1] observed that "If advantage is to be taken of the growth spurt, it is necessary to predict its timing at least 1 or 2 years in advance of peak height velocity (PHtV)." Otherwise, the advanced patients will already be into their pubertal spurt. In the sample of boys in this study, the average ages were 13.2 ± 0.9 years for PHtV and 13.9 ± 1.2 years for peak mandibular velocity (PMdV).

Reports that support the use of skeletal age to forecast whether PMdV will be delayed or advanced include those by Hunter,[2] Bjork and Helm,[3] Helm et al,[4] Bjork,[5] Bowden,[6] Hagg and Taranger,[7,8] and Demirjian et al.[9] All used skeletal age to forecast the timing of PHtV at puberty, assuming that the relationship of PHtV and PMdV is very close. None explained how the use of skeletal age improves treatment or treatment planning.

To examine the relationship between skeletal age and PMdV, we tested the hypothesis that, when skeletal age at 9 years is delayed (or advanced) by 1 year or more, the succeeding PMdV will be similarly delayed (or advanced). We report the results of tests of this hypothesis for a sample of 94 growing boys enrolled in the Burlington Orthodontic Research Centre (BORC).[10] The relationship between skeletal age at 9 and PHtV was also examined.

MATERIAL AND METHODS

The longitudinal records on which this study is based were drawn from the records of the BORC, housed in the Department of Orthodontics, Faculty of Dentistry, University of Toronto. See Popovich and Thompson[10] and Hunter et al[11] for a description of the annual serial sample characteristics. The sample included 85% to 90% of all 3-year-old boys in Burlington when the study began in 1952 and Burlington's population was 9000. Although there were 172 boys at the beginning, that number had decreased significantly by the time they were 18 years of age.

The portion of the available materials relevant to our project included x-ray images and data for 122 boys

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for whom annual lateral headfilms, hand-wrist films, and height measurements had been obtained from 4 to 18 years of age. As part of the ongoing activity of the BORC, the annual headfilms for each subject had been traced previously. Fifty-nine landmarks had been located on each lateral cephalogram and their coordinates stored. Of the 122 subjects, records and data for 17 were not used in this study because the quality of the hand-wrist films was unsatisfactory for our purposes. The required information for another subject was unavailable; this reduced the sample to 104. Ten subjects were subsequently removed from the sample because of data ambiguities, leaving the final sample size of 94.

The general strategy of the study involved 3 steps that used previously stored longitudinal data from lateral cephalograms and hand-wrist x-ray images: The first step involved the determination of skeletal age from the hand-wrist x-ray images at each of a number of chronological ages in the mixed dentition, by using the method of Greulich and Pyle.[12] In step 2, predictions of PMdV were made on the basis of the data from step 1. In step 3, PMdV was determined blindly from the serial annual lateral cephalograms. The predictions from the second step were then tested statistically against the observed data from the third step, yielding the main findings of the study. Corroborative studies of method error were also made and are included.

Because the enlargement factor for midsagittal structures was constant (9.8%), there was no need to correct for it.

Definitions of variables

Chronologic age was the age at the subject's nearest birthday as determined from the demographic records of the BORC. With very few exceptions, these records had been obtained within 1 month of the subject's actual birthday. Annual increments were positioned at the midpoints between birthdays.

Skeletal age was determined twice, to the nearest tenth of a year, from the hand-wrist radiographs with the procedure of Greulich and Pyle.[12] The first set of skeletal ages was obtained in 1973 and is called GP1. The second set, obtained in 1983 independently of the first set, is called GP2. As a check on the findings with the Greulich and Pyle method, skeletal age was also determined by the method of Tanner et al[13] and is called TW or TW2RUS (second method, with radius, ulna, and the small bones of the hand).

Prepubertal mandibular growth minimum (PPM) was defined as the age of minimal annual mandibular growth between the ages of 8 and 14 (2.4 mm or less, with a mean value of 0.90 ± 0.69 mm). The distance between articulare (Ar) and gnathion (Gn) on each annual lateral cephalogram was calculated from the previously recorded coordinates of the 2 landmarks. Annual increments of mandibular growth for each subject were then derived by subtracting each annual value for ArGn from that for the next year. The increments were connected and plotted as shown in Figure 1. Identification of the PPM was included as part of an initial plan to evaluate it as a time to avoid treatment, but that is not part of this report. However, we used PPM to identify the age of PMdV as explained next.

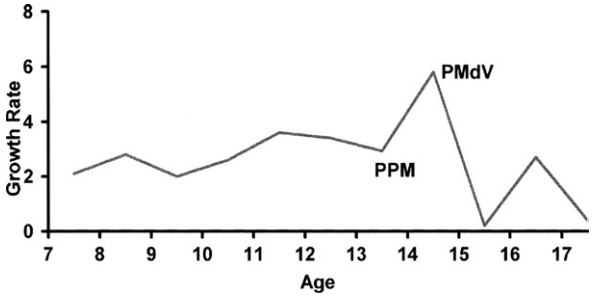

Fig 1. Increment graph for subject 1059. Both PPM (13.5 y) and PMdV (14.4 y) were determined from increment graphs.

Table I. Age of PMdV in years

	n	*Mean*	*SD*	*Advanced*	*Delayed*
PMdV	94	13.9	±1.21	≤12.7	≥15.1

PMdV was the age of maximum annual mandibular growth after the PPM, determined from the plotted increments for ArGn as described above. Because of vagaries in funding, there were 65 subjects for whom it was not possible to obtain records at the 15-year time point, and their 15-year values were estimated by dividing the increments from ages 14 to 16 by 2.

In this study, PMdV was considered advanced if it occurred more than 1 SD before the mean for the entire sample and delayed if it occurred more than 1 SD after the mean for the entire sample (Table I).

PHtV was the age of greatest annual increment in stature between 8 and 15 years. Measurements of stature at annual time points were available directly from the BORC records. Values for the increments were determined as described for PPM.

Using the above definitions, we found that, among the 104 subjects who satisfied the original sampling criteria, 8 had 2 PMdV values for which the mandibular growth increment was equal (± 0.5 mm). Two additional subjects had 2 PPM and 2 PMdV values. Rather than modify the definitions or make ad-hoc changes, we

Table II. Correlations by year

Correlations	9 years	10 years	11 years	12 years	13 years	14 years
GP1 and GP2	.95	.97	.97	.96	.95	.96
GP1 and TW2RUS	.61	.63	.69	.71	.71	.84
GP1 and PMdV	−.01	−.04	−.11	−.28	−.50	−.58
GP1 and PHtV	−.12	−.12	−.23	−.36	−.50	−.55

decided to remove those 10 subjects, yielding our final sample size of 94.

The increments at PPM and PMdV, and for the 2-year period from 13 to 15 years of age, were estimated from the increment graphs and recorded. The correlations between skeletal age, PMdV, and PHtV at each age were calculated. The association between the skeletal age determinations at 9 years of age and the timing of PMdV, categorized as delayed, average, or advanced, was tested by using the Fisher exact test.[14]

RESULTS

Findings for skeletal age and PMdV

The correlations between skeletal age and the timing of PMdV for the GP1 estimates ranged from −.01 at 9 years to −.58 at 14 years, and the correlations between the GP1 estimates for skeletal age and PHtV were −0.12 at 9 to −0.55 at 14 years (Table II). There were 30 boys whose skeletal age at 9 years was 8 years or less; they were categorized as delayed (Table III). In only 4 of those subjects was PMdV delayed (ie, occurred after age 15.1 years). At 10 and 11 years, 29 and 26 boys, respectively, were identified as having delayed skeletal ages. In both cases, PMdV was delayed for the same 4 boys. Similarly, at 9, 10, and 11 years of age, 10, 12, and 9 boys, respectively, were categorized as advanced; of those, only 2 (the same 2) in each case had advanced PMdV.

With regard to the timing of PMdV, using the skeletal age forecasts shown in Table III, the sample can be seen as containing 3 prediction groups consisting of 30 delayed, 54 average, and 10 advanced subjects. We tested the entire sample of 94 to see whether there was any difference in the distribution from what might have been expected to have occurred by chance if the entire group had been homogeneous with no differences between prediction groups. According to the most appropriate statistical test, the Fisher exact test,[14] a relationship as strong as that observed had a probability of 0.92 by chance alone. The more familiar, although not quite so appropriate, chi-square test produced a probability of 0.93. We concluded that the predictions using skeletal age were no better than chance.

Error studies

Skeletal age was estimated independently twice for all subjects by using the Greulich and Pyle methods (GP1 and GP2)[12] and a third time by the TW2RUS[13] method. Correlations between the GP1 and GP2 estimates were obtained and the error variances calculated.

The correlations between GP1 and GP2 ranged from 0.95 to 0.97 (Table II). The measurement error variance was 0.19 years, or 19%, of the skeletal age variance of 1.01 years at age 9; this is well beyond the usual range of 3% to 10%. Nevertheless, the second determination of skeletal age (GP2) identified 28 (all but 3 were the same as the GP1 subjects delayed at 9) as delayed at 9 with the same 4 having delayed PMdV. Because the 2 estimates were completely independent and made by different observers, it is perhaps remarkable that they are so similar. The correlations between the GP1 and TW2RUS estimates of skeletal age (Table II) were smaller than those between GP1 and GP2, most likely because of the reference sample differences as noted below. Although the errors of determination exceed acceptable limits for numerical data, the difference between the number delayed at 9 and the number who experienced delayed PMdV far exceeds what could be attributed to errors of determination.

To evaluate the accuracy of determining the age of PMdV using the graphic method, the headfilms for 20 randomly selected subjects were traced again for each age from 8 years to and including 14 years of age. Ar and Gn were identified and digitized; the increments were calculated as described above, plotted, and graphed as before.

Traditionally, in studies with headfilms, errors of measurement are considered to arise from both landmark identification and the linear or angular measurements derived from those landmarks either directly or through the use of a scanner and x-y coordinates. This study has, as an additional source of error, the effects of converting the linear measurements of ArGn to increment values.

For 7 (35%) of the 20 double determinations of the age of PMdV, the second determination was not the same as the first. Of those, 3 were 1 to 2 years older, and 4 were 1 to 2 years younger. For subject 1009 (Fig 2), the first and second determinations differed by 18 months. If mandibular length at the second measure at 14.0 years had been 0.6 mm larger, the increment at 13.5 years would have been 3.6 mm, and the 15-year increment would have been 3.4 mm,

Table III. Predictions of the timing of PMdV for 9-year-old boys using skeletal age as determined by the Greulich and Pyle method

			3. Measure actual PMdV		
	2. Predict PMdV		*Delayed*	*Average*	*Advanced*
1. Determine skeletal age (y)	*Category*	*N*	*(after age 15.1)*	*(between ages 12.7 and 15.1)*	*(before age 12.7)*
<8.0	Delayed	30	4	18	8
8.0 to 10.0	Average	54	7	36	11
>10.0	Advanced	10	2	6	2
	Totals	94	13	60	21

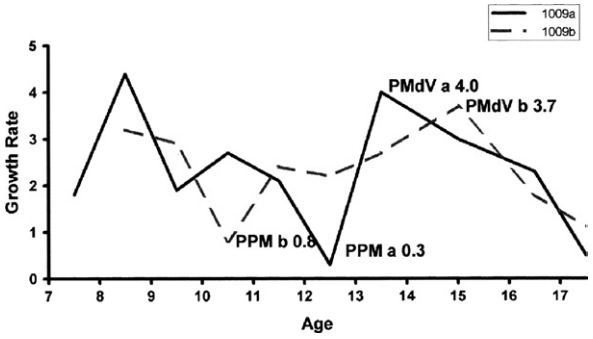

Fig 2. Plots for double determinations for subject 1009: first (*solid line*) and second (*broken line*) determinations. Second determination located PMdV 18 months later than first.

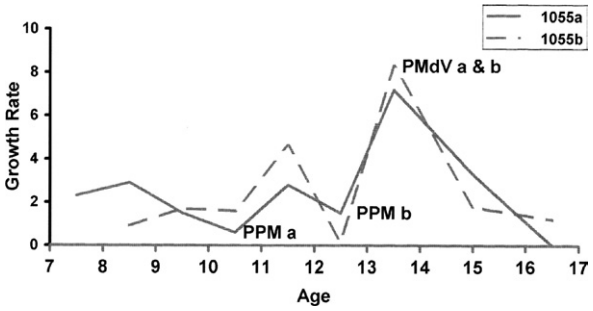

Fig 3. Plots for double determinations for subject 1055: first (*solid line*) and second (*broken line*) determinations. Second determination located PPM at 12.5 years; first determination located PPM at 10.5 years. PMdV was located at 13.5 years by both determinations. Note also increment at 15 rather than at 14.5.

so that PMdV would have coincided with PMdVa at 13.5 years. Thus, a rather small difference can change the location of PMdV by a year or 18 months. Although the second determinations for subject 1055 (Fig 3) were similar to the first, slight differences in opposite directions at 10.5 and 12.5 years resulted in the prepubertal minima differing by 2 years.

DISCUSSION

There is abundant evidence that the rate of bone growth accelerates at puberty and that the acceleration occurs earlier in girls than in boys, with considerable variability in each sex. Because it is a widely held belief that efforts to increase the length of the mandible are enhanced during its pubertal acceleration, the prediction of the timing of that acceleration is of interest to orthodontists.

The concept of skeletal age assumes that all bones of the body develop in concert, or, in this study, that the stages of finger-bone development occur concomitantly with the developmental stages of the mandible with PMdV as a stage marker. That assumption is implicit when the developmental status of the finger bones (determined by the Greulich and Pyle[12] or the TW2RUS[13] method) is called "skeletal age." The concept might have been initiated by Greulich and Pyle in the introduction to their second edition in which they wrote: "The skeleton of the healthy, adequately nourished child develops as a unit, and its various parts tend to keep pace with one another."

However, since 1959, at least 6 investigators have questioned the use of skeletal age at 9 (or 10 or 11) to forecast the timing of PMdV. They include Bambha and Van Natta,[15] Smith,[16] Ekstrom,[17] Lewis et al,[18] and Moore et al. [19] More recently, Hunter et al[20] reported that, in a sample of 104 girls, 20 were classified as having delayed skeletal ages at 9 years of age but only 5 of those experienced delayed PMdV. On the other hand, in a review of 11 reports that related skeletal age to various aspects of facial growth, Flores-Mir et al[21] observed that "overall facial growth velocity was well related to standing height growth velocity and skeletal maturity." No numerical data other than sample sizes were provided.

Errors in the determination of skeletal age

The GP1 data showed that 30 subjects (32%) had delayed skeletal ages at 9 years. The GP2 data labeled 28 (30%) as delayed at 9 years. The differences

between the 2 sets of estimates were not large enough to result in substantially different findings with respect to the hypothesis being tested. As a result of deficient wrist films, skeletal age could be estimated only for 85 subjects (the TW2RUS method requires that the radius and the ulna be seen). Of those, 5 experienced delayed PMdV; only 2 of those were the same as the GP1 delayed. The smaller percentage of delayed skeletal age subjects at age 9 determined by the TW2RUS procedure (26% vs 32%) is probably because the Harpenden reference sample of Tanner et al[13] matured earlier than the Cleveland sample on which the Greulich and Pyle procedure was based.

Errors in location of PMdV

The correlation between the first and second measurements for mandibular length (ArGn) was almost perfect at 0.99, whereas, for the increments that determined the location of PMdV, the correlation was 0.61. It is not surprising then that, of the 20 double determinations of PMdV, only 13 were the same the second time. The error variance for the measure ArGn contributed less than 2% to the variance of ArGn itself, whereas the error variance for the increments accounted for nearly 50% of the variance of the increments. That is because the actual error variance for ArGn (not a percentage) was carried over to the increment values. Thus, an acceptable amount of error for measurements of 100.0 mm becomes quite substantial for measurements of 3.0 to 6.0 mm. However, because the second determinations were as often older as younger, it is not likely that the error component could have created the finding that of 30 boys at age 9 with delayed skeletal ages, only 4 experienced delayed PMdV. Similarly, of the 10 boys with advanced skeletal ages at 9, only 2 experienced advanced PMdV. Thus, although the determination of PMdV by graphic procedure is not exact, there is no indication that the inexactitude changed 26 delayed PMdV values to average or advanced. They did it without our help.

Skeletal age and the timing of PMdV

From the perspective of the orthodontic clinician, the key group in this sample is that comprising the 30 subjects for whom GP1 predicted delayed mandibular growth spurts. If an orthodontist had postponed intervention on the basis of this prediction, he or she would have been in error for 26 of the 30 patients (86.7%) because, as shown in Table III, 18 (60%) subjects had average values for PMdV and 8 more (27%) actually had advanced PMdV values. Only 4 of the 30 delayed subjects (13%) had delayed PMdV.

Of the 54 subjects for whom average peak velocity was predicted, a clinician would not have made such severe errors, but he or she still would not have fared very well. For 11 (20%) of the 54 subjects, the event of peak velocity would have been missed; 36 subjects (67%) would have been treated during peak growth, but, for 7 subjects (13%), intervention would have been premature.

Table IV. Annual skeletal age changes for 30 boys who had delayed skeletal age at 9 years

	Chronologic age (y)					
	9	10	11	12	13	14
Delayed (n)	30	29	26	18	12	7
Average (n)	—	1	4	11	16	21
Advanced (n)	—	—	—	1	2	2

For the 10 subjects for whom advanced PMdV had been predicted, the clinician who believed the prediction would have intervened prematurely in 8 patients (80%), but the interventions would have been well timed in the other 2 (20%).

If skeletal age at 9, 10, or 11 is to predict the timing of PMdV, it must remain the same whether early, average, or late until PMdV has occurred. As Table IV shows, only 7 of the 30 delayed at 9 had delayed skeletal age at 14. The other 23 had become average or advanced. The correlations between skeletal age and PMdV are 0.01 at 9 years and −0.58 at 14. We concluded that the success rate of these predictions leaves much to be desired.

The correlation between PHtV at puberty and PMdV at puberty was 0.36. Similarly, the correlations between the GP1 estimates of skeletal age and PHtV ranged from −0.12 at 9 to −0.58 at 14 (Table II). Thus, the developmental status of the hand bones was not the same as that of the mandible or the stature. We were dealing with finger bone age and PMdV and statural age. For this sample of 94 boys, none of those 3 kept pace with one another closely enough to be useful to forecast PMdV. Hansman and Maresh[22] reported in 1961 that "about 1/3 of the girls and fewer of the boys show a time lag in skeletal maturation during the childhood years but each child 'catches up' at about the time of his or her adolescence." Of the 30 delayed subjects in our sample, all but 7 caught up, as seen in Table IV.

We also considered whether the subjects at 9 who were delayed more than 1 year would be more likely to experience delayed PMdV. According to the GP1 estimates, 9 boys had skeletal ages at 9 of 7 years or less; ie, they were delayed 2 years or more. Only 2 of those 9 experienced delayed PMdV (22%).

Table V. Increments of ArGn growth from 13 to 15 years

Change in ArGn	*n*
Increment of 2.0 mm/y or more from 13th to 15th birthdays	68
Average increment of 3.4 ± 1.2 mm at age 13.9	16
Increment of 1.0-2.0 mm between 13th and 15th birthdays	9
<1.0 mm growth from 13th to 15th birthdays	1

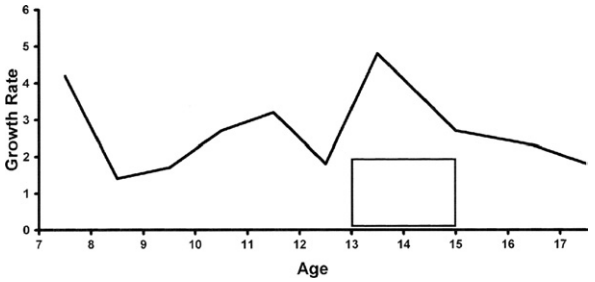

Fig 4. Subject 1053, one of 63 subjects who experienced increments in ArGn of 2.0 mm or more per year from 13th to 15th birthday. He had more than 4.0 mm growth in 14th year. Box between 13 and 15 represents 2.0 mm per year of growth of ArGn. See Table V.

This is a companion study to a previously published examination of the relationship of skeletal age at 9 to the timing of PMdV for 104 girls from the BORC.[20] The findings for this sample of boys confirm our conclusions for the girls, that skeletal age as determined from the maturation of the finger bones at 9, 10, or 11 years of age is not useful for predicting whether PMdV will be early, average, or late.

However, the default option of treating at the average age for PMdV—between the 13th and 15th birthdays—means working with at least 2.0 mm per year of mandibular growth in more than two thirds of the patients. As Table V shows, 68 boys, or 72% of our sample, had at least 2.0 mm per year of increase in the length of ArGn between their 13th and 15th birthdays (Fig 4). Although 26 did not have a 2.0 mm per year increase for the entire 2-year period, 16 of the 26 had an average increment of 3.4 ± 1.2 mm at the average age for PMdV of 13.9 years. Nine more experienced 1.0 to 2.0 mm of mandibular growth between their 13th and 15th birthdays. The remaining subject had only a small amount of growth early in his 13th year (Fig 5), probably because he had experienced PMdV at 11.5 years and had simply stopped growing. However, we cannot identify him prospectively or identify in advance the advanced, average, or delayed status of any subject or patient. Nevertheless, the fact that nearly 90% of our sample experienced substantial mandibular

Fig 5. Incremental graph for subject 1085, showing only slight growth of ArGn between 13 and 15 years.

growth between their 13th and 15th birthdays, suggests that we may exploit that growth without knowing exactly when PMdV will occur.

CONCLUSIONS

The relationship between skeletal age determined from hand-wrist films with the Greulich and Pyle method[12] and PMdV from the plots of the annual increments of mandibular growth was evaluated for 94 boys by using their longitudinal records from 4 to 18 years of age. Thirty boys were found to have delayed skeletal ages at 9 years. Subsequently, of those 30, 4 had delayed PMdV. Although 10 boys had advanced skeletal ages at 9, only 2 had advanced PMdV. Thus, the maturation status of the epiphyses of the hand bones at 9 (or 10 or 11) does not forecast the timing of peak growth of the mandible. It is suggested that treating around the average age for PMdV is a viable alternative to attempting to time treatment to the exact occurrence of PMdV.

REFERENCES

1. Houston WJB, Miller JC, Tanner JM. Prediction of the timing of the adolescent growth spurt from ossification events in hand-wrist films. Br J Orthod 1979;6:145-52.
2. Hunter CJ. The correlation of facial growth with body height and skeletal maturation at adolescence. Angle Orthod 1966;36:44-54.
3. Björk A, Helm S. Prediction of the age of maximum pubertal growth in body height. Angle Orthod 1967;37:134-43.
4. Helm S, Siersbaek-Nielsen S, Skieller V, Björk A. Skeletal maturation of the hand in relation to maximum pubertal growth in body height. Tandlaegebladet 1971;75:1223-34.
5. Bjork A. Timing of interceptive orthodontic measures based on stages of maturation. Trans Eur Orthod Soc 1972;61-74.
6. Bowden BD. Epiphyseal changes in the hand/wrist area as indicators of adolescent stage. Aust Orthod J 1976;4:87-104.
7. Hägg U, Taranger J. Maturation indicators and the pubertal growth spurt. Am J Orthod 1982;82:299-309.
8. Hägg U, Taranger J. Skeletal stages of the hand and wrist as indicators of the pubertal growth spurt. Acta Odont Scand 1980;38:187-200.
9. Demirjian A, Buschang PH, Tanguay R, Patterson DK. Interrelationships among measures of somatic, skeletal, dental and sexual maturity. Am J Orthod 1985;88:433-8.

10. Popovich F, Thompson GW. Evaluation of preventive and interceptive orthodontic treatment between three and eighteen years of age. In: Cook JT, editor. Transactions of the 3rd International Orthodontic Congress; August 13-18, 1973, London, UK. St Louis: Mosby; 1975. p. 260-81.
11. Hunter WS, Baumrind S, Moyers RE. An inventory of United States and Canadian growth record sets. Am J Orthod Dentofacial Orthop 1993;103:545-55.
12. Greulich W, Pyle SI. Radiographic atlas of skeletal development of the hand and wrist. 2nd ed. Stanford, Calif: Stanford University Press; 1959.
13. Tanner JM, Whitehouse RH, Cameron N, Marshall WA, Healy MJR, Goldstein H. Assessment of skeletal maturity and prediction of adult height (TW2 method). 2nd ed. London: Academic Press; 1983.
14. Riffenburgh RH. Statistics in medicine. San Diego: Academic Press; 1999.
15. Bambha JK, Van Natta P. Longitudinal study of facial growth in relation to skeletal maturation during adolescence. Am J Orthod 1963;45:481-93.
16. Smith RJ. Misuse of hand-wrist radiographs. Am J Orthod 1980;77:75-8.
17. Ekstrom C. Facial growth rate and its relation to somatic maturation in healthy children. Swed Dent J 1982;11(Suppl):1-99.
18. Lewis AB, Roche AF, Wagner B. Growth of the mandible during pubescence. Angle Orthod 1982;52:325-42.
19. Moore RN, Moyer BA, DeBois LM. Skeletal maturation and craniofacial growth. Am J Orthod Dentofacial Orthop 1990;98: 33-40.
20. Hunter WS, Popovich F, Baumrind S, Jorgensen G. Skeletal age and peak mandibular growth velocity at puberty. In: McNamara JA Jr, editor. Treatment timing: orthodontics in four dimensions. Craniofacial Growth Series. Ann Arbor: Center for Human Growth and Development; University of Michigan; 2002. p. 185-98.
21. Flores-Mir C, Nebbe B, Major PW. Use of skeletal maturation based on hand-wrist radiographic analysis as a predictor of facial growth: a systematic review. Angle Orthod 2004;74:118-24.
22. Hansman CF, Maresh MM. A longitudinal study of skeletal maturation. Am J Dis Child 1961;101:305-21.