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# Erupted teeth in the newborn

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The presence of erupted teeth in the newborn has been observed and recorded from the earliest time, according to Gates.[7] Probably the greatest notoriety has been accorded the central figure of Shakespeare's play, *King Richard III*, who was referred to as "that dog that had his teeth before his eyes. . ." The child born with teeth has often been regarded with great superstition, both in the past and even in certain present-day communities, and the condition has variously been regarded as an omen for good or ill.

Apart from merely recording the occurrence, however, there was little attempt at scientific analysis or medical assessment of the condition until Massler and Savara[12] reviewed the situation in 1950. Even today certain aspects do not appear to be fully appreciated, and it is the objective of this article to clarify these features or at least to bring them into the open for discussion.

## NOMENCLATURE

In the older literature teeth in the mouth of the new or recently born child were variously referred to as fetal teeth, congenital teeth, or dentitia praecox. Massler and Savara[12] attempted to introduce uniformity by assigning the term *natal* to those teeth which were present at birth and *neonatal* to those which erupted within the so-called neonatal period, or the first 30 days of the child's independent existence. It will be readiy appreciated that such a temporal distinction is a convenient but strictly artificial one and does not really divide the cases into two distinct anatomic, physiologic, pathologic, or clinical entities. This is unfortunate, because, as will be seen, such cases do most certainly appear to divide into two distinct groups with two distinctly different prognoses. The main distinction between the two groups should not be whether the teeth were present at birth or appeared during the first month but, rather, whether or not their structure is mature. One group of these teeth is relatively normal, whereas the

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other group consists of teeth which have reached only various stages of partial completion of their development. It would be most apt to speak of this latter group as comprising "premature" teeth, in the same sense that an infant is spoken of as being premature. This implies that the object described suffered an untimely exposure to independent existence before full development had fitted it to face these rigors.

The terms *natal teeth* and *neonatal teeth*, however, have been widely accepted in terminology and textbooks, and it would appear expedient to retain them. This nomenclature should therefore be given real clinical significance by being further qualified. Such structures are better described as *mature* natal or neonatal teeth, implying that they are fully developed as compared with the remainder of the deciduous series and hence that their prognosis is relatively good, or as *immature* natal or neonatal teeth—an important distinction, since it carries the stigma of an incomplete substandard structure and, even more important, implies a poorer prognosis and the different treatment planning which should necessarily accompany it.

*Predeciduous teeth*, another term which requires further consideration, is applied to supposed representatives of a series of teeth which are reputed to be atavistic relics of our reptilian development. In our opinion, this term perpetuates a fable which should be either substantiated or discarded and would seem, like the fables of old, to have been handed down from author to author with little real substance to support its actual clinical existence. Even such a valuable reference work as that by Thoma and Goldman[16] describes the predeciduous teeth as comprising "white rudimentary epithelial structures sitting on the gingivae in hood-like fashion. They are hornified structures and can easily be removed." Thoma and Goldman refer the reader to the original description by Schröder and Moral,[13] who examined teeth ascribed to this predeciduous series and found them to consist of "scanty enamel with dentine attached at the undersurface. This dentine contained large numbers of interglobular spaces and was lined by a thin layer of osteodentine." This description would not appear to coincide with Thoma and Goldman's description of a hornified structure, but it would correspond reasonably well with the immature natal teeth of the normal deciduous series described by Hals[3] and also, as will be seen, with our own case. It is certainly not tenable as a description distinctive of any predeciduous entity.

The case described by Allwright[1] may or may not have more validity. It is significant that here also, however, a valid and definite histologic description is not available. Despite the valuable contribution to the subject of natal and neonatal teeth which Allwright's article represents, one would hesitate to establish such an entity purely on the basis of this description, for clinical impressions—no matter how genuinely held—can be extremely misleading on occasion. Even the highly scientific and reputable textbook by Shafer, Hine, and Levy[14] repeats the myth, albeit those authors are sufficiently discreet to avoid quoting any authority. It would seem, therefore, that this entity deserves, at best, the Scottish-law verdict of "not proven"—a description which damns by implication—and hence the term *predeciduous teeth* should not be perpetuated in the literature.

## CLINICAL CONSIDERATIONS REGARDING NATAL AND NEONATAL TEETH

The incidence of natal teeth was investigated by Bodenhoff,[3] among others, and probably varies between one in 2,000 and one in 3,500 live births. The incidence of neonatal teeth is much more poorly documented, reflecting the relatively recent interest in pediatric records as compared with obstetrical ones. Massler and Savara[12] observe that "only 5 cases were reported during the past half-century." This figure obviously does not validly reflect the prevalence but merely indicates the interest shown in recording the condition. Allwright[1] assessed a minimum of ten cases out of 6,817 live births. However, it is his clinical opinion that the true figure is probably much higher than this, as the mothers usually left the hospital a few days after delivery and in many positive cases the patients probably did not report back. It would, indeed, be thought that the axes denoting age and incidence of eruption of lower central incisors would probably approximate those of a normal random distribution curve, with a peak around 6 months and reducing to one in 2,000 to 3,500 as birth is approached.

The *cause* of the condition is unknown. It is likely that the most frequent cause is developmental, and it is probably associated with a superficial position of formation of the tooth germ involved. Boyd and Miles[4] showed histologic sections in which such a tooth was developing without any proper bony crypt, being merely associated with a slight hollow on the upper surface of the subjacent alveolar bone. Their case, however, was that of a stillborn cyclops fetus and could not be considered as necessarily representing the bulk of such cases. The likelihood of a developmental etiologic factor is also supported by the strong familial pattern shown in a large number of the reported cases, by the anomaly's occurrence in twins who "both erupted deciduous incisors soon after birth,"[1] and by the bilaterally symmetrical nature of the majority of cases (Allwright's[1] series included nineteen bilaterally symmetrical cases out of twenty-five).

It is possible that other factors are superimposed upon this developmental predisposition. Hormonal influence would appear to be significantly related, and it may be significant that Allwright's series contained twenty females as compared with only six males. Other writers mention various other potentially predisposing causes, such as febrile systemic illnesses, hyperthyroidism, etc. (mostly conditions which tend to raise the basal metabolic rate), but none of these have very good substantiation.

*Symptoms* associated with the presence or eruption of natal or neonatal teeth include those commonly associated with the clinical entity of "teething." These consist of such things as local irritation, salivation, upper respiratory infection, malaise, etc. Ballantyne[2] noted that sublingual ulceration is prone to complicate such cases. Probably because of previous lack of spatial confinement of the tongue, protrusion and "drooling" of that organ are commonly found in young infants and the tongue is thus inclined to become traumatized by the sharp erupted tooth. Occasionally, it is not only the tissues of the child which are traumatized; laceration and infection of the nipples of the nursing mother may also result.

In cases of immature natal and neonatal teeth, the lack of root formation



results in an inadequate degree of support to the structure and the tooth becomes increasingly hypermotile in all directions. Cusick[5] claimed that because of the pain associated with this increased mobility the child may refuse to nurse. The danger of swallowing the loosened tooth is an obvious hazard which has been given prominence in the literature, but undoubtedly such a small object would be quite capable of completing an uneventful journey through the alimentary canal. The more serious complication would be inhalation. However, neither swallowing nor inhalation of one of these teeth has ever been reported in the literature, and the danger is probably more imaginary than real.

*Signs* associated with natal and neonatal teeth may vary from a circumscribed hyperemia of the affected area prior to eruption, to a moundlike elevation of the gingival tissue housing the tooth during eruption, or even to the fait accompli of a fully exposed crown. The site of occurrence is usually the lower anterior region, and the lower incisors are the teeth most commonly involved, although this is not invariably the case.

## CASE REPORT

The patient was an 8-week-old girl with a lower left central incisor which had been present within the oral cavity since birth and was now mobile and interfering with feeding. The tooth was discolored and green but appeared with little doubt to be a regular member of the normal deciduous series, although no radiologic examination was carried out.

The pregnancy had been uneventful and the mother and child were otherwise perfectly healthy, but the child was born prematurely after 34 weeks' gestation and weighed only 4 pounds, 2 ounces at birth. There was no history of any similar dental occurrence in any of three siblings. The tooth was extracted with relative ease and with no noteworthy complication.

### Pathology and histopathology

*Macroscopic examination* was carried out after the tooth was fixed in 10 per cent formalin. The main bulk of the tissue was constituted by the crown, and the soft tissues of the pulp were readily accessible to the fixative through the open base. At one side, on the labial cervical margin, the pulpal opening was somewhat narrowed by a thickening in the hard tissues which encroached in a localized downward and inward diaphragm-like projection. Enamel covered most of the crown but appeared to be thinner than normal, although it was relatively hard to the probe. Two shallow horizontal "grooves" ran along the labial surface and were thought probably to be of a hypoplastic nature.

The tooth was subjected to hard-tissue hemisection in a labiolingual vertical plane, and this procedure was carried out as gently as possible to avoid major disturbance of the soft tissues of the pulp. One of the halves was then prepared as a ground section. The other half (which contained the localized "pseudodiaphragm") was decalcified, and serial histopathologic sections were prepared.

The *hard-tissue section (Fig. 1, A)* confirmed that the *enamel* covering the crown was thin and showed that it appeared to narrow down to a premature knife-edge as it approached the apical extent of the dentine formation. Aside from a quantitative deficiency as compared to the average, however, the enamel appeared relatively normal in quality—a feature markedly different from the case reported by Hals,[8] who described one specimen with grossly substandard enamel.

It would seem from this appearance that the actual rate of tooth development and the timing of eruption may be controlled independently by different factors and need not necessarily correspond. Thus, when one is considering the clinical implications, it is necessary to have some knowledge of the timings associated with the relevant developmental features involved. Stones[15] states that calcification of the crown of the lower central incisor (the tooth

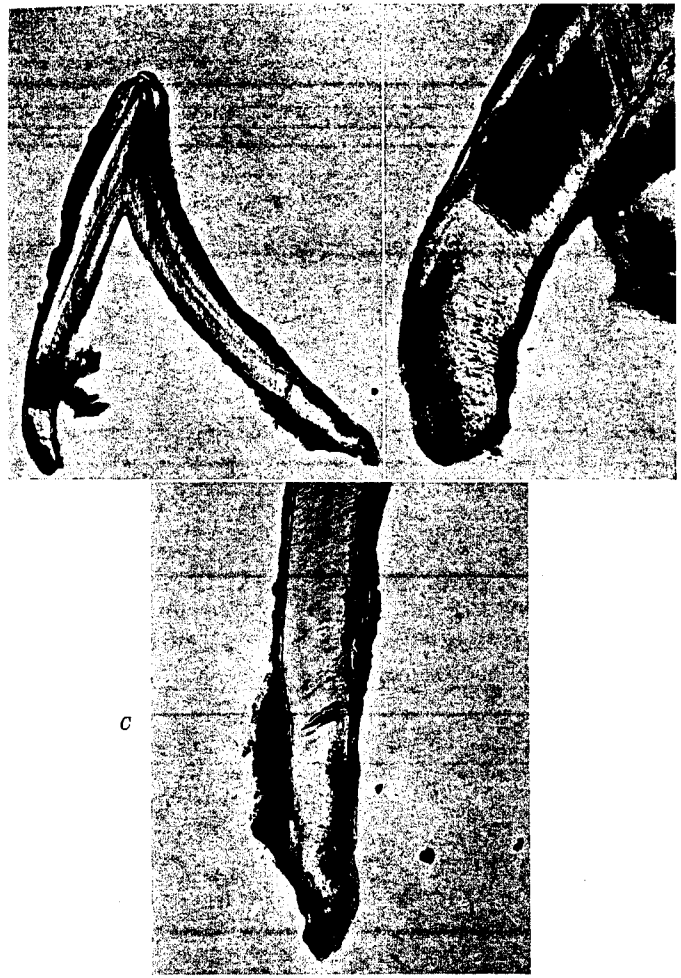


*Fig. 1. A*, Ground section of immature natal tooth showing enamel and dentine. Note the "neonatal" line visible running through the midthickness of the dentine. This forms a valuable landmark by which the timing of incremental deposition of hard tissue may be judged. *B*, High-power view of cervical portion (buccal) of ground section. Note the localized dead-tract zone in the dentine and the more apically lying cementum-like tissue with its pulpal deposition of irregular tubular dentine. *C*, High-power view of cervical portion (lingual) of ground section. Appearances correspond with the description in *B*, but the changes are not so gross.

most frequently involved) commences at approximately 4½ months in utero and is completed at approximately 3 months postnatally, covering a total formative period of 7½ months. Thus, if the tooth erupts at birth, unless development keeps pace and is correspondingly advanced, the crown of any tooth following a normal formative pattern will be only three-fifths completed. Since conditions in the oral cavity imply the simultaneous degeneration of



any still-functioning enamel organ, once eruption has occurred, premature eruption of any tooth means that enamel formation must be jeopardized and, indeed, may not be completed in a certain percentage of cases. Such was the impression afforded by the appearance of our own specimen.

The *dentine* showed evidence of equally severe disturbance. A well-defined junctional line was clearly visible traversing the midthickness zone of most of the dentine (Fig. 1, *A*). This line probably coincided with a period of dentine formation occurring at a time of metabolic upheaval—possibly either at birth or during eruption which, the history indicates, must have been even earlier than birth. Indeed, there is a suggestion in the lingual dentine that there are two lines and not just one—possibly representing both birth and eruption. This line or zone will be referred to somewhat loosely as the *neonatal line* and should be clearly borne in mind, as it forms a reference point of some significance when one considers the timing of formation of the various tissues comprising the tooth. The ends of this line join the amelo-dentinal junction approximately at the cervical extent of the enamel formation—a feature that is compatible with the conditions which would be expected if eruption and birth were reasonably coincident, as active enamel formation would also stop when the tooth entered the oral cavity.

The dentine formed postnatally probably comprises the tissue found pulpally and cervically in relation to this "neonatal line." It would appear to divide qualitatively into three zones. The first comprises that dentine deposited directly in the pulpal area in relation to the neonatal line, which was reasonably regular and normal. This indicates that the odontoblasts not only had originally formed normally but had continued to function in a reasonably normal manner after birth, within the protected area formed by this "cap" of hard tissue.

The dentine immediately adjacent to the cervical ends of the neonatal line showed localized but definite zones of dead-tract formation. It must be assumed that initially conditions here were sufficiently normal to allow regular dentine to form but that subsequent irritation imposed upon this area had resulted in dead-tract formation. The change was most marked in the labial dentine (Fig. 1, *B*), but it was also evident in the lingual section (Fig. 1, *C*).

The third dentine zone of note comprised the most apical portion, immediately cervical to this dead-tract area. Here the outer part of the hard tissue consisted of a highly irregular calcific deposit, with localized lacuna-like defects, although some of these were obviously larger than the usual cementum or bone lacunae, housing a single cell or cementocyte, and grossly irregular tubular dentine had deposited on the pulpal surface of this calcific mass. The gross morphologic configuration of the crown suggested that this doubtful tissue occupied an area which would normally consist of dentine, and it could therefore have represented an abortive but genuine attempt at root formation. This would appear to be the interpretation placed upon such abnormal tissue in previously published cases. However, the other explanation—and, in our opinion, the more likely one—is that this tissue represents an abnormally coarse type of secondary cementum, and its morphologic relationship relative to the outside of the dentine would support this view. It certainly did not appear to contain the first essential of dentinal structure—identifiable tubules.

The *decalcified sections* showed no indication of either remnants of reduced enamel epithelium or Hertwig's sheath. Those sections representing tissue nearest the center of the tooth confirmed the microscopic impression that a calcific ingrowth had, indeed, tended to form a localized apicohorizontal "diaphragm" constricting the pulp (Fig. 2, *A*), and this could be even more clearly visualized in sections from the outer interproximal portion (Fig. 3, *A*).

The coronal two thirds of the dentine was relatively normal, but the cervical third showed gross irregularity. Detailed description will accordingly be focused upon this area. In the most coronal portion of this tissue, the outer part consisted of an irregular calcific material, with no evidence of any dentinal tubules, and contained spaces varying from 10 $\mu$ to 30 $\mu$ in diameter. On the inner part, pulpal to this tissue, the dentine was tubular and relatively normal, although a few large isolated defects were seen. These defects were of a roughly circu-

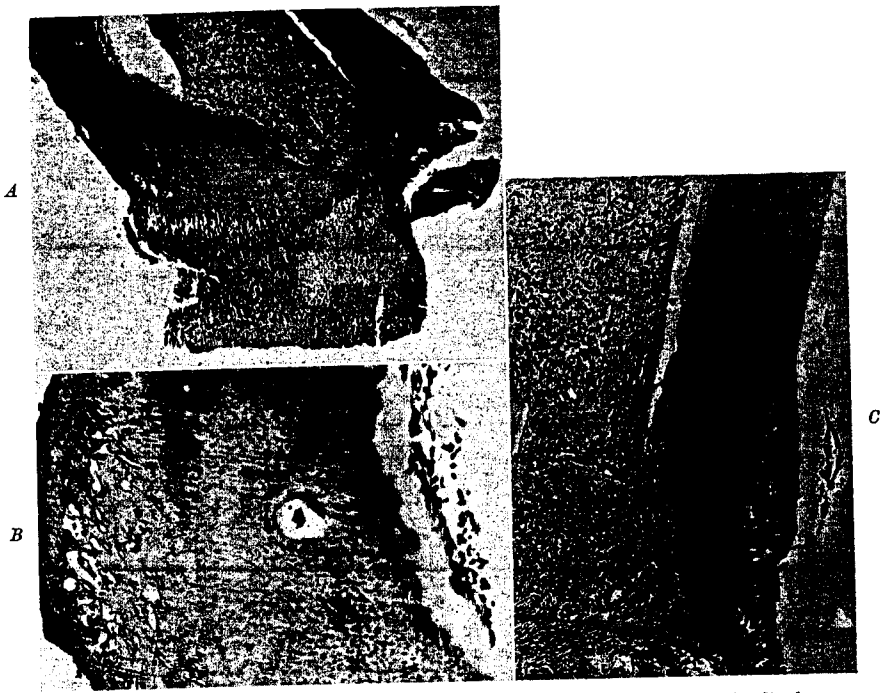


*Fig. 2. A*, Decalcified section close to midtooth. Note the commencement of the diaphragm-like encroachment of hard tissue and also its highly irregular nature. It is possible that it was originally fused with the underying bone. *B*, High-power view of coronal portion (buccal) of section shown in *A*. Note the outer irregular calcific material and the more normal dentine pulpal to it. It is questionable whether the outer tissue represents coarse cementum or dentine. *C*, High-power view of lingual portion of section shown in *A*. Changes correspond with those found in *B* but are not so gross. Note the increased pulpal cellularity in the more apically placed soft tissue. *D*, High-power view of cervical portion (buccal) of section shown in *A*. Note the immature bonelike formation on the outer surface of the dentine and the two distinct zones seen within it. This tissue is possibly a coarse secondary cementum, but it could equally well be an avascular type of bone or (less likely) dentine deposition. *E*, High-power view of the immature cellular tissue seen in *D*.

lar shape, approximately 75 to 100 $\mu$ in diameter, and they appeared to contain cellular debris. There was no evidence of any vascularization within these regions and no indication that they formed vascular channels. They could be seen clearly in various stages of formation (Fig. 3, *A*). The predentine layer was wider and more irregular than normal, and the presence of interglobular spaces reflected the disruption of the orderly sequence of dentinal calcification.

The most apically placed portion of the dentine consisted of the localized hard-tissue "diaphragm" downgrowth already noted. This tissue had developed mainly in continuity with the labial and interproximal wall of the dentine. It contained a large number of irregular spaces in its coronal region (Figs. 2, *A* and 3, *A*), and in the most apical portion it consisted of a highly cellular tissue resembling bone (Fig. 2, *E*). Schröder and Moral[13] noted the presence of a tissue with cellular inclusions, and referred to it as "osteodentine," although they recognized that it was not directly comparable with the more orthodox definition of osteodentine found in fishes and reptiles. Its appearance in our sections strongly suggested two separate periods of gross deposition, rather than any gradually progressive or orderly incre-


*Fig. 2. Cont'd.* For legend see opposite page.

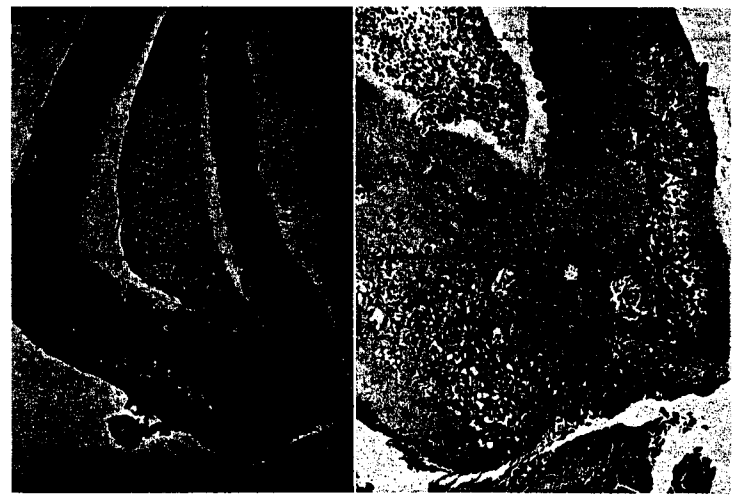


*Fig. 3. A*, Decalcified section close to the interproximal dentine wall. The "diaphragm-like" encroachment of the abnormal tissue is clearly seen. Various stages of formation of the circular soft-tissue inclusions are also apparent in the dentine. *B*, High-power view of section shown in *A*. Note absence of zone of Weil and cell-rich zone—features which merely imply that dentine formation is still actively proceeding.

mental process of the type that might have occurred in a genuine attempt at root formation. The histologic appearance of this tissue closely approximated a coarse secondary cementum or an avascular type of bone. Indeed, it is possible that the tissue was continuous with the subjacent alveolus and that ankylosis had occurred, although there was no definite clinical indication of this. Such a finding would coincide well with the specimens described by Hals[8] and by Schröder and Moral.[13] (The latter described the condition as a "gomphosis.")

The pulp tissue had been somewhat distorted by the initial hard-tissue sectioning. As in the cases decribed by Schröder and Moral[13] and by Hals,[8] our sections also showed that the zone of Weil and the so-called "cell-rich" zone were lacking. These features are not unusual in such specimens, and their absence merely implies that primary dentine formation had not been completed. Increased cellularity was present in the basal portion of the pulp (Fig. 2, *C*), but this may have been associated as much with irritation as with any developmental proliferation in the area.

## DISCUSSION

Boyd and Miles[4] reviewed *eruption theories* on the possible supposition that the natal tooth not only could have been the result of a superficially sited development but may also have been the product of an abnormally fast eruption pattern. In looking for abnormal features that might have produced an unusual eruptive effect, they point especially to the cellular inclusions found in the tissue at the cervical end of the developing dentine and note that similar conditions were observed by Herpin,[9] Howkins,[10] and Schröder and Moral.[13] They speculate, however, that the nonconformity of the structure of the cellular hard tissue with any incremental pattern "makes it difficult to accept what would otherwise be an attractive hypothesis, namely, that the zone corresponds with a period of rapid axial movement of the tooth." Hals[8] also notes such a condition in his specimen but states: "This tissue need not necessarily follow normal lines of growth. The importance of this irregular hard tissue as a contributing factor at the eruption, therefore, should not be disregarded. The inclusion of cells indicates in itself that the formation occurs rapidly."

In our own case it is probable that the cellular inclusions are not so highly significant if the tissue, indeed, represents secondary cementum and not dentine, although it is difficult to judge from printed photo-micrographs whether the previously reported tissues are equivalent. It should be noted, however, that other presumptive evidence is certainly present to support the supposition that this irregular overgrowth of tissue *formed after eruption* and therefore could not, in itself, be a contributory factor in any acceleration of that process. The tissue in question was found deposited in a position cervical and pulpal to the incremental "neonatal" line, which has been previously noted in the dentine, and therefore it must have been deposited subsequent to the formation of this line. As previously stated, it is highly probable that this line corresponds with the metabolic upset of either birth or eruption (which we know, indeed, took place before birth). This cellular calcific overgrowth is almost certainly a posteruptive phenomenon and thus could not correspond with any such "period of rapid axial movement." It is therefore of prime physiologic significance, when reviewing possible factors associated with eruption of teeth, to realize that *root formation could not have contributed significantly to eruption and the presence of this tooth in the mouth.*

Hals[8] also points out that the pulp, as seen by him, was highly vascular and



implies that this vascularity may have contributed to a potential increase in speed of eruption. The pulp seen in our case was not excessively vascularized, as compared with the pulp of any other developing tooth, although vascularity was moderately marked in the basal region. Even here, however, it did not give the impression that it could have made any great contribution to an accelerated eruption pattern.

*Root formation* was grossly deficient over-all, despite the localized outgrowth, and its pattern would have had to change markedly if any functional form of permanent attachment were to be subsequently envisaged. This was considered one of the most highly significant clinical implications to be drawn from the histologic findings.

*Treatment* of an immature (as distinct from a mature) natal or neonatal tooth must obviously be modified, once it is accepted that root formation is unlikely to proceed very far in such circumstances, unless it has already progressed sufficiently prior to eruption to give the tooth stability. In addition, in such immature teeth the enamel has a marked tendency to chip and the teeth are likely to suffer marked attrition and premature death due to both qualitative and quantitative enamel defects, even in certain cases where the root is firm.

Allwright[1] registers a gloomy prognosis for these immature teeth, despite his relatively extensive experience in dealing with such cases. Although his aim is to save such teeth if possible, unless the patient can be kept under close review, he believes that "early extraction of even slightly loose teeth would appear to be justified."

Extraction of such immature teeth presents no more difficulty than any other minor surgical procedure in the newborn child who is otherwise normal and healthy. Allwright[1] extracted twenty-five teeth from fifteen such patients without having to take any special hemostatic measures, but it would seem that the unscientific and emotional superstitions associated with natal teeth are not confined to the lay public. The supposed hazards which the profession tends to associate with such an operation have probably been perpetuated from the death in such circumstances of a patient reported by Magitot[11] in 1883, although it is likely that other complicating factors were present in that case.

The series reported by Gardiner[6] bears out Allwright's clinical impressions. In four of the twelve cases which Gardiner cites, the teeth were extracted surgically; in four other cases in which such teeth were left, the children shed them spontaneously within 3 months; and in only four of the original twelve cases did the teeth ultimately become functional.

Probably the most pertinent and interesting feature of Gardiner's report, however, lies in his discussion of the ultimate prognosis of such a condition, as distinct from the short-term outlook. Reluctance to extract even a grossly dubious tooth in these circumstances stems mainly from the fact that extraction may be associated with space loss and ultimate collapse of the lower arch, with its attendant orthodontic complications. Gardiner (himself an orthodontist) was acutely aware of this possibility and consequently followed up his cases. He found that the spaces originally occupied by the extracted teeth did, indeed, close within a short time, but *in all cases they opened up again at the age of 5 or 5½ years.* His follow-up showed that "in none of the nine cases, where

permanent lower incisors appeared, have these been crowded." Obviously, such a history can be no guarantee against crowding of the permanent teeth, but it would suggest that if irregularity does subsequently occur, it is likely to be due to some other factor and not merely to the loss of the original deciduous incisor. Such a feature would also tend to justify the more radical approach advocated by Allwright[1] where this is indicated.

## SUMMARY

The terms *natal teeth* and *neonatal teeth* constitute a relatively artificial distinction and should be further qualified to provide a more practical clinical significance. It is suggested that the terms *mature* and *immature* are more in keeping with the varying prognoses associated with such cases. We also consider that speculation concerning a *predeciduous* series of teeth is based mainly upon fallacy and believe that usage of this term should be discontinued until more positive proof is forthcoming.

We have presented a case in which an immature natal tooth required extraction at the age of 2 months. The histologic findings strongly suggest that the tooth would never have become truly functional. We stress that the poor immediate prognosis frequently associated with the immature type of natal and neonatal teeth should be recognized by the profession and that treatment should be instituted accordingly. We also stress, however, that even when radical treatment is resorted to, provided that all other factors are normal, then the long-term prognosis is still reasonably good, as orthodontic problems would not seem to be associated per se with such conditions.

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# Fibrous dysplasia and infection of the mandible

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Osteomyelitis superimposed on a fibro-osseous lesion of the jaws appears to be relatively uncommon. The case to be presented in this article is one of fibrous dysplasia of the mandible in which a coexistent infection, originating from an apical abscess of a lower molar, confused the diagnosis and was responsible for delaying treatment.

## REVIEW

Fibrous dysplasia does not occur infrequently, as evidenced by the voluminous literature. It usually occurs in patients who are in good general health, and it appears as a slowly developing expansion of bone in all directions. This expansion usually exhibits a smooth or slightly nodular surface covered with normal oral mucosa. Diffuse involvement is more common in the mandible.[1]

Consultation is often motivated by the occurrence of more rapid growth with a concomitant esthetic change. Generally there is no pain.

In the mandible the buccal sulcus may be obliterated, the lingual fossa may disappear, and the lower border may become convex. All this can take place without affecting the position and function of the teeth, although separation of the teeth and alteration of the occlusal plane is known to occur. Should the condylar process be involved, the affected side may elongate, resulting in asymmetry and probable cross-bite.

Roentgenographically, the coarse mottling of the bone with irregular radiolucent areas (rarely defined or separated from one another) commonly appears in the mandible, especially when the ascending ramus and coronoid process are involved. Rushton[2] thinks that this tendency to patchy sclerosis in the mandible

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