Probiotics are live microorganisms administered in adequate amounts with beneficial health effects on the host. A few conventional foods containing probiotics are yogurt, fermented and unfermented milk, and soya beverages. The term "probiotics" was introduced in 1965 by Lilly and Stillwell. Most often, they come from two groups of bacteria, Lactobacillus or Bifidobacterium, which are commonly found in the oral cavity, including caries lesions. These were the first probiotic species to be introduced into research (Lactobacillus acidophilus by Hull et al., 1984 and Bifidobacterium bifidum). The mechanism involved is shown in Figure 1.

Definition

World Health Organization (WHO) in 2001 defined probiotics as “live microorganisms which when administered in adequate amounts confer a health benefit on the host.” Lilly and Stillwell were the first to use the term “probiotics.” Parker defined probiotics as organisms and substances which contribute to intestinal microbial balance. Fuller redefined probiotics as “a live microbial feed supplement which beneficially affects the host animal by improving its intestinal microbial balance.”

Antibiotics destroy the harmful bacteria that can cause infection, while also destroying the good bacteria that help to fight infection (WHO 2002).

Prebiotics are generally defined as not digestible food ingredients that beneficially affect the host by selectively stimulating the growth and/or activity of one or a limited number of bacterial species already established in colon, and thus in effect improve host health. These prebiotics include inulin, fructooligosaccharides, galactosoligosaccharides, and lactulose (WHO 2002).

Synbiotics are defined as mixtures of probiotics and prebiotics that beneficially affect the host by improving the survival and implantation of live microbial dietary supplements in the gastrointestinal tract of the host.

The term replacement therapy (Bacteriotherapy) is sometimes used interchangeably with probiotics. Although both approaches use live bacteria for prevention or treatment of infectious disease, there are some slight differences (Victor 2010). The concept is “bacterial Interference,” whereby one microorganism can prevent and/or delay the growth and colonization of another member of the same or a different ecosystem.

History

Metchnikoff, 1907: Ingesting yogurt with lactobacilli reduces toxic bacteria of the gut and prolongs life
Kipeloff, 1926: Stressed the importance of L. acidophilus for good health
Rettger, 1930s: Early clinical application of Lactobacillus
Parker, 1974: First to use the term probiotics
Fuller, 1989: Defined probiotics
### Mechanism of Action

Vivek gupta *et al.* (2010) had given the following mechanisms:

- **Direct interaction**
  - Probiotics interact directly with the disease-causing microbes, making it harder for them to cause the disease.

- **Competitive exclusion**
  - Beneficial microbes directly compete with the disease developing microbes for nutrition or enterocyte adhesion sites.

- **Modulation of host immune response**
  - Probiotics interact with and strengthen the immune system and help prevent disease.

### Uses

- Elimination of lactose intolerance
- Anti-diarrheal
- Immunomodulatory
- Antidiabetic
- Anticarcinogenic
- Hypocholesterolemic
- Antihypertensive
- Anticarcinogenic: Colon, breast, and others
- Antiallergic
- Anti-inflammatory diseases: Inflammatory bowel disease (IBD), ulcerative colitis, Crohn’s disease, pouchitis, and postoperative complications

### Probiotics: Competitive Inhibition

They help to restore the balance of “good” bacteria and “bad” bacteria and facilitate the growth of healthy bacteria, i.e. *Bifidobacterium* and *Lactobacillus*. *Bifidobacterium infantis* inhibits the growth of *Salmonella* (O’Mahony, 2004).

### Probiotics: Barrier Protection

Intestinal permeability to bacteria is increased with inflammation, i.e. Crohn’s, ischemia (Nejdfors *et al.*, 1998). Pretreatment with *Lactobacillus plantarum* 299v inhibits *Escherichia coli* intestinal permeability (Mangell *et al.*, 2002). *B. infantis* prevents bacterial (*Salmonella*) translocation (O’Mahony, 2004).

### Probiotics and Immune Function

Mononuclear cells incubated with lactobacilli produce higher levels of interferon (IFN)-γ, tumor necrosis factor (TNF)-α, and interleukin (IL)-1 (MacFarlane and Cummings, 1999). Bifidobacteria suppressed the proinflammatory mediators (TNF-α, IFN-γ, IL-12) in a murine model of IBD (IL-10 knockout) (McCarthy *et al.*, 2003). In healthy volunteers, *Lactobacillus rhamnosus* increased phagocytic activity and natural killer (NK) tumor cell killing activity (Sheih *et al.*, 2001).

### Probiotics: Potential Uses

- Infectious diarrhea in children (i.e. rotavirus)
- Traveler’s diarrhea
- Antibiotic associated diarrhea
- *Clostridium difficile*

### Periodontal Diseases

Periodontal diseases are classified into two major types – gingivitis and periodontitis. Gingivitis is characterized by inflammation of gingiva, whereas periodontitis is a progressive, destructive disease that affects all supporting tissues of teeth, including the alveolar bone. The main pathogenic agents associated with periodontitis are *P. gingivalis*, *Treponema denticola*, *Tannerella forsythia*, and *Aggregatibacter actinomycetemcomitans* (Socransky). The treatment strategies conferred by probiotics against periodontal diseases are mainly thought to be either by inhibition of specific pathogens or by altering the host immune response through multifactorial causes.

Various probiotic organisms used in periodontal therapy are *Lactobacillus*, *Bifidobacterium* species, and *Streptococcus* species. These organisms can be delivered as food products (cheese, milk, yogurt) or supplements as chewing gum, lozenges, capsules, tablets, mouth rinses, sprays, etc. Probiotics can be used in treatment of various periodontal conditions like, gingivitis, periodontitis, and halitosis.
**Lactobacillus reuteri** is known for its secretion of two bacteriocins, reuterin and reutericyclin, that inhibit the growth of a wide variety of pathogens. It has a strong capacity to adhere to host tissues, thereby competing with pathogenic bacteria. The anti-inflammatory effects on the intestinal mucosa lead to the inhibition of secretion of proinflammatory cytokines, or a direct or indirect beneficial effect of this bacterium on people with periodontal disease (Krasse). Consumption of probiotic milk drink containing Lactobacillus Casei reduced the MMP-3, elastase activity in students with plaque induced gingivitis.

Riccia and colleagues in 2007 studied the anti-inflammatory effects of *Lactobacillus brevis* in a group of patients with chronic periodontitis. It has the capacity to prevent the production of nitric oxide and, consequently, the release of prostaglandin E2 (PGE2) and the activation of MMPs induced by nitric oxide. During the fermentation process in milk, *Lactobacillus helveticus* produces short peptides that act on osteoblasts and increase their activity in bone formation. These bioactive peptides could thereby contribute in reducing the bone resorption associated with periodontitis.[7]

Chewing Gum “perio balance” is the first probiotic specifically formulated to fight periodontal disease. It contains a combination of two strains of *L. reuteri*, specially selected for their synergetic properties in fighting cariogenic bacteria and periodontopathogens. Each dose of lozenge contains at least 2 × 10⁸ living cells of *L. reuteri* Prodentin. Users are advised to use a lozenge every day, either after a meal or in the evening after brushing their teeth, to allow the probiotics to spread throughout the oral cavity and attach to the various dental surfaces. Additional studies are required to evaluate the long-term effects of using these products.

Probiotics lower the pH so that plaque bacteria cannot form dental plaque and calculus that causes the periodontal disease. They produce antioxidants which prevent plaque formation by neutralizing the free electrons that are needed for the mineral formation. Probiotics are able to break down putrescence odors by fixating on the toxic gases (volatile sulfur compounds) and changing them to gases needed for metabolism. The most common probiotic strains belong to the genera *Lactobacillus* and *Bifidobacterium*. The species are *L. acidophilus*, *Lactobacillus johnsonii*, *L. casei*, *L. rhamnosus*, *Lactobacillus gasseri*, and *L. reuteri.*[8] Similarly, the *Bifidobacterium* strains include *B. bifidum*, *Bifidobacterium longum*, and *B. infantis*. Lactobacilli can produce different antimicrobial components including organic acids, hydrogen peroxide, low-molecular weight antimicrobial substances, bacteriocins, and adhesion inhibitors, and have gained prominence as probiotics. *Streptococcus oralis* and *Streptococcus uberis* have been shown to inhibit the growth of pathogens both in the laboratory and animal models.

Presence of *S. oralis* and *S. uberis* provides a good indication of health of periodontium. When these bacteria are absent from sites in the periodontal tissues, those sites are more prone to disease. The probiotic tablets (Wakamate D®), containing 6.7 × 10⁸ colony forming units (CFU)/tablet of *Lactobacillus salivarius* WB21 and xylitol (280 mg/tablet), were originally prepared to contribute for the intestinal microbial balance by providing acid-tolerant *L. salivarius* WB21. Using these tablets, it was found that orally administered *L. salivarius* WB21 significantly decreased the plaque index and probing pocket depth of subjects who were smokers, suggesting clinical improvement of the periodontal condition by probiotic intervention. A significant reduction in salivaary lactoferrin (Lf) levels was also observed for smokers at 8 weeks.

Probiotic species alter the balance of proinflammatory and anti-inflammatory cytokines secreted by epithelial cells. Elevated levels of TNF-α, IL-1, IL-6, and IL-8 are regarded as hallmarks of the inflammatory response in the intestine. The potentially protective role of probiotics in periodontal disease might benefit from adopting methods from studies in the gastrointestinal tract. However, so far, there have been no studies in this interesting area. Example for commercially available probiotic dietary supplement shown in the Figure 2.

*L. reuteri* and *L. brevis* are among the species able to affect gingivitis and plaque composition positively as well as being specific markers for periodontal disease. A significant decrease in gingival bleeding and a reduction in gingivitis were observed after a 2-week intake of probiotic species. This is due to the effective colonization of the probiotic bacteria within the oral cavity. The oral administration of a tablet containing *L. salivarius* WB21 was able to decrease the plaque index significantly and the pocket probing depth markedly in subjects who were smokers. *L. salivarius* WB21 reduces the prevalence of periodontal pathogens. This study stresses that a probiotic intervention could be a useful tool for the treatment of inflammation and the clinical symptoms of periodontitis ([Mayanagi, 2009]).[9] *L. acidophilus* present in a tablet named Acilact was first clinically tested by Pozharitskaia *et al.* in 1994 and they found improved clinical parameters in periodontitis patients and shifts in local microflora toward gram-positive cocci and lactobacilli.

Grudianov *et al.* in 2002 carried out a clinical study where they obtained a probiotic mix in the tablet forms, Acilact and Bifidumbacterin, and found normalization of microflora and reduction of signs of gingivitis and periodontitis. Shimazaki and colleagues used epidemiological data to assess the relationship...
between periodontal health and the consumption of dairy products such as cheese, milk, and yoghurt. The authors found that individuals, particularly nonsmokers, who regularly consumed yoghurt or beverages containing lactic acid, exhibited lower probing depths and less loss of clinical attachment than individuals who consumed few of these dairy products. By controlling the growth of the pathogens responsible for periodontitis, the lactic acid bacteria present in yoghurt would be in part responsible for the beneficial effects observed.

Breath malodor is a considerable social problem and majority of the pathologies (85%) causing halitosis are present in the oropharynx (tongue coating, gingivitis, periodontitis, tonsillitis). The common organisms implicated in halitosis are *Fusobacterium nucleatum*, *P. gingivalis*, *P. intermedia*, and *T. denticola*. These organisms degrade salivary and food proteins, and generate amino acids, which are in turn transformed into volatile sulfur compounds (VSCs). There will be re-colonization of halitosis-causing bacteria after treatment is stopped. To prevent the regrowth of odor-causing organisms, pre-emptive colonization of the oral cavity with probiotics might have a potential application as adjuncts for both the treatment and prevention of halitosis.

*Streptococcus salivarius* was detected most frequently among people without halitosis and is therefore considered a commensal bacterium of the oral cavity. It produces bacteriocins which reduce the number of bacteria that produce VSCs. The use of gum or lozenges containing *S. salivarius* K12 reduced the levels of VSCs among patients diagnosed with halitosis. However, additional studies with larger patient cohorts are needed to confirm the long-term potential of probiotics in preventing and/or treating halitosis (Burton et al.).[10]

**Products Available**

- Wakamata D
- Perio balance–Chewing gum
- Acilact
- Align – A digestive care probiotic supplement
- Cuturelle – A probiotic for digestive health
- Ganeden Sustenex – A dietary probiotic used to boost the immune system and digestive health
- Nature Made – A line of vitamins and supplements that has an acidophilus tablet
- Other brands: Natrol, Nature’s Bounty, Schiff, BioGaia, Sundown, Windmill, and more
- Central Food Technology and Research Institute, Mysore
- National Dairy Research Institute (NDRI), Karnal
- Institute of Microbial Technology, Chandigarh
- National Dairy Development Board, Anand
- Nestle Pvt. Ltd., Panipat

**Conclusion**

Probiotics represent a new area of research in periodontal therapy. However, longitudinal studies are required to clarify the observed relationship between regular consumption of products containing probiotics and periodontal health. Realizing the immense potential of probiotics and their relevance in meeting the nutritional and health care requirements from the national perspective, NDRI, Karnal, took an initiative and formed a national core group on probiotics and its first meeting was held at NDRI, Karnal, on 5 March 2010 to discuss some pertinent issues related to probiotic status in India. The 2nd meeting of the core group was held at NASC complex, New Delhi, on 15 November 2010.

**References**

1. Deepa D, Mehta DS. Is the role of probiotics friendly in the treatment of periodontal diseases! J Indian Soc Periodontol 2009;13:30-1.
2. Teughels W, Esswe MV, Siesiew I, Quirynen M. Probiotics and oral health care. Periodontology 2000 2008;48:111-47.
3. Victor DJ, Liu DTC, Anupama T, Devapriya AM. Role of probiotics and bacterial replacement therapy in periodontal disease management. SRM University J Dent Sci 2010;1:99-102.
4. Gupta V, Gupta B. Probiotics and periodontal disease: A current update. J Oral Health Comm Dent 2010:4(Spl):35-7.
5. Newman MG, Takei H, Curranza FA. Clinical Periodontology, 10th ed. Philadelphia: Saunders; 2006.
6. Socransky SS, Haffajee AD. The bacterial etiology of destructive periodontal disease current concepts. J Periodontol 1992;63:322-33.
7. Kazor CE, Mitchell PM, Lee AM, Stokes LN, Loesche WJ. Dewhirst FE, et al., Diversity of bacterial populations on the tongue dorsa of patients with halitosis and healthy patients. J Clin Microbiol 2003;41; 558-563.
8. Teanpaisan R, Dahlem G. Use of polymerase chain reaction techniques and sodium dodecyl sulphate-polyacrylamide gel electrophoresis for differentiation of oral Lactobacillus species. Oral Microbiol Immunol 2006;21:79-83.
9. Mayanagi G, Kimura M, Nakaya S, Hirata H. Probiotic effects of orally administered Lactobacillus salivarius WB21- containing tablets on periodontopathic bacteria: A double blinded, placebo-controlled, randomized clinical trial. J Clin Periodontol 2009;13:145-47.
10. Burton JP, Chilcott CN, Tagg J. The rationale and potential for the reduction of oral malodour using Streptococcus salivarius K 12 on oral malodour parameters. J Appl Microbiol 2006;100:754-64.

**Source of Support:** Nil, **Conflict of Interest:** None declared.
Significance of curve of Spee: An orthodontic review

Senthil Kumar K. P., Tamizharasi S.

ABSTRACT
Exaggerated curve of Spee is frequently observed in dental malocclusions with deep overbites. Such excessive curve of Spee alters the muscle imbalance, ultimately leading to the improper functional occlusion. It has been proposed that an imbalance between the anterior and the posterior components of occlusal force can cause the lower incisors to overerupt, the premolars to infraerupt, and the lower molars to be mesially inclined. This altered condition requires specialized skills for the practitioner. It would be useful if we have a thorough knowledge of how and when this curve of Spee develops, so that it will aid us in our treatment. The understanding of why the curve of Spee develops is limited in literature. The purpose of this article is to increase our knowledge regarding the development and its effect on dentition and its treatment in exaggerated cases.

KEY WORDS: Curve of Spee, leveling, occlusion

He used skulls with abraded teeth to define the line of occlusion as the line on a cylinder tangent to the anterior border of the condyle, the occlusal surface of the second molar, and the incisal edges of the mandibular incisors.

Most of Spee’s predictions were made from a view of skulls perpendicular to the midsagittal plane. He based his study using three propositions.

Proposition one: Spee indicated that from a profile view, the molar surfaces lie on the arc of a circle which, continued posteriorly, touches the anterior border of the condyle.

Proposition two: It is easy to demonstrate the curve in cases with marked attrition than in cases with well-preserved cusps.

Proposition three: When other points besides molars were included in measurements from the line of occlusion, they, along with the condyle, could be on a common arc.

Spee suggested that this geometric arrangement defined the most efficient pattern for maintaining maximum tooth contacts during chewing and considered it an important tenet in denture construction. This description became the basis for Monson’s spherical theory on the ideal arrangement of teeth in the dental arch.

Curve of Spee – Today

Today, in orthodontics, the curve of Spee commonly refers to

Department of Orthodontics, KSR Institute of Dental Science and Research, Tiruchengode, Namakkal (Dt), Tamil Nadu, India

Address for correspondence: Dr. S Tamizharasi, E-mail: senarasi@yahoo.co.in
the arc of a curved plane that is tangent to the incisal edges and the buccal cusp tips of the mandibular dentition viewed in the sagittal plane.\textsuperscript{14,16}

This anteroposterior curve, or curve of Spee, was defined as the anatomical curve established by the occlusal alignment of the teeth, as projected onto the median plane, beginning with the cusp tip of the mandibular canine and following the buccal cusp tips of the premolar and molar teeth, continuing through the anterior border of the mandibular ramus and ending at the anterior aspect of the mandibular condyle (Glossary of Prosthodontic terms 1994).\textsuperscript{19} The curvature of the arc would relate, on average, to part of a circle with a 4-inch radius.

More recently, it was suggested that the curve of Spee has a biomechanical function during food processing by increasing the crush/shear ratio between the posterior teeth and the efficiency of occlusal forces during mastication.\textsuperscript{10}

Development

Viewed in the sagittal plane, occlusal curvature is a naturally occurring phenomenon in the human dentition. Found in the dentitions of other mammals and fossil humans, this curvature was termed the curve of Spee in 1890 when a German anatomist, Ferdinand Graff Spee described it in humans.

The understanding of how the curve of Spee develops is limited in literature. Some suggest that its development probably results from a combination of factors including growth of orofacial structures, eruption of teeth, and development of the neuromuscular system.\textsuperscript{11} It has been suggested that the mandibular sagittal and vertical position relative to the cranium is related to the curve of Spee, which is present in various forms in mammals.\textsuperscript{9} In humans, an increased curve of Spee is often seen in brachycephalic facial patterns\textsuperscript{12,13} and associated with short mandibular bodies.\textsuperscript{14}

In a mechanical sense, the presence of a curve of Spee may make it possible for a dentition to resist the forces of occlusion during mastication.\textsuperscript{15-21} Although several theories have been proposed to explain the presence of a curve of Spee in natural dentitions, its role during normal mandibular function has been questioned.\textsuperscript{16,22,23} It has been proposed that an imbalance between the anterior and the posterior components of occlusal force can cause the lower incisors to overerupt, the premolars to infraerupt, and the lower molars to be mesially inclined.\textsuperscript{24,25} According to Root and Fidler et al.\textsuperscript{26} when a skeletal open bite is not present, the curve of Spee in Class II malocclusions is deeper than in other malocclusions.

Andrews\textsuperscript{27} noted that the occlusal planes in 120 non-orthodontically treated and ostensibly normal occlusions varied from being generally flat to having a slight curve of Spee. This finding led him to believe that the presence of a curve of Spee could be associated with post-orthodontic treatment relapse. Andrews concluded, “even though not all of the orthodontic normals had flat planes of occlusion, I believe that a flat plane should be a treatment goal as a form of overtreatment.” A deep curve of Spee may make it almost impossible to achieve a Class I canine relationship, though it may also result in occlusal interferences that will manifest during mandibular function.

It is perhaps worthwhile noting that very little research has been undertaken to determine the most effective method of leveling and to evaluate the long-term stability of leveling the curve of Spee.

Curve of Spee – From Flat to Mild

It has been suggested that the deciduous dentition has a curve of Spee ranging from flat to mild, whereas the adult curve of Spee is more pronounced. The findings were supported by Ash.\textsuperscript{28} Its greatest increase occurs in the early mixed dentition as a result of permanent first molar and central incisor eruption; it maintains this depth until it increases to maximum depth with eruption of the permanent second molars and then remains relatively stable into late adolescence and early adulthood. These findings also support those of Carter\textsuperscript{29} and McNamara\textsuperscript{29} and Bishara et al.\textsuperscript{30} that once established in adolescence, the curve of Spee appears to be relatively stable.

Certain cephalometric and dental factors are associated with individual variations in the curve of Spee, but they do not predict its biologic variance unequivocally. It appears that craniofacial morphology is just one of the many factors influencing its development.\textsuperscript{11,13} The curve of Spee is only influenced to a minor extent by craniofacial morphology. The curve is greatly influenced by the horizontal position of the condyle and is weakly influenced by the vertical craniofacial dimension and by the position of the mandible with respect to the anterior cranial base.

Mew\textsuperscript{31} quotes that whenever the curve of Spee is increased, the margins of the tongue will be seen to overlay the lingual cusps of the mandibular premolar, and the greater the curve, the more likely it is to overlay both the lingual and buccal cusps, often with scalloping.\textsuperscript{13,36} This is because that the tongue adapts to dental and skeletal forms, but there is no evidence to suggest that tongue posture is one of the determining factors of arch form.

Andrews in describing the six characteristics of normal occlusion found that the curve of Spee in subjects with good occlusion ranged from flat to mild, noting that the best static intercuspation occurred when the occlusal plane was relatively flat. He proposed that flattening the occlusal plane should be a treatment goal in orthodontics. This concept, especially as applied to deep overbite patients, has been supported by others\textsuperscript{37-42} and produces variable results with regard to maintaining a level after treatment.\textsuperscript{14,43-45}

Construction of Curve of Spee

Various authors have used various techniques to measure the depth of curve of Spee. The curve of Spee was universally likened to a part of a circle. In 1899, Bonwill proposed 4 inches (101.6mm) for the dimension of his “mandibular triangle.” Later, Monson (1932) proposed 4 inches as the radius of this
circle. However, Christensen (1959) reminds us that Wilson, in 1920, after measuring 300 mandibles, found only 6% of them in agreement with the 4-inch radius proposed by Bonwill. In fact, the mean radius of the curve, initially proposed by Spee himself, was much lower, 65–70 mm in adults. Similar values was obtained by Hitchcock (1983): 69.1 mm ± and Orthlieb (1997): 83.5 mm.

However, there is little consensus in the literature concerning the measurement of the curve of the Spee. Baldridge[45] used the perpendicular distances on both sides. Balridge and Garcia found the ratio to be more accurately expressed by the formulae: Y = 0.488x - 0.51 and Y = 0.657x + 1.34, respectively, where Y is the arch length differential in millimeters and x is the sum of right and left side maximum depths of the curve of Spee in millimeters.[46,47]

Bishara et al.[50] used the average of the sum of the perpendicular distances to each cusp tip. Sondhi et al.[51] used the sum of the perpendiculars. Braun et al.[50] and Braun and Schmidt[49] used the sum of the maximum depth on both sides. Traditionally, these measurements are taken from study models or photographs with a divider or caliper[44] and a coordinate measuring machine.[48]

The curve of Spee can also be determined by using a simplified occlusal plane analyzer (SOPA).[50] An SOPA is preset at 4 inches from the condylar axis. The SOPA works with Denar articulators. It is an excellent aid for establishing an ideal occlusal plane if all posterior teeth are to be restored.

Dawson (1989) described reconstruction of the curve of Spee[51] with a flag technique (The Broadrick Occlusal Plane Analyzer) which incorporated the same radius for almost all patients. The flag technique was recently redescribed by Lynch and McConnell (2002).

As technology advanced, new measuring devices became available, e.g. 3-dimensional (3D) optical digitizers that accurately measure small changes. At present, 3D virtual models are available for clinicians, supplemented by dedicated software to perform the necessary measurements.

**Leveling the curve of Spee**

A review of literature reveals that there is disagreement among the proponents of the various orthodontic techniques that are used to level deep curves of Spee.[12,52-55] The discussion involves around which leveling technique produces the most effective overbite correction as well as the most stable long-term treatment outcomes. Clinicians who adhere to the Tweed philosophy of orthodontic treatment use continuous archwires that incorporate reverse curve of Spee to produce flat occlusal planes.

Accordingly, arch leveling occurs mostly by an extrusion of the lower premolar teeth in conjunction with a minimal intrusion of the mandibular incisor teeth. In contrast to the earlier approach, advocates of sectional arch orthodontics treat deep curve of Spee by intrusion of mandibular incisors while usually allowing the lower premolars to erupt into occlusion. These people believe that extruding posteriors will cause an increase in lower facial height. They further believe that in individuals with strong muscles of mastication, the orthodontically extruded buccal segments will tend to relapse after the orthodontic treatment, which will lead to recurrence of anterior deep bites.[52,56,57]

But a study conducted by Carcara et al.[11] with cases treated by Wick Alexander by his Alexander Discipline showed that curve of Spee could be leveled successfully and results were stable when continuous archwires mechanics were used. It must be kept in mind that not every straight wire appliance has the unique prescription that is part of the Alexander Discipline, namely the -5° torque in the mandibular incisor and the -6° distal tip built into the molar tubes. This unique appliance prescription may play a large role in allowing for an effective, and controlled, mandibular arch leveling. In addition, the mechanical principles of actively tying back a heat-treated curved archwire may contribute to the success of arch leveling.

**Correction of Exaggerated Curve of Spee**

Correction of exaggerated curve of Spee can be achieved by the following tooth movements:

1. Extrusion of molars
2. Intrusion of incisors
3. Combination of both movements

**Extrusion of posterior teeth**

One millimeter of upper or lower molar extrusion effectively reduces the incisor overlap by 1.5–2.5 mm. A very common method is the use of continuous archwires.[58] A close variation of this technique is to use mandibular reverse curve of Spee and/or maxillary exaggerated curve of Spee wires. Progressively increasing step bends in an archwire also levels the curve of Spee. Other common methods include the use of a bite plate, which allows the posterior teeth to erupt.

**Indications**

Indicated in patients with short lower facial height, excessive curve of Spee, and moderate-to-minimal incisor display.

**Disadvantages**

The stability is questionable in non-growing patients. Major disadvantages include excessive incisor display, increase in the interlabial gap, and worsening of gingival smile.[59,56] Flaring of incisors is a common disadvantage with reverse curve wires. The primary drawback of using step bends in archwires to level curve of Spee is the change in cant of the occlusal plane toward a deeper bite.

**Intrusion of incisors**

Intrusion of upper and/or lower incisors is a desirable method to
level curve of Spee in many adolescent and adult patients.\textsuperscript{[60-62]} The four common methods to facilitate intrusion of the upper incisors are:

- Burstone\textsuperscript{[61]}
- Begg and Kesling\textsuperscript{[64]}
- Ricketts\textsuperscript{[65]}
- Greig\textsuperscript{[66]}

All four designs apply tipback bends at the molars to provide an intrusive force at the incisors. All of them recognize the need for a light and continuous force application.

**Indications**

Intrusion is particularly indicated in patients with a large vertical dimension, excessive incision–stomion distance, and a large interlabial gap.

**Disadvantages**

A major risk factor associated with orthodontic treatment is external apical root resorption.\textsuperscript{[67-71]} Many clinicians seem to have a subjective opinion that incisor intrusion increases the risk of apical root resorption. Many recent clinical studies\textsuperscript{[72-78]} have proven that the use of intrusion arches with average force provide a healthy biologic response with negligible root resorption.

**Effects of Curve of Spee Leveling**

A study conducted by Pandis \textit{et al}.\textsuperscript{[79]} showed that Curve of Spee (COS) is mainly flattened by proclining the mandibular incisors. For 1 mm of leveling, the mandibular incisors were proclined 4°, without increasing the arch width. But Afzal and Ahmed\textsuperscript{[80]} measured the pretreatment and posttreatment plaster models and found that 1 mm of arch circumference necessary to level each 1 mm of COS was only an overestimation.

**Continuous archwire**

Bernstein \textit{et al}.\textsuperscript{[81]} performed a long-term cephalometric study and found that leveling of COS with the continuous archwire technique takes place by a combination of premolar extrusion and, to a lesser extent, by incisor extrusion. It is very effective in leveling the COS in patients with Class II Division I deep bite malocclusions treated without extractions when the initial COS is 2–4 mm.

**Comparison between rectangular and round archwires**

AlQabandi \textit{et al}.\textsuperscript{[82]} evaluated the effects of full continuous archwire, rectangular and round, in leveling and showed that in both groups, the lower incisors proclined with uncontrolled tipping, which can be probably attributed to the intrusive force introduced by the archwire being labial to the center of resistance of the lower incisors.

**Age changes**

The curve of Spee may get altered physiologically with age or pathologically in situations resulting from rotation, tipping, and extrusion of teeth.

As the age advances, there is a significant change in the curve of Spee and decrease in posterior disclusion during mandibular protrusion.\textsuperscript{[77]} Hence, as patients grow older, clinicians should be aware that the occlusal adjustments with age have gradually altered the curve of Spee of youth toward a more favorable individual occlusal curvature. Thus, if the curve of Spee is not maintained in these dentitions during full mouth rehabilitation, it may lead to interferences along the mandibular movements which will jeopardize the health of the masticatory system.

**Long-term stability**

The stability of leveling curve of Spee may be dependent on the specific nature of its correction. Additionally, various factors, such as growth and neuromuscular adaptation, may play a role in relapse. Simons and Joondeph,\textsuperscript{[78]} in a 10-year post-retention study, reported that proclination of lower incisors and a clockwise rotation of the occlusal plane during treatment were significant relapse factors. The stability of posterior extrusion is controversial. Variables such as the amount of growth and the patient’s age during treatment, muscle strength, adaptation, and the original malocclusion have all been postulated as factors contributing to the long-term stability of correction of curve of Spee.\textsuperscript{[81]}

Burzin and Nanda\textsuperscript{[84]} specifically investigated the stability of incisor intrusion and found that maxillary incisor showed insignificant relapse.

According to Praeter \textit{et al}.\textsuperscript{[85]} leveling the curve of Spee during orthodontic treatment seems to be very stable on a long-term basis.

**In maxillary arch**

Very few studies have examined the characteristics of the curve of Spee in the maxillary arch. A study conducted by Xu \textit{et al}.\textsuperscript{[86]} showed that the curve was significantly flatter in maxillary arch than in mandibular arch.

**Muscle force**

A highly significant correlation is demonstrated between the forward inclination of the superficial masseter muscle and the forward tilt of molar teeth in the sagittal plane, conforming to the posterior end of the curve of Spee. The tilt of the curve of Spee increases the crush/shear ratio of the force produced on food between the posterior molars.\textsuperscript{[87-89]}

**Sexual variation**

Marshall \textit{et al}.\textsuperscript{[4,90]} have shown in their study there are no significant differences in maximum depth of curve of Spee between either the right and left sides of the mandibular arch or the sexes.
Discussion

The study was performed to gain a thorough knowledge of the curve of Spee from orthodontic aspect. The articles were searched in relation to orthodontic field from the year of 1970. But more importance was given to the articles in the 2000 group. Of the 186 articles reviewed, 106 articles were omitted as they did not match with the study purpose. The 90 articles used for this article are given as references. In the 2000 group, most of the articles were based on construction of Spee and leveling. We found that importance to its development or prevention was very less.

Conclusion

The understanding of curve of Spee in the field of orthodontics is very important as orthodontists deal with it in virtually every patient they treat. But, however, articles offering an in-depth understanding of its cause and development, and influencing factors are very few in the literature. It starts its journey from the deciduous dentition and travels taking variable forms influenced by various factors till the edentulous condition of an individual. Hence, clinicians should be aware that the occlusal adjustments with age gradually alter the curve of Spee of youth toward a more favorable individual occlusal curvature.

The correction of curve of Spee in a non-growing individual always poses a great problem to the orthodontists. Hence, in future, more studies should be aimed at predicting the right age for the correction of exaggerated curve of Spee. Studies should also be aimed at preventing the exaggerated curve of Spee in younger age group.

References

1. Sal Carcara C, Preston B, Jureyda O. The relationship between the curve of spee, relapse, and the Alexander discipline. Semin orthod 2001;7:90-9.
2. Spee FG. The gliding path of the mandible along the skull. J Am Dent Assoc 1980;100:670-5.
3. Hitchcock HP. The curve of Spee in Stone Age man. Am J Orthod 1953;39:779-801.
4. Marshall SD, Caspersen M, Hardinger RR, Franciscom RG, Aquilino SA, Southard TE. Development of the curve of Spee. Am J Orthod 2008;134:344-52.
5. Ramford SP, Ash MM. Occlusion. 3rd ed. Philadelphia: W. B. Saunders; 1971.
6. Ekesson J. Management of temporomandibular disorders and occlusion. In: 5th ed. St Louis: Mosby; 2003. p. 67-197.
7. Van Blarcom CW. The glossary of prosthodontic terms. 8th ed. St Louis: Mosby; 2005.
8. 34. Ramford SP, Ash MM. Occlusion. 2nd ed. Philadelphia: W. B. Saunders; 1971.
9. Van Blarcom CW. Glossary of Prosthodontics. 6th ed. J Prosthet Dent 1994;71:43-104.
10. Monson GS. Applied mechanics to the theory of mandibular movements. Dent Cosmos 1932;74:1039-53.
11. Osborn JW. Orientation of the masseter muscle and the curve of Spee in relation to crushing forces on the molar teeth of primates. Am J Phys Anthropol 1993;92:99-106.
12. Wylie WL. Overbite and vertical facial dimensions in terms of muscle balance. Angle Orthod 1994;19:13-7.
13. Bjork A. Variability and age changes in overjet and overbite. Am J Orthod 1963;39:779-801.
14. Salem OH, Al-Sehaibany F, Preston CB. Aspects of mandibular morphology, with specific reference to the antagonel notch and the curve of spee. J Clin Pediatr Dent 2002;27:261-5.
15. Root T. Level Anchorage. Monrovia, CA: Unitek Corp, 1988.
16. Sicher H. Oral Anatomy. St. Louis: CV Mosby, 1949.
17. Hemley S. Orthodontic theory and practice. 2nd ed. New York: Grune and Stratton; 1953.
18. Wheeler RC. A textbook of dental anatomy and physiology. 2nd ed. Philadelphia: W.B. Saunders; 1950.
19. Ash MM. Wheeler’s dental anatomy, physiology and occlusion. 6th ed. Philadelphia: W.B. Saunders; 1984.
20. Osborn JW. Orientation of the masseter muscle and the curve of Spee in relation to crushing forces on the molar teeth of primates. Am J Phys Anthropol 1993;92:99-106.
21. Mohl ND, Zarb GA, Carlsson GE, Rugh JD. A textbook of occlusion. Copenhagen: Munksgaard; 1988.
22. Dawson P. Evaluation, diagnosis and treatment of occlusal problems. St. Louis: CV Mosby; 1974.
23. Diamond M. Dental Anatomy. 3rd ed. New York: McMillan; 1952.
24. Strang RH. A textbook of orthodontia. 3rd ed. Philadelphia: Lea and Febiger; 1950.
25. Gresham H. A manual of orthodontics. Christ Church, New Zealand: N.M. Peryer; 1957.
26. Fidler BC, Artun J, Joondeph DR, Little RM. Long-term stability of angle class II, Division I malocclusions with successful occlusal results at the end of active treatment. Am J Orthod 1995;107:276-85.
27. Andrews PL. The six keys to normal occlusion. Am J Orthod 1972;62:296-309.
28. Ash M. Wheeler’s dental anatomy, physiology and occlusion. 7th ed. Philadelphia: W.B. Saunders; 1993.
29. Carter GA, McNamara JA. Longitudinal dental arch changes in adults. Am J Orthod Dentofacial Orthop 1998;114:88-99.
30. Bishara S, Jakobsen J, Treder J, Stassi M. Changes in the maxillary and mandibular tooth size–arch length relationship from early adolescence to early adulthood (A longitudinal study). Am J Orthod Dentofacial Orthop 1989;95:46-59.
31. Farella M, Michelotti A, Martina R. The curve of Spee and craniofacial morphology: A multiple regression analysis. Eur J Oral Sci 2002;110:277-81.
32. Shannon KR, Nanda R. Changes in the curve of Spee with treatment and at 2 years posttreatment. Am J Orthod Dentofacial Orthop 2004;125:589-96.
33. Baydas B, Yavuz I, Atasarli N, Ceylan T, Dagsuyu I. Investigation of the changes in the positions of upper and lower incisors, overjet, overbite, and irregularity index in subjects with different depths of curve of Spee. Angle Orthod 2004;74:349-55.
34. Mew J. The curve of Spee. Am J Orthod Dentofacial Orthop 2009;135:3.
35. Mew JR. The aetiology of malocclusion: can the tropic premise assist our understanding. Br Dent J 1981;151:296-302.
36. Mew JR. The postural basis of malocclusion: A philosophical overview. Am J Orthod Dentofacial Orthop 2004;126:729-38.
37. Tweed CH. Clinical orthodontics. In: St Louis: Mosby; 1966. p. 84-180.
38. Schudy FF. The control of vertical overbite in clinical orthodontics. Angle Orthod 1968;38:19-38.
39. Burstone CR. Deep overbite correction by intrusion. Am J Orthod 1977;72:1-22.
40. Koyama TA. Comparative analysis of the curve of Spee (lateral aspect) before and after orthodontic treatment with particular reference to overbite patients. J Nihon Univ Sch Dent 1979;21:25-34.
41. Otto RL. Anholm JM, Engel GA. A comparative analysis of intrusion of incisor teeth achieved in adults and children according to facial types. Am J Orthod 1980;77:437-46.
42. Garcia R. Leveling the curve of Spee: A new prediction formula. J Orthod 1999;26:101-113.
43. Osborn JW. Relationship between the mandibular condyle and the occlusal plane during hominid evolution: Some of its effects on jaw mechanics. Am J Phys Anthropol 1987;73:193-207.
44. De Praeter J, Dermant L, Martens G, Kuipers-Jagtman AM. Long-term stability of the leveling of the curve of Spee. Am J Orthod Dentofacial Orthop 2002;121:269-72.
45. Baldridge DW. Leveling the curve of Spee. J Clin Pediatr Dent 2002;27:261-5.
46. Root T. Level anchorage. Monrovia, CA: Unitek Corp, 1988.
47. Sicher H. Oral Anatomy. St. Louis: CV Mosby, 1949.
48. Hemley S. Orthodontic theory and practice. 2nd ed. New York: Grune and Stratton; 1953.
49. Wheeler RC. A textbook of dental anatomy and physiology. 2nd ed. Philadelphia: W.B. Saunders; 1950.
50. Osborn JW. Orientation of the masseter muscle and the curve of Spee in relation to crushing forces on the molar teeth of primates. Am J Phys Anthropol 1993;92:99-106.
51. Mohl ND, Zarb GA, Carlsson GE, Rugh JD. A textbook of occlusion. Copenhagen: Munksgaard; 1988.
52. Dawson P. Evaluation, diagnosis and treatment of occlusal problems. St. Louis: CV Mosby; 1974.
53. Diamond M. Dental Anatomy. 3rd ed. New York: McMillan; 1952.
54. Strang RH. A textbook of orthodontia. 3rd ed. Philadelphia: Lea and Febiger; 1950.
Kumar and Tamizharasi: Curve of Spee

70. Harry MR, Sims MR. Root resorption in bicuspid intrusion. A scanning electron microscopic study. Angle Orthod 1982;52:235-68.

71. Ketcham A. A progress report of an investigation of apical root resorption of vital permanent teeth. Int J Orthod Oral Surg Rad 1929;25:310-28.

72. Baumrind S, Korn EL, Boyd RL. Apical root resorption in orthodontically treated adults. Am J Orthod Dentofacial Orthop 1996;110:311-20.

73. Kaley JP, Phillips C. Factors related to root resorption in edgewise practice. Angle Orthod 1991;61:125-32.

74. Goerigk BD, Wehrbein H. Intrusion of anterior teeth with the segmented arch technique of Burstone – a clinical study. Fort der Kieferorthopadie 1992;53:16-25.

75. Dermatt LR, De Munck A. Apical root resorption of upper incisors caused by intrusive tooth movement: a radiographic study. Am J Orthod Dentofacial Orthop 1986;90:321-6.

76. Costopoulos G, Nanda R. An evaluation of root resorption incident to orthodontic intrusion. Am J Orthod Dentofacial Orthop 1996;109:543-8.

77. Ahmed I, Nazir R, Gul-e-Erum, Ahsan T. Influence of malocclusion on the depth of curve of Spee. J Pak Med Assoc 2011;61:1056-9.

78. Simons ME. Joondarph DR. Change in overbite: A ten-year postretention study. Am J Orthod 1973;64:349-67.

79. Pandis N, Polychronopoulos A, Sifakis I, Makou M, Eliades T. Effects of leveling of the curve of Spee on the proclination of mandibular incisors and expansion of dental arches: A prospective clinical trial. Aust Orthod J 2010;26:61-5.

80. Afzal A, Ahmed I. Leveling curve of Spee and its effect on mandibular arch length. J Coll Physicians Surg Pak 2006;16:709-11.

81. Bernstein RL, Preston CB, Lampassso J. Leveling the curve of Spee with a continuous archwire technique: A long term cephalometric study. Am J Orthod Dentofacial Orthop 2007;131:363-71.

82. AlQabandi AK, Sadowsky C, Begole EA. A comparison of the effects of rectangular and round arch wires in leveling the curve of Spee. Am J Orthod Dentofacial Orthop 1999;116:522-9.

83. Berg R. Stability of deep overbite correction. Eur J Orthod 1993;5:75-83.

84. Burzin J, Nanda R. The stability of deep overbite correction. In: Nanda R, editor. Retention and stability. Philadelphia: WB Saunders; 1993.

85. De Praeter J, Dermaut L, Martens G, Kuijpers-Jagtman A-M. Long-term stability of the leveling of the curve of Spee. Am J Orthod Dentofacial Orthop 2002;121:266-72.

86. Xu H, Suzuki T, Muronai M, Ooya K. An evaluation of the curve of Spee in the maxilla and mandible of permanent healthy dentitions. J Prosth Dent 2004;92:536-9.

87. Woods M. A reassessment of space requirements for lower arch leveling. J Clin Orthod 1986;20:770-8.

88. Lynch CD, McConnell RJ. Prosthodontic management of the curve of Spee: Use of the Broadrick flag. J Prostheth Dent 2002;87:593-97.

89. Garcia R. Leveling the curve of Spee: A new prediction formula. J Prosthet Dent 2004;92:536-9.

90. Source of Support: Nil, Conflict of Interest: None declared.
“Early baby teeth”: Folklore and facts

Uma Maheswari N., Kumar B. P., Karunakaran1, Thanga Kumaran S.2

ABSTRACT

Variations in the newborn’s oral cavity have been an enduring interest to the pediatric dentist. The occurrence of natal and neonatal teeth is a rare anomaly, which for centuries has been associated with diverse superstitions among many different ethnic groups. Natal teeth are more frequent than neonatal teeth, the ratio being approximately 3:1. The purpose of this case report is to review the literature related to the natal teeth folklore and misconceptions and discuss their possible etiology and treatment.

Case Report

A 2-day-old male infant was referred with the complaint of two teeth in the lower jaw since birth, continuous crying, and refusal to suck milk. Oral examination revealed two crowns of the teeth in the mandibular anterior region [Figure 1], whitish opaque in color and exhibiting grade III mobility. The crown size was normal; the gingiva was of normal appearance. A diagnosis of natal tooth was made. Since immediate extraction was the treatment of choice, the pediatrician was consulted and vitamin K was administered intramuscularly as a part of immediate medical care to prevent hemorrhage; and the teeth were extracted under topical local anesthesia [Figure 2], which the patient tolerated well. The extracted teeth had a crown but were devoid of roots [Figure 3]. The patient was reevaluated after 7 days, and the recovery was found to be uneventful [Figure 4].

Review of Literature

Folklore and fact

The occurrence of natal and neonatal teeth for centuries has been associated with diverse superstitions among many different ethnic groups. In some cultures like Malaysian communities, a natal tooth is believed to herald good fortune. Chinese community considers presence of these teeth as a bad omen and the affected children are considered to be monsters and beavers of misfortune. Shakespeare contributed his thoughts on natal teeth in “King Henry the Sixth” when he refers to Richard the Third in his quotation, “teeth hadst thou in thy head when thou wast born to riguity thou camest to bite the word.”[1] In England, the belief was that this condition would guarantee the conquest of the world.[2]
Massler and Savara (1950)\(^3\) defined these teeth as natal and neonatal teeth, taking only the time of eruption as a reference. This definition has been widely accepted and followed. Natal teeth are those teeth that are present at the time of birth and neonatal teeth are those teeth that erupt within the first 30 days of life. Terms such as congenital teeth, fetal teeth, predeciduous teeth, and precocious dentition, as well as Dentitia praecox and dens cannatalis, have been used to describe these teeth.

**Classification**

Spoug and Feasby (1966) have suggested that clinically, natal and neonatal teeth are further classified according to their degree of maturity.\(^2\)

1. A mature natal or neonatal tooth is the one which is nearly or fully developed and has relatively good prognosis for maintenance.
2. The term immature natal or neonatal teeth, on the other hand, implies a tooth with incomplete or substandard structure; it also implies a poor prognosis.
3. The appearance of each natal tooth into the oral cavity can be classified into four categories given below, as the teeth emerge into the oral cavity.\(^2,4\)

4. Shell-shaped crown poorly fixed to the alveolus by the gingival tissue and absence of a root.
5. Solid crown poorly fixed to the alveolus by the gingival tissue and little or no root.
6. Eruption of the incisal margin of the crown through the gingival tissues.
7. Edema of the gingival tissue with an unerupted but palpable tooth.

If the degree of mobility is more than 2 mm, the natal teeth of category (1) or (2) usually need extraction.\(^4\)

**Incidence and prevalence**

The incidence of natal and neonatal teeth has been estimated to be 1:1000 and 1:30,000\(^5,6\). Reports about significant difference in males and females are conflicting, with females, in general, being more affected. Natal teeth are more frequent, approximately three times more common than neonatal teeth,\(^1\) with the most common localization being the mandibular region of the central incisors (85%), followed by maxillary incisors (11%), mandibular cuspids or molars (3%) and the maxillary cuspids and molars (1%).\(^3\) Natal or neonatal cuspids are extremely rare.\(^7\) As has been noted, the natal and neonatal teeth are more frequently seen in the
mandibular incisor regions and are more frequently bilateral. Most commonly, these teeth are precociously erupted from the normal complement of primary teeth (90–99%). Only 1-10% of natal and neonatal teeth are supernumerary.\(^{[8,9]}\)

**Etiology**

The variety of natal and neonatal descriptions suggests the lingering controversy regarding this condition and its etiological aspect. In fine the law in this regard is yet to be resolved.

1. The rate at which baby’s teeth comes through will depend on its “genetic blueprint,” i.e., hereditary transmission of a dominant autosomal gene appears to be an important factor.\(^{[2,10]}\)

2. Endocrine disturbances: It is thought to be because of excessive secretion of pituitary, thyroid, or gonads.

3. Eruption of natal and neonatal teeth could be dependent on osteoblastic activity within the area of the tooth germ.\(^{[1,10]}\)

4. Infection: For example, congenital syphilis appears to have varying effect. In some cases, the teeth has erupted early, while in others the eruption has been retarded.\(^{[10]}\)

5. Nutritional deficiency, for example, hypovitaminosis (which in turn is caused by poor maternal health, endocrine disturbances, febrile episodes, pyelitis during pregnancy, and congenital syphilis).\(^{[1,10]}\)

6. Febrile status: Fever and exanthemata during pregnancy tend to accelerate eruption as they do in various other processes.

7. Superficial position of the tooth germ.

8. Environmental factors: Polychlorinated biphenyls (PCBs) and dibenofuran\(^{[11]}\) seem to increase the incidence of natal teeth. These children usually show other associated symptoms such as dystrophic finger nails, hyperpigmentation, etc.

9. The most acceptable theory is based upon the result of a superficial localization of the dental follicles, probably related to the hereditary factor.\(^{[5,8,12]}\)

Natal teeth and neonatal teeth are frequently found associated with developmental abnormalities and recognized syndromes. These syndromes include Ellis–van Creveld, pachyonychia congenita, Hallemann–Streiff, Rubinstein–Taybi, steatoctystoma multiplex, Pierre–Robin, cyclopia, Pallister–Hall, short rib-polydactyly type II, Wiedeman–Rautenstrauch, cleft lip and palate, Pfeiffer, ectodermal dysplasia, craniofacial dysostosis, multiple steacytoma, Sotos, adenogenital, epidermolysis bullosa simplex including van der Woude and Walker–Warburg syndromes.\(^{[1,13]}\)

**Complications**

1. Potential risk of the infant inhaling the tooth into his/her airway and lungs if the tooth becomes dislodged during nursing, due to its great mobility.

2. Ulceration to ventral surface of the tongue: Coldrallin first described this condition in 1857. Riga and Fede histologically described the lesion, which was then started to be called Riga–Fede disease.\(^{[2,14]}\)

3. Difficulty in feeding or refusal to feed due to pain.

4. Ulceration to the nipple of the mother and interference with breast feeding.

**Clinical aspects**

Clinically, the natal teeth are small, or of normal size, conical, or of normal shape. They may reveal an immature appearance with enamel hypoplasia and small root formation. Natal teeth may exhibit a brown-yellowish/whitish opaque color. They are attached to a pad of soft tissue above the alveolar ridge, occasionally covered by mucosa, and as a result, have an exaggerated mobility, with the reason of being swallowed or aspirated, in most of the cases.\(^{[5,15]}\) Bigeard et al. revealed that the dimensions of the crown of these teeth are smaller than those for the primary teeth under normal conditions.

**Histological features**

In this study, ground section of natal and neonatal teeth demonstrated varying thickness of enamel and almost straight dentino-enamel junction. Dentin demonstrated irregular branching of dentinal tubules and Tomes granular layer [Figures 5–7].

The first report on microscopic observation of natal and neonatal teeth was given by Howkins in 1932. Histological investigations of natal teeth have been well detailed by Boyd and Miles.\(^{[16]}\) The histological aspect shows a thin enamel layer, with varying degrees of mineralization and/or hypoplasticity to total absence of enamel in some regions. Friend et al. demonstrated that the alteration in amelogenesis was detected due to premature exposure of the tooth to oral cavity, which resulted in metaplastic alteration of the epithelium of the normally columnar enamel to a stratified squamous configuration.\(^{[4]}\) Atubular osteodentin, such as that observed in the occusal central fossa, is equivalent to the irregular tertiary dentin deposited in response to untoward stimuli such as caries or attrition.\(^{[14]}\) This suggests that odontoblasts in the central fossa were exposed to the oral environment before developing a covering enamel and normal tubular dentin and responded by depositing the atubular substance. The dentin may show alterations with atypical deposition of dentinal tubules, chiefly in the cervical third, and occasionally of osteodentin, which is attributed to stimulation by movement of the teeth. It has been further postulated that the mobility may cause degeneration of Hertwig’s sheath, thus preventing root development and stabilization.\(^{[12,15,17]}\)

The usually increased mobility causes histological changes in the cervical dentin and cementum.\(^{[6]}\) The pulp cavity and radicular canals are wider, although the pulp shows normal development.\(^{[11]}\) Weil’s zone and cell-rich zone are missing.\(^{[16]}\) Absence of root formation, lack of cementum formation, lack of pulp chamber, and an irregular dentin formation are also observed. In the polarized light and micro-radiographic studies, these teeth showed enamel hypoplasia and dentinal disturbances including the formation of osteodentin and
irregular dentin in the cervical portions and interglobular dentin in the coronal region. Natal teeth with no enamel formation are extremely rare; there has been only one case reported, in which cartilage-like teeth erupted prematurely at birth.

**Diagnosis**

The diagnosis of the teeth is done based on a complete history, physical examination of the infant, and by the clinical and radiographic findings to rule out being the part of normal dentition or supernumerary, so that indiscriminate extractions would be performed. A proper examination can reveal the relationship between a natal/neonatal tooth and adjacent structures, nearby teeth, and presence or absence of a tooth germ in the primary dentition would determine whether or not later belongs to normal dentition. Investigators have observed that most of these teeth are primary teeth of normal dentition and not supernumerary teeth. According to the citations, diagnosis is important to plan treatment, keeping in view the maintenance of the normal dental occlusion.

**Treatment and management**

In confronting a typical variation in the newborn’s oral cavity, the pediatric dentist must decide between “early treatment” and the other extreme “should never be treated.” If the erupted natal and neonatal tooth is diagnosed as a tooth of normal dentition, the maintenance of these teeth in the mouth is the first treatment option, unless this would cause injury to the baby or mother. Spouge and Feasby have pointed out that prematurely erupted teeth are often well formed and normal in all respects except that they may be somewhat mobile.

Grinding or smoothening the incisal edges of the teeth was advocated by Allwright and Martins et al. in 1998. To prevent the injury to the maternal breast, feeding splint was the option reported by Bjuggren (1973). Goho (1996) reported his treatment of natal teeth by covering the incisal margin with composite resin. Tomizawa et al. (1989) reported two cases of treatment of Riga–Fede disease by covering the incisal margin with photopolymerizable resin, which aided rapid healing of the ulcers. This petty tooth can sometimes become pretty serious. Removal of natal teeth is indicated when they are poorly developed, interfere with feeding, highly mobile, and associated with soft tissue growth.

Kates et al. suggested extraction as a treatment as they thought despite initial space loss, the space was regained and crowding of permanent mandibular incisors was not apparent. If extraction is carried out, it is necessary to ensure that the underlying dental papilla and Hertwig’s epithelial root sheath are removed by gentle curettage as root development can continue if these structures are left in situ. The prophylactic administration of vitamin K (0.5–1.0 mg i.m.) is advocated because of the risk of hemorrhage as the commensal flora of the intestine might not have been established until the child is 10 days old and since vitamin K is essential for the production of prothrombin in the liver.

**Conclusion**

Pediatric dentists should make every effort to educate the parents and the medical community on the preferred treatment for the natal teeth. If the extraction of the natal tooth is indicated, then it should be performed by the pediatric dentist.
to avoid unnecessary trauma to the area. Periodic follow-up by a pediatric dentist to ensure preventive oral health is very essential. Hence, to avoid any complication, early diagnosis and adequate treatment should be a prime concern in the management of natal teeth.

References

1. Alvarez MP, Crespi PV, Shanske AL. Natal molars in Pfeiffer syndrome type 3: A case report. J Clin Pediatr Dent 1993;18:21-4.
2. Anegundi RT, Sudha P, Kaveri H, Sadanand K. Natal and neonatal teeth: A report of four cases. J Indian Soc Pedo Prev Dent 2002;20:86-92.
3. Massler M, Savara BS. Natal and neonatal teeth: A review of 24 Cases reported in the literature. J Pediatr 1960;36:349-59.
4. Singh S, Subbba Reddy VV, Dhananjaya G, Patuk R. Reactive fibrous hyperplasia associated with a natal tooth: A case report. J Indian Soc Pedo Prev Dent 2004;22:183-6.
5. Bodenhoff J. Natal and Neonatal teeth. J Odontal Tidskr 1959;67:646-85.
6. Bodenhoff J, Gortin RJ. Natal and neonatal teeth: Folklore and fact. Pediat 1963;32:1087-93.
7. Goncalves FA, Birmani EG, Sugayai NN, Melo AM. Natal teeth. Review of literature and report of an unusual case. Braz Dent J 1998;9:53-6.
8. Available from: http://www.newdao.com/natal-teeth-baby-born. Html. (last updated on 2007 Nov. 9)
9. El Khatib K, Abouchadi A, Nassih M, Rziz A, Jidal B, Danino A, et al. Natal teeth: Study of five cases. Rev Stomatol Chir Maxillofac 2005;106:325-7.
10. McDonkd RD, Abouchadi A, Nassih M, Rziz A, Jidal B, Danino A, et al. Natal teeth: Study of five cases. Rev Stomatol Chir Maxillofac 2005;106:325-7.
11. Alaluusua S, Kiviranta H, Leppanieni A, Holtti P, Lukinmaa PL, Lope L, et al. Natal and neonatal teeth in relation to environmental toxicants. Pediat Res 2002;52:652-6.
12. Portela MB, Damasceno L, Primo LG. Unusual case of multiple natal teeth. J Clin Pediatr Dent 2004;29:37-9.
13. Darwisha S, Sastry RH, Ruprecht A. Natal teeth, bifid tongue and deaf mutism. J Oral Med 1987;42:49-53.
14. Sigal MJ, Mock D, Weinberg S. Bilateral mandibular hamartomas and familial natal teeth. Oral Surg Oral Med Oral Pathol 1988;65:731-5.
15. Delbem AC, Fraraco Junior IM, Percinot C, Delbem AC. Natal teeth: Case report. J Clin Pediatr Dent 1996;20:325-7.
16. Anderson RA. Natal and neonatal teeth: Histologic investigation of two black females. ASDC J Dent Child 1982;49:300-3.
17. Masatomi Y, Abe K, Ooshima T. Unusual multiple natal teeth: Case report. Pediatr Dent 1991;13:170-2.
18. Uzamis M, Olmez S, Ozturk H, Celik H. Clinical and ultrastructural study of natal and neonatal teeth. J Clin Pediatr Dent 1999;23:173-7.
19. Robson C, Farli A, Parecida CB, Dione DT, Wanda TG. Natal and Neonatal teeth: Review of the literature. J Pedo Dent 2001;23:158-62.
20. Chow MH. Natal and Neonatal teeth. JADA 1980;100:215-6.
21. Spouge JD, Feasby WH. Erupted teeth in the newborn. Oral Surg Oral Med Oral Path 966;22:198-208.
22. Allwright WC. Natal and neonatal teeth. A review of 50 cases. J India Soc Pedo Prev Dent 1996;21-3.
23. Kates GA, Needleman HL, Holmes LB. Natal and Neo natal teeth- a clinical study. JADA 1984;109:441-3.
24. Bodenhoff J. Natal and neonatal teeth. Dental Abstr 1960;5:485-8.
25. Goho C. Neonatal sublingual traumatic ulceration (Rega –Fede disease) : Reports of cases. J Dent child 1996:63:362-364
