AMERICAN JOURNAL OF PHYSICAL ANTHROPOLOGY 104:89–103 (1997)

Relationship of Enamel Hypoplasia to the Pattern of Tooth Crown Growth: A Discussion

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KEY WORDS hypoplasia; dental development; dental enamel defects; perikymata

ABSTRACT The defects of enamel hypoplasia can be related to the layered structure of enamel which represents the sequence of development in tooth crowns. From such studies, it is possible to see that furrow-type enamel defects (the most common form of hypoplasia seen with the naked eye) are just the most prominent expression of a continuum which extends ever smaller, down to a microscopic disturbance to a single layer in the crown formation sequence. Furthermore, the progressive decrease in spacing between development layers which occurs down the crown sides, from occlusal to cervical, affects both the prominence and apparent width of the defects. This makes it difficult to use measurements as a means of estimating the duration of the disturbance causing a particular defect. The difficulty is even greater for the less common pitted or exposed-plane-type defects, for which the apparent width bears very little relationship with the duration of the growth disturbance. The defects of enamel hypoplasia can therefore be understood clearly only when examined under the microscope in relation to the structures which mark the development sequence of the tooth crown. Am J Phys Anthropol 104:89–103, 1997.

Enamel hypoplasia (Fig. 1) is routinely recorded in the teeth of both living children and archaeological remains (Buikstra and Ubelaker, 1994; Goodman and Rose, 1990; Skinner and Goodman, 1992), and its pitted and furrowed defects form a recognised part of epidemiological standards such as the DDE index of the Commission on Oral Health, Research and Epidemiology (1982). Hundreds of records of hypoplasia have been made in many projects worldwide, but the minimum level of defect recordable as hypoplasia has never been defined, and, without this, it is impossible to standardise records for comparison between studies. It may seem surprising that so crucial a part of the definition has been left out, but the answer is not a simple one. Some types of hypoplasia, such as pitting or layers of missing enamel, are clear-cut and appear in an all-or-nothing manner. Furrowing, which is by far the commonest form of hypoplasia, is, however, part of a continuum of defects which extend down into a microscopic scale, to features that would not be recognised as hypoplasia in a clinical setting. Here, the definition of a minimum level is highly problematic, and this is one of the major concerns of the present paper.

Some hypoplastic defects are related to inherited conditions, but these are very rare in most parts of the world. By far the majority of defects are caused instead by growth disruptions (childhood fevers, dietary deficiencies, etc.) to the cells forming enamel matrix in growing teeth, which in

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Received 6 January 1997; accepted 17 June 1997.

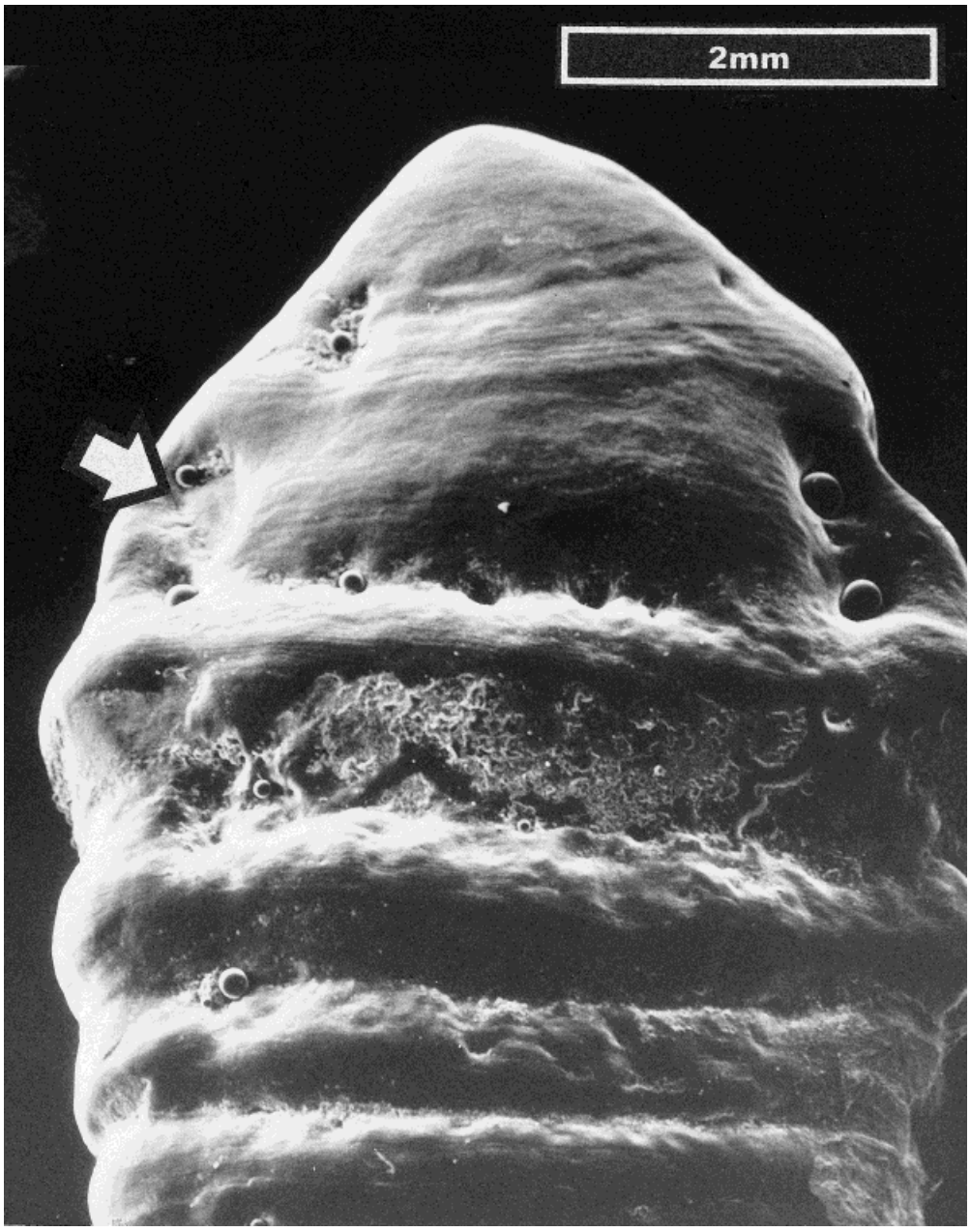

Fig. 1. Repeated and strongly marked developmental defects of dental enamel in a human lower right canine (Odontological Museum D.35.1) reproduced by kind permission of the Royal College of Surgeons of England. Epoxy replica made from an impression taken in the museum, examined in a Hitachi S570 scanning electron microscope, operated at 20kV using the Everhart-Thornley detector. The field of view shows the buccal crown surface with the occlusal tip of the canine uppermost, and the spherical bodies are air bubbles trapped when the initial impression was taken, a problem which is hard to avoid. Almost all types of defect can be seen in this specimen, ranging from minor furrow-form defects in the upper part of the crown, associated with large and small pits, and major plane-form defects which produce a series of steps down the lower half of the picture. The pit-form defect marked by an arrow is shown in more detail in Fig. 8. Just below this is a sharp furrow-form defect containing a row of small pits.

turn cause groups of these cells to cease secreting matrix earlier than normal (Hillson, 1996). Most of the factors that initiate hypoplasia are thus episodic in nature, so one of the main aims of research into hypoplasia is to establish the timing and duration of these initiating factors. In many recent studies (Bermúdez de Castro and Pérez, 1995; Blakey and Armelagos, 1997; Blakey et al., 1994; Ensor and Irish, 1995, 1997; Goodman et al., 1984; Hutchinson and Larsen, 1988, 1990; Moggi-Cecchi et al., 1994; Ogilvie et al., 1989), this timing has been established by taking measurements to the occlusal and the cervical margins of each defect, from the cement-enamel junction of the crown (the point at which it joins the root and at which crown formation ended), and these measurements are converted into ages by the use of standard tables developed initially by Swärdstedt (1966) or into estimates of the duration of growth disruption as a proportion of the crown height involved. When examined under the microscope, however, tooth crowns bear evidence of the process and sequence of formation, which makes it possible to examine the defects of enamel hypoplasia against a highly detailed background of layered crown growth and raises difficult questions about the recording and interpretation of the defects.

INCREMENTAL STRUCTURES AT THE CROWN SURFACE

Amelogenesis, or the formation of enamel during the development of the tooth crown, is one of the most complex processes in developmental biology, and there is not enough space in this short paper to give a detailed account of all aspects. Only those details which are directly relevant to the discussion are described, but further explanation can be found in a range of textbooks (Boyde, 1989; Hillson, 1996; Osborn, 1981; Osborn and Ten Cate, 1983; Ten Cate, 1985). On the surface of an unworn tooth crown, the pattern of its formation is recorded by a sequence of regularly spaced grooving which runs around the circumference. These grooves are known variously as perikymata or imbrication lines. The two names arise out of differences in their appearance in different parts of the crown, and they can be divided into three main types (Boyde, 1971):

1. Occlusal type. These are the classic perikymata form, which takes its name from the Greek *peri* (around) and *kymata* (waves). They consist of broad, shallow waves, with the troughs between them some 100 µm or more apart (Fig. 2). There is a relatively smooth transition between the crests of the waves and the troughs in between. The whole is decorated by a diffuse pattern of small pits, each some 4 µm in diameter, with their centres spaced a little over 5 µm apart and which, under low magnification, give a surf-like appearance which undeniably adds to the impression of breaking waves. They are known as Tomes' process pits—each marking the position of one enamel-forming cell (ameloblast) and signifying the end of enamel matrix production by that particular cell. The Tomes' process pits are more strongly developed in the grooves than along the wave crests.
2. Mid-crown type. The wave-like impression is retained in this type, but there is a relatively sharp line in each of the grooves between the waves (Fig. 3A). The Tomes' process pits are still to some extent scattered, but they concentrate much more tightly in a band which marks out the groove just to occlusal of the line. The spacing between the grooves is rather closer than in the occlusal type, in the order of 70 µm.
3. Cervical type. These are the classic imbrication line form, taking their name from the Latin *imbrex* (a tile), and *imbricare-atum* (to tile). Here, the lines in the grooves are much sharper, and the ridges in between are much less wave-like (Fig. 3B). Instead, the impression is one of overlapping sheets or, if irregular, tiles, and this gives the pattern its name. The spacing between the lines in the grooves is shorter again, at 50 µm or less.

These types grade into one another but are clearly discernible on most crown surfaces. The occlusal type is confined to the occlusal one-half to one-third of the crown, depending upon the tooth, and the cervical type is confined to the more cervical one-quarter to one-third in a similar way. In between, the mid-crown type is found. The

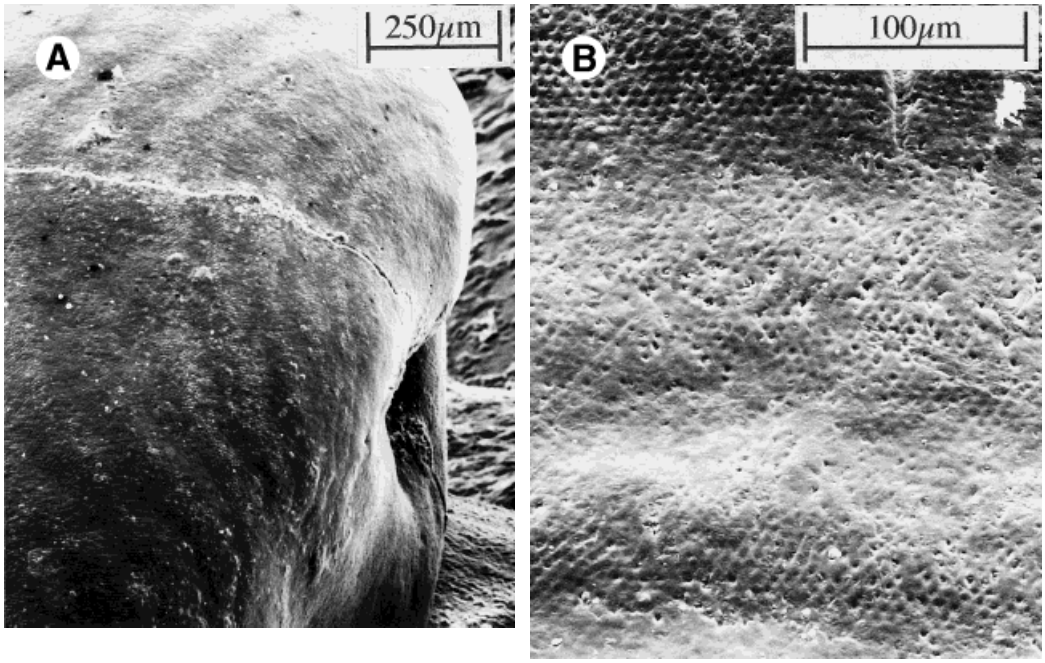

Fig. 2. Occlusal type perikymata. **A:** Tip of a canine crown showing the first perikymata to be formed at the crown surface—all of them exhibiting a marked wave-like form. Epoxy replica of buccal crown surface, examined as in Fig. 1 but using a different specimen. **B:** Detail of occlusal perikymata on the same crown, with the occlusal tip situated above the top of the picture. As is conventional in scanning electron micrographs, the image can be interpreted as though it were obliquely illuminated by a light source at the top of the field of view, although in fact it is formed in a different way, and care needs to be taken with this analogy (Goldstein et al., 1992). Tomes' process pits decorate most of the surface, but the perikyma grooves are outlined by the diffuse dark bands of apparent shadowing which mark their occlusal sides. In the floor of each perikyma groove is an irregular, poorly defined horizontal line which marks the position of a brown stria of Retzius as it emerges at the crown surface. Specimen and microscopy as for panel A.

prominence and form of these structures varies among individuals (and teeth), but often the broadly spaced occlusal type can easily be made out with the naked eye, especially in the little worn permanent incisors of young children. Gradual abrasion, particularly tooth brush abrasion, makes the structures much less prominent in later life, although they can often still be followed under the microscope by looking for the telltale bands of Tomes' process pits which lie protected in the grooves and are therefore retained for longer.

In an anterior tooth such as an incisor or (particularly) a canine, most of the crown side is occupied by perikymata of the mid-crown type (Hillson, 1992a, 1992b). The broad, shallow occlusal type is confined at most to the occlusal third of the crown. Similarly, the cervical type occupies just the cervical one-quarter or less. Cheek teeth (particularly molars) have much more of their crown height occupied by perikymata of the occlusal type which, in many cases, occupies the occlusal half of the crown, and some of the grooves are very widely spaced—up to 150 μm or even 200 μm. These differences between anterior and cheek teeth represent a fundamental difference in the underlying geometry of the way in which their crowns were formed.

In spite of the variation, all these types of surface patterning are commonly referred to as perikymata. The groove part of each is usually known as the perikyma groove (pkg for short).

PATTERN OF GROWTH INSIDE THE CROWN

The perikymata and imbrication lines are surface expressions of a system of incremen-

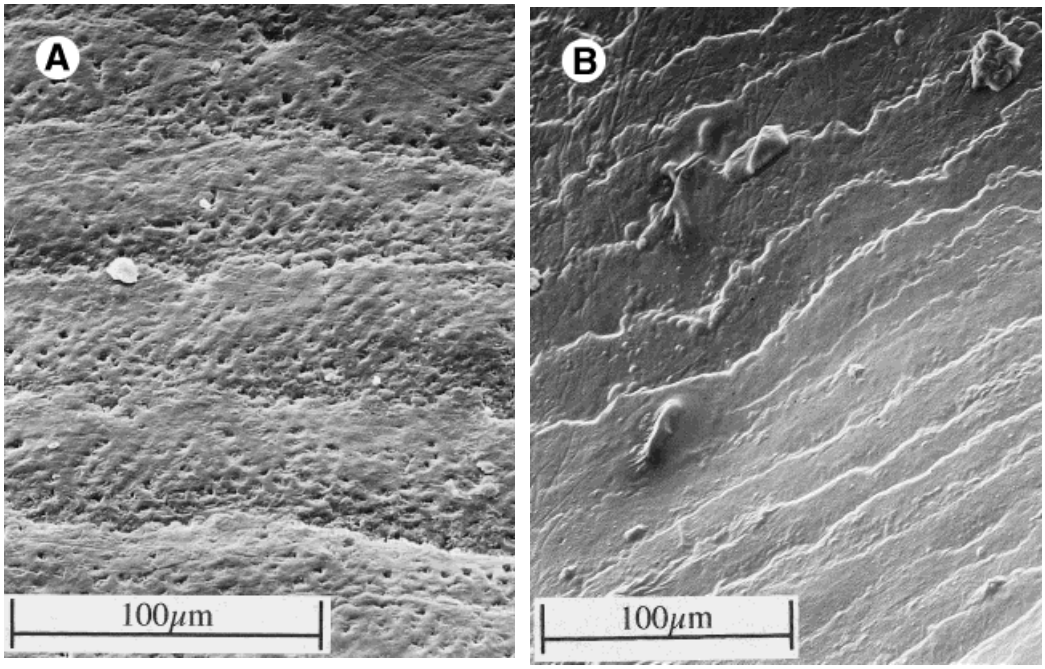

Fig. 3. Perikymata further down the crown side. **A:** Mid-crown perikymata type, with sharp lines marking the edge of each perikyma groove and with Tomes' process pits more clearly confined to the grooves. Specimen and microscopy as for Fig. 2A. **B:** Cervical imbrication line type of perikymata with very prominent lines giving an overlapping sheet-like form. Tomes' process pits not usually strongly marked in the cervical zone of the crown and cannot be made out in this image. Specimen and microscopy as for Fig. 2A.

tal structures that permeates the crown. If the enamel is fractured in a particular way, then it can be made to break up along the planes of weakness which are caused by these structures (Fig. 4A). The floors of the pkg can be seen as the edges of the planes of weakness, whose exposed surfaces show a similar pattern of Tomes' process pits, indicating their origin in a brief and temporary interruption to production of enamel matrix by the ameloblasts. These planes of crown layering are, however, best seen using light microscopy on ground and polished sections cut through the main axis of a cusp or tooth, where they are seen as dark, fuzzy lines (Fig. 4B). The darker appearance results from a scattering of light as it passes through the section in the microscope, caused by increased pore space along the disrupted planes. Seen in this way, the layers bear the name of the Swedish anatomist Anders Retzius (Retzius, 1836, 1837), and they have gone into the literature as the brown striae of Retzius. Their particular interest lies in the fact that they mark out successive positions of the enamel matrix forming front, so they record in great detail the way in which the crown developed. Just underneath the crown surface they are fairly sharply defined, with a clearly regular spacing. Deeper into the crown, they become a little fuzzier, but it is still possible to follow the sequence of development.

Underneath the cusp of the tooth, the brown striae represent a series of dome-like increments, piled one on top of another. This has often been labelled the appositional zone of crown formation (Dean and Beynon, 1991), and the same terminology is used here with the meaning that the cusp height is built up by apposition of complete increments. The number of increments in the appositional zone can be rather difficult to establish, but there are now some reasonably consistent results from a number of studies (Table 1). Under the main central cusp of a canine tooth or the central mamelon of an incisor there may be the equiva-

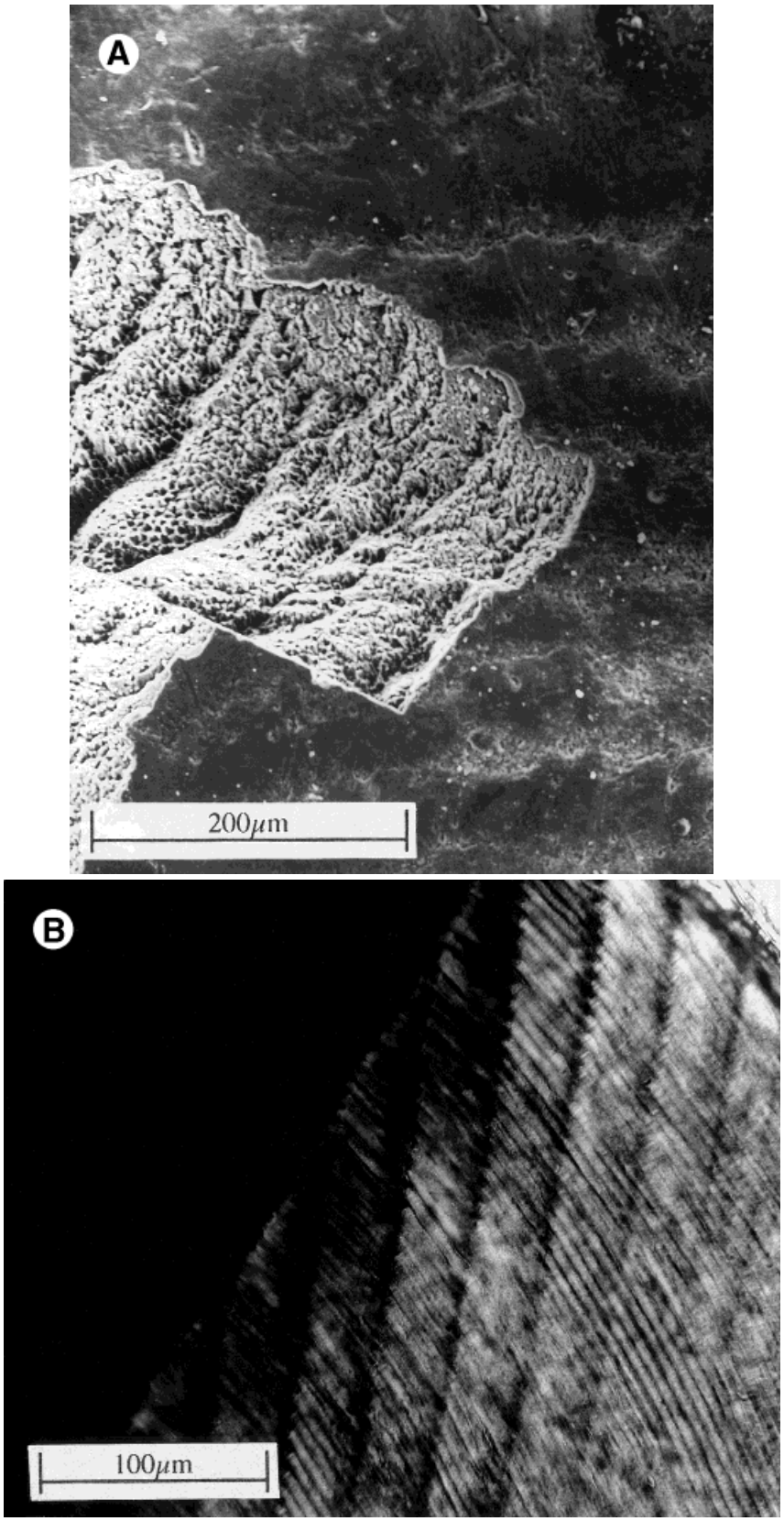

Fig. 4.

TABLE 1. Appositional and imbricational increments in crown formation

Tooth type	Number of teeth	Appositional zone—count of brown striae of Retzius (or the equivalent in cross-striation)	Imbricational zone—count of brown striae of Retzius (or pkg)	Reference
First incisor	9	26–34	121–193	Bullion, 1987
Upper and lower canine	9	32–35	93–318	Bullion, 1987
Upper second incisor	4	40–45	109–127	Bullion, 1987
First premolar	8	36–43	94–192	Bullion, 1987
Second premolar	7	41–51	75–148	Bullion, 1987
First molar	4	47–60	70–92	Bullion, 1987
Second molar	7	69–82	69–106	Bullion, 1987
Lower incisor	10	20–30	165–202	Bromage and Dean, 1985
Lower incisor	2	31–34	162–224	Dean and Beynon, 1991
Upper first molar (mesiobuccal cusp)	1	60–61	120	Dean and Beynon, 1991
Upper first molar (mesiolingual cusp)	1	76	85	Dean and Beynon, 1991
Upper first incisor	1	—	185	Stringer et al., 1990
Lower incisor	2	—	200–270	Stringer et al., 1990

lent of 20–35 of the increments represented by brown striae in the appositional zone (Fig. 5). By contrast, in a molar there are more likely to be 50–80 increments in the appositional zone, whereas premolars seem to occupy an intermediate position, with 35–50 increments. It therefore seems that there is a fundamental difference between tooth classes. This is accentuated when the zone of formation outside the main cusp is considered. This is the imbricational zone (Dean and Beynon, 1991), where the increments are more sleeve-like in nature. Each increment is positioned slightly more to cervical than its predecessor and the side of the crown is formed by an overlapping series, from the tip of the cusp down to the cement-enamel junction at its base. It is only in the imbricational zone that the edges of the planes of the brown striae of Retzius reach the crown surface to form pkg. This is so because the stria planes in the appositional zone are dome-like structures with a closed top, and so there is no surface evidence for them. The numbers of striae in the imbricational zone can be evaluated without sectioning in most crowns by counting the pkg. They again show a systematic difference between tooth classes, although the numbers of increments seem to be more variable than those in the appositional zone. For an incisor, there would typically be 100–250 or so imbricational increments. A canine, with its tall crown, may well have up to 300 or more. Molars may have over 100 imbricational increments, but more typically this number is 70–90. In summary, therefore, the molars seem to have a larger proportion of the formation process confined to the appositional zone (typically between 40 and 50% of the increments), whereas anterior teeth have more of their formation exposed in the imbricational zone (typically around 15–20% of increments may be appositional).

The differences between the three types of perikymata/imbrication lines are generated by the geometry of crown formation. Under-

Fig. 4. Brown striae of Retzius. **A:** Fractured preparation of an original molar crown surface, examined using similar microscopy to Fig. 2A. The surface of the crown is the darker area occupying the right hand side of the frame, and it is marked by the horizontal lines of mid-crown type perikyma grooves, with finely stippled bands above the lines indicating the position of the prominent Tomes' process pits in the floors of the perikyma grooves (the magnification is much lower than in Figs. 2–3). In the irregularly fractured area on the left of the picture, rough, step-like features, also marked with Tomes' process pits, angle up towards the crown surface. The part of each step which faces the viewer is a plane of weakness marking a brown stria of Retzius and rises to meet the line in its corresponding perikyma groove. **B:** Brown striae of Retzius in a ground section, examined by polarised light microscopy, angling up to meet the crown surface. The surface itself is marked by a line running diagonally from the scale bar up to the middle of the top frame of the picture, with a uniformly black area on its left. The brown striae are the fuzzy lines running almost vertically from bottom to top, whilst the much finer, slightly curved lines running from lower right to top left are the so-called prisms, which are bundles of tiny crystals making up the fundamental units of enamel structure (Boyde, 1989; Hillson, 1996; Osborn, 1981; Osborn and Ten Cate, 1983; Ten Cate, 1985).

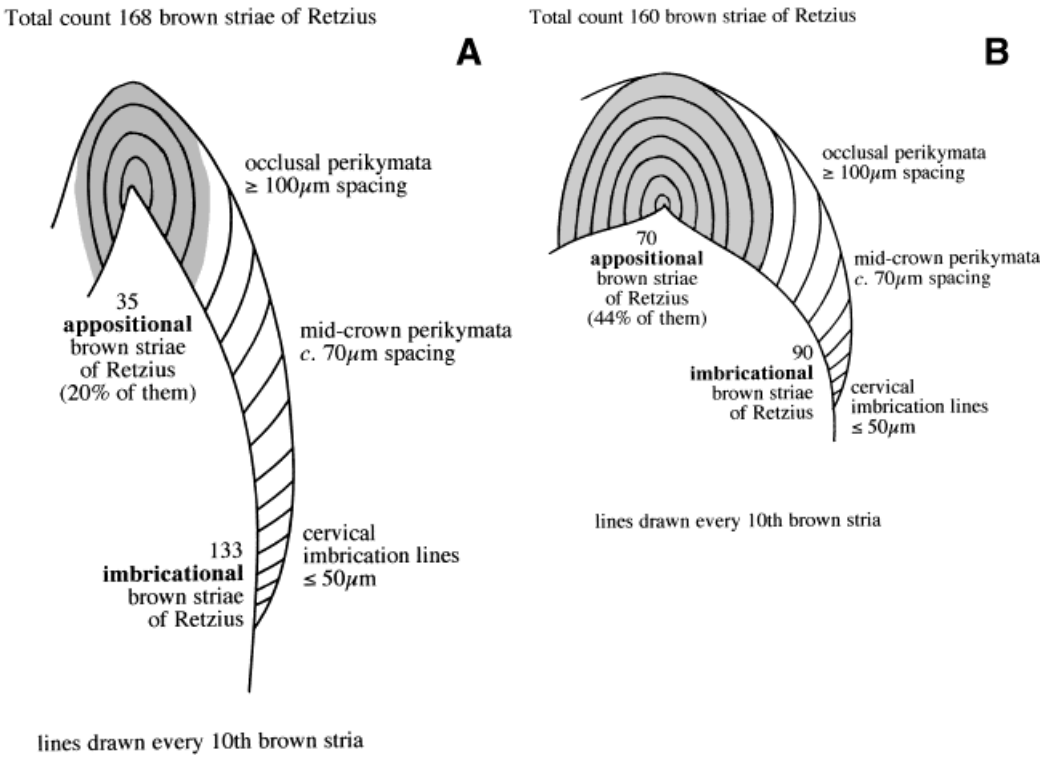

Fig. 5. Diagrammatic representations of crown growth. Shaded areas mark appositional enamel. **A:** Permanent upper central incisor with 35 appositional and 133 imbricational brown striae of Retzius. **B:** Permanent lower second molar with 70 appositional and 90 imbricational brown striae of Retzius.

neath the occlusal type, the planes of the brown striae make an angle of about 15° with the surface on their cervical side. This shallow angle causes the pkg to be relatively far apart and gives rise to their rather low relief. The angle increases into the mid-crown zone, to perhaps 30° or 40°, so that the spacing between pkg decreases and the relief becomes sharper, and increases further into the cervical type zone, where it is often nearer 60°. In addition, the spacing between brown striae decreases, from 30–40 μm in the occlusal zone down to 15–20 μm in the cervical. Both factors produce the closer spacing and much sharper, overlapping sheet-like structure of the imbrication lines in the cervical zone (Fig. 3B).

This sequence of crown formation is the background to enamel hypoplasia. Both the internal layering and external features exert a strong controlling influence on the form of the hypoplastic defects.

HYPOPLASTIC DEVELOPMENTAL DEFECTS OF ENAMEL

The work of J. Berten (1895) remains one of the best general descriptions of enamel hypoplasia. Berten defined three main types of defect: *Fürchenforming,* or furrow form, *Grübchenforming,* or pit form, and *Flächenformig,* or plane form. These divisions continue to work well at the present day, because they describe real differences in the way in which the defects were formed. Berten's other major contribution was to demonstrate the way in which the defects could be matched between different teeth in the same individual, thus showing clearly that the defects are related to some systemic factor which disrupts all ameloblasts secreting matrix at the time.

Furrow-form defects

Furrows are the commonest form of hypoplastic defect in enamel (Fig. 6). They may

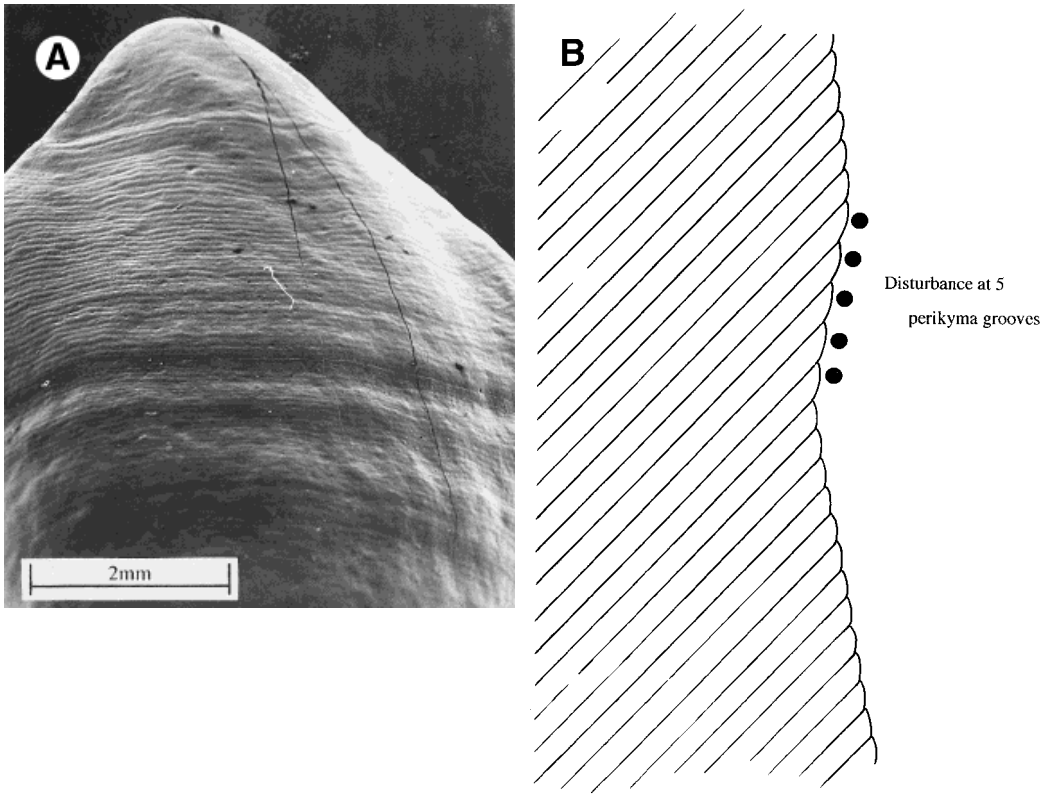

Fig. 6. Furrow-form defects. **A:** Various sizes of furrow-form defect in the buccal surface of an upper canine, with its occlusal crown tip uppermost. The defects, seen at this magnification, are not sharply defined, and the most marked in this specimen are the three broad but irregular furrows outlined by apparent shadowing in the lower half of the image. Other disturbances to the spacing of the perikyma grooves can, however, also be seen higher up the crown—some involving only a pair of adjacent perikyma grooves. Epoxy resin replica, with microscopy as for Fig. 1. **B:** Model for perikyma groove spacing changes in a hypothetical furrow-form defect, shown as a buccolingually orientated radial section (Hillson, 1996) through the crown surface, with occlusal to the top and cervical to the bottom. Each line marks a brown stria of Retzius plane, and the dots mark the slightly wider spacing between five consecutive pairs of perikyma grooves, in comparison with the rest of the crown surface. These produce the occlusal wall of the defect, whilst the floor is produced by a return to normal spacing.

be single or multiple and may occur, on occasion, in association with the other types of defect. Under the microscope they can be seen to arise from local variation in the spacing of pkg, superimposed over the normal progression. Furrows are therefore not a form of defect that can appear in response to growth disruptions which occur during the formation of the appositional zone. This is an important distinction because a large part of the crown formation sequence (up to 50% in molars) may thus be unrepresented. A furrow-form defect may be divided into three elements: its occlusal wall, its floor, and its cervical wall. The spacing between pkg can be measured under the microscope (Hillson and Jones, 1989), and the grooves are farther apart than is normal for that particular zone of the crown in the occlusal wall of the furrow. Effectively, a wider margin of each brown stria plane is exposed than normal, suggesting that a larger band of ameloblasts ceased matrix production than would usually be expected at a pkg. The spacing returns to normal in the floor and cervical wall. It thus appears that only the occlusal wall actually represents a growth disruption, as expressed by the increase in pkg spacing. The floor and the cervical wall seem instead to represent a recovery to the

normal pattern of crown formation. Even when a furrow-form defect is examined with the naked eye or low magnification, the occlusal wall is usually more sharply defined than the cervical, and the apparent breadth of the floor and cervical wall are often strongly related to the angle of the illumination. In summary it appears that, in a pure furrow-form defect, only the pkg in the occlusal wall represent the growth disturbance so that the apparent full width of the furrow gives a misleading impression of the duration of the disturbance.

This being so, the duration of the disruption causing a furrow defect is best defined by the pkg count in its occlusal wall. A chronic disruption may involve 20 or more pkg in this way, whilst a short one may involve 5 pkg or so. The minimum expression is just 1 pkg, spaced more widely than normal from its occlusal neighbour. The defects thus form a continuum from a 1 pkg defect up to the largest at 20, 30, or more. All of them can be matched between teeth from the same individual, right down to the smallest (Dean et al., 1993; Hillson, 1992a, 1993). It is therefore apparent that all have similar systemic causes. The smaller furrows cannot really be seen without a microscope and will certainly be missed in a routine clinical examination, yet they are just as much indicators of a disturbance to growth as the large defects. This raises fundamental questions about what the minimum level of recording should be and whether or not it can be defined except under the microscope.

This problem is further increased because the same-sized furrow (in terms of the count of pkg in the occlusal wall) varies in width and prominence with its position on the crown. A 10 pkg furrow in the mid-crown region would have an occlusal wall some 700 µm wide. A similar 10 pkg furrow in the occlusal region of the crown would be much wider because the pkg are in any case much more widely spaced. It would come out at about 1,500 µm in width and would produce a shallower defect because the pkg are themselves shallower than in the mid-crown region. In the cervical region, the same 10 pkg furrow would have a 300 µm wide occlusal wall and would be relatively more sharply defined. All this is purely a function of the variation in spacing and form of the pkg in different parts of the crown which is, in turn, a function of normal crown growth geometry. It follows that a sharply defined furrow defect in the mid-crown part of the canine may be difficult to match with a defect caused by the same disruption in the occlusal part of the premolar because, here, it seems much wider and shallower and less well defined. Thus, if the prominence of the defect is taken to indicate the severity and the width of the furrow (as measured by a pair of calipers) is taken to indicate its duration, then both parameters will be very greatly affected by the position of the defect on the crown, regardless of the disturbance that caused it. This raises a further fundamental question about the baseline for defect recording. Should some attempt be made to define a mid-crown average? Once again, the answer may be to express the defects in terms of the number of pkg involved rather than the apparent size of the furrow.

Pit-form defects

The pits (Figs. 1, 7, 8) may be substantial—over 500 µm in diameter—or any one of a range of sizes down to a few tens of micrometres across in the pit floor. Berten (1895) distinguished the smallest as *punktforming*, or point form, and sometimes the pits line up in a row around the circumference of the crown, looking a little like a row of stitching. More often, however, the pits are found in a broader band spreading round the crown. All such pits mark a disturbance at one former position of the enamel forming front at the plane of a brown stria of Retzius. For some reason which is not understood, little clusters of ameloblasts stop forming enamel matrix; in small pits, a group of ten or so ameloblasts may be involved, but large pits may involve hundreds. In many cases, the exposed Tomes' process pits left in the pit floor are evidence of a sharp and final disruption to the ameloblasts, but in others there is evidence of some continued deposition of a thin layer of irregular enamel matrix. The ameloblasts in between the disrupted clusters seem to continue undisturbed and produce perfectly normal enamel matrix, so that brown striae of Retzius form in the normal way, with their edges marked by pkg

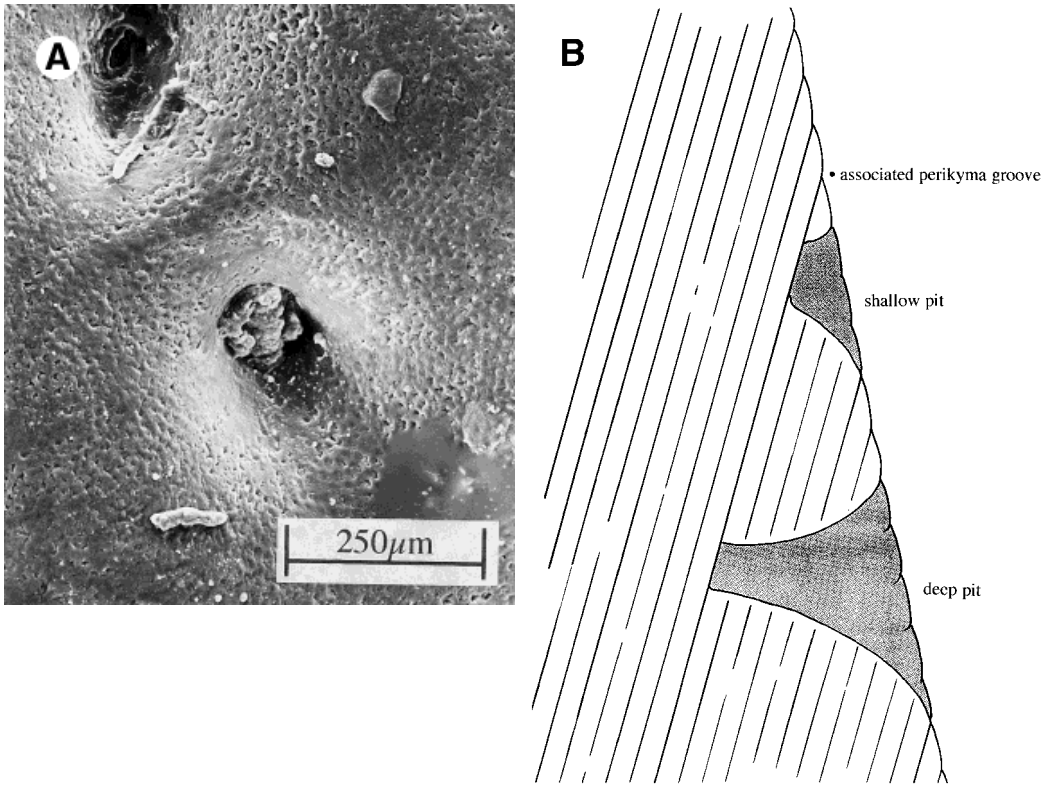

Fig. 7. Pit-form defect. **A:** Pit defects, with some irregular enamel matrix formation continuing in their floors after the growth disruption that caused them. Epoxy resin replica; microscopy as for Fig. 1. **B:** Model for pit formation, shown as a section through the crown, orientated as for Fig. 6B. A growth disruption has stopped enamel matrix secretion at two points along the brown stria of Retzius plane, without causing a disturbance at the associated perikyma groove. One growth disruption has therefore caused two depths of pit.

at the surface (Figs. 7B, 8A). When viewed from the surface, the pits are interspersed with normally spaced pkg which curve down into the pit sides. The depth of the pits must be related to their position on the plane of the brown stria of Retzius which marks the growth disruption, relative to the pkg which forms the edge of that brown stria. A shallow pit is not far from the associated pkg, whereas a deep pit is much further from the associated pkg, and, when the pit is at the occlusal end of the crown, its associated brown stria plane might in fact be one of those buried in the appositional zone. Such pits may persist through tens of enamel increments before the surface around their outer opening is formed. From this, it is apparent that the position at which a defect appears on the crown surface may be some distance to cervical of the pkg which is associated with the brown stria of Retzius at which the pit was initiated. To put it another way, if it is intended to place the growth disruption which caused the pitting into its correct place in the crown formation sequence, then the position of the pits at the crown surface can be very misleading. It is not even clear that the associated pkg will of itself show any evidence of growth disruption. Whilst shallow pits are occasionally associated with a furrow-form defect (a neat row of pits may be dotted along the furrow floor, as in Fig. 1) or large pits may form a cervical fringe to an exposed-plane-form defect, many bands of pits appear to be unrelated to any other defects.

The model shown in Figure 7B shows that even a substantial band of pits may represent a disturbance to only one brown stria of Retzius plane. In fact, just by looking at the

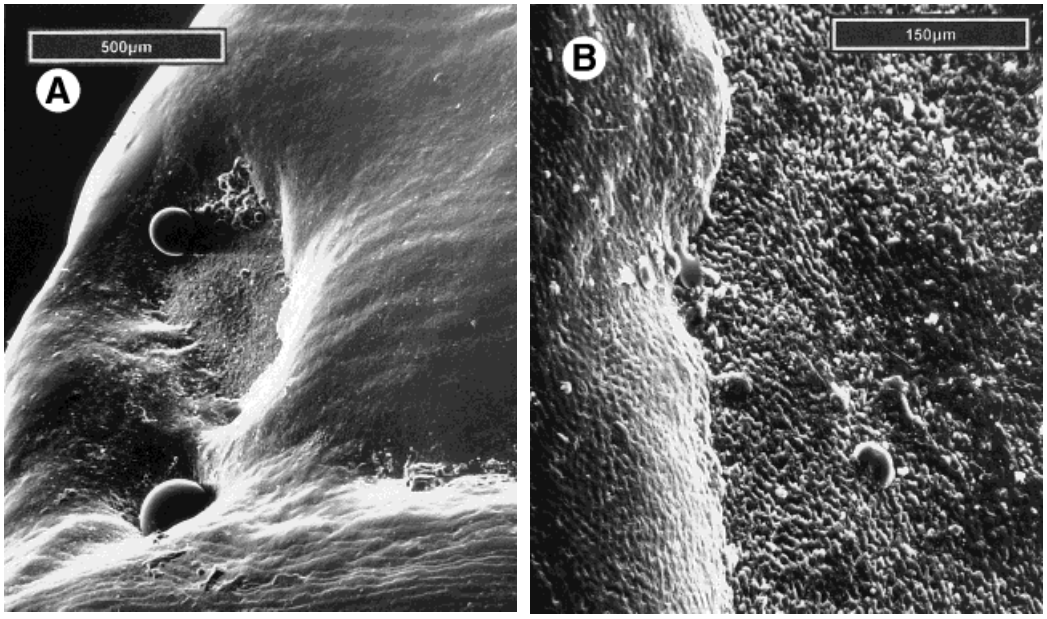

Fig. 8. Large pit-form defect, marked in Fig. 1. **A:** Lower magnification view of the pit and its surroundings. The normal crown surface around the pit is marked by occlusal and mid-crown types of perikyma grooves, and a prominent furrow-form defect runs across the picture just above the lower frame. The floor of the pit is formed by an exposed brown stria plane, with Tomes' process pits (seen at this magnification as a fine stippling on the exposed plane) showing that the ameloblasts ceased matrix secretion abruptly. The sides of the pit rise up steeply, and the perikyma grooves of the surrounding crown surface curve down into them. **B:** Higher magnification view of the pit floor and wall showing details of the Tomes' process pits in the floor and the abrupt transition which marks the base of the wall on the left hand side of the picture. Specimen and microscopy as for Fig. 1. Lower right canine (Odontological Museum D.35.1) reproduced by kind permission of the Royal College of Surgeons of England.

surface, one cannot easily establish this other than by making an estimate based upon the depth of the pits and the likely angle of the plane in the enamel underneath. It is, however, perfectly possible that many clusters of ameloblasts could be switched off simultaneously—a broad band of them for only one disturbance at one brown stria plane. This disturbance may be serious in that it causes a large amount of disruption in the subsequent enamel formation, but, if it involves only one brown stria, it is likely to be of short duration. From this it is clear that neither the diameter of the pits nor the width of pit band as it appears at the surface is a good measure of the duration of the disturbance.

Exposed-plane-form defect

In some cases it is not just isolated clusters of ameloblasts that cease enamel matrix formation prematurely. Here, the plane of a brown stria of Retzius is left wholly or partially exposed (Fig. 9) and can be identified by its decoration of Tomes' process pits. There is usually a marked step at the cervical margin of the defect, where normal enamel with perikymata resumes. Quite frequently a defect of this kind involves several planes of brown striae, to give a stepped appearance. The junction between the normal enamel and the exposed plane may also undulate irregularly, or, as noted above, the defect may merge into a band of pits beyond its cervical margin (Fig. 9).

These defects are most frequently found in the occlusal part of the crown and expose a brown stria plane in the appositional zone. Further down the crown, they are occasionally found in the floor of a furrow-form defect (Boyde, 1970), leaving a marked step partway down the crown (Fig. 1). Altogether, they are the most marked and noticeable manifestation of enamel hypoplasia, but it is

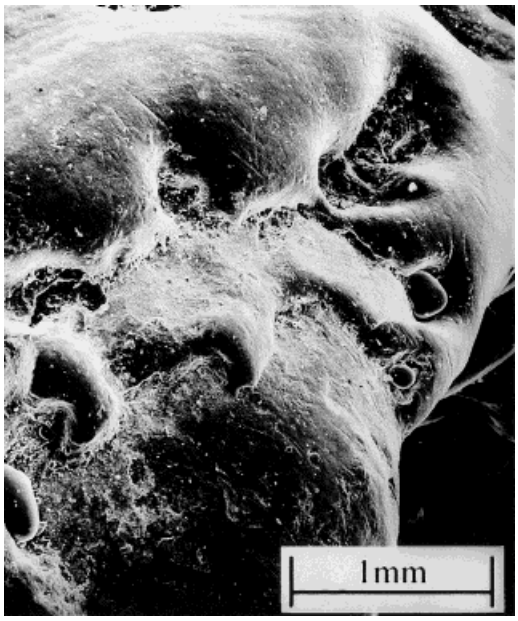

Fig. 9. Plane-form defect in a molar. The crown is shown cusp tip downwards. A first growth disruption has caused the top layers of enamel to be missing from the cusp tip, exposing the plane of a brown stria of Retzius which now forms the highest point of the cusp. A second growth disruption, following soon after the first, has exposed the edge of another brown stria of Retzius plane to create a step running from lower left to upper right across the middle of the image. The smooth bulge of enamel (marked with perikyma grooves) which defines the outer edge of this step marks the return to normal enamel matrix formation at the base of the cusp. The boundary between this bulge and the second step is irregular, because it is marked by occasional pit-type defects. Epoxy resin replica; microscopy as for Fig. 1.

important to note that they frequently involve just one brown stria plane and are often followed immediately by perfectly normal enamel. This means that the disturbance that initiated the defect was of very short duration. Once again, the defect's position on the crown and its size in terms of the crown height it occupies may bear little relation to either the timing of the disturbance or its duration.

CONCLUSIONS

These observations point to a number of issues which have relevance to the recording of hypoplasia.

1. All histological studies so far undertaken suggest that the appositional zone of a tooth crown hides a considerable proportion of enamel layers. These layers do not appear on the surface, and therefore, unless a growth disruption generates a plane-form or pit-type defect, no evidence of hypoplasia is produced during this part of the crown's development. For anterior teeth, the appositional zone hides some 10–20% of crown formation time in this way. For cheek teeth, up to one-half of crown formation time may be hidden. This is a major consideration for the construction of recording systems for enamel hypoplasia. It should come as no surprise, because the fundamental work of Massler and colleagues (1941) includes a diagram (their Figure 6, part B) which implies that about 7 months of incisor formation time lies hidden in an appositional zone, whilst well over 10 months lies under the molar cusps (Hillson, 1992b).

2. Furrow-form defects are found only in the imbricational zone and include a continuum which ranges from microscopic lines up to furrows which may readily be made out with the naked eye. All sizes can be matched between teeth from the same individual and are therefore all the result of some systemic disturbance to growth, so that there is no reason why the microscopic defects should be seen as a different phenomenon to the macroscopic. Indeed, in any one tooth crown, the bulk of defects are usually of microscopic size and would not be detected in a routine clinical examination, so most studies must therefore not have recorded them. It is clear that a full study of hypoplasia must involve the use of low level microscopy.

3. The size and prominence of a furrow-form defect are greatly affected by the arrangement of lighting (or apparent illumination in the scanning electron microscope). The disturbance to pkg spacing which generates the defect affects only pkg in the occlusal wall of the defect, whereas the floor and cervical wall are produced by a return to normal spacing. Thus, depending upon the lighting, the apparent width of the defect often includes more than just the enamel layers which are affected, and therefore measurements of the defect's full width must inevitably lead to an overestimate of the duration of the disturbance to growth.

4. When crowns of different teeth from one individual are matched, it is clear that one growth disruption causes defects in a different place in each of the different tooth crowns being formed at the time, with a different size and prominence in each. This has been noted before as a difference in "susceptibility" between tooth classes (Goodman and Armelagos, 1985a,b), and, whilst there may indeed be differences in the resistance to disruption of ameloblasts at different stages of their matrix secreting career, there is little doubt that the geometry of crown growth described above must be a major factor. Any recording system must take into account this variation, and it would almost certainly be better to base assessments of defect duration on pkg counts rather than measurements of apparent furrow width.

5. The surface position of pit-form defects may be quite unrelated to the point in the crown growth sequence at which they were initiated by a growth disruption. The pits are always located to cervical of the pkg which marks the edge of the disturbed growth layer. That pkg itself may be associated with evidence of a disturbance in the form of a furrow-form or plane-form defect to occlusal. This is not, however, necessarily the case, and there may be no indication which pkg represents the relevant growth layer and therefore the timing of the growth disruption that generated the pits. All this makes it very difficult to devise a simple means for assigning a band of pit-type defects to an age.

6. The extent of a pit band or of an exposed-plane-form defect often bears no relation to the duration of the disturbance that caused it. The exposed plane is the surface of just one growth layer. It presumably represents a pronounced disruption but not a long one.

All these are comments which can be made simply by considering the literature of enamel histology and by examining a few specimens. If anything is to be done to alleviate the problems to which such observations point, this will require concentrated effort on a large number of specimens to define what is normal in terms of crown formation sequence and timing worldwide. Alternatively, the study of hypoplasia could become a purely microscopic one, in which sequences of pkg counts determine the relative timing and sequence of defects.

ACKNOWLEDGMENTS

The authors are grateful to Dr. Caroline Grigson, principal curator of the Museums of the Royal College of Surgeons of England, for allowing impressions to be made from specimen D.35.1 in the Odontological Museum and to the president and council of the College for permission to publish the resulting scanning electron micrographs.

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