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Oral complications associated with neonatal oral tracheal intubation: a critical review

George M. Angelos, DMD David R. Smith, MD
R. Jorgenson, DDS, PhD Edward A. Sweeney, DMD

Abstract

This paper summarizes and evaluates the oral complications associated with orotracheal intubation in neonates. The palatal defect resulting from orotracheal intubation is best described as palatal grooving, rather than clefting since no oral nasal communication has been demonstrated. Palatal grooving may be caused by the inhibition of the molding tongue forces on the lateral palatine shelves. The incidence of palatal grooving increases with duration of intubation and reportedly resolves following extubation. However, posterior cross-bites, high palatal vaults, and poor speech intelligibility have been reported in children who previously have been intubated. Impingement of an orotracheal tube on the alveolus rather than on the palate may cause alveolar grooving which can cause dilaceration of primary teeth.

Bilateral linear enamel hypoplasia in premature neonates is caused by an interruption in amelogenesis from intrauterine disturbances. However, gross unilateral incisal enamel hypoplasia in children who have been intubated is probably due to traumatic intubation. Avoiding excessive pressure on the maxillary alveolus during intubation is suggested. An appliance is available which secures oral tubes and protects the palate and alveolus.

Premature neonates frequently require assisted ventilation using nasotracheal or orotracheal tubes. Since neonates are obligate nasal breathers with small nasal airways in proportion to their ventilatory needs, they are unable to supplement nasal ventilation by oral breathing if one of the nostrils is occupied by a nasotracheal tube. Consequently, they are forced to increase breathing efforts to maintain a constant tidal volume (Sullivan 1982).

Orotracheal tubes have been used to avoid the septal and respiratory impairments associated with nasotracheal tubes (Jung and Thomas 1974; Gowdar et al. 1980). However, orotracheal intubation is not free of complication. Movements of tubes in the airway have been associated with frank extubation, histopathologic changes to the airway mucosa, damage to the larynx, subglottic and bronchial stenosis, and subglottic cysts (Symchych and Cadotte 1967; Rasche and Kuhns 1972; Hengerer et al. 1975; Nagaraj et al. 1980; Papsidero and Pashley 1980; Couriel and Phelan 1981; Jones et al. 1981; Ratner and Whitfield 1983). In addition, cleft palate, palatal grooving, alveolar grooving, primary tooth dilaceration, cross-bites, high palatal vaults, poor speech intelligibility, and enamel hypoplasia have been associated with the use of oral tubes (Boice et al. 1976; Duke et al. 1976; Saunders et al. 1976; Krous 1980; Moylan et al. 1980; Wetzel 1980; Erenberg and Nowak 1984b; Seow et al. 1984; Seow et al. 1986; Kopra et al. 1988).

Literature Review

Alveolar Grooving

Boice et al. (1976) were the first to report oral complications following orotracheal intubation in a neonate. They reported a "noticeable concavity" of the anterior alveolar ridge at the site of the orotracheal tube after 3 days. At autopsy, microscopic sections of tissues taken from the areas exhibiting the most noticeable concavity showed disruption of the enamel organ with a cystic space around the organ and deviation of the long axis of the tooth. The enamel organs for the succedaneous teeth reportedly were normal in all sections examined as were teeth on the unaffected side.

Wetzel (1980) reported that 5 orally intubated children developed erosions of the maxillary anterior alveolar ridge. He observed that neonates who dragged the orotracheal tube over the alveolar ridge during respiratory movements rapidly developed a pale ischemic area under the tube. This dragging motion traumatized the mucosa with resultant ulceration and "pressure necrosis" of the alveolar ridge and underlying tooth buds. The deepest of the grooves observed was 7 mm and entirely divided the alveolar ridge.

Krous (1980) observed that orotracheal tubes secured with tape to the upper lip caused gingival excavation in the maxillary anterior region. The excavation of the alveolar ridge was the negative image of the tube. The gingival excavation rarely was observed in infants surviving only a few hours but was prominent in infants surviving several days.

Palatal Grooving

Duke et al. (1976) reported that cleft palate was associated with prolonged orotracheal intubation in 2 neonates. A cleft of the hard palate extending from the incisive foramen to the soft palate was noted at 70 days and 50 days from intubation. After extubation the palatal deformity decreased in size with apparent closure from both superior and lateral aspects in 1 neonate, while 4 months later they reported no noticeable closure of the deformity in a second neonate. They also reported that 300 other neonates requiring orotracheal tubes for periods shorter than the 2 cases presented were free of palatal deformities. In addition, 7 infants intubated for greater than 50 days did not develop the palatal deformity.

Saunders et al. (1976) also described palatal grooving following orotracheal intubation. Two neonates extubated at 70 and 78 days developed a deep palatal groove. The term "palatal grooving" has been used by most authors since this report.

Behrstock et al. (1977) questioned whether prolonged pressure suspected by Duke et al. (1976) and Saunders et al. (1976) from an orotracheal tube could cause palatal grooving or clefting. He postulated that the palatal grooving described was formed by the prominence of the lateral palatine ridges with medial hypertrophy adjacent to the tube. He referred to a paper by Hanson et al. (1976) which described the normal prominence of the lateral palatine ridges at birth. The lateral palatine prominences (a normal anatomic structure in neonates) were said to remodel with time as a result of normal tongue function. Behrstock et al. (1977) believed that the tube inhibited the molding effect of the tongue on the palate, leaving a groove in the midline. They reported that once the tube was removed, the tongue's motion against the palate caused the groove to diminish in prominence by age 2.

Saunders and Easa (1977) agreed that palatal grooving might be associated with interference of the tongue's molding effect on the lateral palatine ridges with the tube contributing to the shape and severity of the groove.

Biskinis and Herz (1978) described the formation of a palatal groove after 44 days of orotracheal intubation in a neonate. A deep groove extending to the left of the midline from behind the alveolar ridge to the soft palate was noted at extubation. A follow-up examination at an undisclosed time revealed almost complete resolution of the groove.

Erenberg and Nowak (1984b) reported the frequency of palatal grooves in a group of neonates and infants with orotracheal tubes in place from 1 to 62 days. Maxillary casts were made of neonates requiring orogastric or orotracheal tubes. Sixty-three neonates had orotracheal tubes placed from 1 to 62 days prior to the making of the maxillary casts. Forty-two of the neonates (68%) had impressions taken during the first week of life. Palatal grooves were observed in 30 (47.6%) of the infants at the time of the impression. The longer the period of intubation, the greater the frequency with which palatal grooving was observed. Neonates intubated for less than 7 days showed a 39.5% frequency, while those requiring intubation for 15 or more days showed an 87.5% frequency. No relationship between body weight at the time of impression and the formation of palatal grooving could be found.

Maxillary casts also were made of another 106 neonates, all of whom had orogastric tubes and 6 of whom also had orotracheal tubes. No palatal grooves were observed in the 100 with orogastric tubes regardless of duration of intubation (50-day minimum). Palatal grooving was observed in the 6 neonates who had both orotracheal and orogastric tubes. These results indicated that the frequency of palatal grooving increased significantly in neonates who required an orotracheal tube for 15 or more days.

Orogastric tubes appeared not to be associated with palatal grooving even after 50 days, perhaps due to their smaller diameter and different flexibility. They also suggested that the palatal groove formed due to continuous pressure of the tube against the median palatine suture.

Ginther (1986) noted that "the groove represents thickening of the alveolar ridges because of restriction of tongue movement" due to the presence of the oral tube. Broadening of the "alveolar ridge then creates a false impression that the palate has been eroded by a groove," he wrote.

Arch Symmetry

Seow et al. (1985) compared the symmetries of palatal and dental arches between a group of children previously intubated at birth with a group of nonintubated controls. Maxillary casts of 18 intubated and 31 control children ranging in age from 26 months to 5 years, 5 months were made. Symmetry was evaluated using the median palatine raphe as the baseline and comparing the distance from it to antimeric teeth. No palatal grooves were observed in any of the

casts of the intubated neonates. Symmetry of the palate in intubated and control children showed no significant differences. They also studied the effect of duration of intubation on palatal arch symmetry by comparing 13 children who had previously been intubated for 3-10 days to 9 children intubated for more than 20 days. No significant differences in arch symmetry were noted between the 2 groups.

In contrast, Kopra et al. (1988) reported that children who had been intubated had a characteristic high palatal vault, posterior cross-bites (25%), and a higher incidence of poor speech intelligibility compared to controls. These authors concluded that there are long-term oral sequellae due to neonatal orotracheal intubation.

Prevention of Palatal Grooves

Because palatal grooving reportedly was associated with pressure from orotracheal tubes, Molteni and Bumstead (1986a) studied the incidence of palatal grooves when a soft, flexible tube was used. Of the 103 neonates included in their study, 57 received standard hard tubes and 46 received soft tubes. Neonates were examined weekly for the appearance of a palatal groove which, if present, was measured in millimeters from the floor of the groove to the surface of the palate. Only neonates intubated for 8 days or longer developed a significant palatal groove of > 0.5 mm. The development of a significant palatal groove was closely related to birth weight, with a 50% prevalence of grooving in neonates of < 1000 g compared to only 17% in neonates of > 1000 g. The presence of grooves also was related to the duration of intubation. No significant difference could be demonstrated between using hard tubes and soft tubes relative to the incidence of palatal grooving; therefore, the authors concluded that soft tubes offered no advantage in reducing grooves.

Sullivan (1982) described an appliance to support oral tubes in premature infants. He said that infants born after 26-36 weeks' gestation had neither sucking nor a coordinated swallowing reflex; thus, the normal pattern of oral feeding was impossible because of the risk of inhalation and asphyxia. He stated that the daily food required by a premature child dictated that continuous feeding be utilized. Nasogastric tubes typically have been used for continuous feeding, but as noted earlier, compromise breathing. Orotracheal tubes can be used in place of nasotracheal tubes, but they cause an increase in jaw and tongue movement which tends to dislodge the tubes and cause tissue irritation.

The appliance that Sullivan (1982) described permitted oral tubes to be positioned securely. The appliance was constructed by taking an impression of the maxilla, from which a stone model was poured and a denture-like appliance constructed with grooves extending anteriorly-posteriorly to secure the tubes. However, rapid postnatal growth resulted in the loss of the peripheral seal and required that the appliance be relined frequently. Relining may extend the use of the appliance for up to 6-8 weeks. The author noted no deleterious side effects from the use of the appliance and suggested that it resulted in reduced breathing efforts by the neonates.

Erenberg and Nowak (1984a) also described an appliance for stabilizing orogastric and orotracheal tubes in infants, crediting Sullivan and Haringman (1981) with the original design. Their appliance was identical to the one described earlier except for the use of a nontoxic denture adhesive to provide increased retention. The authors recommended that the appliance be made for any infant requiring an oral tube for more than 24 hr, since in their experience 12 hr was the shortest period recorded for palatal groove formation.

Enamel Hypoplasia

Moylan et al. (1980) published the results of a study of 158 children who had required oral or nasal tracheal tubes as neonates. Of the 158 children, 28 (17.7%) had 90 defective teeth. Seventy-seven defective teeth were maxillary incisors (85.6%). The right maxillary incisors were involved 1.7 times as frequently as the left; all affected subjects had defects of the right maxillary central or lateral incisor. Fourteen of the affected subjects (50%) had defective development of all 4 maxillary incisors. The high frequency of dental defects on the maxillary right side was postulated to be due to trauma from laryngoscopy, since routine laryngoscopy may involve force by the laryngoscope on the tissues of the right side of the alveolus.

Wetzel (1980) explained that the high prevalence of right-sided dental defects reported by Moylan et al. (1980) was due to the custom of securing the orotracheal tube to the right side of the mouth, not trauma from laryngoscopy. Once secured with tape, dragging of the tube resulted in pressure necrosis of the alveolus and underlying tooth bud accounting for the dental defects. Variability in the position in which tubes were secured could cause erosions and subsequent dental defects in different sites.

Seow et al. (1984) reported on developmental defects of the primary dentition of low birth-weight infants who had prolonged endotracheal intubation. Of 40 intubated neonates, 34 (85%) had defects of the maxillary anterior teeth (56 total defective maxillary anterior teeth). Of the 23 control neonates, only 5 (21.7%) showed defects of the maxillary anterior teeth (19 total defective maxillary teeth). The difference was statistically significant ($P < 0.001$).

The duration of intubation and the prevalence of dental defects also were studied by Seow et al. (1984). Dental defects occurred in 66.7% (6/9) intubated for less than 1 day (usually less than 2 hr) compared to 74.2% (23/31) intubated from 2 to 64 days. Because the prevalence of hypoplasia seemed to be unrelated to the duration of intubation and since 2 hr seemed too short a period for an oral tube to cause hypoplasia, the authors speculated that the direct trauma from the laryngoscope was responsible.

In contrast to the Moylan et al. (1980) findings, Seow et al. (1984) reported that 37 (66.1%) of the defects occurred on the left side, compared to 19 (33.9%) on the right side. Of the 19 defective maxillary anterior teeth in control neonates, 9 (47.4%) occurred on the left side and 10 (52.6%) on the right side, an essentially identical distribution.

Seow et al. (1987) attempted to clarify the cause of enamel defects in prematurely born children by studying the prevalence of enamel defects in children with different birth weights: 77 very low birth weight (VLBW < 1500 g), 33 low birth weight (LBW 1500-2500 g) and 47 normal birth weight (NBW > 2500 g). At the time of examination the children ranged in ages from 9 to 42 months. The presence of enamel opacities, dental caries, and enamel hypoplasia along with postnatal medical and dental histories were analyzed. Enamel hypoplasia was found in 51.9% of the VLBW children, 21.1% of the LBW children, and 6.4% in the NBW children. In the VLBW group 63.1% of the enamel hypoplasia was observed on the maxillary left side and 36.9% on the maxillary right side. In the LBW and the NBW groups enamel hypoplasia appeared fairly consistent from side to side: LBW (58.3% left, 41.7% right), NBW (52.2% left, 47.8% right). Maxillary anterior teeth were affected twice as often on the left side as the right (67.2 vs. 32.8%) in intubated children from the VLBW group when compared to controls. Based on this evidence, the authors suggested that unilateral distribution of enamel defects in intubated neonates is most likely related to trauma from laryngoscopy and endotracheal intubation. Kopra et al. (1988) also reported maxillary incisor hypoplasia and a higher involvement of the left central incisor in 23% of neonates who had been intubated.

Discussion

Palatal Grooves/Arch Symmetry

Duke et al. (1980) described the palatal deformity resulting from orotracheal intubation as "cleft palate" even though there were no communications between the oral and nasal cavities since dye inserted into the nose in one of their cases did not discolor cotton placed in the palatal defect. Biskinis and Herz (1978) also reported no communication between the oral and nasal cavity in a neonate with palatal grooving. This apparent misnomer has been perpetuated in the literature with several publications listing cleft palate as a complication of orotracheal intubation. Recognizing this misnomer Molteni and Bumstead (1986b) wrote that the term "groove" does not imply a palatal defect or cleft, but a transient mechanical obstruction of normal growth and remodeling of the lateral palatine ridges.

More likely, the palatal deformity is related to normal morphology of the palate. The lateral palatine processes lie between the alveolar ridges and median palatine ridges in neonates. Hanson et al. (1976) suggested that the lateral palatine ridges are normal anatomic structures in the neonate and that the combination of molding pressure by the tongue and growth of the alveolus reduces the prominence of these structures over time. They indicated that individuals with neuromuscular defects tend to have reduced tongue movements and reduced pressure on the palatal vault, and thus maintain prominent lateral palatine ridges. What Ginther (1986) had described as "alveolar ridges" are in actuality the lateral palatine ridges and he merely reiterated what Behrstock et al. (1977) had described. While early reports associated palatal grooving with prolonged pressure from the orotracheal tube (Duke et al. 1976; Saunders et al. 1976), Behrstock et al. (1977) were the first to suggest that the inaction of the tongue was an etiologic factor in failure of remodeling of the palate.

Duke et al. (1976) first suggested that the duration of orotracheal intubation might be related to the formation of palatal grooves. Erenberg and Nowak (1984b) confirmed this suggestion, showing that neonates intubated for less than 7 days showed a 39.5% frequency of palatal grooving while those intubated for 15 or more days showed an 87.5% frequency. They attributed their higher reported incidence of groove formation in neonates intubated less than 7 days to a clear identification of the groove from their maxillary cast techniques rather than the simple intraoral evaluation used by others (Erenberg and Nowak 1986). The shortest period in which a palatal groove has been reported to have formed was 12 hr (Erenberg and Nowak 1984b). Molteni and Bumstead (1986c) observed the development of palatal grooves after only 7 days of intubation. The lack of grooving in neonates intubated less than 7 days has been attributed to the gestational age of the study population: all subjects were older than those in the group reported with 39.5% grooving before 7 days (Erenberg and Nowak 1984b).

If the presence of the tube on the palate inhibits a normal growth process, it is conceivable that the more premature, lower birth-weight neonates would

experience a higher incidence and severity of palatal grooving. However, Erenberg and Nowak (1984b) found no relationship between body weight and formation of palatal grooves. Sixty-three neonates and infants weighing 580-4400 g were represented in their sample. This range includes very premature, low birth-weight neonates and normal birth-weight infants. In contrast, Molteni and Bumstead (1986a) reported that development of palatal grooves was related closely to birth weight, with a 50% prevalence of grooving in neonates of < 1000 g as compared to 17% in neonates > 1000 g.

The incidence of palatal grooving appears to increase with increasing time of orotracheal intubation. The relationship of body weight and prematurity to palatal groove formation is less clear and deserves further investigation. Study of the incidence of palatal grooving as a function of prematurity and birth weight also may help to clarify the etiology of palatal grooving.

Duke et al. (1976) were the first to report apparent remodeling and decrease in size of a palatal groove 4 months postextubation. Behrstock et al. (1977) observed that the grooves diminished in prominence by age 2 years. Biskinis and Herz (1978) reported almost complete resolution after an unstated amount of time. Seow et al. (1985) reported that the lack of palatal asymmetry and previously intubated neonates suggested that remodeling occurred regardless of the intubation duration. Although the method of evaluating symmetry seems adequate, no data were given as to how many of the previously intubated neonates had grooves originally. Kopra et al. (1988), on the other hand, described high palatal vaults and palatal grooves (21%) in previously intubated 3- to 5-year-old children. Furthermore, 25% of the previously intubated children had posterior cross-bites while only 7-10% of the general population reportedly have posterior cross-bites (Woodside 1968; Kutin and Hawes 1969; U.S. Department of Health, Education, and Welfare 1973; Infante 1975). These data (Kopra et al. 1988) are the first to suggest that palatal grooving may have long-term effects on the development of the palate and symmetry of the dental arches. Because these results have been published only in abstract form, they await further review. It is recommended that previously intubated neonates be examined by a pediatric dentist for interarch evaluation and cross-bite correction where indicated.

Given the malleability, thinness, and open state of the palatine midline suture, it seems conceivable that direct pressure from the tube could distort the palate. The etiology of the observed grooving is better explained however by a combination of pressure from the tube, inhibition of the molding effect of the tongue on the lateral palatine ridges, and molding of the tissues adjacent to the tube. These factors may account for the formation of the groove in the midline of the palate and also explain the disappearance of grooves in some neonates after extubation.

Alveolar Grooves

Some orally intubated neonates acquire palatal grooving, while others acquire grooving of the alveolus. No patient has been reported to have shown both conditions.

Grooving of the alveolus cannot be explained by the proximity of the tube to the lateral palatine ridges as in palatal grooving. If Wetzel's (1980) report is accurate, the 7-mm alveolar defect he described probably was associated with pressure necrosis or the lateral displacement of the crypts of the primary incisors. An insult of this type to the alveolar ridge might cause the defects in the dentition described by Boice et al. (1976).

The position of the tube in the oral cavity may account for the lack of development of both palatal and alveolar grooving in the same patient. Wetzel (1980) felt that the practice of securing the tube to the upper lip accounted for alveolar grooving. It is possible that if the tube impinges on the palate, the pressure exerted causes palatal grooving. If, however, the tube is flexed away from the palate by external fixation the pressure of the tube may come to rest on the alveolus rather than the palate, thus causing alveolar grooving.

Only 3 cases of alveolar grooving (Boice et al. 1976; Krous 1980; Wetzel 1980) have been reported and no evidence concerning the resolution of the defects with time has been given. Additional studies of alveolar grooving and the long-term effects on the alveolus and dentition are merited.

Prevention of Grooves

The appliance described by Sullivan (1982) and recommended by Erenberg and Nowak (1984a) appears capable of securing oral tubes. The appliance appears capable of imparting protection to the oropharyngeal cavity from histologic changes associated with tube movement, palatal and alveolus grooves. Although the appliance requires continuous relining and refabrication due to rapid growth, no deleterious side effects have been reported.

Presently there is no consensus as to which neonates should receive the appliance. Erenberg and Nowak (1984b) reported the formation of a palatal groove in as little as 12 hr. However, no information concerning the size, depth, or severity of the groove was reported. The birth weight, age (degree of prematurity), and expected length of intubation all merit consideration.

Furthermore, utilization of such an appliance would require interdisciplinary cooperation among pediatric dentist, physicians, and nurses. If successful, the use of such a stabilizing appliance could become the standard of care for orally intubated neonates.

Enamel Hypoplasia

Two types of hypoplasia have been described. One follows a chronologic linear distribution pattern affecting many teeth forming simultaneously, whereas gross enamel hypoplasia is nonlinear in distribution and usually affects single teeth. Linear hypoplasia follows lines of tooth formation and the width of the defective enamel band is indicative of the duration of the insult if the ameloblasts recover their secretory function after the hypoplastic insult. The birth event, whether complicated or not, is recorded as a microscopic defect in the enamel of the forming primary dentition. The neonatal line may become visible at the enamel surface depending on the duration and severity of the insult (Schour 1936).

Enamel hypoplasia has been reported in 18-42% of preterm neonates (Stein 1947; Grahnen and Larssan 1958; Funakoshi et al. 1970; Grahnen et al. 1974; Mellander et al. 1982) with a preponderance of defects occurring in the maxillary incisor region. Although not previously described for primary teeth, certain permanent teeth have been reported to have a greater susceptibility to hypoplasia than others forming at the same time (Goodman and Armelagos 1985). The maxillary central incisors, lateral incisors, and canines are affected in order of decreasing susceptibility. Perhaps the same susceptibility to hypoplasia exists for primary teeth.

Johnsen et al. (1984) reported on the distribution of enamel defects and their association with respiratory distress in very low birth-weight infants. They found a significant amount of enamel hypoplasia in the incisal and middle thirds of low birth-weight infants born with respiratory distress. No hypoplasia was noted in the postnatally formed cervical third. These data show significantly greater hypoplasia in low birth-weight infants compared to controls of normal birth weight ($P < 0.05$). Enamel hypoplasia was more common in neonates suffering from severe respiratory distress syndrome and requiring mechanical ventilation with an orotracheal tube ($P < 0.01$). This seems plausible since neonates requiring intubation not only have the greatest number of neonatal risk factors, but also may have increased hypoplasia potentially associated with oral intubation itself. Seow et al. (1987) found hypoplasia in 51.9% of neonates with birth weights less than 1500 g, 21.2% of those from 1500 to 2500 g and 6.4% of those > 2500 g.

It is reasonable that the more premature the neonate the lower the birth weight and hence the greater the number of neonatal risk factors. Hypoplasia would be expected to increase then as a function of decreasing birth weight. This fact would not, however, explain the unilateral distribution of enamel hypoplasia that Moylan et al. (1980), Seow et al. (1984, 1985, 1986, 1987), and Kopra et al. (1988) have described. In addition, the defect described by these authors involves gross hypoplasia of the incisal edge, not the linear defects associated with prematurity. Their gross hypoplastic lesions would be far more reasonably ascribed to local trauma than systemic insult.

Seow et al. (1984) associated the unilateral distribution of hypoplastic maxillary incisors with traumatic intubation. Their paper merits some additional consideration since they grouped linear hypoplastic defects with opacity in their analysis. Yet no difference in opacities between full-term and premature infants has been described by others (Johnsen et al. 1984).

The inclusion of enamel opacities in their paper may have obscured the significance of the Seow et al. (1984) findings, particularly because many opacities were described in the primary first and second molars, teeth not in the region of the orotracheal tube or in the field susceptible to trauma from laryngoscopy. Retabulation of their data (eliminating opacities) reveals hypoplasia to be more prevalent in primary centrals, laterals, and canines which is consistent with the findings of others who have noted the same relationship in the permanent dentition. These retabulated data (Seow et al. 1984) show an almost threefold difference in the number of hypoplastic teeth on the left side (28) compared to the right side (10) in the intubated group. There is an almost fourfold difference in hypoplasia of the maxillary teeth of intubated compared to nonintubated neonates. Seow et al. (1987) reported a twofold difference in hypoplastic maxillary incisors from left to right side in intubated neonates.

The unilateral localization of hypoplasia in the maxillary left incisor region seems to implicate trauma from laryngoscopy as the etiology. Moylan et al. (1980), however, found 1.7 times as much hypoplasia of the right primary central incisor compared to the left. Differences in individual techniques of laryngoscopy may account for these apparently inconsistent data. Both investigators attributed the hypoplasia to the technique of laryngoscopy with the reported gross unilateral dental defects. Both described the placement of the laryngoscope from the right side, but Seow et al. (1984) claim the instrument is repositioned beyond the midline to the left side and leverage then exerted causes trauma on that side. Moylan et al. (1980) describes

damage to the right side which may reflect the side on which leverage is exerted prior to movement of the laryngoscope to the left, a somewhat different technique. However, Wetzel's (1980) explanation that the unilateral dental defects were due to the custom of securing the orotracheal tube to one side of the mouth and not from traumatic laryngoscopy cannot be entirely ruled out.

Studies of aborted fetuses by Kraus and Jordan (1965) suggest that calcification of the maxillary primary central incisor begins as early as 13 weeks or as late as 16 weeks in utero. At term, the maxillary primary central incisor crown is approximately 5/6 calcified (Lunt and Law 1974) at the height of the dentinoenamel junction, but by reason of the oblique calcification fronts associated with amelogenesis the central incisor is only 2/3 completely calcified at the enamel surface. Thus, while most charts of tooth formation list enamel crown formation of the primary maxillary central incisor at birth to be 5/6 complete, surface enamel defects of these teeth at birth are found at the level of 2/3 crown formation. With this in mind it becomes difficult to explain the dental defects of the incisal edges that some reports have associated with orotracheal intubation as a pure developmental defect. These reports (Moylan et al. 1980; Seow et al. 1984) presented photos of primary incisors with gross amounts of incisal tooth structure missing and implied that gross loss of enamel structure was due to interruption in amelogenesis. But, gross inspection of incisors from expired neonates, while revealing a fragile eggshell-like structure, show complete incisal enamel formation. However, lower birth weight and more premature neonates would be expected to have less developed, more fragile incisors. Given the fragility of the developing teeth, their incomplete calcification, and the close proximity of the teeth to the surface of the oral mucosa, it is quite possible that pressure and/or trauma from a laryngoscope is responsible for the gross tissue destruction.

Bilateral linear enamel hypoplasia of the incisal edges probably is associated with systemic disturbances related to early intrauterine insults, but certainly not associated with the timing of orotracheal intubation which results in gross unilateral hypoplasias directly due to the forces exerted during laryngoscopy.

The etiology of enamel hypoplasia has focused on metabolic disturbances during amelogenesis. To date no one has entertained seriously the notion that osteoclastic activity adjacent to injured, unerupted enamel surfaces might produce enamel defects. The ability of osteoclasts to destroy tooth structure cannot be denied, however, as much destruction of cementum, dentin, and enamel is evident in internal and external resorption.

Conclusion

Although continued research is encouraged, it appears that palatal grooving may have long-term sequellae affecting the symmetry of the palate and dental arches. The appliance advocated by Erenberg and Nowak (1984a) is capable of securing oral tubes that have been associated with histologic changes in the mucosa and protect the palate from grooving. The actual intubation of a neonate is complicated by the small size of the oropharyngeal complex and the critical health status. However, cautious avoidance of excessive pressure on the maxillary alveolus during intubation is suggested.

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Dr. Angelos is a commissioned officer with the U.S. Public Health Service and dental director, Brownsville Community Health Center, Brownsville, Texas; at the time of writing he was a pediatric dentistry resident at UTHSC-San Antonio. Dr. Jorgenson is a professor and Dr. Sweeney is interim chairman, pediatric dentistry, UTHSC-San Antonio, and Dr. Smith is a commissioned officer, U.S. Public Health Service, Rockville, Maryland. Reprint requests should be sent to: Dr. George M. Angelos, Brownsville Community Health Center, Dental Dept., 2137 E. 22nd St., Brownsville, TX 78520.

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