

## Dental enamel growth, perikymata and hypoplasia in ancient tooth crowns

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### Summary

This paper describes the hypoplastic defects commonly seen on the surface of ancient human tooth crowns, excavated from archaeological sites, and presents a new method for estimating the ages at which these defects were initiated during life. The method is based upon examination of microscopic incremental structures on the enamel surface and it is possible also to apply it to reconstruction of the sequence and timing of dental crown development. The method of examination is non-destructive and allows full use to be made of the large numbers of complete, unworn dentitions which are found amongst archaeological remains.

### Introduction

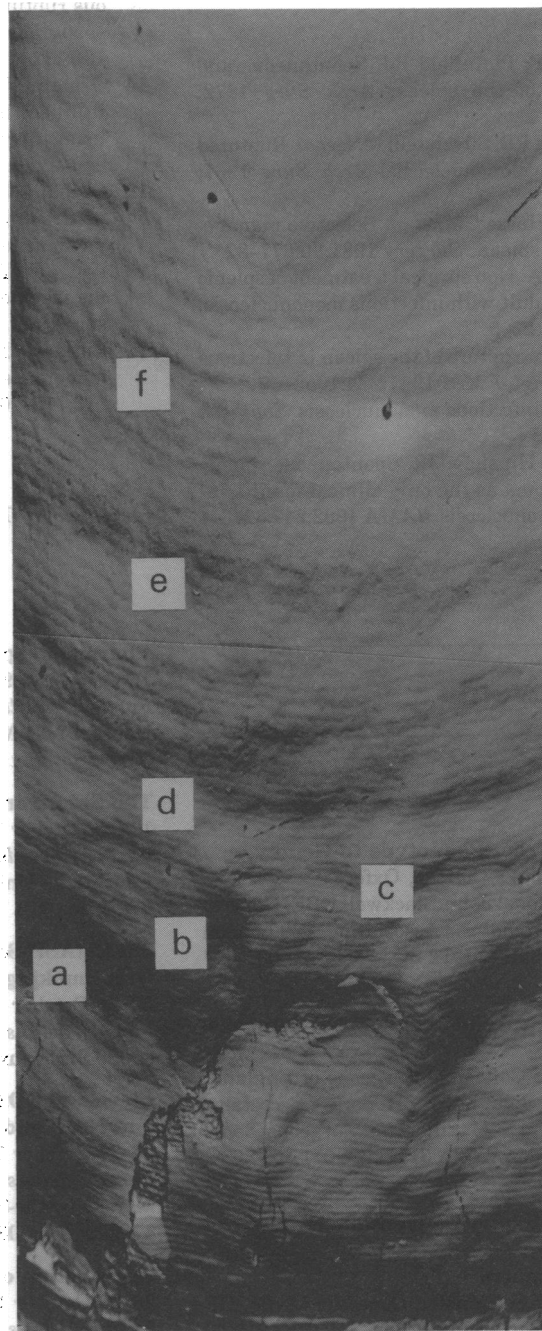
Very large numbers of excavated burials are available for study in museums and excavation archives. Tooth crowns are the most resistant, and therefore the best preserved, elements of human remains under most conditions of burial. They form a potentially very valuable resource for the study, not only of dental pathology, but also the processes of dental development.

#### *Defects of dental enamel in archaeology*

Defects of the crown surface are very commonly seen in archaeological dentitions<sup>1</sup>. Almost entirely, they are defects of the enamel surface contour (hypoplasia), ranging from minor perturbations to major disruptions (Figure 1) and even patches devoid of enamel. The opaque white patches normally known as hypocalcifications, which are so common amongst living people, have not been described in archaeological material. This may partly result from archaeological processes because, if such white patches are due to deficient mineralization of enamel, the mineralogical changes occurring during burial could well obliterate them.

The defects of enamel most commonly reported in archaeological tooth crowns have the following general characteristics:

- They are a clear alteration to the profile of the crown, which can be seen in oblique lighting and felt with a fine probe, or demonstrated by rubbing a soft graphite pencil lead over the surface.
- They follow the pattern of crown surface layering (the perikymata, see below) by running around the circumference of the crown. In most studies, defects which cannot be followed round at least two sides of the crown would not be recorded.
- Each defect represents a clearly delimited episode, marking out a band of enamel within the growth sequence of the crown.



*Figure 1. Perikymata and developmental defects on the buccal crown surface of the permanent lower second molar of a young man buried at the Predynastic cemetery of Badari, in Egypt. The labels a, b, c, d, e and f mark defects described in the text. Crown surface replica, examined in a Hitachi S570 scanning electron microscope, operated in secondary electron mode at 15 kV. The specimen comes from the collection of the Duckworth Laboratory, Faculty of Archaeology & Anthropology, University of Cambridge. Width of field is 2.3 mm*

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- Defects can be matched between the crowns of teeth, from the same individual, which would have been growing at the same age. Most researchers now ignore any defect which cannot be matched in this way, at least with the corresponding tooth from the other side of the jaw.

From these characteristics, the defects are clearly related to the varying physiology of tooth crown growth and development. The traditional name for them is enamel hypoplasia, which implies defective growth. Few of the defects so far described in archaeological material are likely to be related to genetically inherited disorders. Such genetically determined defects (amelogenesis imperfecta) are rare in living human populations<sup>2</sup> although locally they may be somewhat more abundant. Unless an excavation had uncovered a group of closely related people who shared a condition of this type, statistically significant frequencies of amelogenesis imperfecta could not be expected in archaeology. Most types of amelogenesis imperfecta also differ from the form of defect described above.

Since the first published descriptions of developmental defects of enamel, various forms have been recognized. Dr Otto Zsigmondy of Vienna, who first proposed the phrase 'hypoplasia of the enamel' in his paper to the 1893 World's Columbian Dental Congress at Chicago<sup>3</sup>, described furrow like defects, bands of small pits, and zones of missing enamel which exposed the dentine underneath. Bertin<sup>4</sup> added plane-form defects, where a whole layer of enamel was missing, without necessarily exposing the dentine. Lady May Mellanby, who carried out a classic series of experiments on hypoplasia in beagle dogs, grouped the defects involving pitting and the deficient formation of large areas of enamel as gross hypoplasia<sup>5</sup>. Since that time, it has become common to use the term M-hypoplasia (M for Mellanby) for furrow-like defects and G-hypoplasia to describe the gross defects<sup>1</sup>. M-hypoplasia may involve a single furrow around the crown, or a whole group of them in a wide band (Mellanby's washboard pattern). G-hypoplasia may be just a single line of small pits, a band several pits wide, a band of pits which have coalesced together, or the various types of defect where whole layers of enamel are missing. This classification is really one of convenience and, in spite of more than two centuries of epidemiological and experimental study, it is still not known what the different types of defect signify in terms of the level of disturbance which occurred during crown growth.

One of the difficulties in finding the answer to such questions is in the level of recording. The defects which are routinely recorded are those large enough to see clearly with the naked eye, or feel with a probe. They represent, however, just one end of a continuum which ranges from a microscopic perturbation of just one or two crown growth layers up to defects which involve almost the whole crown. Different observers and different conditions of observation produce different results. Standards for recording such as the Commission on Oral Health, Research and Epidemiology's<sup>6</sup> developmental defects of dental enamel (DDE) index, do not specify the size a defect must be before it is recorded<sup>7</sup>. One of the reasons that enamel hypoplasia is so common in archaeological material could well be that the teeth are seen dry, in ideal conditions of lighting where the jaw can be

oriented so that the light falls obliquely across the crown, and often with magnification such as a low-power stereomicroscope. Under these conditions, it may be that smaller defects are more easily observed than in living subjects.

Yet another problem is that defects, which apparently represent the same disturbance to growth, vary in appearance and prominence between the crowns of different tooth types, between antimeres of the same tooth type and even between different surfaces of the same tooth crown. In such a case it may be hard to decide which level of prominence should be recorded.

If the defects of enamel hypoplasia are related to disturbances experienced during the development of tooth crowns then, where dental development follows a reasonably constant pattern in different individuals (and traditionally it is assumed to do so) it should be possible to estimate approximately the age at which the disruption which caused an individual defect occurred. Various methods have been established for this, the most widely used of which trace their ancestry back to the work of Logan and Kronfeld<sup>8</sup>, who examined decalcified, stained whole jaw sections from the post-mortem remains of 25 children. Schour and Massler<sup>9</sup> published a table, based on Logan and Kronfeld's results, giving age ranges for the start and finish of tooth crown and root formation, followed by line drawings<sup>10</sup> defining four growth 'rings' in the layered enamel structure of tooth crowns - neonatal (birth to 2 weeks of age), infancy (10 months), early childhood (2.5 years) and later childhood (5 years). The positions of these rings on the surfaces of different tooth crowns have been used by many to estimate the age at which defects occurred<sup>7</sup>. Logan and Kronfeld clearly indicated<sup>8</sup> the statistical uncertainties involved in estimating normal crown formation timing from 25 terminally ill children, only 10 of whom were over one year of age at death, but all the large-scale studies of tooth development in modern children since theirs have been based upon radiography. This is not suitable for precise determination of crown surface formation timing and no new standards have been defined which could be used to study hypoplasia. An alternative approach is needed.

#### *The epidemiology of developmental defects*

Many experiments, largely carried out during the first half of the 20th century, have demonstrated that the defects of enamel hypoplasia can be linked to both diet and disease. Lady May Mellanby<sup>5</sup> demonstrated that vitamin A and D deficient diets could cause hypoplasia in beagle dogs. Kreshover's<sup>11</sup> work showed that experimentally induced diabetes and fever in pregnant rats disrupted enamel formation in their offspring and, when rats were inoculated with pathogenic viruses and bacteria, defective enamel was formed.

Clinical studies have long linked the defects with dietary deficiencies and childhood infectious diseases. In 1746 Robert Bunon<sup>12</sup> published a study in which the defects were found in the unerupted teeth of dead children who had suffered from rickets, scurvy, measles and smallpox. Studies during the present century have concentrated largely on defects formed around birth and during early infancy. Schour and Kronfeld<sup>13</sup> noted the neonatal ring, a defect which could be linked to the time of birth, and was more pronounced in children with birth injury. The

deciduous incisors of children from the less fortunate socioeconomic groups in many parts of the world are marked by a prominent furrow-form defect, called linear enamel hypoplasia, which also appears to correspond to the time just before or around birth<sup>14</sup>. Other studies have linked hypoplasia in deciduous teeth with hypocalcaemia of the kind caused by vitamin D deficiency, allergies, premature birth, various congenital defects, neonatal haemolytic anaemia, maternal rubella during pregnancy and maternal diabetes<sup>14</sup>. Hypoplastic defects of permanent teeth are amongst the classic stigmata of congenital syphilis - Hutchinson's incisors, Moon's molars and Mulberry molars - all in parts of teeth that would start to form at birth or during the first postnatal year.

It is more difficult to find clinical evidence of a relationship between the diseases of later childhood and defects of enamel hypoplasia. Children with severe rickets have both higher frequencies of defects and more pronounced defects than non-rachitic children, and defects are found more frequently than normal in children with a history of non-specific diarrhoea<sup>14</sup>. In some instances episodes of diarrhoea can be matched with particular crown defects. The classic study attempting to match defects in the teeth with medical records is that of Sarnat and Schour<sup>15</sup>. Their records included rickets, chickenpox, convulsions, diarrhoea, diphtheria, measles, pneumonia, scarlet fever, vomiting and whooping cough, but in only 29 out of 60 children did Sarnat and Schour match these episodes with dental defects. The poor degree of concordance in this study has undoubtedly discouraged further work, but much depends upon the level of defect which is recorded as hypoplasia and the methodology by which the age at which the defect occurred is estimated. An unpublished study by the author and Dr R M Hillson suggested that 70% of defects could be matched to episodes in the medical records of young men. The question must, however, remain open until more reliable and objective methods for recording and ageing defects can be applied.

#### *Incremental structures within the enamel and on the crown surface*

Possible solutions to the difficulties outlined above may lie in studies of the layered patterning of enamel growth. Growth layering is seen both within the enamel and at the surface. In ground and polished sections of enamel, examined in transmitted light microscopy, the most prominent layering is marked out by dark, fuzzy bands known as the brown striae of Retzius<sup>16</sup>. They represent a regular growth rhythm<sup>17</sup> whose physiological basis is not fully understood, but which is regular enough to use as the basis of crown growth studies. In any tooth, the first increments of enamel are laid down on top of one another, each successive layer hiding the previous layers so that there is no evidence of them at the surface of the crown. The number of increments included in this hidden zone varies between teeth, but at some point in the growth of each crown, the tops of the layers open out and the most occlusal edge of each layer becomes visible at the surface. In each layer, this edge is laid down slightly to cervical of the preceding edge, giving rise to the pattern of crests and grooves (perikymata or imbrication lines) seen at the surface. Each brown stria of Retzius (in the non-

hidden zone) is represented by a perikyma groove which extends around the whole circumference of the tooth crown<sup>18</sup>. Counts of perikyma grooves may therefore be used to represent counts of the underlying brown striae. The proportion of brown striae which are represented by perikyma grooves varies between tooth classes. Work with sections from a large number of freshly extracted teeth<sup>19</sup> has demonstrated that, in permanent incisors or canines, the last 80-90% of the total count of brown striae within the crown emerge at the surface to produce matching perikyma grooves. In permanent molars, only 50-70% of brown striae reach the crown surface in this way. Work with replicas of archaeological specimens has shown that the different tooth classes are also characterized by differences in the pattern of perikyma groove spacing down the crown (below).

Crown development chronologies based upon counts of brown striae and perikyma grooves have been used to estimate age-at-death in both archaeological and fossil hominid specimens. Gustafson<sup>20</sup> found that the brown striae varied in prominence to produce a consistent pattern that could be matched between different teeth from one individual. Boyde<sup>21</sup> examined the teeth of an Anglo-Saxon child, whose permanent first molar crown had been completed, but whose permanent first incisor was still being formed at the point of death. In sections of these teeth, it was possible to recognize the neonatal line, a particularly prominent brown stria corresponding to the point of birth, under the cusps of the first molar. Counting from this, an estimate could be formed of the age at which the first molar crown had been completed. The pattern shown in the later part of brown stria sequence in the first molar could be matched with the earlier part of the sequence seen in the first incisor and, counting on from this, an estimate could be made of the age at which this crown ceased formation, and thus the age at which death occurred.

This general idea has been applied to perikymata by Dean and colleagues<sup>22,23</sup>. Sections of modern extracted first incisor teeth were used to establish the mean number of brown striae from the first phase of crown formation which were concealed under the incisal edge of the crown. This combined with an estimate for the start of crown formation taken from the literature, provided an estimate for the mean age at which the first perikyma groove appeared on the surface of first incisors. Replicas of the surface of first incisor crowns were prepared for a number of immature hominid fossils and perikyma grooves were counted up to the point at which crown formation stopped. From this, it was possible to calculate an estimate of the age-at-death.

In the present research, perikyma groove counts are used in a similar way to provide a chronology for the sequence of defects seen on crown surfaces, and to build up a chronology of the sequence of crown formation.

#### **Methods of study**

Archaeological specimens have particular advantages for the study of perikymata and crown surface defects. All teeth can be examined in microscopic detail, and can be cleaned very thoroughly to present an excellent surface for examination. They do not have the severe toothbrush abrasion seen in living dentitions, so that perikymata are clearly visible even in young adults. Occlusal attrition is usually more severe than in

living dentitions but, as the perikymata are less densely spaced in the occlusal part of the tooth crown (see below), this destroys fewer perikymata and defects than toothbrush abrasion of the cervical region does. The best specimens are yielded from young individuals, whose teeth were just erupting when they died, or whose crowns can be dissected from the jaws with little damage. An almost complete set of unworn crowns is often available. All these factors allow detailed studies that would not be possible outside archaeological material. Large numbers of specimens are available in Britain and the present study has examined Egyptian material from the Faculty of Archaeology and Anthropology at the University of Cambridge, together with Roman, Medieval and Post-Medieval material from London, made available by the Greater London Environmental Archaeology Section of the Museum of London.

After initial examination using a low-power stereomicroscope at magnifications of  $\times 15$  and  $\times 30$ , specimens were selected for a low degree of attrition and abrasion, and for the presence of prominent defects for study. After cleaning with absolute alcohol swabs and pointed hardwood sticks (to avoid scratching), impressions of the crown surfaces were taken using Coltène President, a widely employed dental impression material. Replicas were cast in these impressions with Ciba-Geigy Araldite MY 753 resin with XD 716 hardener. The method was based upon Beynon<sup>24</sup>, although some different materials and procedures were used. The resolution of these replicas is better than  $1\ \mu\text{m}$ , which is ample for the present study. Two sets of replicas were made for each specimen. One set was sputter coated with gold and examined in a Hitachi S 570 scanning electron microscope. The other set was left in its original translucent state and examined by both transmitted and reflected light microscopy. Profiles of perikymata spacing were produced by a modified engineers' measuring microscope, described in Hillson and Jones<sup>25</sup>.

## Observations

### *The overall pattern of crown surface growth*

Figure 2 shows a perikyma groove spacing profile taken from the permanent lower second molar of a young man from the Predynastic cemetery of Badari in Upper Egypt. The crown is only slightly worn at the occlusal cusp tips and the full series of grooves

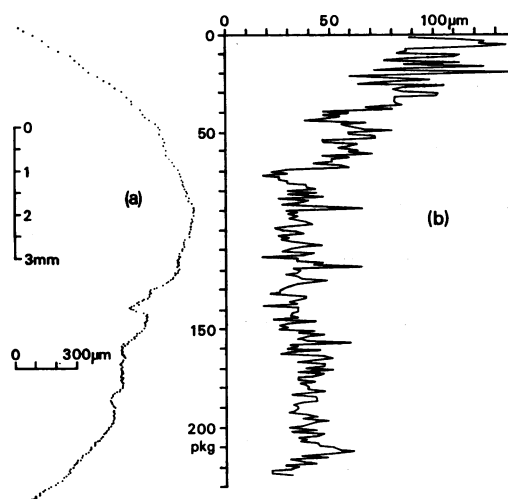


Figure 3. (a) Perikyma groove profile and (b) spacing graph for a permanent lower canine from the same individual as the second molar in Figures 1-2. The plots represent the labial crown surface. Scales and organization as for Figure 2

seems to be present. A maximum of 106 perikyma grooves could be counted in the second molar crown. In the profile chosen to represent this crown, the first four grooves to appear at the occlusal tip of the cusp are widely spaced - over  $150\ \mu\text{m}$  apart - and easily visible with the naked eye. After this, the spacing decreases to around  $40\text{--}60\ \mu\text{m}$  at the 23rd perikyma groove and  $20\text{--}30\ \mu\text{m}$  at the 45th. The remaining 61 grooves all remain at approximately this spacing, to form the cervical third of the crown surface.

Figure 3 shows a similar profile taken from the permanent lower canine of the same individual. The first perikyma grooves are more modestly spaced - never more than  $135\ \mu\text{m}$  - and their spacing decreases gradually to  $30\text{--}50\ \mu\text{m}$  at about the 70th groove (with some departures outside this range). The remaining 157 grooves continue at approximately this spacing, to form the cervical two-thirds of the crown surface.

Observations similar to these have been made in many other crown surface profiles, with a pattern similar to the second molar being shown in other molars and a pattern similar to the canine being shown in both canines and incisors.

### *Irregular fluctuations in perikyma groove spacing and defects*

In the profiles shown in Figures 2 and 3, minor and irregular changes in spacing are superimposed over the regular pattern of spacing. Many of these have a random appearance, but others can be related to defects seen on the surface of the crown. Figure 1 is a scanning electron micrograph of the crown surface of the second molar plotted in Figure 2. Six clear furrow-type defects, labelled a to f, can be seen. The same defects can be made out in the profile and spacing plots for this tooth. Defects f and e are only minor interruptions to the regular progress of crown surface formation, each involving between two and five perikyma grooves. The three defects b to d are close together, forming a corrugated band, and each involving between five and seven perikyma grooves. a is probably the only defect that would be recorded at a clinical level as M-hypoplasia and it involves 22 grooves. The way in which each furrow-type defect has been formed is apparent from the spacing graph, which shows perikyma grooves to be wider apart on

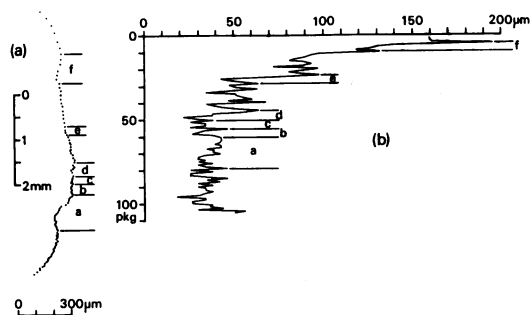


Figure 2. (a) Perikyma groove profile and (b) spacing graph for the buccal crown surface of the permanent lower second molar shown in Figure 1. The most occlusal point of the cusp is at the top of the profile and the cervical margin at the bottom. Each dot in (a) shows the point at which a perikyma groove was encountered along a line down the crown replica. The vertical axis of (b) shows perikyma groove pkg counts. Labelling as in Figure 1

the occlusal face of the furrow, and closer together on the cervical face. This is particularly clear in defect a, where the first 13 grooves of the defect are considerably further apart than the remainder.

Many other specimens have shown a similar variation in the number of perikyma grooves included in furrow-type defects. Even the smallest of these defects can be followed round onto other surfaces of the tooth and matched on the crown surfaces of other teeth which were formed at the same time. They therefore comply with the definition of developmental defects given above, even though they would not be scored as such in a routine clinical study.

*Matching defect and perikyma groove sequences*

Figure 4 is a scanning electron micrograph of a crown surface replica taken from the permanent upper canine of a child buried in the Medieval cemetery of the Dominican Priory at Carter Lane in London. There are several furrow-type defects visible on the micrograph, but few of them would be diagnosed as

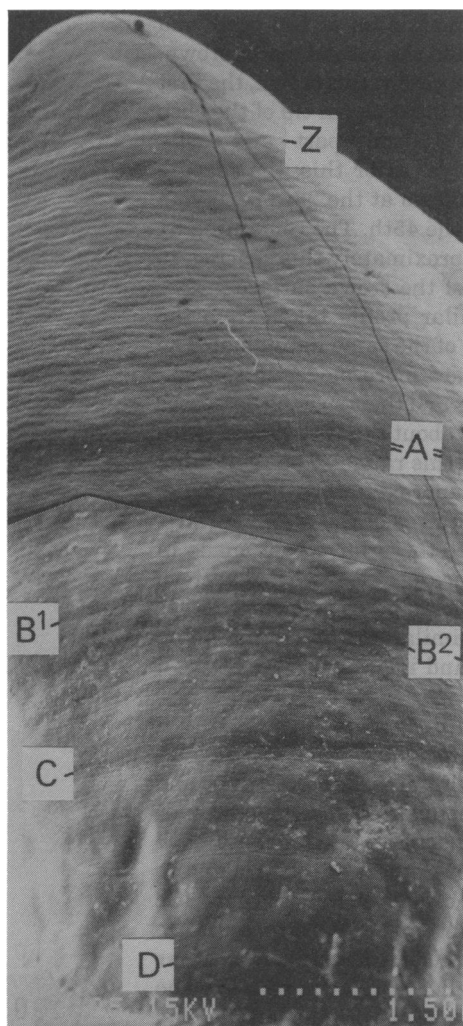


Figure 4. Perikymata and developmental defects on the labial crown surface of the permanent upper canine of a child (PIC87 106) from the Medieval cemetery of the Dominican Priory at Carter Lane in London. It forms part of the collection of the Greater London Environmental Archaeology Section at the Museum of London. The most occlusal point is towards the top of the photograph. The labels Z, A, B<sup>1</sup>, B<sup>2</sup>, C and D mark small developmental defects described in the text. Crown surface replica, examined in a Hitachi S570 scanning electron microscope operated in secondary electron mode at 15 kV. Width of field is 3.8mm

defects from routine surface examination. Just below the cusp is a minor defect labelled Z involving only two perikyma grooves. Further down to cervical is a slightly larger defect whose occlusal edge is marked by two very prominent grooves which have been labelled A. The ensuing defect involves some eight perikyma grooves. Further still to cervical, two defects are labelled B<sup>1</sup> and B<sup>2</sup>. Each is little more than single accentuated perikyma groove, separated by five more normal grooves. Ten perikyma grooves to cervical of B<sup>2</sup> is a further small defect called C, involving three grooves. Finally, just above the cement-enamel junction is a single accentuated perikyma groove labelled D, at a count of 38 grooves after C. The full canine sequence involves 145 perikyma grooves.

Parts of the canine sequence are matched very closely in the permanent upper central incisor, upper first molar, lateral incisor, first premolar and second molar of the same individual. The count of perikyma grooves both within the defects, and in between them, coincides almost exactly (Figure 5). Adding together the matched sequences, a combined sequence of 231 perikyma grooves can be constructed for this individual, extending through three intervening tooth crowns from the first perikyma groove to be formed on the permanent first molar to the last perikyma groove to be formed on the second molar.

Similar matched sequences have been constructed for a number of other individuals, and more are being constructed. As the method involves minor defects as well as very prominent defects, only very rarely are there too few defects for the sequences to be established. The example given here involves only furrow-type (M-hypoplasia) defects, but pit-type (G-hypoplasia) defects can be matched in the same way.

**Interpretation**

It has long been clear that large defects of the type routinely diagnosed as enamel hypoplasia can be

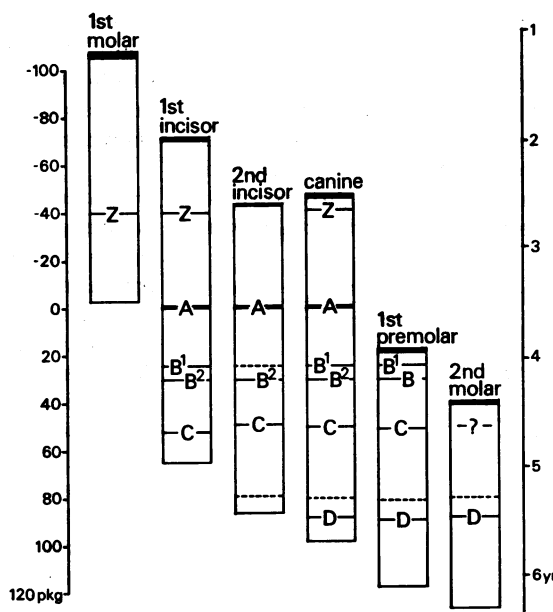


Figure 5. Perikyma groove counts and defect matches for the permanent upper first molar, central incisor, lateral incisor, canine, first premolar and second molar of the individual PIC87 106 shown in Figure 4. Left hand vertical scale gives perikyma groove pkg counts relative to the prominent pair of perikyma grooves labelled A. Right hand vertical scale gives estimated age in years after birth

matched between different teeth from one individual, demonstrating the systemic nature of the disturbance causing the defects. Zsigmondy<sup>3</sup> and Berten<sup>4</sup> both noted this phenomenon. The observations described above, together with others carried out by the author on archaeological material, demonstrate that the large defects are part of a continuum which includes defects involving as few as one single perikyma groove. These are unlikely to be diagnosed at a routine clinical level as enamel hypoplasia, but it has nevertheless proved possible to match consistently between teeth not only the defects, but also the counts of perikyma grooves within and between them. This indicates that the microscopic defects are also related to systemic disturbances and, in view of the continuum between them and larger defects, are likely to be related to growth disturbances similar to those discussed above.

The perikymata, to which the defects are related, represent a consistent growth rhythm. It is possible to suggest that the rhythm is a regular one from recent studies of the regular pattern of light and dark banding (at 2–5  $\mu\text{m}$  intervals) known as prism cross striations, seen in transmitted light microscopy of ground, polished sections of enamel. Cross striations are seen in a variety of different types of microscopy and appear to be represented in scanning electron microscope images of fractured enamel surfaces by an alternate constriction and varicosity of the enamel prisms<sup>16</sup>. There is both circumstantial and experimental evidence that the cross striations so defined represent a 24 hour rhythm of enamel growth<sup>16,17</sup>. Three recent studies<sup>19,22,26</sup> have made counts, in ground polished enamel sections, of cross striations along prisms which pass through a number of brown striae of Retzius. The overall arithmetic mean count between pairs of brown striae is 7.87 cross striations (with a standard error of 0.219), suggesting a rhythm of approximately 8 days for the layering represented by the brown striae and perikymata. The minimum cross striation count in the three studies was six and the maximum, 10. This variation might be due to fluctuations in the underlying growth rhythm, but it is as likely to be due to difficulties in observation. Whilst they can themselves readily be counted, the brown striae as seen in transmitted light microscopy are ill defined, rather fuzzy structures that become less clearly visible at higher powers of magnification. It is often difficult to identify precisely the point at which to stop counting. There is also a possibility of misidentification of cross striations but, with careful observation, this can be minimized<sup>16</sup>. When all observations are taken into account, the variation shown in the studies summarized above is in any case relatively small, because the standard error yields a 95% confidence interval of 7.4–8.3 cross striations.

One way to test the periodicity of brown striae is to apply the mean 7.87 cross striations rhythm to the perikyma groove counts shown in Figure 5. The 232 perikyma groove sequence starts with the first groove to be formed on the permanent upper first molar. Bullion<sup>19</sup> counted the brown striae of Retzius hidden under the cusps of three upper first molars, and found that the number ranged between 57 and 60. Assuming 7.87 days between each pair of brown striae, this gives 448–472 days (14.7–15.5 months) for the time taken to form the hidden increments of the first molars in Bullion's study. Permanent first molar crowns start

to form within a few weeks of birth<sup>27</sup> and it can therefore be suggested that the first perikyma groove in these teeth, representing the next brown stria to be formed after those in the hidden zone, was formed at a count of 58–61 brown striae after birth, or 457–480 days (15–15.8 months) of age. If it is assumed that this estimate can be applied to the upper first molar of the Carter Lane specimen then ages can be determined for the first perikyma groove and for the completion of the crown in all the teeth in the series (Figure 5).

These estimates for age of crown completion are plotted against four independent estimates in Figure 6. The estimates of Schour and Massler<sup>9</sup> are based mainly upon their interpretation of the histological evidence of Logan and Kronfeld<sup>8</sup>, whilst the estimates of Anderson *et al.*<sup>28</sup>, Nolla<sup>29</sup> and Fass<sup>30</sup> are all based upon the radiography of children's jaws. In the first molar, all four independent estimates for the completion coincide fairly well, and the estimate based upon perikyma groove counts in the Carter Lane child lies comfortably in the middle. In the later developing tooth crowns, the independent estimates coincide less closely, but the perikyma groove based estimate always falls within some part of the overall range. For the first premolar and second molar, the perikyma groove based estimate is very close to the mean ages of Andersen *et al.* This result has been repeated in a number of other dentitions and lends support not only to the idea that the brown striae and perikyma grooves are formed in response to a regular rhythm, but also that the estimate for the periodicity of this rhythm is not far from the truth. Over the period of more than 6.25 years from birth to the estimated time of second molar crown completion, if the periodicity deviated on average from 7.87 days by only one day then the final estimate could be up to 290 days in error. If the periodicity deviated by two

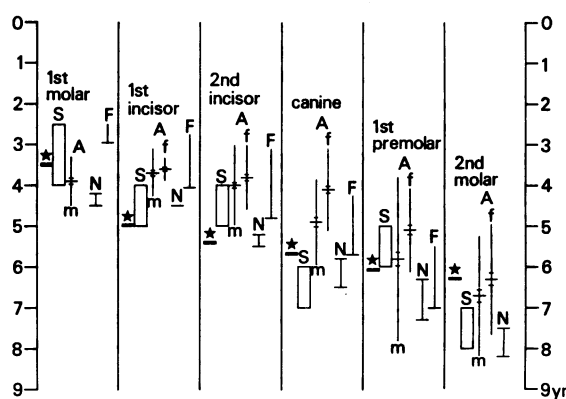


Figure 6. Crown completion ages estimated from the perikyma groove sequence of PIC87 106 shown in Figure 5, compared with widely used standards for crown completion in permanent teeth. The bar surmounted by a star is the estimate based on perikyma groove counts. The box labelled S represents the range given by Schour and Massler<sup>9</sup>. The two vertical bars m and f, surmounted by the label A give 95% ranges for boys and girls, calculated from the figures of Anderson *et al.*<sup>28</sup>. The central tick of these bars represents the arithmetic mean age given by Anderson *et al.* and the small ticks above and below represent the 95% confidence limits of the mean. The bar and ticks labelled N represent the 'norms' for boys and girls given by Nolla<sup>29</sup>. In this case, the girls are always the younger of the two, except for the 1st incisor, where the norms coincide. The tick and bar labelled F are the median and lower limit for crown completion given by Fass<sup>30</sup>. The vertical scales of the plot are in years after birth

days, then the maximum error would be over a year and a half in error.

### Conclusion

The method presented here allows estimates to be made for the age at which the growth disruptions which caused developmental defects occurred, and for the duration of these disruptions. It also allows estimates to be made for the ages at which the crowns of different teeth in a dentition were completed. In young individuals, who died before their permanent tooth crowns were completed, it will be possible to provide estimates of the age at death. All of these are based upon a histological examination of the specimens themselves, and make it possible to move away from the use of standard tables. This is particularly useful for archaeological material, but the method may also have applications in the forensic field.

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