

Dental Maturation in Children With Shunt-Treated Hydrocephalus

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Objective: The aim of this study was to examine the relationship between dental maturation and somatic development and to discover their possible deviations in children with shunt-treated hydrocephalus.

Design: Radiographs (orthopantomogram and hand-wrist radiograph) from children with shunt-treated hydrocephalus were analyzed with respect to the dental maturation and compared with values of an age- and sex-matched control group and population standards.

Patients: Forty-one children with shunt-treated hydrocephalus (27 boys and 14 girls) aged from 5 to 16 years (median age 11.0 years).

Results: The mean deviation of dental age from chronological age was significantly greater in the hydrocephalic group than in the control group. The mean value of dental age deviation was advanced in early pubertal stages but reduced in later pubertal stages.

Conclusions: These findings can be explained by changes in the endocrinological conditions, which are due to disturbed pituitary secretion. The present results could also partly reflect an adaptable developmental pathway in an otherwise strictly genetically determined process.

KEY WORDS: *dental maturation, hydrocephalus, shunt treatment*

Children with shunted hydrocephalus (HC) have various abnormalities in their general somatic growth and timing of puberty. It has long been known that somatic growth shows much greater variation in children with HC than in normal children (Klauschie and Rose, 1996; Cholley et al., 2001). The prepubertal linear growth of children with HC is slow, whereas their pubertal maturation is accelerated with a reduced final height. Levels of basal growth hormone and pituitary sex hormones have been shown to be altered. It has been suggested that the explanation for this could lie in the raised or otherwise abnormal intracranial pressure resulting in pituitary dysfunction (Löppönen et al., 1996, 1997).

Reports on aberrations affecting the craniofacial area in children with HC reveal a progressive thickening of the calvarium associated with shunt treatment (Anderson et al., 1970; Griscom and Oh, 1970; Huggare et al., 1986, 1989, 1992). Significant changes in cranial size, alterations in skull base morphology, and the size and location of the sella have also been

observed (Griscom and Oh, 1970; Kantomaa et al., 1987; Huggare et al., 1989, 1990; Lestrel and Huggare, 1997). Facial changes, however, have been reported to be minimal (Forrester et al., 1966). However, there are no reports on dental maturation in children with HC.

The relationship between dental maturation and somatic growth is normally found to correlate weakly, although alterations in humoral levels controlling general growth have some detectable effects on dental maturation status. Factors that significantly contribute to dental development have been reported to include growth hormone; sex hormones; and, to a lesser degree, corticosteroids and thyroid hormones. Myllärniemi et al. (1978) studied dental development in relation to somatic growth in cases of hypopituitarism and found that growth retardation was greatest in skeletal maturation and smallest in dental development. When children with short stature and with or without growth hormone deficiency were examined, no difference was found between the two groups in dental development. However, both groups with short stature exhibited a delayed dental maturity when compared with healthy controls (Krekmanova et al., 1997).

A common finding in a variety of craniofacial anomalies, such as cleft lip/palate, is an increased deviation of growth timing and dental development (Harris and Hullings, 1990). This has been assumed to result from decreased integration among organ and tissue systems in which the development of dentition is one of the variables involved.

The aim of the present study was to examine the relationship

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between dental maturation and somatic development and to discover their possible deviations from normal development in children with shunt-treated HC.

SUBJECTS AND METHODS

The subjects were 41 children with shunt-treated HC (27 boys, 14 girls) aged from 5 to 16 years (mean age 11.4 years; median age 11.0 years; SD = 3.39 years) who were primarily operated on in early childhood at the Department of Pediatrics in the Oulu University Hospital (Finland). Patients with tumors and achondroplasia were excluded from the study, as were those with an intelligence quotient below 50 because factors other than the HC itself may influence their growth and pubertal maturation. Nine patients in the study population (21.9%) had epilepsy. The median age during the first operation was 0.5 years (range 0.0 to 8.6 years).

The control group for the tooth development examination consisted of age- and sex-matched children (27 boys and 14 girls, mean age 11.3 years, median age 10.8 years, SD = 3.11 years) from the neighboring child welfare clinics and schools. The children were normal healthy children, and their regular panoramic x-rays of dentition had been taken for the treatment of minor crowding of dentition. The control group was matched so that the chronological age of each control differed by no more than 3 months from the chronological age of the corresponding child in the hydrocephalic group.

The assessment of dental maturity and dental age was performed from panoramic radiographs using the seven-teeth system (Demirijan et al., 1973; Demirijan and Goldstein, 1976). Maturity scores were converted to dental age using Finnish standards (Kataja et al., 1989), and were compared with the chronological age.

Analysis of the bone age of children with HC was based on radiographs of the left hand and the wrist using the method reported by Greulich and Pyle (1959). The clinical evaluation was carried out at the Institute of Dentistry in the University of Oulu and in the Department of Pediatrics at Oulu University Hospital. The clinical evaluation method is explained in detail by Löppönen et al. (1995, 1996, 1997). The stage of puberty was assessed according to Tanner and Whitehouse (1976). If there were no signs of puberty, the subjects were classified as prepubertal (pubic hair 1 [PH1] and genitals 1 [G1] in boys or PH1 and breast 1 [B1] in girls). Subjects were classified to be sexually fully mature when the Tanner stage in boys for pubic hair was 5 (PH5) and genitals 5 (G5) or in girls the PH5 stage and breast 5 (B5). Subjects with adult bone age were classified as postpubertal.

Informed consent was obtained from the subjects, their parents, or both. The study was conducted according to the Declaration of Helsinki. For ethical reasons, the hand-wrist radiograph could not be taken from the children in the control group. Tanner staging for this group was unobtainable because the control group had been examined earlier because of orthodontic reasons.

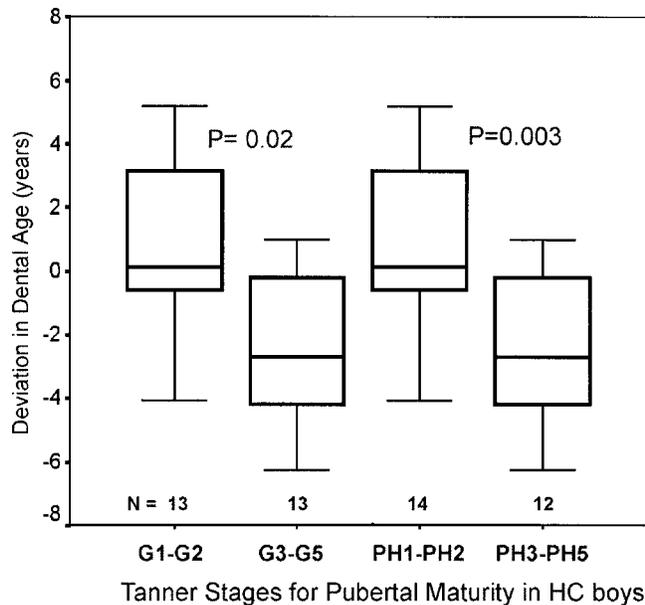


FIGURE 1 Box-plot (means, 25th and 75th percentiles, and ranges) showing the deviation of dental age from chronological age in prepubertal or early pubertal stages (genital stage 1 or 2 and pubic hair stage 1 or 2) and later stages (genital stages 3 through 5 and pubic hair stages 3 through 5) of puberty in boys with shunt-treated hydrocephalus. One male subject with adult bone age was classified as postpubertal and was not included in the analysis.

Statistics

Statistical evaluation was performed using (1) cross-tabulation and chi-square statistics, (2) Mann-Whitney *U* test, and (3) Spearman's correlation. SPSS 10.0 statistical packet (SPSS Inc., Chicago, IL) was used for the analyses.

RESULTS

The mean deviation between chronological age and dental age was significantly greater in the hydrocephalic group than in the control group. The mean deviation was 1.1 years (SD = 0.98, range 0.2 to 6.2) in the hydrocephalic group and 0.8 (SD = 0.64, range 0 to 2.8) in the control group ($p < .05$, Mann-Whitney *U* test). The portion of those children whose deviation in dental age exceeded 2 SD of age-specific population standards was 9.7% in the hydrocephalic group and 2.4% in the control group ($p < .05$, chi-square test).

When the deviation in dental age was analyzed with respect to the registered pubertal stage in the children with HC, it was found that the mean value of dental age deviation was advanced in early pubertal stages (Tanner stages G1-G2 and PH1-PH2 in boys and B1-B2 and PH1-PH2 in girls) but reduced in later pubertal stages (Tanner stages G3-G5 and PH3-PH5 in boys and B3-B5 and PH3-PH5 in girls). The difference between these characteristics was seen in both sexes (Figs. 1 and 2) but was more significant in boys ($p = .02$ and $.003$) than in girls ($p = .086$ and $.046$; Mann-Whitney *U* test).

The individual deviation in dental age in children with HC

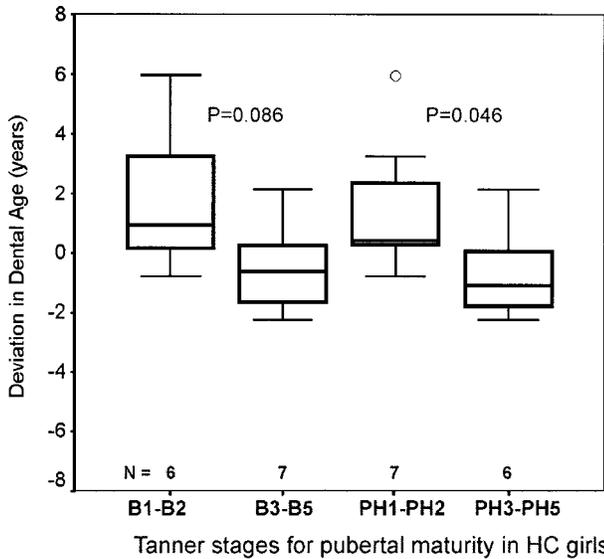


FIGURE 2 Box-plot (means, 25 and 75th percentiles, and ranges) showing the deviation of dental age from chronological age in prepubertal or early pubertal stages (breast stages 1 through 2 and pubic hair stages 1 through 2) and later stages (breast stages 3 through 5 and pubic hair stages 3 through 5) of puberty in girls with shunt-treated hydrocephalus. One female subject with adult bone age was classified as postpubertal and was not included in the analysis.

was compared with the deviation in bone age. The correlation between individual deviations in dental and bone ages was weak ($r = .29$, $p = .06$, Spearman's correlation).

DISCUSSION

HC is defined as a condition in which the volume of cerebrospinal fluid is increased, resulting in excessive intracranial pressure (Aicardi et al., 1992). The etiology of HC is estimated to be prenatal in 60% of cases, perinatal in 20%, postnatal in 7%, and unknown in 13% (Voutilainen, 1992). Ventriculoperitoneal shunting is the most commonly used technique in the treatment of HC (Hirsch, 1992). The mortality rate has decreased markedly, from about 50% prior to 1960 to about 15% in the 1980s (Amacher and Wellington, 1984). The outcome of the children with HC has improved since the introduction of shunt treatment in the 1950s and patients treated for HC from early childhood have, for instance, known to become pregnant and given birth to healthy children (Hirsch, 1992; Stevens, 1996).

Dental age assessment should preferably be designed to meet population differences in tooth maturation (Nyström et al., 1988). Therefore, our age- and sex-matched controls were selected to represent similar ethnic and environmental backgrounds with respect to the place of residence. The method used to determine dental maturation has been tested and found accurate (Hägg and Mattson, 1985). This method is especially useful when proper population correction has been performed (Krekmanova et al., 1997). Data required to make the population correction were available for the present survey (Kataja et al., 1989), and, therefore, the possibility for systematic bias

in dental age estimation was diminished. The results indicate that dental development in children with HC shows great variation but relatively little association with general growth. The fact that dental age was advanced in the children with HC during early pubertal stages but delayed during later stages is interesting. This finding is very clear in spite of the relatively small study group and a wide age range of the children with HC. Earlier findings from the same patient population showed an analogous earlier onset of puberty and decreased postpubertal growth (Löppönen et al., 1996).

Moreover, Löppönen et al. (1996) found that in this hydrocephalic population there were significantly higher concentrations of anterior pituitary hormone (follicle-stimulating hormone and luteinizing hormone) and lower concentrations of basal growth hormone (GH) than in the corresponding healthy population. These findings may explain the general somatic growth characteristics of the examined children with HC.

The alteration of humoral function has been speculated to follow from increased pressure around the hypophysis (Stanhope and Brook, 1989). Alterations in pituitary morphology are visible in those treated for HC as their morphology turns to normal in the course of treatment. On the other hand, prolonged shunt treatment is often associated with a considerable reduction in size of the sella (Kaufman et al., 1970, 1973). So far, we do not know whether the increased intracranial pressure, caused by the primary disease, or the later shunt failures with periods of cerebrospinal fluid overdrainage is the causative factor of disturbed pituitary gland secretion. On the other hand, without the function of the shunt, the condition would be fatal in most cases, emphasizing advances in the treatment of HC.

It might be speculated that the advanced dental maturation during early pubertal stages and delayed development during the later stages could be a result of progression in the course of the disease. No such apparent progression could be seen clinically in this patient group, however. Although the correlation of humoral factors with dental maturation is generally low, it is interesting that the hormones shown to most significantly affect dental maturation, the sex hormones and GH, were found to be altered in this same patient group by Löppönen et al. (1995, 1996, 1997). Thus, the early dental maturation could be associated with early pubertal maturation because of increased levels of anterior pituitary sex hormones. The low levels of GH could partly explain the later delay in dental maturation.

The difference between the relative timing of dental maturation in children with HC in early and later pubertal stages could be explained, at least in part, by these humoral factors. This view is supported by the findings of Keller et al. (1970), who discovered that patients with endocrine and metabolic diseases leading to precocious puberty have a significantly advanced dental development, whereas in the case of anterior pituitary insufficiency, a short stature associated with retarded dental development is prevalent. The latter finding has been confirmed in subsequent studies (Myllärniemi et al., 1978; Krekmanova et al., 1997).

Dental development is a good marker of genetic regulation or developmental homeostasis because it is highly autonomous in nature and less prone to external influences than, for example, bone growth (Pirinen, 1995; Pelsmaekers et al., 1997; Van Erum et al., 1998). This phenomenon is seen in the very low correlation of dental and somatic development in the current study. It is likely that the greater deviation in the timing of dental maturation found in the present study is to a great deal dependent on the less strict genetic regulation of dental development.

In conclusion, dental development in shunt-treated children with HC deviates from that of healthy age- and sex-matched children. Dental maturation in children with HC was advanced in the early pubertal stages but retarded in the later pubertal stages.

It is suggested that the findings related to dental maturation are due to changes in endocrinological conditions but partly also an indication of less control of the developmental pathway in an otherwise strictly genetically determined process.

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