

Nursing caries: a comprehensive review*

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Nursing caries is a specific form of rampant decay of the primary teeth of infants. The distinguishing features of rampant caries are: (1) many teeth are involved; (2) lesion development is rapid; and (3) carious lesions occur on surfaces generally considered to be at low risk to decay, such as proximal surfaces of mandibular anterior teeth, facial surfaces of maxillary anterior teeth, and lingual surfaces of posterior teeth. A key feature of nursing caries is the usual absence of decay of the mandibular incisors, thus differentiating this condition from classical rampant caries.

Terminology

Nursing caries has been called by a number of names including nursing bottle caries, nursing bottle syndrome, milk bottle syndrome, baby bottle caries, and baby bottle tooth decay. The latter term has been endorsed by the Healthy Mothers-Healthy Babies coalition. The term "baby bottle tooth decay" is easily understood by nonprofessionals and, therefore, is useful in a program that educates parents about this condition. Nevertheless, it is not inclusive. Although improper nursing bottle habits are the most frequent cause of this condition, it also has been reported in infants who have been breast fed and those who have used a sweetened pacifier. While "nursing caries" is not an inclusive appellation because it fails to include the sweetened pacifier habit, it does not give the erroneous impression that a bottle always is necessary. This report, therefore, will use the more precise term "nursing caries."

Clinical Appearance

The intraoral decay pattern of nursing caries is characteristic and pathognomonic of the condition. The 4 maxillary incisors are most affected, while the 4 mandibular incisors usually remain sound. The other primary teeth, the canines, first molars, and second molars may

exhibit involvement depending upon how long the carious process remains active, but the extensiveness of the lesions usually is not as severe as those of the maxillary incisors.

Initially, the maxillary incisors develop a band of dull white demineralization along the gum line that goes undetected by the parents. As the condition progresses, the white lesions develop into cavities that girdle the necks of the teeth in a brown or black collar (Fig 1). In advanced cases, the crowns of the 4 maxillary incisors may be destroyed completely leaving decayed brownish-black root stumps (Fig 2). Conversely, the 4 mandibular incisors remain unaffected.

Table 1 indicates the surfaces of the primary teeth involved in children with nursing caries, reported by several authors (Fass 1962; Michal 1969; Picton and Wiltshear 1970; Dilley et al. 1980). All of the teeth listed in the table need not be affected and the canines and second molars often are sound. The symmetrical distribution of decay between the maxillary and mandibular teeth, with the exception of the mandibular incisors, is evident.

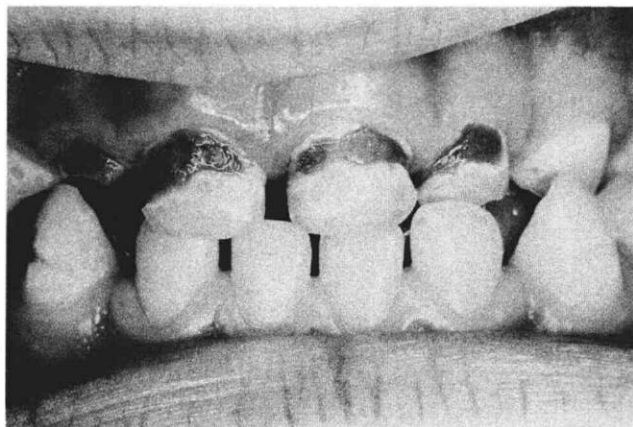


FIG 1. Intermediate stage of nursing caries showing circumferential lesions on the gingival third of the maxillary incisors. The mandibular incisors are unaffected (compliments of Dr. Robert Musselman).

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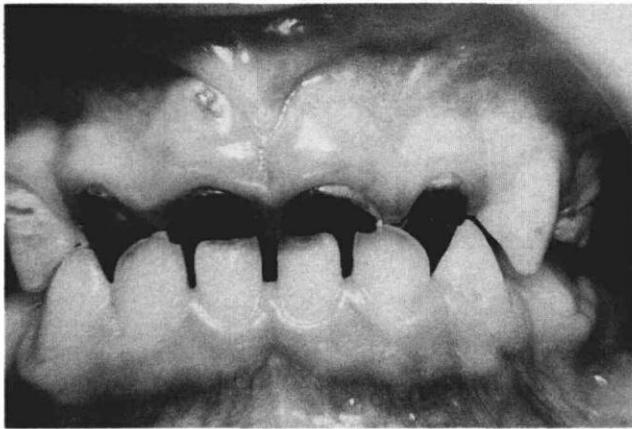


FIG 2. Advanced stage of nursing caries. There is complete destruction of the crowns of the maxillary incisors. The mandibular incisors are unaffected.

Explanation of the Caries Pattern

The reason for the unique distribution of caries between the maxillary and mandibular incisors and the unequal severity of the lesions between the incisors and the other teeth is related to 3 factors: the chronology of primary tooth eruption; the duration of the deleterious habit; and the muscular pattern of infant sucking.

A potentially cariogenic oral habit that begins soon after the child is born will affect the primary teeth as soon as they enter the mouth. The primary incisors erupt by 1 year, the canines and first molars before 2, and

the second primary molars by 2 years, 6 months (Table 2; Lunt and Law 1974). The maxillary incisors, which are among the first to erupt, will be the first to experience the cariogenic challenge and will suffer the longest caries attack. If the habit continues, the other teeth will be subjected to the cariogenic challenge in sequence with their eruption order.

Conversely, if the habit is discontinued by 1 year, 6 months to 2 years, newly erupted teeth, such as the canines and first molars, may be minimally affected, and the unerupted second molars would not be affected at all.

During sucking, the natural or artificial nipple rests against the palate, while the tongue is extended over the lower incisors. Liquid from the nursing bottle or a mother's breast will bathe all of the teeth except the lower incisors which are physically protected by the tongue. If the liquid is consumed frequently and for prolonged periods during the day or night, the liquid will pool around the teeth (Kroll and Stone 1967; Picton and Wiltshear 1970; Dilley et al. 1980). If the liquid contains a fermentable carbohydrate it will be metabolized by oral microorganisms into organic acids that demineralize the teeth. In this stagnant acid environment lesions can develop quickly and have been reported in children under 12 months (Suher et al. 1953; Brown et al. 1985). The maxillary incisors will be the most severely affected because of their early eruption. The mandibular incisors, protected by the tongue and washed by saliva from the mandibular salivary glands, remain sound.

TABLE 1. Caries Pattern in the Primary Dentition of Children with Nursing Caries

Surface	Central Incisor		Lateral Incisor		Canine		First Molar*		Second Molar*	
	Max	Mand	Max	Mand	Max	Mand	Max	Mand	Max	Mand
Facial	X		X		X	X				
Lingual	X		X		X	X				
Mesial	X		X		X	X				
Distal	X		X		X	X				
Occlusal	-	-	-	-	-	-	X	X	X	X

* Other surfaces will be involved as the duration of the nursing habit increases.

TABLE 2. Chronology of Eruption of the Primary Teeth in Months

Teeth	Mandibular	Maxillary
Central incisors	8	10
Lateral incisors	13	11
First molars	16	16
Canines	20	19
Second molars	27	29

One standard deviation is ± 2 or 3 months for the incisors, first molars, and canines, and ± 4 months for the second molars.

Source: Lunt RC, Law DB: J Am Dent Assoc, 1974.

Etiology

All carious lesions, including those associated with nursing caries, result from the interaction among 3 variables: (1) pathogenic microorganisms in the mouth; (2) fermentable carbohydrates that the microorganisms metabolize to organic acids; and (3) tooth surfaces that are susceptible to acid dissolution. In order for the lesions to progress and to be clinically diagnosed, these 3 variables

must interact over a suitable period of time (Fig 3, next page).

Microorganisms

Microbiologic studies clearly demonstrate that one of the variables necessary for caries initiation and progression can be present in infants' mouths shortly after the eruption of their first primary teeth.

Streptococcus mutans is the principal microorganism responsible for coronal caries in humans (Loesche 1985;

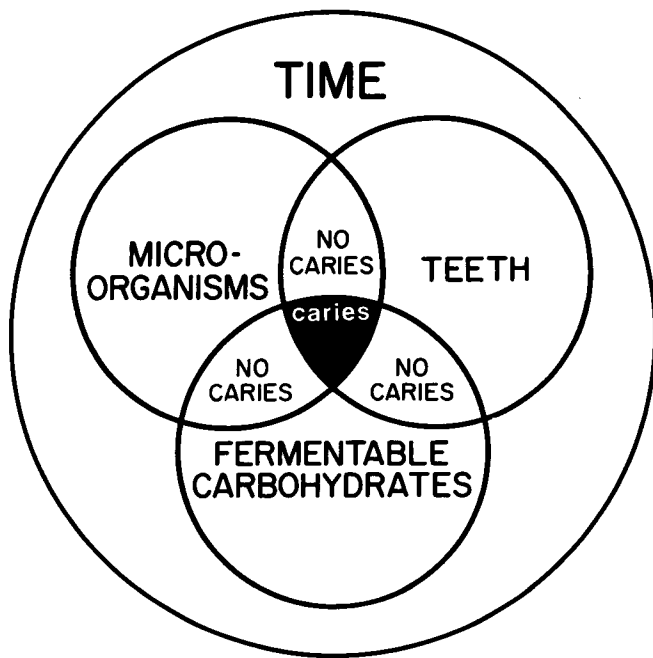


FIG 3. The four variables necessary for the initiation and progression of dental caries (modified from Newbrun).

1986). Because *S. mutans* primarily colonizes tooth surfaces (Krasse and Edwardsson 1966), the microorganism establishes itself in the mouth after teeth are present (Berkowitz 1985). Several clinical studies have failed to isolate *S. mutans* from the mouths of normal preerupted infants (Berkowitz et al. 1975; Carlsson et al. 1975; Catalanotto et al. 1975; Stiles et al. 1976; Berkowitz 1985). Berkowitz et al. (1975) found that 91 normal preerupted infants were free of *S. mutans*. Conversely, of 17 infants with erupted incisors, 8 (47%) harbored *S. mutans* in their plaque. Carlsson et al. (1975) studied 25 healthy infants until they were 5 years old. They found *S. mutans* in only a few 1 year olds, but as the children continued to erupt more teeth, more of them tested positive. By the age of 5 years, *S. mutans* had been identified in 21 of the 25 children.

The microorganisms responsible for dental caries can be transmitted from one individual to another. Several studies have indicated that the transmission of *S. mutans* to human infants is usually from their mothers (Berkowitz and Jordan 1975; Berkowitz et al. 1975; Masuda et al. 1980; Berkowitz et al. 1981). Maternal intrafamilial transmission should not be considered unusual since the mother is usually the "significant adult" caring for the infant; although, in one study maternal and paternal transfer of *S. mutans* was found with a similar frequency (Rogers 1981).

Saliva is the vehicle by which the transfer occurs (Duchin and van Houte 1978; Kohler and Bratthall

1978). Berkowitz et al. (1981) found a significant association between maternal salivary levels of *S. mutans* and the risk of infant infection. The frequency of infant infection was 9 times greater when maternal salivary levels of *S. mutans* exceeded 10^5 colony forming units (CFU)/ml compared to when maternal salivary levels were equal to or less than 10^3 CFU/ml. Likewise, Kohler and Bratthall (1978) found a correlation between the number of *S. mutans* in mothers and their 4 1/2 to 5-year-old children. This correlation was not as strong when the *S. mutans* counts of the children and their fathers were compared. Kohler and Bratthall (1978) also studied the transfer of *S. mutans* from adults to a flat metal surface resembling a common household spoon. Because *S. mutans* survived on the metal surface, the investigators postulated a scenario whereby *S. mutans* could be transferred from parent to infant:

"A mother with high numbers of *S. mutans* in her saliva is a source for infection in the close vicinity of the child. If she, for example, uses her own spoon to feed the child, she may introduce, each time, several hundreds of CFU into the mouth of the child. This is more than 100 times the number that a low infected mother will do. If she at the same time feeds the child with a sucrose-containing dish, she will fulfill another requirement for implantation. Objects such as glasses and forks, which the mother had had in her mouth, may harbor *S. mutans* for several hours."

S. mutans usually comprises less than 1% of the flora of children with negligible caries activity (Loesche et al. 1975; van Houte and Duchin 1975). In children with nursing caries, *S. mutans* is the predominant microorganism associated with the lesions (van Houte et al. 1982; Berkowitz et al. 1984; Milnes and Bowden 1985), and the proportion of *S. mutans* in the plaque and/or saliva is high. van Houte et al. (1982) and Berkowitz et al. (1984) studied a total of 13 children, ranging from 18 to 42 months old, with nursing caries. van Houte et al. (1982) reported that the concentration of *S. mutans* in the dental plaque of these children regularly exceeded 50% of the total cultivable flora, and in the saliva the concentration was 10%. Berkowitz et al. (1984) found the average proportion of *S. mutans* from plaque samples to be 30%. Both investigating teams commented on the high concentrations of *S. mutans* obtained from both carious and sound tooth surfaces.

Recruiting from maternal and child health clinics in Brisbane, Australia, Brown et al. (1985) took salivary samples from 112 infants younger than 2 years old and their mothers. They found statistically significant relationships between the *S. mutans* counts of the mothers and the infants, and between the *S. mutans* counts and number of erupted primary teeth. Eight infants in this

group had caries, 6 of whom were diagnosed as having nursing caries. Five of the 6 infants had moderate or high *S. mutans* counts, and all of their mothers' *S. mutans* counts were moderate or high (Table 3).

Fermentable Carbohydrates

The carbohydrate component of the diet is associated with the formation of dental caries. Certain carbohydrates are utilized by oral microorganisms, especially *S. mutans*, to form a sticky plaque matrix that enables the microorganisms to adhere to the teeth. The carbohydrates also serve as metabolites in the production of organic acids that demineralize the teeth (Kleinberg 1974; Loesche 1986).

Sucrose, or common table sugar, is a disaccharide composed of glucose and fructose. Sucrose is considered to be the major cariogenic food in the human diet (Newbrun 1969; Makinen 1972), and the intraoral colonization of *S. mutans* is highly dependent upon dietary sucrose levels (Loesche 1985). Many studies and case reports of children with nursing caries found that the nursing bottle was adulterated by the addition of table sugar or other cariogenic sweetening agents (James et al. 1957; Winter et al. 1966; Goose 1967; Goose and Gittus 1968; Curzon and Curzon 1970; Picton and Wiltshire 1970; Powell 1976; Dilley et al. 1980; Kammerman and Starkey 1981; van Houte et al. 1982). A common practice, especially in England, although the practice is reported to be declining (Holt et al. 1982), is the use of a pacifier (also called a "dummy" or "comforter") which has been dipped in a sugar solution or honey, a mixture of glucose and fructose (Shannon et al. 1979). Numerous reports have indicated an association between use of a sweetened pacifier and nursing caries in preschool children (James et al. 1957; Winter et al. 1966; Goose 1967; Goose and Gittus 1968; Winter et al. 1971; Derkson and Ponti 1982). Another practice, also prominent in reports from England, that has been implicated in nursing caries involves syrupy, sweet vitamin preparations added to nursing bottles or used with infant feeders, composed of a rubber nipple and small plastic container (James et al. 1957; Guggenheim et al. 1966; Goose and Gittus 1968; Winter et al. 1971; Roberts and Roberts 1979).

Bovine and human milk contain the carbohydrate lactose (Table 4). Laboratory studies have shown that lactose, a disaccharide composed of glucose and galactose, will enhance the oral implantation of cariogenic bacteria in animals, produce caries when fed to animals, and demineralize tooth enamel when acted upon by bacteria (Krasse 1965; Guggenheim et al. 1966; Koulourides et al. 1976; Brown et al. 1977; Schemmel et al. 1982). In humans, acid production in dental plaque will increase after frequent use of either lactose or milk (Birkhed

TABLE 3. *Streptococcus mutans* Scores of Mothers and Infants Younger Than Two Years Old with Nursing Caries

Age (mo)	No. of Erupted Teeth	No. of Carious Surfaces	<i>S. Mutans</i> Score*	
			Infant	Mother
11	6	4	High	Moderate
12	6	4	Moderate	High
13	4	2	High	High
13	10	8	High	High
19	16	3	High	High
22	16	13	Low	Moderate

* Estimated CFU/ml of paraffin stimulated saliva

low 0 - 10⁵

moderate 10⁵ - 10⁶

high > 10⁶

Source: Brown et al: Aust Dent J, 1985.

et al. 1981). Therefore, it is not surprising that the clinical reports, although rare, that children who consumed only bovine milk in their nursing bottle (Winter et al. 1966; Michal 1969; Powell 1976; Dilley et al. 1980) or were breast fed on demand (Gardiner et al. 1977; Kotlow 1977; Curzon and Drummond 1987) developed nursing caries.

In spite of these reports, the issue of either plain bovine milk or human milk being linked to nursing caries is complex (Finn 1969). This is because milk itself is a complex fluid, and, in addition to its potentially cariogenic lactose content, it contains ingredients that may protect against caries (Dreizen et al. 1961; Jenkins and Ferguson 1966; Weiss and Bibby 1966a, 1966b; Frustell 1970; Brown et al. 1977; McDougall 1977; Mor and McDougall 1977; Bibby et al. 1980; Reynolds and Del Rio 1984; Rugg-Gunn et al. 1985).

Bovine milk contains high concentrations of calcium and phosphorous (Table 4). Both are bound to organic and inorganic molecules in milk and are present in ionic form. By virtue of its calcium and phosphorous content, milk could contribute to the remineralization of enamel. Moreover, milk contains a number of proteins including casein (a milk phosphoprotein) and whey (the non-phosphorylated milk protein group) which could provide a protective organic coating on the enamel surface.

Jenkins and Ferguson (1966) showed that although

TABLE 4. Nutrient Content of Different Milk Sources

Milk Type	Constituents per 100 ml				
	Protein g	Fat g	Lactose g	Ca mg	P mg
Human	1.2	3.8	7.0	36	18
Bovine	3.3	3.7	4.0	120	95
Milk-formula	1.8	3.6	7.0	40	20
Soy-formula	2.0	3.5	0.0*	60	50

All types contain approximately 87% water.

* Lactose free, but approximately 7.0 g total carbohydrate.

bacterial action would produce acid from lactose, the amount of enamel dissolution in saliva was significantly less when milk provided the lactose to the incubation mixture compared to the same amount of lactose alone. By isolating the individual constituents of milk, they concluded that calcium and phosphate provided the resistance to the demineralization. In a separate study, Weiss and Bibby (1966a) also found that milk protected enamel against demineralization. They reported that enamel initially exposed to bovine milk was 20% less soluble in acetic acid buffer than unexposed enamel. The effect of the milk remained after the enamel had been washed, which would have removed inorganic calcium and phosphate, and in a subsequent report, Weiss and Bibby (1966b) identified the milk protein casein as being responsible for the acid protection.

Because it would be unethical to conduct human trials designed to measure caries produced by human or bovine milk, alternative approaches have been used. These include: (1) testing for cariogenicity in laboratory animals; (2) monitoring plaque pH in situ; and (3) conducting demineralization-remineralization laboratory experiments. These studies have shown the low cariogenic potential of milk or verified its anticariogenic properties. Studies on humans have found milk is slightly acidogenic in plaque (Frostell 1970; Mor and McDougall 1977); however, milk is less acidogenic than the same amount of lactose or sucrose alone (Mor and McDougall 1977) or milk supplemented with sucrose (Birkhed et al. 1981). Studies on caries production in laboratory animals found that the addition of milk to the diet decreased caries (Dreizen et al. 1961; Reynolds and Del Rio 1984). The addition of milk solids to laboratory cakes composed of sucrose and starch decreased enamel demineralization in an artificial mouth (Bibby et al. 1980). The exposure of enamel to milk between periods of demineralization in lactate buffer produced less demineralization than controls which only were subjected to the acid buffer (McDougall 1977). In the same report, the exposure of artificially demineralized lesions to milk for 50 hr resulted in remineralization.

This experimental evidence suggests that, under usual dietary conditions, milk is not very cariogenic and may be caries protective. This conclusion does not contradict the clinical evidence indicting milk in cases of nursing caries, because in children with this condition the usual dietary conditions do not prevail. Exposure to the milk is frequent and prolonged, resulting in pooling and stagnation around the necks of the teeth, especially the maxillary incisors. Under these conditions, human milk can be a greater offender than bovine milk because it contains nearly twice as much lactose (Table 4) and can

produce a greater drop in plaque pH and more enamel demineralization (Rugg-Gunn et al. 1985). As recorded in Table 4, the concentrations of the constituents in baby formula are similar to that of human milk, including the lactose content. Soy-protein formula is lactose free but contains an equivalent concentration of total carbohydrate, which, depending upon the brand, may be mostly sucrose.

Fruit juices and carbonated beverages also have been reported to be ingredients in the bottles of children diagnosed with nursing caries (Winter et al. 1966; Powell 1976; Dilley et al. 1980). Fruit juices naturally contain a sugar (fructose) and are intrinsically acidic. Carbonated beverages may have a sugar sweetening agent (frequently fructose) and an acid pH. Several studies have demonstrated the erosive effects of fruit drinks on enamel (Wynn and Haldi 1948; Holloway et al. 1958; Hartles and Wagg 1962; Smith 1987), and Winter et al. (1966) have suggested that when fruit juices are involved in nursing caries, erosion may be the primary enamel change preceding the rampant caries. In testing the effects on human plaque of orange, apple, and lemon juices, Frostell (1970) found that the fruit juices will produce an acid attack of short duration in which the pH decrease is comparable to that caused by a 25% sucrose rinse. He also reported a significant decrease in plaque pH from carbonated beverages.

Teeth

It is axiomatic that for dental caries to develop teeth must be present. However, the tooth vector of the caries tetralogy (Fig 3) assumes special significance in nursing caries because the individuals affected are infants who, only a few months previously, were without teeth.

Implantation of *S. mutans* can occur only when teeth are present because the teeth provide a nonshedding surface for colonization of the microorganisms. As previously noted, *S. mutans* counts in children are related to the number of erupted primary teeth (Berkowitz et al. 1980; Brown et al. 1985). In the study by Brown et al. (1985), there was a significant increase in the *S. mutans* scores of infants as the number of erupted teeth increased from one or more incisors to the full complement of primary teeth (Fig 4).

Reports of nursing caries have not indicated that the involved teeth have a greater intrinsic susceptibility to demineralization than teeth of unaffected infants. Nevertheless, progression of the lesions is quite rapid considering that the maxillary incisors erupt at 10-11 months of age on average, and nursing caries has been reported in children as young as 11-14 months (Suher et al. 1953; Kotlow 1977; Brown et al. 1985). One reason for the rapid progression of the lesions into dentin is the very thin enamel layer in primary incisors (~ 0.5 mm

RELATIONSHIP BETWEEN THE NUMBER OF ERUPTED PRIMARY TEETH AND SALIVARY STREPTOCOCCUS MUTANS SCORES IN INFANTS

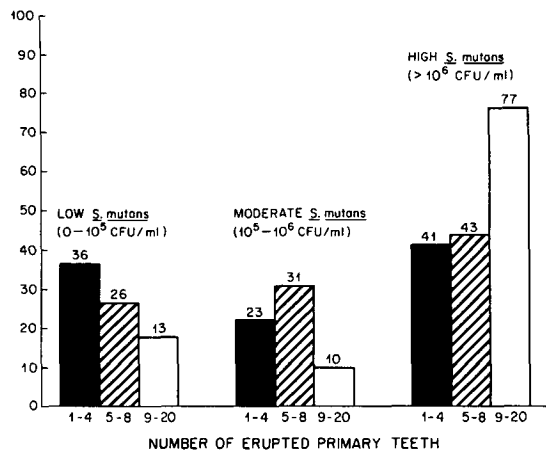


FIG 4. As more primary teeth erupt, the percentage of infants having high *S. mutans* scores increases (Brown et al: Aust Dent J 1985).

compared to more than 1.0 mm in permanent incisors). Nevertheless, it must be assumed that the principal reason for the rapidity of lesion development and progression is the result of the unremitting caries attack caused by the presence of *S. mutans* and the constant supply of fermentable carbohydrates.

Time

The temporal factor in nursing caries already has been implied in relation to the implantation and colonization of *S. mutans*. With few exceptions (Zinner and Jablon 1969; Masuda et al. 1979), *S. mutans* has not been identified in children until approximately 12 months of age. This circumstance, however, relates more to the eruption of primary teeth as a prerequisite for the implantation and colonization of the microbiologic vector of nursing caries rather than the passage of time per se (Berkowitz 1985).

Time is important in nursing caries in relation to the frequency and amount of exposure of the offending liquid. Derkson and Ponti (1982) have reported that children with nursing caries practiced the nursing habit 8.3 hr/day compared to only 2.2 hr/day for children without nursing caries. Such excessive exposure should influence both the initiation and progression of lesions. The nursing habit is continued intermittently during a 24-hr period, and it is believed that frequency of contact of the substrate has a major role in cariogenicity (Gustafsson et al. 1954). Recently, it also has been shown that when milk is taken frequently over a period of 4-6 weeks, there will be a greater decrease in plaque pH from subsequent milk ingestions (Birkhed et al. 1981).

Time is also important in relation to the duration of the deleterious habit. Duration should affect both the

severity of the lesions and the number of teeth involved. In Western civilization, infants generally are weaned from the breast or bottle by 1 year of age (Illingsworth 1975; Rudolph 1977), although weaning, especially from the breast, may be either prolonged or shortened due to social (Winter et al. 1971) or cultural influences (Richardson et al. 1981). Most reports of nursing caries stress the duration of the habit, be it bottle, breast, or sweetened pacifier, beyond the normal weaning period. For example, Winter et al. (1971) reported that children with nursing caries had maintained their habit for a mean period of 18.0 months compared to a caries-free group in which the mean period was 14.2 months. A 4-month difference may not seem large until it is realized that since the maxillary incisors erupt at approximately 10 months of age, this difference doubles the time of the cariogenic challenge. Dilley et al. (1980) reported that children with nursing caries discontinued their habit at a mean age of 23.4 months, but the upward range was as high as 54 months. Berkowitz et al. (1984) described 7 children with nursing caries. The duration of their habit ranged from 15 to 30 months. Three children who discontinued their habit between 15 and 21 months each had 6 carious teeth, the maxillary incisors and first molars. The children who discontinued their habit between 24 and 30 months had 8 or more carious teeth.

Also stressed in the reports of nursing caries is the practice of the habit at night (Kroll and Stone 1967; Picton and Wiltshear 1970; Powell 1976; Dilley et al. 1980) when the flow of saliva is diminished (Scheneyer et al. 1956) and demineralization can occur without the salutary physical and chemical influences of the saliva. The case reports of nursing caries in breast fed infants cite not only that the breast was available on demand, but, in most instances, that the child slept with the mother so that nursing could continue at will during the night (Gardiner et al. 1977; Kotlow 1977; Curzon and Drummond 1987).

Recently Babeely et al. (1987) documented the relationship between the severity of nursing caries and abusive feeding patterns. A nursing caries severity index was developed based upon the carious surface distribution and number of primary teeth involved. The severity of nursing caries was compared to the infants' feeding patterns, mostly time related, in which the average number of feeding times in a 24-hr period, the frequency of administering a bottle or breast at night, and the duration, in months, of the habit were considerations. In the 70 cases examined, the investigators found a strong and significant relationship between the severity of nursing caries and the degree of feeding abuse, indicating that the disease severity is a function of a combination of time-related deleterious feeding practices.

Prevalence

Determining the true prevalence of nursing caries is difficult. First, since the children are of preschool age, they are not as accessible for examination as are older children. Thus, those chosen for examination may not represent the general population for that age group. For instance, evaluating children who present for treatment to a dental facility (Powell 1976) will bias the sample by including many children whose parents are aware that a dental problem exists. Examining children at maternal and child health facilities (Currier and Glinka 1977; Brown et al. 1985) or Head Start centers (Johnsen et al. 1984b; Kelly and Bruerd 1987) will bias the sample by including a disproportionate number of a particular socioeconomic class. Second, because infant feeding habits are influenced by cultural and ethnic factors, the results of examining children of a similar cultural or ethnic type will be representative only for that type (Kelly and Bruerd 1987). Thus, nursing caries prevalence figures for children in one country usually cannot be extrapolated to another (Cleaton-Jones et al. 1978b; Aldy et al. 1979; Richardson et al. 1981). Moreover, the results compiled from one ethnic type cannot be extrapolated beyond that type, even within the same country (Richardson et al. 1981). Third, since infants are difficult to examine, examinations for nursing caries are less rigorous than those conducted on older children. When caries is diagnosed, the investigator must decide whether the condition is nursing caries. Criteria for this judgment differ. Some decide on the basis of at least 3 carious maxillary incisors (Kelly and Bruerd 1987), others on a minimum of 2 (Winter et al. 1966, 1971) or 1 (Cleaton-Jones et al. 1978b), and still others on the labial-lingual pattern of decay (Richardson et al. 1981).

With these caveats in mind, Table 5 presents prevalence figures for nursing caries from several countries throughout the world (Winter et al. 1966; Goose 1967; Goose and Gittus 1968; Winter et al. 1971; Powell 1976; Currier and Glinka 1977; Cleaton-Jones et al. 1978a, 1978b; Aldy et al. 1979; Richardson et al. 1981; Derkson and Ponti 1982; Holt et al. 1982; Johnsen et al. 1984; Brown et al. 1985; Kelly and Bruerd 1987).

The first country to approach nursing caries from an epidemiologic perspective was England. In 1967, Goose conducted a pilot study of 309 infants randomly selected from two locations and reported a nursing caries prevalence of 6.8%. That study was expanded to nearly 7000 children randomly selected from 72 locations in England and Wales (Goose and Gittus 1968). They found a prevalence of 5.9%. At about the same time, Winter et al. (1966) examined 100 children attending child welfare centers in London and found a prevalence

for nursing caries of 12%. An expanded follow-up study in the London borough of Camden by Winter et al. (1971) reported an 8.0% prevalence. A principal factor associated with nursing caries in these studies was the use of a sweetened pacifier or infant feeder. Approximately 13 years later, Holt et al. (1982) repeated the Camden study. They found a decline in the prevalence and severity of caries. The prevalence of nursing caries was 3.1% which they attributed, in part, to a decreased use of sweetened pacifiers.

Although not an actual prevalence study, a report by Powell (1976) provided one of the first indications of the extent of nursing caries in a sample of American children. Powell stated that, based upon an examination of more than 4000 children in the Los Angeles area, 40 or 1.0% had nursing caries. Although many of the details for which this observation was based are not provided, it is of interest that nursing caries was diagnosed by the clinical appearance of the teeth *and* a history of the child's nursing habits. This is important since others have made the observation that not all rampant caries in preschool children can be classified as nursing caries (Currier and Glinka 1977; Aldy et al. 1979; Brown et al. 1985), and, therefore, an appropriate diet history and review of feeding practices may be necessary for a definitive diagnosis. Currier and Glinka (1977) found a prevalence for nursing caries of 5.0% in predominantly black children attending a child health clinic in optimally fluoridated Richmond, Virginia. Johnsen et al. (1984b) reported a nursing caries prevalence of 11.0% in Head Start children from two fluoridated communities in Ohio. Since these children presented to a local dental school clinic from an initial sample of 350, the prevalence of nursing caries may be inflated because of a biased sample. Kelly and Bruerd (1987) examined 514 native American children attending Head Start programs in Alaska and Oklahoma and found the prevalence of nursing caries was 67 and 42%, respectively (average 53.1%). Their extremely high findings support anecdotal comments that the prevalence of nursing caries in native American children is high and are in agreement with earlier reports of high caries activity in Canadian Eskimo preschool children reported by Curzon and Curzon (1970), and in preschool Apache Indian children reported by Infante et al. (1975). By age 5, the Eskimo children had a mean deft of 10.7. More than half of their primary teeth were cariously involved. The high caries was attributed to the Eskimo habit of carrying their children on their back in a garment while at the same time providing them with a nursing bottle of condensed or powdered milk sweetened with sugar. The Apache children had a mean deft of 9.9 by age 5. By age 2, nearly 4 teeth were decayed, suggesting that nursing caries might be a factor in the condition, although the investigators considered enamel hypoplasia

TABLE 5. Prevalence of Nursing Caries in Different Populations

Country	Yr. of Publication	Investigator	Sample	Nursing Caries Criteria	Nursing Caries Prevalence
England	1967	Goose	Random selection of 309 1-2 year olds in 2 counties	Comparison with photographs of nursing caries	6.8%
	1968	Goose and Gittus	Random selection of 5549 1-2 year olds from 72 locations in England and Wales	Comparison with photographs of nursing caries	5.9%
	1966	Winter et al.	100 1-5 year olds attending a child welfare center in London	Minimum of 2 maxillary incisors with labial or lingual lesions	12.0%
	1971	Winter et al.	601 12-60 month olds attending child welfare centers in London borough of Camden	Minimum of 2 maxillary incisors with labial or lingual lesions	8.0%
	1982	Holt et al.	555 12-60 month olds presenting to 10 maternal and child welfare centers in Camden and Islington	Minimum of 2 maxillary incisors with labial or lingual lesions	3.1%
United States	1976	Powell	>4000 children seen at a Los Angeles hospital, dental school, clinic, public health service clinics, and private offices	Clinical appearance and history of nursing habits	1.0%
	1977	Currier and Glinka	180 predominantly black children in a child health clinic in Richmond, VA	Anterior smooth surface caries activity	5.0%
	1984b	Johnsen et al.	200 3.5 to 5-year-old Head Start children in 2 Ohio communities	Labiolingual lesions compatible with nursing caries	11.0%
	1987	Kelly and Bruerd	514 native American Head Start children in 18 locations in Alaska and Oklahoma	Cavitation of at least 3 maxillary incisors	53.1%
Canada	1982	Derkson and Ponti	594 children 9 months to 6 years old randomly selected from public health clinics and community centers in Vancouver	Labiolingual lesions on maxillary incisors	3.2%
Australia	1985	Brown et al.	112 children younger than 2 years attending maternal and child health clinics in lower middle-class suburbs of Brisbane	Bottle caries pattern	5.4%
South Africa	1978a	Cleaton-Jones et al.	499 1 to 5-year-old urban whites in Johannesburg	Caries on the labial surface of one or more incisor teeth	11.4%
				Caries on the labial surface of two or more incisor teeth	8.6%
	1978b	Cleaton-Jones et al.	439 rural black villagers; 192 urban blacks from Soweto, Johannesburg; 1-5 years old	Caries on the labial surface of one or more incisor teeth	13.7% (rural) 3.1% (urban)
	1981	Richardson et al.	437 rural black villagers; 192 urban blacks from Soweto, Johannesburg; 468 urban whites from Johannesburg	Caries on the labial surface of one or more incisor teeth	11.7% (rural) 4.0% (urban black) 12.2% (urban white)
Indonesia	1979	Aldy et al.	100 children younger than 5 years who visited a hospital clinic	Bottle caries pattern	48.0%

of the anterior teeth to be associated with the high caries rate.

Also presented in Table 5 are reports on the prevalence of nursing caries in children from Canada (Derkson and Ponti 1982), Australia (Brown et al. 1985), South Africa (Cleaton-Jones et al. 1978a, 1978b; Richardson et al. 1981), and Indonesia (Aldy et al. 1979). Only the figure from Indonesia approaches the high prevalence reported by Kelly and Bruerd (1987) in native Americans. It is probably not coincidental that a perusal of the recent studies listed in Table 5 involving children from predominantly Western-type cultures, (Powell 1976; Currier and Glinka 1977; Cleaton-Jones et al. 1978a, 1978b; Richardson et al. 1981; Derkson and Ponti 1982; Holt et al. 1982; Johnsen et al. 1984b; Brown et al. 1985) found, with only 3 exceptions (Cleaton-Jones et al. 1978; Richardson et al. 1981; Johnsen et al. 1984), the nursing caries prevalence to be approximately 5% or less (Fig 5). Based upon these observations, it is probable that the prevalence of nursing caries in the United States and other Western-type countries is no higher than 5%.

Prevention

The main strategy for preventing nursing caries is to alert prospective parents and new parents about the condition and its causes. In order to prevent the development of parental behavior that leads to nursing caries, prevention-oriented educational programs must reach prospective parents in their prenatal classes and other prenatal groups and in obstetricians' offices. Prospective parents should be taught that they can demonstrate love for their children without putting them to bed with a bottle or honey-dipped pacifier (Phillips and Stubbs 1987). As part of a Head Start program to prevent nursing caries, several activities were encouraged as alternatives to a nocturnal nursing bottle: the use of a security blanket or teddy bear, singing or playing music, and holding or rocking the child (Phillips and Stubbs 1987).

Information on nursing caries can be distributed to new parents from dental offices and clinics, pediatricians' offices, maternal and baby clinics, and birthing centers (Goepferd 1986). Ideally, families with infants who are at a high risk of developing nursing caries should be targeted. Unfortunately, studies have failed to identify a unique family profile that would predispose to the condition (Dilley et al. 1980; Johnsen 1982; Johnsen et al. 1984a). A common finding in families with nursing caries is parental overindulgence (Johnsen 1982; Johnsen et al. 1984a). While this finding is consistent with the perception that nursing caries is fundamentally a problem of poor parenting, parental overindulgence is not an uncommon trait and cannot be considered differential for nursing caries. Johnsen and

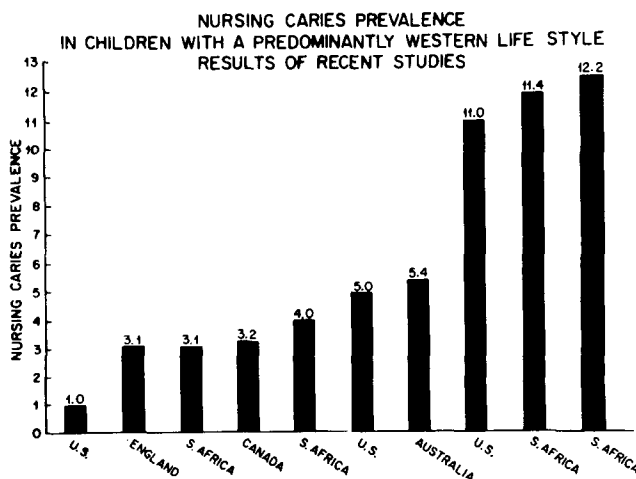


Fig 5. The majority of recent reports of nursing caries in Western-type cultures indicate the prevalence to be approximately 5% or less.

Dixon (1984) observed that children with clefts of the lip and palate are highly susceptible to caries of the primary incisors, but such children are already recognized as needing special dental care.

Common sense dictates that in communities with extremely high nursing caries rates all child bearing-age parents should be exposed to a personal educational program on this topic. Where nursing caries rates are not excessive, information should be disseminated to all prospective and new parents, but only those with a history of a nursing caries child need be the recipients of a more personal educational program. Although information on nursing caries has been available for years, it is disconcerting that in 1986 Tsamtsouris et al. reported that of 179 expectant parents attending prenatal classes at a hospital in the Boston area, 54% thought that a bottle of milk at other than regular feeding times would not harm the teeth of the infant, and 84% had never heard of nursing caries.

Schuman and Mills (1981) have cautioned that an educational program whose ultimate objective is to lower the incidence of nursing caries should not use high fear arousal techniques. Rather, there should be an appeal to parental concern and the promotion of the concept that the parents are contributing to the better health of their children. It is reasonable for parents to expect their infants not to be incessant criers. Health care advocates, therefore, must empathize with the parents by offering practical alternatives for a child who has already become accustomed to the bedtime bottle or breast. Weaning methods include progressively diluting the liquid in the nursing bottle until it is completely replaced with water or offering a pacifier that has not been dipped in honey or other sweet substances (Phillips and Stubbs 1987).

Several articles have described the contents of an education program on nursing caries that is directed to prospective or new parents (Gardiner et al. 1977; Shelton et al. 1977; Tsamtsouris and White 1977; Shearer et al. 1978; Kammerman and Starkey 1981).

1. The parents should be questioned about their child's feeding habits, especially regarding use of a nocturnal bottle, demand breast feeding, and use of a sweetened, e.g., honey-dipped pacifier.
2. The parents should be educated about the relationship between rampant caries and prolonged bottle or breast feeding, the nocturnal nursing habit, and use of a sweetened pacifier. These habits should be discouraged.
3. The parents should be encouraged to feed the infant only while the infant is being held.
4. If the parents insist that a night bottle is necessary for the child to sleep, suggest that the bottle contain only water.
5. The parents should be instructed to clean the infant's teeth after each feeding.
6. Weaning from the breast or bottle should begin at 6 months of age.

Nursing caries is the result of improper parenting. Feeding habits that produce carious destruction of the teeth are used to keep children pacified. It is likely that parents or other caretakers (e.g., baby sitters, grandparents) who resort to this method of behavior control also will use other methods that produce adverse dental consequences. Using sweetened beverages in the infant's spill-proof cup, or plying the infant with cookies, candy, or other sweets are examples of such methods. It is important, therefore, that an educational program about nursing caries be broad enough to include a discussion of other dentally related pernicious food habits.

Berkowitz (1985) lists 3 stages in the development of nursing caries: (1) primary infection; (2) accumulation of the organisms secondary to frequent carbohydrate ingestion via the nursing bottle; and (3) demineralization and cavitation of the tooth structure. An educational program directed at prospective and new parents intercedes at the second stage by eliminating frequent carbohydrate ingestion. It is theoretically possible to intercede in the first stage by preventing or delaying the primary infection (Berkowitz 1985). This may be accomplished by altering the *S. mutans* count in the significant adult caring for the child, usually the mother. Kohler et al. (1982) have reduced high salivary counts of *S. mutans* in mothers by a program consisting of: (1) dietary counseling; (2) professional tooth cleaning with a fluoridated prophylaxis paste, oral hygiene instruction, and topical fluoride application; (3) at-home use of a 0.05% sodium fluoride mouthrinse; and (4) excavation and

restoration of large carious lesions. Unresponding cases also used a chlorhexidine gel administered in mouth trays once-a-day for 2 weeks. Salivary *S. mutans* counts decreased from 10^6 CFU/ml to below a selected threshold of 3×10^5 CFU/ml, which represented an approximate 10-fold decrease. In a separate study, Kohler et al. (1983) showed that a reduction of *S. mutans* in mothers delayed or prevented the establishment of *S. mutans* in their infants during the observation period. The ultimate objective of such measures in parents is to reduce the caries incidence in their offspring. To date, however, no study has been reported in which an alteration of *S. mutans* in mothers has reduced the caries rates of their children.

Treatment

The treatment of children with nursing caries depends upon the extent of the lesions, the age and behavioral level of the child, and the degree of cooperation of the parents. Regardless of these factors, the first step in treating children with nursing caries is identifying the offending habit and eliminating it. It is surprising that Dilley et al. (1980) found that of 75 parents of children treated for nursing caries, 78% reported that they received no instructions to discontinue the bottle. Even when informed about the cause of the condition, many parents are resistant to the message. Johnsen (1982) reported that 40% of parents of children with incisor lesions were previously aware of the cariogenic potential of prolonged nursing, and several case reports of nursing caries associated with breast feeding have documented the reluctance of the parents to discontinue the nursing habit (Gardiner et al. 1977; Kotlow 1977; Curzon and Drummond 1987).

If the lesions are identified while still in the incipient or "white spot" stage, with minimal or no loss of enamel surface integrity, it is theoretically possible that they can be arrested. Since infants showing the early stages of nursing caries would be very young, their cooperation could not be expected and a dentist would need to rely primarily on the cooperation of the parents. Because scientific reports of caries arrest in infants are lacking, treatment recommendations for this age group must be empirical.

Treatment should be aimed at modifying all 4 factors in the caries tetralogy (Fig 3). Eliminating the deleterious habit will reduce the substrate and time factors. An analysis of the child's diet also should be performed to identify other cariogenic food habits and steps should be taken to correct them. If possible, the few teeth in the infant should be polished professionally. The parents must be taught how to clean their child's teeth using a child-size toothbrush and ADA-accepted fluoride den-

tifrice. The surfaces of the teeth should be carefully scrubbed after each feeding using a small amount of dentifrice on the bristles. Burnishing the fluoride dentifrice over the surface of the white spot lesions with cotton-tipped applicators also is desirable. Following dentifrice use, the teeth should be wiped with a gauze or cloth to remove residual dentifrice from the teeth so that it is not swallowed. The frequent brushings should disrupt plaque and reduce the number of bacteria on the teeth, and use of a fluoride dentifrice will result in the incorporation of fluoride into the white spot areas, promoting remineralization and rehardening of the enamel surface. Attention should also be directed to the principal adult caretaker of the child, usually the mother. She should be educated about those habits, such as using the same cups and utensils as the child, that could lead to reinfection of *S. mutans* in the child. Additionally, steps should be taken to reduce her *S. mutans* count (Kohler et al. 1982).

Unfortunately, early intervention and lesion arrest can only be utilized in a few cases; 20-23 months is the average age when parents recognize that their children have a rampant caries problem and by then the condition is usually advanced (Johnsen et al. 1984a).

When cavitation has occurred, more definitive treatment is required. Early stages of cavitation can be treated with intracoronal restorations. Advanced cases will need more complicated procedures including full crown coverage with polycarbonate crowns for the maxillary anterior teeth and stainless steel crowns for the posterior teeth. Depending on the extensiveness of the lesions, pulpotomies or extractions may be indicated. If teeth are extracted, a prosthesis for space maintenance and/or esthetics should be considered (Tsamtsouris and White 1977).

Treating a child with extensive lesions can be expensive. Kelly and Bruerd (1987) reported that the USPHS Indian Health Service and Head Start Bureau estimated that treatment costs for a single child with nursing caries was between \$700 and \$1200, exclusive of hospital costs if the child required general anesthesia. It is possible that in children who have discontinued the habit before professional intervention occurred, the lesions may be arrested. Considering such factors as cost of treatment, time until exfoliation, esthetics and function, treatment might be modified in cases where the lesions are no longer active.

In addition to treating existing carious teeth, future lesions must be prevented. Comparing two groups of preschool children, one of which had been treated for nursing caries and the other caries free, Johnsen et al. (1986) found that at later recall visits 53% of the nursing

caries group had one or more approximal molar lesions compared to 15% of the initially caries-free children. Caries preventive measures should include (Ripa and Whall 1986):

1. Diet analysis and modification of the diet by reducing the amount and frequency of sucrose consumption
2. Sealing all caries-free pits and fissures
3. Professional topical fluoride treatments every 3 months (treatment intervals should be changed to every 6 months when the child remains caries free for a 2-year period)
4. Daily home use of a 0.05% sodium fluoride mouthrinse by children 6 years and older
5. Systemic fluoride supplements if there is a suboptimal concentration of fluoride in the drinking water
6. Oral hygiene instruction to the child and parent and daily use of an ADA-accepted fluoride dentifrice.

Consideration also should be given to reducing potentially high *S. mutans* counts. Klock and Krasse (1978) tested a regimen for reducing the *S. mutans* counts of children with high levels of the microorganism. The regimen included professional toothcleaning once or twice a month with a fluoride prophylaxis paste, dietary and oral hygiene instruction, topical fluoride application, and sealing of pits and fissures. The children also participated in a school-based fluoride mouthrinsing program. (Most of these procedures are included in the above list of caries-preventive measures). The children in Klock and Krasse's treatment groups had their *S. mutans* counts lowered and, compared to a control group, developed fewer carious lesions during the 2-year study. However, the mean number of new carious lesions in the treatment group was ~ 1-1.5, indicating that even with a rigorous caries preventive program, it was not possible to completely inhibit new caries formation in children who, because of their high *S. mutans* counts, were deemed highly caries susceptible.

Summary

Nursing caries is a specific form of rampant decay in preschool children. Its clinical appearance is unique because the maxillary incisors show the greatest carious destruction while the mandibular incisors show the least. The confluence of 4 factors are necessary for nursing caries to occur — susceptible teeth, specific microflora, fermentable substrates, and time. While these 4 factors always are essential for the initiation and progression of dental caries, they are most obvious and identifiable in children with nursing caries.

The contents of the nursing bottle responsible for nursing caries can include milk sweetened with sugar, sugared water, fruit juices, carbonated or noncarbonated beverages, and bovine milk. Milk-based baby for-

mula, because of its lactose content, and soy-based formula, which is lactose free but contains sucrose or other sugars, also are potential promoters of nursing caries. Although numerous studies have demonstrated that bovine milk contains both organic and inorganic caries protective factors, it also contains 4% lactose that can serve as a substrate for *S. mutans*.

Apparently, the quantity and duration of the supply of lactose, resulting from the persistent nursing habit, turns a usually noncariogenic and potentially caries protective infant food staple into a cariogenic source. Case reports have also shown that human breast milk can cause nursing caries when breast feeding occurs on demand for many hours during the day and night. Another habit that can result in nursing caries is the prolonged use of a pacifier that has been sweetened with a substance such as honey.

The prevalence of nursing caries can range from 1 to more than 50%. However, in Western-type cultures, recent studies show it is usually about 5% or less.

Prevention of nursing caries requires an appropriate educational program to prospective and new parents. Treatment of nursing caries must begin by eliminating the deleterious habit. Fluorides should be used to decrease the acid solubility of the enamel surfaces and antimicrobial therapy should be instituted for the child and parent. For cases that are diagnosed early, the aim of treatment should be to arrest the lesions. In advanced cases, complex and usually costly dental procedures are required. Obviously, educational efforts to prevent nursing caries must be ongoing, especially in populations in which the prevalence is high.

Recommendations

Based upon this review, several considerations and recommendations concerning research in nursing caries and its management are appropriate.

Diagnostic Criteria

A common set of criteria must be established to identify nursing caries. How many maxillary primary incisors should have carious involvement for a child to be recognized as having nursing caries? Should the facial-lingual pattern of decay be a consideration? Is it necessary to obtain a positive history of a contributory habit in order to confirm a diagnosis of nursing caries?

Examination Procedures

Because many children with nursing caries are too young to give their full cooperation, accepted caries examination procedures are inappropriate. A universally acceptable examination method for this age group must be devised.

Prevalence Studies

The prevalence of nursing caries in the general American population as well as within targeted subgroups in the United States needs to be established. The relationship between the prevalence of nursing caries and such factors as socioeconomic status, rural vs. urban environments, the presence or absence of optimal water fluoride levels, cultural background, and dietary habits should be investigated in greater detail than heretofore published. It is noteworthy that national caries prevalence studies have been conducted on all age groups in the United States with the exception of preschool children.

Educational Preventive Programs

Priority needs to be given for a major national educational program directed toward educating the public about nursing caries. Although, based upon limited evidence, the prevalence of nursing caries may not be high in the general population, the age of victims of this condition as well as the cost of treatment provide potent incentives for such a program. For population subgroups in which the prevalence of nursing caries is known to be high, educational programs involving direct contact with pregnant women, parents, and other caretakers, such as baby sitters and grandparents, are essential.

Treatment

Because traditional treatment is so costly, alternative methods of handling children with nursing caries need to be explored. The use of fluoride to remineralize early lesions should be considered in relation to the potential for fluorosis of the developing permanent teeth from inadvertent chronic retention of the topical agent. The value of antimicrobial therapy in both the affected child and the parents should be investigated.

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Pediatric dental emergencies

Researchers studying pediatric dental emergencies in children age one month to 18 years found the following:

- Forty-six per cent of the 1456 emergency visits during the one-year study (almost four per day) were related to trauma. Male patients had a significantly higher percentage of trauma episodes than girls (53 vs. 37%).
- The greatest number of trauma emergencies (325) occurred in the youngest age group (birth to 3 years). Injuries in this group were almost entirely caused by accidental falls, while older children's injuries were related to athletics.
- Lacerations and abrasions were the most common trauma diagnosis. Actual dental trauma accounted for 35% while the incidence of bone fractures and TMJ injuries was 3%.
- Of the total emergency visits, 830 (57%) were nontraumatic events, with the most common diagnosis being dental caries with abscess formation. Tooth eruption anomalies and related complications were the second most frequent complaint