HYPOCALCIFICATION AND HYPOPLASIA IN PERMANENT TEETH OF CHILDREN FROM DIFFERENT ETHNIC GROUPS IN SOUTH AFRICA ASSESSED WITH A NEW INDEX

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ABSTRACT

A new descriptive index, the HHI (hypocalcification-hypoplasia index), is described for comparing enamel defects in groups of people. The index was used in a study completed in 1986, in which 1251 11-year-old children from different ethnic groups resident in South Africa were examined: 210 rural black, 203 urban black, 206 urban colored, 426 urban Indian, and 206 urban white. The index can be used as a screening examination, and the results from these different ethnic groups are presented.

INTRODUCTION

In assessments of dental diseases or conditions in children, the development of the teeth with defects caused by illness or trauma, or from general metabolic conditions, including nutrition, is of importance. Mellanby (1934), in defining her index of hypoplasia, related her findings to vitamin D and nutritional deficiencies. These studies have been taken further in recent years with the work of Nikiforuk and Fraser (1979) and Pimlott et al. (1985), who examined enamel defects in primary and permanent teeth of premature and low-birth-weight children.

Enamel opacities and hypoplasia have been shown to occur with excessive fluoride ingestion (enamel fluorosis) [Dean (1934, 1942); Thylstrup and Fejerskov (1978); and Horowitz et al. (1984)]. Russell (1961), Iizuka and Yasaki (1976), and Murray and Shaw (1979), among others, have examined the differential diagnosis of fluoride and non-fluoride enamel defects. Trauma to the primary teeth has been documented in the possible disturbance of the developing permanent teeth, resulting in localized hypoplasia or hypocalcification (Andreasen and Ravn, 1973; Hargreaves et al., 1981).

Cutress and Suckling (1982) gave an overview of indices that had been developed for the assessment of non-carious defects of enamel, and the Commission on Oral Health, Research and Epidemiology of the FDI suggested an index of enamel defects, the DDE index (1982). Results from studies which used this index have been given recently (Dummer et al., 1986; Suckling and Thurley, 1984; Suckling et al., 1985, 1987). Based on these studies, and on concerns raised for examination of enamel defects, a new descriptive index was developed and used in a study of different ethnic groups in South Africa.

MATERIALS AND METHODS

In 1986, 1251 11-year-old children from different ethnic groups resident in South Africa were examined — 210 rural black, 203 urban black, 206 urban colored, 426 urban Indian, and 206 urban white. The urban children all lived within the Greater Johannesburg metropolitan area, and all communities shared the same piped water supply (0.3 ppm fluoride), while rural black children were in five villages in the Ge-
# Hypocalcification/Hypoplasia Index

<table>
<thead>
<tr>
<th>Classification</th>
<th>Description</th>
<th>Anterior Teeth</th>
<th>Posterior Teeth</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No Hypoplasia or Hypocalcification</td>
<td><img src="image1.png" alt="Anterior Tooth" /></td>
<td><img src="image2.png" alt="Posterior Tooth" /></td>
</tr>
<tr>
<td>1</td>
<td>Hypocalcification in incisal or occlusal half of tooth crown</td>
<td><img src="image3.png" alt="Anterior Tooth" /></td>
<td><img src="image4.png" alt="Posterior Tooth" /></td>
</tr>
<tr>
<td>2</td>
<td>Hypocalcification in cervical half of tooth crown</td>
<td><img src="image5.png" alt="Anterior Tooth" /></td>
<td><img src="image6.png" alt="Posterior Tooth" /></td>
</tr>
<tr>
<td>3</td>
<td>Hypoplasia in incisal or occlusal half of tooth crown</td>
<td><img src="image7.png" alt="Anterior Tooth" /></td>
<td><img src="image8.png" alt="Posterior Tooth" /></td>
</tr>
<tr>
<td>4</td>
<td>Hypoplasia in cervical half of tooth crown</td>
<td><img src="image9.png" alt="Anterior Tooth" /></td>
<td><img src="image10.png" alt="Posterior Tooth" /></td>
</tr>
<tr>
<td>5</td>
<td>Hypocalcification &lt; ½ facial surface of a tooth crown</td>
<td><img src="image11.png" alt="Anterior Tooth" /></td>
<td><img src="image12.png" alt="Posterior Tooth" /></td>
</tr>
<tr>
<td>6</td>
<td>Hypocalcification &gt; ½ facial surface of a tooth crown or involving more than one surface of a tooth</td>
<td><img src="image13.png" alt="Anterior Tooth" /></td>
<td><img src="image14.png" alt="Posterior Tooth" /></td>
</tr>
<tr>
<td>7</td>
<td>Hypoplasia &lt; ½ facial surface of a tooth crown</td>
<td><img src="image15.png" alt="Anterior Tooth" /></td>
<td><img src="image16.png" alt="Posterior Tooth" /></td>
</tr>
<tr>
<td>8</td>
<td>Hypoplasia &gt; ½ facial surface of a tooth crown or involving more than one surface of a tooth</td>
<td><img src="image17.png" alt="Anterior Tooth" /></td>
<td><img src="image18.png" alt="Posterior Tooth" /></td>
</tr>
<tr>
<td>9</td>
<td>Hypocalcification/Hypoplasia not included in the above, e.g. diffuse hypoplasia restricted to a single surface other than facial</td>
<td><img src="image19.png" alt="Anterior Tooth" /></td>
<td><img src="image20.png" alt="Posterior Tooth" /></td>
</tr>
</tbody>
</table>

luksan district of Bophuthatswana some 350 km west of Johannesburg. Here, all the borehole water was sampled, and fluoride levels ranged from 0.18 to 0.35 ppm, which were within the same range when compared with findings obtained in another survey some six years earlier. Dental caries, enamel defects, height and weight, and details of each child’s diet were recorded. The nutritional and caries data will be reported elsewhere. The reason 11-year-olds were selected is that this was a base-line age group being
TABLE 1
HHI: CHILDREN 11 YEARS OF AGE

<table>
<thead>
<tr>
<th></th>
<th>n</th>
<th>% of children with hypocalcification/hypoplasia</th>
<th>% of children with &gt;4 teeth involved</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rural black</td>
<td>210</td>
<td>9.5</td>
<td>4.5</td>
</tr>
<tr>
<td>Urban black</td>
<td>203</td>
<td>19.7</td>
<td>7.0</td>
</tr>
<tr>
<td>Urban colored</td>
<td>206</td>
<td>16.0</td>
<td>6.3</td>
</tr>
<tr>
<td>Urban Indian</td>
<td>426</td>
<td>8.2</td>
<td>1.9</td>
</tr>
<tr>
<td>Urban white</td>
<td>206</td>
<td>8.7</td>
<td>3.0</td>
</tr>
</tbody>
</table>

used in a series of longitudinal assessments by the authors (Cleaton-Jones and Hargreaves, 1988).

Enamel defects were assessed by use of the new index, the HHI (hypocalcification-hypoplasia index), which is described in the Fig. The index was applied to each erupted permanent tooth and recorded enamel defects as either linear or as general (diffuse). In the format described, the recorders found no difficulty in interpreting or applying the criteria.

The teeth were examined under either natural light or with anglepoise lamps with 60-watt bulbs, depending on field conditions, and after the teeth were air-dried but not cleaned. Replaceable sickle probes were used, as previously described by Cleaton-Jones et al. (1989), to facilitate tactile detection of surface defects of the enamel.

When any doubt occurred about the presence or absence of a defect, the tooth surface was recorded as normal.

RESULTS

Of the children seen, the urban black and the urban colored children were most commonly affected by enamel defects and had prevalence levels approximately twice those in the other groups. In the different racial/ethnic groups, from 1.9 to 7.0% of children had more than four teeth with defects (Table 1). The percentages of teeth affected by each of the nine categories of HHI by ethnic group are shown in Table 2; since little variation was seen in homologous teeth, the data for the homologies have been combined. The most common lesions found were linear hypocalcifications or hypoplasias of the incisal or occlusal half of the tooth crowns (categories 1 and 3).

The percentage of total teeth involved in any of the ethnic groups was less than 5%, but up to 13.4% of specific teeth were affected. For example, maxillary central incisors showed the most enamel defects (Table 3).

DISCUSSION

The index was found to be easy to use and allowed for a rapid assessment of the children. As a screening tool, it can give an assessment of the number of children who have hypocalcified or hypoplastic teeth. In this study with the five different ethnic groups, the urban black and urban colored children had the most defects compared with the number of children affected; both of these groups had approximately twice as many children with defects compared with the other three groups. When the index was used to examine the teeth involved by category, the rural black, urban black, and urban colored had the highest percentage of teeth with hypocalcification or hypoplasia; the findings are based only on the teeth erupted. A further evaluation, examining the specific teeth grouped in homologous pairs, showed the urban black and urban colored to have the highest percentage of teeth affected in the maxilla, with over 13% of the central incisor teeth involved, and in the mandible the rural black and urban black had the most teeth involved. No consistency of defects in the maxilla and the mandible is shown, particularly the findings in the group of colored children.

The 11-year-old children examined in this study are a key group in current longitudinal assessments by the authors, but because not all permanent teeth are erupted in all children, a slight increase in teeth affected may be shown during future longitudinal studies with these same children. If only the anterior teeth and the first molars are assessed, all of these teeth being erupted in all children, again no obvious pattern is shown. For example, in the urban colored group, a higher involvement of maxillary anterior and first molar teeth is shown when compared with the mandibular teeth, although in all racial groups the maxillary teeth are more commonly involved than the mandibular teeth. An examination of the categories of the HHI for the urban black and urban colored children shows that enamel hypocalcification or hypoplasia of the incisal or occlusal third of a tooth was
the most dominant condition found with the maxillary incisors, the most common teeth involved. These teeth commenced development during the first year of life, and the position of enamel defects on the incisal portions involved the first areas of enamel to be formed.

These findings suggest, specifically if hypoplasia was present, that the most vulnerable time for the development of enamel defects in these communities is during the first year of life.

None of the children resided in an optimum fluoride region (all had water supplies of less than 0.35 ppm F), and fluorosis was not considered to be one of the contributing factors.

The children in the white and Indian communities resided in areas with good food availability and housing — conditions typical of European and North American residential areas. The rural black children lived in the homeland region of Bophuthatswana and maintained many of their traditional customs, including housing and diet. Many of these children were breast-fed for at least one year (Richardson et al., 1981).

The colored and black urban dwellers were essentially those with standardized sub-economic housing with some overcrowding. Food conditions, with many of their cultural traditions abandoned as they settled in urban regions close to the cities, had deteriorated. It is these two groups that showed the greatest number of enamel defects in their permanent dentition.

The index used, the HHI, was developed to make comparisons among children of different ethnic groups in South Africa, and direct comparison with other indices is not possible.

Many other results in the literature include the possibility of fluoride involvement (fluorosis), which is not a factor with this group of children. Wilson and Cleaton-Jones (1978), in examining enamel defects and infectious exanthemata, found no correlation between defects and the onset time of the illness. Suckling et al. (1987), in a study of more than 1000 New Zealand children, reviewed the literature of etiological factors that could influence the prevalence of developmental defects of enamel. They highlighted the influence of fluoride. Despite extensive statistical testing, positive and strong associations were few.

Their study illustrated the difficulty of establishing an etiology of enamel defects, even when medical and dental histories were available. Pimlott et al. (1985) speculated that the high prevalence of enamel hypocalcification in permanent teeth of premature and low-birth-weight infants is the result of metabolic stresses during the first few months post-term.

In the present study, the position of enamel defect supports an early onset age for the majority of defects found. Pimlott et al. (1985) described a much higher involvement of maxillary than of mandibular teeth; this relationship was also observed in the present investigation and has been reported by other workers (Grahnen et al., 1969, 1974; Funakoshi et al., 1981; Johnsen et al., 1984; Dummer et al., 1986). No satisfactory explanation for the greater involvement of maxillary teeth than of mandibular teeth has been reported. Thylstrup and Fejerskov (1978) have related fluoride defects to the thickness of enamel in different tooth types, and this may be a possible reason for maxillary incisors having more defects than mandibular incisors. Localized enamel and dentin defects in particular permanent teeth, following trauma or periapical infection in the primary tooth predecessor, are, however, well-documented (Hargreaves et al., 1981). Defects in enamel from areas with high fluoride levels in the drinking water are also well-documented and have recently been reported by Horowitz et al. (1984) in their assessments of dental fluorosis using a new fluorosis index. The mechanisms of fluoride in producing all types of enamel defects are not completely understood, nor are studies available that fully address them.
It was not the purpose of this study to examine medical records of the children or to relate defects to specific early nutritional deficiencies. It is clear that different patterns of dental defects of enamel are being found from these investigations, but firm etiological factors have not been detailed nor discrepancies resolved.

The need for further study in different communities to record numbers of people and individual teeth affected by enamel defects is apparent. Additional studies could give indications for further research to improve our understanding of the cause and possible prevention of enamel defects in developing and developed communities.

The results presented in this paper add to this body of information, and the HHI could be useful in allowing comparisons to be made within and among population groups.

REFERENCES


