

# Growing Pains: the Interpretation of Stress Indicators

MARY LEWIS<sup>†</sup> AND CHARLOTTE ROBERTS

*The Calvin Wells Laboratory, Department of Archaeological Sciences, University of Bradford, Bradford BD7 1DP, UK*

**ABSTRACT** An emphasis on the study of stress indicators in biological anthropology represents a move away from the identification of specific diseases to a more general analysis of malnutrition and infection in past populations. This paper reviews the current literature and discusses the methodological problems behind scoring and recording these conditions. Suggestions are made on how these problems may be addressed in the future. © 1997 by John Wiley & Sons, Ltd.

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## Introduction

An emphasis on the study of multiple indicators of stress in skeletal and dental material represents a move away from the identification of specific diseases to a more general analysis of factors such as malnutrition and infection. The stress indicators most commonly recorded in biological anthropology include: enamel hypoplasias and Harris's lines, defined as defects formed on the teeth and long bones during a growth disturbance; cribra orbitalia and porotic hyperostosis, most likely indicative of iron deficiency anaemia in Northern European populations;<sup>1</sup> non-specific periostitis and, more recently, endocranial lesions.<sup>2</sup> These bone lesions have all been associated with disease and malnutrition in modern populations.<sup>3–5</sup>

During an analysis of non-adult skeletons from past rural and urban Medieval populations, certain problems with the aetiology, methods and interpretation of stress indicators have

become apparent. This paper examines these problems and suggests some ways in which they may be overcome in the future.

## Dental enamel hypoplasias

Enamel hypoplasias are recorded as grooves or pits on the deciduous and permanent dentition. The exact aetiology of these markers is unknown, but in modern studies they have been linked to fever, starvation, congenital infections, low birthweight and parasitic infection.<sup>6–9</sup>

Enamel hypoplasias have attracted a lot of attention in the literature because the nature of enamel means that defects cannot be remodelled and, therefore, they represent a permanent chronological record of a stressful incident during the first 7 years of life.<sup>5</sup> The age of the individual at the time that a defect was formed is usually calculated by taking a measurement of the distance from the cemento-enamel junction (CEJ) to the line. These measurements are then converted into biological age by estimating the number of years it takes the growth of the crown to be completed and into which half-year of development the defect falls.

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<sup>†</sup>Author to whom correspondence should be addressed.  
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Numerous studies of American and European populations indicate that the peak in development of these defects is 2–4 years; this time period fits neatly with the age of weaning in most developing societies. Therefore, stress caused by a change in the quality and quantity of the food supply has been implicated in the formation of these lines.<sup>10–13</sup> However, doubts about this interpretation have been raised. In 1994, Blakey and colleagues published a study carried out on the skeletons of nineteenth century African American slaves.<sup>14</sup> Although it is known, from documentary evidence of similar sites, that the females were forced to wean their children between the ages of 9 and 12 months, the highest frequency of these defects occurred up to 3 years after that period. The study by Blakey and colleagues has raised questions about the validity of the weaning hypothesis, and proposes that other environmental factors may also be responsible and should be considered.

It has also been suggested that during the 2–4 year developmental period the enamel is more susceptible to environmental disturbance. In 1984, Suckling and Thurley indicated that the longer an ameloblast secretes the enamel matrix, the more vulnerable it is to an insult.<sup>15</sup> It may be that between the second and fourth year of enamel development the activity of the ameloblasts is slowing.

Even if the weaning hypothesis is accepted, problems with the age assessment of the defects exist. In 1980, Goodman and colleagues constructed a table with mean ages for crown development based on data published by Swärdstedt and Massler.<sup>16–18</sup> This method is now widely used for assessing the age at formation of a defect but assumes that tooth development rates in *deceased* ancient children are similar to today's modern *healthy* populations, that this development rate is consistent between individuals and populations and, that tooth size variation has no impact on the age estimation of the hypoplasia.

Comparison between ancient and modern populations is an unavoidable problem in archaeology but can be reduced by using a standard of dental development comparable to the archaeological sample being studied. Children who entered the archaeological record,

i.e. died before they reached adulthood may be expected to have a delayed development, reflecting the impact of the environmental stresses that eventually killed them. However, the majority of children probably died as a result of acute infections and accidental death, which would not be expected to affect their dental development patterns.<sup>19</sup>

Goodman and colleagues recommend Massler's standards for crown completion in order to ensure comparability between studies.<sup>20</sup> However, if these defects are to be incorporated into a wider study of stress indicators in a given population, the standard used for estimating crown height completion should be the same as the standard used for age estimation of the sample and for constructing the mortality profile.

In 1990, Hodges and Wilkinson<sup>21</sup> concluded that tooth size variation between teeth and populations does have an impact on the age assigned to the hypoplastic defect.<sup>21</sup> In a non-adult sample, the crown height of the affected tooth could be used to age each individual hypoplasia. Unfortunately, in an adult sample these measurements may be hindered due to attrition, which would alter the original dimensions of the crown. Therefore, for an adult sample it is advised that, rather than using a fixed conversion chart that assumes an equal tooth size, the mean crown height for each sample be used to estimate the age of line formation. The mean should be derived from measurements of teeth unaffected by attrition and applied to the following equation:<sup>20</sup>

$$\text{Age at formation} = \text{age at crown completion} - [(9 \text{ years of formation/crown height}) \times \text{defect height (from CEJ)}]$$

### Harris's lines

Work incorporating Harris's (transverse) lines as indicators of stress has been hindered in adult skeletal populations due to the unpredictable and frequent remodelling of the lines.<sup>22,23</sup> For this reason, a juvenile population is preferable when investigating the frequency and age distribution of line formation within a population.

The use of Harris's lines to estimate the frequency of stress episodes is still fraught with problems. In 1969, Gindhart showed that diseases were only followed by a line in 25 per cent of cases, and 10 per cent of the lines occurred when no stressful episode was documented.<sup>24</sup> Males and females have different rates of remodelling, and stresses as mild as an inoculation or as severe as malnutrition may cause a cessation of growth and the development of a line.<sup>25</sup> It has yet to be decided whether the thickness of a line is a measure of stress severity and longevity.<sup>26</sup> Also, the individual would need to recover from the stress in order to display a line. Therefore, continually stressed people may not have visible transverse lines.

The method of determining the age of an individual at the time of line development is very similar to that employed in the age estimation of enamel defects, where the growth rate is estimated using modern standards. Although the method has recently been refined by Maat, it employs the same assumptions as the hypoplasia ageing method.<sup>27</sup>

Perhaps the most worrying aspect of studying Harris's lines was demonstrated by a study carried out by Macchiarelli and colleagues who showed that there are major discrepancies in both inter-observer and intra-observer analysis when counting the number of lines present on a bone.<sup>28</sup> Until stricter rules can be applied to the identification of these lines, any attempt to compare the number of episodes of stress in different populations and from previous studies seems futile. It may be better to merely score presence and absence of these lines in zones relating to different periods of development and, if possible, to limit the study to non-adult populations.

### **Cribræ orbitalia and porotic hyperostosis**

Porous lesions found on the cranial vault known as 'porotic hyperostosis' and on the superior aspects of the orbit termed 'cribræ orbitalia' are thought to be indicative of iron deficiency anaemia as a result of malnutrition, chronic blood loss, parasitic infestation or an increased pathogen load.<sup>1,29</sup>

Increased pathogen load is the most recent area of investigation into the aetiology of these lesions. It is suggested that acute and chronic infections may stimulate the immune system to withhold iron from invading microorganisms as a defence mechanism.<sup>30</sup> Until recently, these lesions were thought to be useful indicators of a dietary deficiency of iron in past populations making the transition from a hunter-gatherer to an agricultural subsistence.<sup>31-33</sup> However, clinical studies and trace element analyses have begun to question the validity of this association.<sup>34</sup>

One of the major problems in investigating the frequency of these lesions in other populations concerns the use of terminology. Some researchers do not make a distinction between the two lesions and refer to both as 'porotic hyperostosis', a phrase first coined by Angel in 1966.<sup>35</sup>

Few European studies have recorded vault lesions of similar severity or frequency compared to the North American population studies, even though orbital lesions are common. In 1991, Wiggins assessed the association of these lesions in some British skeletal populations but found no significant correlation between vault and orbital lesions.<sup>36</sup> Wiggins concluded that orbital lesions may represent a milder form of iron deficiency anaemia, that the orbital and vault lesions found in the USA are of a different aetiology to those found in Europe or that orbital and vault lesions were not of the same aetiology. It is recommended that the vault and orbital lesions be recorded under different names until the association between them can be clarified.

### **Non-specific periostitis and endocranial lesions**

Periostitis and lesions on the endocranial surface of the skull are both used as indicators of non-specific infection or trauma but share similar difficulties with interpretation and identification in non-adult populations.

Non-specific periostitis is defined as a new layer of bone on the cortical surface deposited under an inflamed periosteum as the result of trauma or infection. Endocranial lesions are a

more recent area of investigation and can appear as 'worm-like' deposits of new bone, vascular depressions or 'hair-on-end' formations.

In the long bones, appositional growth involves the deposition of immature disorganized bone on the periosteal surface. This new bone is macroscopically identical to the 'woven' bone deposited during an infection or after trauma. Similarly, during the neonatal and infant periods the skull is expanding rapidly to accommodate the developing brain and new layers of bone will be deposited on the surface of the skull vault. In order to progress with studies into infant mortality, there is a need to be able to distinguish between new bone deposited during growth and that deposited during an inflammatory insult.

In the few studies that mention non-adult periostitis it is recognized as a unilateral, isolated patch of bone raised above the cortex.<sup>37,38</sup> However, if the deposits are the result of a more widespread infection, the diffuse deposits of periostitis will be indistinguishable from rapid appositional growth. Birth trauma, child abuse, syphilis, rickets, hypervitaminosis A and cortical hyperostosis (Caffey's disease) can all occur in the newborn and infant but are rarely recognized archaeologically.<sup>39-43</sup>

The various appearances of endocranial lesions may indicate different aetiologies; meningitis, epidural haematomas, birth trauma, scurvy, venous drainage problems and tuberculosis may all cause inflammation or haemorrhage of the meningeal vessels.<sup>2,40</sup> However, until modern or museum specimens with these conditions associated with clinical histories can be located and described, it will be difficult to identify or interpret endocranial lesions and periostitis in the youngest skeletons.

Scanning electron microscopy and histological analysis of the lesions to aid interpretation is not always practical, and therefore there is a need to establish a more detailed account of when and where bone growth occurs in order to ascertain the 'normal' appearance of bone for the different age groups. For the present it may be useful to subgroup those individuals suspected of displaying endocranial or periosteal lesions by age. Thus, new bone deposits laid down during the growth period should occur in the same area of the skeleton for that age group, and any

unusually placed deposits may then be more accurately identified as pathological.

## Conclusions

The examination of stress indicators in skeletal populations is an important area of research in biological anthropology. However, there is a need to reassess some of the basic assumptions about their aetiology and to be aware of the multiple factors that often contribute to their appearance. It is advisable to be conservative with the interpretations and to understand the origins of the methods used. The choice of modern standards derived from populations of similar geographical origin to the skeletal sample is essential. A standard for terminology would allow cross-cultural comparisons to be undertaken and, in the future, help to expand our knowledge of childhood morbidity and mortality and the experience of stress in past populations.

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