Oral and dental diseases: Causes, prevention and treatment strategies

NASEEM SHAH

DENTAL CARIES

Dental caries is an infectious microbiological disease of the teeth that results in localized dissolution and destruction of the calcified tissues. It is the second most common cause of tooth loss and is found universally, irrespective of age, sex, caste, creed or geographic location. It is considered to be a disease of civilized society, related to lifestyle factors, but heredity also plays a role. In the late stages, it causes severe pain, is expensive to treat and leads to loss of precious man-hours. However, it is preventable to a certain extent. The prevalence of dental caries in India is 50%–60%.

Aetiology

An interplay of three principal factors is responsible for this multifactorial disease.

- Host (teeth and saliva)
- Microorganisms in the form of dental plaque
- Substrate (diet)

Thus, caries requires a susceptible host, cariogenic oral flora and a suitable substrate, which must be present for a sufficient length of time.

Host factors

Teeth[1–4]

- *Composition:* Deficiency in fluorine, zinc, lead and iron content of the enamel is associated with increased caries.
- *Morphological characteristics:* Deep, narrow occlusal fissures, and lingual and buccal pits tend to trap food debris and bacteria, which can cause caries. As teeth get worn (attrition), caries declines.
- *Position:* The interdental areas are more susceptible to dental caries. Malalignment of the teeth such as crowding, abnormal spacing, etc. can increase the susceptibility to caries.

Division of Conservative Dentistry and Endodontics
Centre for Dental Education and Research
All India Institute of Medical Sciences, New Delhi 110029
e-mail: nshah@aiims.ac.in

Saliva[5–8]

Saliva has a cleansing effect on the teeth. Normally, 700–800 ml of saliva is secreted per day. Caries activity increases as the viscosity of the saliva increases. Eating fibrous food and chewing vigorously increases salivation, which helps in digestion as well as improves cleansing of the teeth. The quantity as well as composition, pH, viscosity and buffering capacity of the saliva plays a role in dental caries.

- *Quantity:* Reduced salivary secretion as found in xerostomia and salivary gland aplasia gives rise to increased caries activity.
- *Composition:* Inorganic—fluoride, chloride, sodium, magnesium, potassium, iron, calcium and phosphorus are inversely related to caries.
  Organic—ammonia retards plaque formation and neutralizes the acid.
- *pH:* A neutral or alkaline pH can neutralize acids formed by the action of microorganisms on carbohydrate food substances.
- *Antibacterial factors:* Saliva contains enzymes such as lactoperoxidase, lypozyme, lactoferrin and immunoglobulin (Ig)A, which can inhibit plaque bacteria.

Dental plaque[9–12]

Dental plaque is a thin, tenacious microbial film that forms on the tooth surfaces. Microorganisms in the dental plaque ferment carbohydrate foodstuffs, especially the disaccharide sucrose, to produce acids that cause demineralization of inorganic substances and furnish various proteolytic enzymes to cause disintegration of the organic substances of the teeth, the processes involved in the initiation and progression of dental caries. The dental plaque holds the acids produced in close contact with the tooth surfaces and prevents them from contact with the cleansing action of saliva.

Table 1. Causes of dental caries

Direct	Indirect	Distant
1. Tooth • Structure—fluoride content and other trace elements such as zinc, lead, iron • Morphology—deep pits and fissures • Alignment—crowding 2. Microorganisms—dental plaque accumulation due to poor oral hygiene 3. Diet • Intake of refined carbohydrates such as sucrose, maltose, lactose, glucose, fructose, cooked sticky starch, etc. —quantity; frequency, physical form; oral clearance rate • Saliva (quantity and quality) —reduced secretion (xerostomia) increases caries —Viscosity: more viscous, more caries —pH: alkaline pH neutralizes acid, less caries —enzymes: lactoperoxidase, lysozyme lactoferrins —immunoglobulins IgA	• Poor contact between the teeth resulting in food impaction and caries due to the following causes —malalignment of the teeth (crowding) —loss of some teeth and failure to replace them • Gingival recession leading to root caries	• Socioeconomic status • Literacy level • Location—urban, rural • Age • Sex • Dietary habits • Climatic conditions and soil type • Social and cultural practices • Availability/access to health care facility • Health insurance

Substrate[13–16]

The role of refined carbohydrates, especially the disaccharide sucrose, in the aetiology of dental caries is well established. The total amount consumed as well as the physical form, its oral clearance rate and frequency of consumption are important factors in the aetiology. Vitamins A, D, K, B complex (B6), calcium, phosphorus, fluorine, amino acids such as lysine and fats have an inhibitory effect on dental caries.

Indirect causes[17,18]

- Loss of some natural teeth and failure to replace them results in drifting of the teeth in the edentulous space. This leads to increased food impaction between the teeth and formation of new carious lesions.
- Malalignment of the teeth, especially crowding, does not allow proper cleaning between the teeth and leads to an increased incidence of caries.
- Gingival recession, abrasion and abfraction defects at the neck of the tooth increase root caries.
- Selenium in the soil increases the formation of caries while molybdenum and vanadium decrease it.
- A high temperature is associated with a lower prevalence of caries. Water has a cleansing effect on the teeth. If the fluoride content of the water is at an optimum concentration, it will also exert an anticaries effect.

Distant causes[19,20]

- A low socioeconomic and literacy status is associated with caries.
- Urbanization is linked to an increased incidence of caries.
- Caries is more common in childhood and adolescence, and after 60 years of age, when the incidence of root caries is higher.
- Females develop caries more often than males.
- Non-vegetarians develop caries more often than vegetarians.
- Availability/access to a health care facility can affect utilization of health care services.
- Lack of oral health insurance promotes oral neglect and increases disease levels.

Table 1 summarizes the causes of dental caries.

Prevention and control of dental caries

1. Increase the resistance of the teeth.[21–25]

Systemic use of fluoride: (i) Fluoridation of water, milk and salt; (ii) fluoride supplementation in the form of tablets and lozenges; and (iii) consuming a fluoride-rich diet such as tea, fish, etc.

Topical: (i) Use of fluoridated toothpaste and mouth wash; (ii) use of fluoride varnishes (in-office application, longer duration of action, high fluoride content); (iii) use of casein phosphopeptide–amorphous calcium phosphate (CPP–ACP), which is available as tooth mousse, helps to remineralize the soft initial carious, demineralized areas of the teeth.

2. Combat the microbial plaque by physical and chemical methods.

(i) *Physical methods*[26–30]

The correct method and frequency of brushing should be followed—in the morning and before going to bed and preferably after every major meal.

Tongue cleaning and the use of indigenous agents such as the bark of neem or mango (where toothbrush and paste are unaffordable) should be encouraged. The use of coarse

toothpowder and tobacco-containing dentifrices should be avoided.

The use of various interdental cleaning aids such as dental floss, interdental brush, water pik, etc. supplements the cleansing effect of a toothbrush. Use of an electronic toothbrush in children and persons with decreased manual dexterity is recommended.

(ii) *Chemical methods*

These include the use of a fluoride-containing toothpaste, mouth rinses and 0.2% chlorhexidine and povidine–iodine mouthwash. These should be used on prescription of a dental surgeon.

3. Modify the diet.[31–34]

Reduce the intake and frequency of refined carbohydrates. Avoid sticky foods and replace refined with unrefined natural food. Increase the intake of fibrous food to stimulate salivary flow, which is protective against caries. Consume caries-protective foods such as cheese, nuts, raw vegetables, fruits, etc. Stimulate salivary flow with sugar-free chewing gum. Xylitol (a sugar substitute)-containing chewing gum, if chewed between meals, produces an anti-caries effect by stimulating salivary flow.

Preventive interventions[35–43]

The use of pit and fissure sealants[35,36] and application of fluoride varnish[37,38] help in slowing down the development of caries.

Preventive restorations should be carried out[39,40] and atraumatic restorative treatment (ART) should be used as a community-based approach for the treatment and prevention of dental caries.[41–43]

Treatment of dental caries

Treatment comprises removal of decay by operative procedures and restoration with appropriate materials such as silver fillings, gold inlays, composite resin, glass ionomer cement, full metal or porcelain crowns, etc. In advanced cases, where the pulp of the tooth is involved, endodontic treatment may be required. Where there is extensive destruction of the tooth structure or when endodontic treatment is not feasible, extraction of the tooth and replacement by an artificial prosthesis may be required.

Miscellaneous measures

These include the following:

- Prevention of malocclusion (especially crowding of the teeth)
- Prevention of premature loss of deciduous teeth
- Restoration of missing permanent teeth by prostheses (dentures)
- Making sugar-free chewing gum freely available and affordable in the country

Table 2. Prevention and treatment of dental caries

Medical interventions	Non-medical interventions	Other interventions
• Use of systemic and topical fluorides • Use of pit and fissure sealants • Preventive restorations • Different types of restorations and endodontic treatment • Regular dental check-up	• Oral health education • Nutrition and diet • Proper methods of maintaining oral hygiene —use of fluoride tooth-paste and brush —use of dental floss and interdental brushes, etc. —antiseptic mouth washes (under prescription)	• Make oral health care more accessible and affordable • Improve the socioeconomic and literacy level of the population • Include oral health care in general health insurance

- Using sugar substitutes such as saccharine, xylitol, mannitol, aspartame, etc. in paediatric medicinal syrups, bakery products, jams, marmalade, etc.
- Making toothbrushes and fluoridated toothpaste available to the masses at low cost. Regular use of fluoridated toothpaste is proven to reduce the incidence of dental caries by 30%.

Table 2 summarizes the prevention and treatment strategies for dental caries.

References

1. Babaahmady KG, Marsh PD, Challacombe SJ, Newman HN. Variations in the predominant cultivable microflora of dental plaque at defined subsites on approximal tooth surfaces in children. *Arch Oral Biol* 1997;**42:**101–11.
2. Liu F. [The relation between the resistance distribution on crown surface and caries.] *Zhonghua Kou Qiang Yi Xue Za Zhi* 1993;**28:**47–9.
3. Marcucci M, Bandettini MV. Dental caries in the rat in relation to the chemical composition of the teeth and diet. Variations in the diet of the Ca/P ratio obtained by changes in the phosphorus content. *Minerva Stomatol* 1981;**30:**17–20.
4. Haldi J, Wynn W, Bentley KD, Law ML. Dental caries in the albino rat in relation to the chemical composition of the teeth and of the diet. IV. Variations in the Ca/P ratio of the diet induced by changing the calcium content. *J Nutr* 1959;**67:**645–53.
5. Daniels TS, Silverman S, Michalski JP, Greenspan JS, Sylvester RA, Talal N. The oral component of Sjogren's syndrome. *Oral Surg* 1975;**39:**875–85.
6. Finn SB, Klapper CE, Voker JF. Intra-oral effects upon experimental hamster caries. In: RF Sognnaes (ed). *Advances in experimental caries research*. Washington, DC: American Association for the Advancement of Sciences; 1955:155–68.
7. Frank RM, Herdly J, Phillippe E. Acquired dental defects and salivary gland lesions after irradiation for carcinoma. *J Am Dent Assoc* 1965;**70:**868–83.
8. Kermiol M, Walsh RF. Dental caries after radiotherapy of the oral regions. *J Am Dent Assoc* 1975;**91:**838–45.
9. Fitzgerald RJ, Keyes PH. Demonstration of the etiologic role of streptococci in experimental caries in the hamster. *J Am Dent Assoc* 1960;**61:**9–19.
10. Keyes PH. The infection and transmissible nature of experimental dental caries. *Arch Oral Biol* 1960;**1:**304–20.

11. Orland FJ, Blayney JR, Harrison RW, Reyniers JA, Trexler PD, Ervin RF, *et al.* Experimental caries in germ-free rats inoculated with enterococci. *J Am Dent Assoc* 1955;**50:**259–72.
12. Rosen S, Kolstad RA. Dental caries in gnotobiotic rats inoculated with a strain of *Peptostreptococcus intermedius*. *J Dent Res* 1977;**56:**187.
13. Burt BA, Eklund Sa, Morgan KJ, Larkin FE, Guire KE, Brown LO, *et al.* The effects of sugar intake and frequency of ingestion on dental caries increment in a three-year longitudinal study. *J Dent Res* 1988;**67:**1422–9.
14. Caldwell RC. Physical properties of foods and their caries-producing potential. *J Dent Res* 1970;**49:**1293–8.
15. Harris RS. Minerals: Calcium and phosphates. In: RF Gould (ed). *Dietary chemicals vs. dental caries. Advances in chemistry services* 94. Washington, DC: American Chemical Society; 1970:116–22.
16. Nizel AE. *Nutrition in preventive dentistry*: *Sciences and practice*. 2nd ed. Philadelphia: WB Saunders; 1981:417–52.
17. Helm S, Petersen PE. Causal relation between malocclusion and caries. *Acta Odontol Scand* 1989;**47:**217–21.
18. Warren JJ, Slayton RL, Yonezu T, Kanellis MJ, Levy SM. Interdental spacing and caries in primary dentition. *Pediatr Dent* 2003;**25:**109–13.
19. Ellwood RP, Davies GM, Worthington HV, Blinkhorn AS, Taylor GO, Davies RM. Relationship between area deprivation and the anticaries benefit of an oral health programme providing free fluoride toothpaste to young children. *Commun Dent Oral Epidemiol* 2004;**32:**159–65.
20. Shah N, Sundaram KR. Impact of socio-demographic variables, oral hygiene practices, oral habits and diet on dental caries experience of Indian elderly: A community-based study. *Gerodontology* 2004;**21:**43–50.
21. Hicks J, Garcia-Godoy F, Flaitz C. Biological factors in dental caries: Role of remineralization and fluoride in the dynamic process of demineralization and remineralization (Part 3). *J Clin Pediatr Dent* 2004;**28:**203–14.
22. Kargul B, Caglar E, Tanboga I. History of water fluoridation. *J Clin Pediatr Dent* 2003;**27:**213–17.
23. Featherstone JD. Prevention and reversal of dental caries: Role of low level fluoride. *Commun Dent Oral Epidemiol* 1999;**27:**31–40.
24. Stephen KW. Systemic fluorides: Drops and tablets. *Caries Res* 1993;**27**(Suppl. 1):9–15.
25. Cai F, Shen P, Morgan MV, Reynolds EC. Remineralization of enamel subsurface lesions in situ by sugar-free lozenges containing casein phosphopeptide–amorphous calcium phosphate. *Aust Dent J* 2003;**48:**240–3.
26. Klock B. Krasse B. Effect of caries preventive measures in children with high numbers of *S. mutans* and lactobacilli. *Scand J Dent Res* 1978;**86:**221.
27. Krasse B. *Caries risk*: *A practical guide for assessment and control*. Chicago: Quintessence Publishing Co. Inc; 1985.
28. Loe H. Human research model for the production and prevention of gingivitis. *J Dent Res* 1971;**50:**256.
29. Emilson CG. Potential efficacy of chlorhexidine against mutant streptococci and human dental caries. *J Dent Res* 1994;**73:** 682–91.
30. Twetman S. Antimicrobials in future caries control? A review with special reference to chlorhexidine treatment. *Caries Res* 2004;**38:**223–9.
31. Marshall TA. Carries prevention in pediatrics: Dietary guidelines. *Quintessence Int* 2004;**35:**332–5.
32. van Loveren C, Duggal MS. Experts' opinions on the role of diet in caries prevention. *Caries Res* 2004;**38** (Suppl. 1):16–23.
33. Vanobbergen J, Declerck D, Mwalili S, Martens L. The effectiveness of a 6-year oral health education programme for primary schoolchildren. *Commun Dent Oral Epidemiol* 2004;**32:**173–82.
34. TanzerJM. Xylitol chewing gum and dental caries. *Int Dent J* 1995;**45**(Suppl. 1): 65–76.
35. Kumar J, Siegal MD. Workshop on guidelines for sealant use: Recommendations. *J Pub Health Dent* 1955;**5**(Special issue): 263–73.
36. Swift EJ Jr. The effect of sealants on dental caries: A review. *J Am Dent Assoc* 1988;**116:**700–4.
37. Beltran-Aguilar ED, Goldstein JW, Lockwood SA. Fluoride varnishes—a review of their clinical use, cariostatic mechanism, efficacy and safety. *J Am Dent Assoc* 2000;**131:**589–96.
38. Savanberg M, Westergren G. Effect of SnF2, administered as mouth rinses or topically applied, on *Streptococcus mutans*, *Streptococcus sanguis* and lactobacilli in dental plaque and saliva. *Scand J Dent Res* 1983;**91:**123.
39. Simonsen RJ. Preventive resin restoration. *Quintessence Int* 1978;**9:**69–76.
40. Simonsen RJ. Preventive resin restorations: Three year results. *J Am Dent Assoc* 1980;**100:**535–9.
41. Frencken JE. [Atraumatic restorative treatment (ART). A special tissue preservative and patient-friendly approach.] *Ned Tijdschr Tandheelkd* 2003;**110:**218–22.
42. Carvalho CK, Bezerra AC. Microbiological assessment of saliva from children subsequent to atraumatic restorative treatment (ART). *Int J Paediatr Dent* 2003;**13:**186–92.
43. Smales RJ, Gao W. *In vitro* caries inhibition at the enamel margins of glass ionomer restorations developed for the ART technique. *J Dent* 2000;**28:**249–56.

DENTOFACIAL ANOMALIES OR MALOCCLUSION

Dentofacial anomalies include hereditary, developmental and acquired malocclusion or malalignment of the teeth. Worldwide, the average prevalence of malocclusion in the 10–12 years' age group is reported to be 30%–35%.

Aetiology

Direct causes[1–17]

- *Heredity:* Hereditary factors play an important role in conditions such as cleft lip and palate, facial asymmetries, variations in tooth shape and size, deep bites, discrepancies in jaw size.[1–4]
- *Congenital:* These include cleft lip and palate, and syndromes associated with anomalies of craniofacial structures, cerebral palsy, torticollis, cleidocranial dysostosis, congenital syphilis, etc.[5,6]
- *Abnormal pressure habits and functional aberrations:* These include abnormal suckling, thumb and finger sucking, tongue thrusting and sucking, lip and nail biting, mouth breathing, enlarged tonsils and adenoids, trauma and accidents.[7–13]

Table 3. Causes of dentofacial anomalies and malocclusion

Direct	Indirect	Distant
• Hereditary/congenital • Abnormal pressure habits and functional aberrations —abnormal suckling —mouth breathing —thumb and finger sucking —tongue thrusting and sucking —abnormal swallowing • Trauma and accidents • Local factors —abnormalities of number (supernumerary teeth, missing teeth) —abnormalities of tooth size and shape —abnormal labial frenum and mucosal barriers —premature tooth loss —prolonged retention of deciduous teeth —delayed eruption of permanent teeth —abnormal eruptive path —untreated dental caries and improper dental restorations, especially on the proximal surfaces	• Environmental factors —prenatal causes such as trauma, maternal diet and metabolism, German measles, certain drugs, and position *in utero* —postnatal causes such as birth injury, cerebral palsy, temporomandibular joint injury	• Poor nutritional status—deficiency of vitamin D, calcium and phosphates • Endocrine imbalance such as hypothyroidism • Metabolic disturbances and muscular dystrophies • Infectious diseases such as poliomyelitis • Functional aberrations —psychogenic tics and bruxism —posture

- *Local factors:* These include abnormalities of number such as supernumerary and missing teeth, abnormalities of tooth size and shape, abnormal labial frenum causing spacing between the upper anterior teeth, premature tooth loss with drifting of the adjoining and opposite teeth, prolonged retention of the milk teeth, delayed eruption of the permanent teeth, abnormal eruptive path, dental caries, and improper dental restorations.[14–17]

Indirect causes[18–25]

Environmental

—Prenatal: trauma, maternal diet and metabolism, German measles, certain drugs and position *in utero*

—Postnatal: birth injury, cerebral palsy, temporomandibular joint injury

Distant causes [26,27]

- *Endocrine imbalance:* Hypothroidism is related to an abnormal resorption pattern, delayed eruption and gingival disturbances. Retained deciduous teeth may be due to hypothroidism.
- *Metabolic disturbance and infectious diseases:* Acute febrile conditions delay growth and development. Diseases such as poliomyelitis, muscular dystrophy and cerebral palsy have a characteristic deforming effect on the dental arch.
- *Nutritional:* Vitamin D, calcium and phosphorus are associated with bone metabolism and their deficiency could lead to growth disturbances.
- *Abnormal muscle function and posture:* Psychogenic tics and abnormal head posture can contribute towards malrelation of the jaws.

Factors responsible for causing dentofacial anomalies and malocclusion are summarized in Table 3.

Prevention and treatment[28–33]

The prevention and treatment of dentofacial anomalies can be undertaken at three levels (Table 4).

- Primary prevention—Preventive orthodontics
- Secondary prevention—Interceptive orthodontics
- Tertiary prevention—Corrective orthodontic treatment by removable and fixed appliances, and surgical orthodontics

Table 4. Strategies for the prevention and treatment of dentofacial anomalies and malocclusion

Medical interventions	Non-medical interventions
• Habit-breaking appliances • Serial extractions • Space-maintainers and -regainers • Functional appliances in developing malocclusion to correct jaw relations • Frenectomies and simple appliances to correct anterior cross-bites • Removable and fixed appliances • Orthognathic and plastic surgery • Speech therapy • Regular dental check-up for early intervention • Counselling • Preservation and restoration of primary and permanent teeth	• Control harmful oral habits • Prenatal and perinatal care • Genetic counselling

Primary prevention

This includes control of harmful oral habits, and preservation and restoration of primary and permanent dentition.

Secondary prevention

Habit-breaking appliances should be used. Serial extractions, space maintainers/regainers, and functional appliances to correct jaw relations are other modalities. Frenectomies and simple appliances can be used to correct anterior crossbites.

Tertiary prevention

Corrective orthodontic treatment includes the use of fixed and removal appliances and surgical orthodontics in cases of severe malocclsion.

References

1. Mossey PA. The heritability of malocclusion: Part 1. Genetics, principles and terminology. *Br J Orthod* 1999;**26:**103–13.
2. Mossey PA. The heritability of malocclusion: Part 2. The influence of genetics in malocclusion. *Br J Orthod* 1999;**26:**195–203.
3. Varrela J. Genetic and epigenetic regulation of craniofacial development. *Proc Finn Dent Soc* 1991;**87:**239–44.
4. Moss ML. Genetics, epigenetics, and causation. *Am J Orthod* 1981;**80:**366–75.
5. Golan I, Baumert U, Hrala BP, Mussig D. Early craniofacial signs of cleidocranial dysplasia. *Int J Paediatr Dent* 2004;**14:**49–53.
6. Ortiz-Posadas MR, Vega-Alvarado L, Toni B. A similarity function to evaluate the orthodontic condition in patients with cleft lip and palate. *Med Hypotheses* 2004;**63:**35–41.
7. Chen QR, Zhong HL. [Lower lip biting habits and malocclusions.] *Shanghai Kou Qiang Yi Xue* 1994;**3:**3–6.
8. Yamaguchi H, Sueishi K. Malocclusion associated with abnormal posture. *Bull Tokyo Dent Coll* 2003;**44:**43–54.
9. daCosta OO, Orenuga OO. Dentofacial anomalies related to the digit sucking habit. *Afr J Med Med Sci* 2002;**31:**239–42.
10. Massler M. Oral habits: Development and management. *J Pedod* 1983;**7:**109–19.
11. Popovich F. The prevalence of sucking habit and its relationship to oral malformations. *Appl Ther* 1966;**8:**689–91.
12. Hatzakis S, Toutountzakis N. Speech defects and malocclusion. *Hell Stomatol Chron* 1984;**28:**97–106.
13. Hawkins AC. Mouth breathing and its relationship to malocclusion and facial abnormalities. *N M Dent J* 1969;**20:**18–21.
14. Nik-Hussein NN. Supernumerary teeth in the premaxillary region: Its effects on the eruption and occlusion of the permanent incisors. *Aust Orthod J* 1990;**11:**247–50.
15. Northway WM, Wainright RL, Demirjian A. Effects of premature loss of deciduous molars. *Angle Orthod* 1984;**54:**295–329.
16. Basdra EK, Kiokpasoglou MN, Komposch G. Congenital tooth anomalies and malocclusions: A genetic link? *Eur J Orthod* 2001;**23:**145–51.
17. Forsberg CM, Tedestam G. Etiological and predisposing factors related to traumatic injuries to permanent teeth. *Swed Dent J* 1993;**17:**183–90.
18. Proffit WR. On the aetiology of malocclusion. The Northcroft lecture, 1985 presented to the British Society for the Study of Orthodontics, Oxford, 18 April, 1985. *Br J Orthod* 1986; **13:**1–11.
19. Defabianis P. Post-traumatic TMJ internal derangement: Impact on facial growth (findings in a pediatric age group). *J Clin Pediatr Dent* 2003;**27:**297–303.
20. Schoenwetter RF. A possible relationship between certain malocclusions and difficult or instrumental deliveries. *Angle Orthod* 1974;**44:**336–40.
21. Vittek J, Winik S, Winik A, Sioris C, Tarangelo AM, Chou M. Analysis of orthodontic anomalies in mentally retarded developmentally disabled (MRDD) persons. *Spec Care Dentist* 1994;**14:**198–202.
22. Strodel BJ. The effects of spastic cerebral palsy on occlusion. *ASDC J Dent Child* 1987;**54:**255–60.
23. Matsumoto S, Morinushi T, Ogura T. Time dependent changes of variables associated with malocclusion in patients with Duchenne muscular dystrophy. *J Clin Pediatr Dent* 2002;**27:**53–61.
24. Singh GD, Rivera-Robles J, de Jesus-Vinas J. Longitudinal craniofacial growth patterns in patients with orofacial clefts: Geometric morphometrics. *Cleft Palate Craniofac J* 2004;**41:** 136–43.
25. Mg'ang'a PM, Chindia ML. Dental and skeletal changes in juvenile hypothyroidism following treatment: Case report. *Odontostomatol Trop* 1990;**13:**25–7.
26. Gola G. [Dietetic factors in the development of the facial bones and in the etiology of malocclusion.] *Riv Odontostomatol Implantoprotesi* 1983;**3:**25–9, 31.
27. Iwamoto J, Takeda T, Ichimura S, Sato Y, Yeh JK. [Differential effect of vitamin K and vitamin D supplementation on bone mass in young rats fed normal or low calcium diet.] *Yonsei Med J* 2004;**45:**314–24.
28. Kerosuo H. The role of prevention and simple interceptive measures in reducing the need for orthodontic treatment. *Med Princ Pract* 2002;**11:**16–21.
29. Varrela J, Alanen P. Prevention and early treatment in orthodontics: A perspective. *J Dent Res* 1995;**74:**1436–8.
30. Sapino S. Space maintenance devices. *Minerva Stomatol* 1989;**38:**981–7.
31. Binder RE. Serial extraction in preventive dentistry. *Clin Prev Dent* 1979;**1:**21–2.
32. Richard JP. Superior labial frenectomies in the child. *Pedod Fr* 1977;**11:**171–6.
33. Taylor PM, Mason RM. An orthodontist's perspective on the use of habit appliances. *Int J Orofacial Myology* 2002;**28:**3–4.

PERIODONTAL DISEASES

Periodontal diseases are one of the major causes of tooth loss in India. These include pathological conditions of the supporting structures of the teeth, i.e. gingiva, alveolar bone, periodontal ligament and cementum. Gingival and periodontal diseases affect 90% of the population. Gingival disease progresses to periodontal disease, if not checked in time.

Aetiology

Direct causes[1–6]

These include poor oral hygiene leading to accumulation of dental plaque and calculus, and traumatic occlusion.

Indirect factors[7–18]

- Malnutrition (deficiency of vitamins A and C, niacin and protein) is associated with a higher prevalence of periodontal diseases.
- Endocrine disturbances including physiological causes such as puberty, pregnancy, menopause, and pathological causes such as hyperthyroidism, hyperparathyroidism and diabetes may aggravate existing periodontal disease.
- Decreased immunity as in persons with HIV and those on immunosuppressive drugs.
- Blood disorders such as acute monocytic leukaemia and pernicious anaemia can lead to periodontal diseases.
- Malalignment of the teeth interferes with proper plaque control.
- Tobacco smoking and chewing reduce tissue resistance and increase the susceptibility to periodontal diseases.
- An improper brushing technique, besides resulting in inadequate plaque removal, can also cause gingival recession.
- Drugs—certain drugs such as phenytoin sodium and nifedipine can cause gingival hyperplasia.

Distant causes[19–25]

These include low socioeconomic and literacy level, difficult access to an oral health care facility, poor oral health awareness, and lack of oral health insurance. Stress is known to predispose to acute necrotizing ulcerative gingivitis.

Table 5. Causes of periodontal diseases

Direct	Indirect	Distant
• Poor oral hygiene resulting in accumulation of dental plaque and calculus • Traumatic occlusion	• Food impaction • Chewing and smoking of tobacco • Malnutrition—deficiency of vitamins A and C • Endocrine disturbances —physiological (puberty, pregnancy and the menopause) —pathological (hyperthyroidism, hyperparathyroidism and diabetes mellitus) • Decreased immunity—HIV infection, persons on immunosuppressive drugs • Blood disorders—anaemia, leukaemia • Idiopathic—gingival fibromatosis • Drug induced—phenytoin sodium, nifedipine, etc.	• Socioeconomic status • Literacy level • Access to oral health care facility • Oral health knowledge and awareness • Health insurance

The various causes of periodontal diseases are summarized in Table 5.

Prevention and treatment

These are the same as for dental caries.[26–38] Oral health education is required for the maintenance of oral hygiene (brushing, flossing, rinsing, etc.). The use of chemical mouthwashes (under prescription) and improved nutrition, as well as removal or treatment of aggravating factors are additional strategies. Interventions for the prevention and treatment of periodontal diseases are given in Table 6.

Table 6. Prevention and treatment of periodontal diseases

Medical interventions	Non-medical interventions	Other interventions
• Scaling and polishing of teeth • Oral and systemic antibiotics • Use of mouth washes • Gingival and periodontal surgery —gingivoplasty, gingivectomy, flap surgery, mucogingival surgeries, guided tissue regeneration, synthetic bone grafts, etc.	• Oral health education • Nutrition and diet • Proper methods of oral hygiene maintenance —use of toothpaste and tooth brush —use of inter-proximal cleaning devices such as interdental brushes, dental floss and water pik, etc. • Regular dental check-up	• Make oral health care more accessible and affordable • Improve the socioeconomic and literacy level of the population • Include oral health care in general health insurance

References

1. Lovegrove JM. Dental plaque revisited: Bacteria associated with periodontal disease. *J N Z Soc Periodontol* 2004;**87:**7–21.
2. Listgarten MA. Pathogenesis of periodontitis. *J Clin Periodontol* 1986;**13:**418–30.
3. Higgins TJ, Hunter N, Knox KW. Current concepts in periodontal diseases. *Med J Aust* 1985;**142:**590–4.
4. Overman PR. Biofilm: A new view of plaque. *J Contemp Dent Pract* 2000;**1:**18–29.
5. Genco RJ. Current view of risk factors for periodontal diseases. *J Periodontol* 1996; **67**(Suppl.):1041–9.
6. Checchi L, D'Achille C, Montella A. Tartar and periodontal disease—a cofactor in etiopathogenesis. *Dent Cadmos* 1991;**59:**80–4, 87–90, 93–5.
7. Verma S, Bhat KM. Diabetes mellitus—a modifier of periodontal disease expression. *J Int Acad Periodontol* 2004;**6:**13–20.
8. Genco RJ, Grossi SG. Is estrogen deficiency a risk factor for periodontal disease? *Compend Contin Educ Dent Suppl* 1998;**22:**S23–S29.
9. Genco R, Offenbacher S, Beck J. Periodontal disease and cardiovascular disease: Epidemiology and possible mechanisms. *J Am Dent Assoc* 2002;**133**(Suppl.):14S–22S.
10. Gera I. [Osteoporosis: A risk factor for periodontal disease (literature review).] *Fogorv Sz* 2002;**95:**49–54.
11. Kinane DF, Marshall GJ. Periodontal manifestations of systemic disease. *Aust Dent J* 2001;**46:**2–12.
12. Johnson GK, Slach NA. Impact of tobacco use on periodontal status. *J Dent Educ* 2001;**65:**313–21.
13. Slots J, Contreras A. Herpesviruses: A unifying causative factor in periodontitis? *Oral Microbiol Immunol* 2000;**15:**277–80.
14. Seymour RA, Ellis JS, Thomason JM. Risk factors for drug-induced gingival overgrowth. *J Clin Periodontol* 2000;**27:**217–23.
15. Hennig BJ, Parkhill JM, Chapple IL, Heasman PA, Taylor JJ. Association of a vitamin D receptor gene polymorphism with localized early-onset periodontal diseases. *J Periodontol* 1999;**70:**1032–8.
16. Enwonwu CO. Interface of malnutrition and periodontal diseases. *Am J Clin Nutr* 1995;**61:**430S–436S.
17. Turnbull B. Smoking and periodontal disease. A review. *J N Z Soc Periodontol* 1995;**79:**10–15.
18. Murray PA. HIV disease as a risk factor for periodontal disease. *Compendium* 1994;**15:**1052, 1054–63; quiz 1064.
19. Schenkein HA, Burmeister JA, Koertge TE, Brooks CN, Best AM, Moore LV, *et al.* The influence of race and gender on periodontal microflora. *J Periodontol* 1993;**64:**292–6.
20. Dougherty MA, Slots J. Periodontal diseases in young individuals. *J Calif Dent Assoc* 1993;**21:**55–69.
21. Genco RJ. Host responses in periodontal diseases: Current concepts. *J Periodontol* 1992;**63** (Suppl.):338–55.
22. Hung HC, Willett W, Ascherio A, Rosner BA, Rimm E, Joshipura KJ. Tooth loss and dietary intake. *J Am Dent Assoc* 2003;**134:** 1185–92.
23. Page RC. Current understanding of the aetiology and progression of periodontal disease. *Int Dent J* 1986;**36:**153–61.
24. Forrest JL, Miller SA. Manual versus powered toothbrushes: A summary of the Cochrane Oral Health Group's Systematic Review. Part II. *J Dent Hyg* 2004;**78:**349–54.
25. Borrell LN, Burt BA, Neighbors HW, Taylor GW. Social factors and periodontitis in an older population. *Am J Public Health* 2004;**94:**748–54.
26. Bsoul SA, Terezhalmy GT. Vitamin C in health and disease. *J Contemp Dent Pract* 2004;**5:**1–13.
27. Nield-Gehrig JS, Daniels AH. Improving awareness and dental care of diabetic patients. *Pract Proceed Aesthet Dent* 2004;**16:**85–7.
28. Deery C, Heanue M, Deacon S, Robinson PG, Walmsley AD, Worthington H, *et al.* The effectiveness of manual versus powered toothbrushes for dental health: A systematic review. *J Dent* 2004;**32:**197–211.
29. Glassman P, Miller CE. Preventing dental disease for people with special needs: The need for practical preventive protocols for use in community settings. *Spec Care Dentist* 2003;**23:**165–7.
30. Glassman P. Practical protocols for the prevention of dental disease in community settings for people with special needs: Preface. *Spec Care Dentist* 2003;**23:**157–9.
31. Kendall KH, Marshall RI. Antibiotics for periodontal therapy—where are we now and where are we going? Prophylaxis and systemic antibiotics. *Ann R Australas Coll Dent Surg* 2002;**16:**93–4.
32. Venezia E, Shapira L. Use of antimicrobial agents during supportive periodontal therapy. *Oral Dis* 2003;**9**(Suppl. 1):63–70.
33. Baehni PC, Takeuchi Y. Anti-plaque agents in the prevention of biofilm-associated oral diseases. *Oral Dis* 2003;**9**(Suppl. 1):23–9.
34. Haffajee AD, Arguello EI, Ximenez-Fyvie LA, Socransky SS. Controlling the plaque biofilm. *Int Dent J* 2003;**53**(Suppl. 3): 191–9.
35. Ower P. The role of self-administered plaque control in the management of periodontal diseases. 2: Motivation, techniques and assessment. *Dent Update* 2003;**30:**110–16.
36. Frentzen M, Ploenes K, Braun A. Clinical and microbiological effects of local chlorhexidine applications. *Int Dent J* 2002;**52:**325–9.
37. Hancock EB, Newell DH. The role of periodontal maintenance in dental practice. *J Indiana Dent Assoc* 2002;**81:**25–30.
38. Cobb CM. Clinical significance of non-surgical periodontal therapy: An evidence-based perspective of scaling and root planing. *J Clin Periodontol* 2002;**29**(Suppl. 2):6–16.

ORAL CANCER

India has the highest prevalence of oral cancer in the world (19/100,000 population). It is the most common cancer in men and the fourth most common cancer in women, and constitutes 13%–16% of all cancers. Of all the oral cancers, 95% are related to the use of tobacco.

Oral cancer has a high morbidity and mortality. The 5-year survival rate is 75% for local lesions but only 17% for those with distant metastasis. Therefore, early diagnosis of oral cancer is important. Since the oral cavity is easily accessible for examination and the cancer is always preceded by some pre-cancerous lesion or condition such as a white or red patch, an ulcer or restricted mouth opening, it is preventable to a great extent. Unfortunately, in India, most cancers are diagnosed at a very late stage, when treatment not only becomes more expensive, but the morbidity and mortality also increase.

Aetiology

Direct causes

- Tobacco—Many forms of tobacco are used in India—smoking (78%); chewing of betel quid, *paan masala*, *gutka*, etc. (19%); inhalation of snuff (2%); and dentifrices (>1%)

Table 7. Causes of oral cancer

Direct	Indirect	Distant
• Tobacco smoking/chewing • *Paan masala/gutka* chewing • Infections—HPV, HSV, AIDS, syphilis, candidiasis • Chronic irritation—faulty prosthesis, sharp teeth • Exposure to radiation	• Industrial pollution—asbestos, lead, leather and textile industries • Compromised immune status • Nutritional deficiencies (vitamins A and B complex, and zinc)	• Low socioeconomic and literacy level • Poor access to oral health care facilities for prevention and early detection • Poor oral health awareness

- Alcohol[6,7]
- Bacterial infections such as syphilis, and fungal (candidiasis) and viral (HPV, HSV, AIDS) infections[8–10]
- Chronic irritation due to sharp teeth and faulty prosthesis[11,12]
- Exposure to radiation[16,17]

Indirect causes

- Industrial pollution due to asbestos, lead[13–15]
- Nutritional deficiencies such as those due to vitamins A, B complex, and iron deficiency[18–19]

Distant causes

- Low socioeconomic and literacy level
- Poor oral health awareness
- Poor access to oral health care facilities for prevention and early detection

Table 7 lists the direct, indirect and distant causes of oral cancer.

Prevention and treatment

Strategies for prevention and treatment of oral cancer are summarized in Table 8.

References

1. Gupta PC. *Gutka*: A major new tobacco hazard in India. *Tob Control* 1999;**8:**134.
2. Dharkar D. Oral cancer in India: Need for fresh approaches. *Cancer Detect Prev* 1988;**11:**267–70.
3. Shiu MN, Chen TH. Impact of betel quid, tobacco and alcohol on three-stage disease natural history of oral leukoplakia and cancer: Implications for prevention of oral cancer. *Eur J Cancer Prev* 2004;**13:**39–45.
4. Gerson SJ. Oral cancer. *Crit Rev Oral Biol Med* 1990;**1:**153–66.
5. Maier H, Weidauer H. Alcohol drinking and tobacco smoking are the chief risk factors for ENT tumors. Increased incidence of mouth cavity, pharyngeal and laryngeal carcinomas. *Fortschr Med* 1995;**113:**157–60.
6. Kabat GC, Wynder EL. Type of alcoholic beverage and oral cancer. *Int J Cancer* 1989;**43:**190–4.
7. Weinstein RL, Francetti L, Maggiore E, Marchesi G. Alcohol and smoking. The risk factors for the oral cavity. *Minerva Stomatol* 1996;**45:**405–13.
8. Dickenson AJ, Currie WJ, Avery BS. Screening for syphilis in patients with carcinoma of the tongue. *Br J Oral Maxillofac Surg* 1995;**33:**319–20.
9. Iamaroon A, Pongsiriwet S, Mahanupab P, Kitikamthon R, Pintong J. Oral non-Hodgkin lymphomas: Studies of EBV and p53 expression. *Oral Dis* 2003;**9:**14–18.
10. Glick M, Muzyka BC, Lurie D, Salkin LM. Oral manifestations associated with HIV-related disease as markers for immune suppression and AIDS. *Oral Surg Oral Med Oral Pathol* 1994;**77:**344–9.
11. Llewellyn CD, Linklater K, Bell J, Johnson NW, Warnakulasuriya S. An analysis of risk factors for oral cancer in young people: A case–control study. *Oral Oncol* 2004;**40:**304–13.
12. Lockhart PB, Norris CM Jr, Pulliam C. Dental factors in the genesis of squamous cell carcinoma of the oral cavity. *Oral Oncol* 1998;**34:**133–9.
13. Mose E, Lee WR. Occurrence of oral and pharyngeal cancers in textile workers. *Br J Ind Med* 1974;**31:**224.
14. Varghese I, Rajendran R, Sugathan CK, Vijayakumar T. Prevalence of oral submucous fibrosis among the cashew workers of Kerala, South India. *Indian J Cancer* 1986;**23:**101–4.
15. Kennedy AR, Billings PC, Maki PA, Newberne P. Effects of various preparations of dietary protease inhibitors on oral carcinogenesis in hamsters induced by DMBA. *Nutr Cancer* 1993;**19:**191–200.
16. Pogoda JM, Preston-Martin S. Solar radiation, lip protection, and lip cancer risk in Los Angeles County women (California, United States). *Cancer Causes Control* 1996;**7:**458–63.
17. Elwood JM. Epidemiological studies of radiofrequency exposures and human cancer. *Bioelectromagnetics* 2003;Suppl. 6:S63–S73.
18. McLaughlin JK, Gridley G, Block G, Winn DM, Preston-Martin S, Schoenberg JB, *et al.* Dietary factors in oral and pharyngeal cancer. *J Natl Cancer Inst* 1988;**80:**1237–43.
19. La Vecchia C, Negri E, D'Avanzo B, Boyle P, Franceschi S. Dietary indicators of oral and pharyngeal cancer. *Int J Epidemiol* 1991;**20:**39–44.

Table 8. Prevention and treatment of oral cancer

Medical interventions	Non-medical interventions	Other interventions
• Biopsy of pre-malignant lesions • Surgery • Radiotherapy • Chemotherapy • Combination treatment	• Stop all oral abusive habits such as tobacco smoking and chewing • Improve oral hygiene • Remove all irritants from the mouth • Improve the nutritional status • Undergo regular oral check-up	• Self-examination of the oral cavity • Prevent initiation of harmful habits • Industrial safety legislation and protection of the health of workers

DENTAL FLUOROSIS[1–6]

Fluorine is a trace element which has a caries-preventive effect. The optimum level of fluorine in drinking water is 0.75–1 ppm. A fluoride content higher than 1 ppm is known to cause dental and skeletal fluorosis. Dental fluorosis is also known as 'mottled enamel'. It manifests as unsightly, chalky white or yellowish-brownish discoloration of the teeth, sometimes with structural defects in the enamel such as pitting of the surface. Table 9 lists the direct, indirect and distant causes of dental flourosis.

Fluoride toxicity depends upon several factors such as (i) the total quantity of ingested fluoride from all sources—water, food and drugs with a high fluoride content, (ii) climatic conditions of the region—in tropical countries such as India, water consumption can be high causing higher ingestion of fluoride-containing water, (iii) nutritional status of the individual—deficiency of vitamin D, calcium and phosphate can aggravate the manifestations of fluoride toxicity, (iv) presence of advanced kidney disease and hyperthyroidism are associated with manifestations of fluoride toxicity.

The prevention of dental fluorosis can be undertaken at three levels (Table 10).

References

1. Hodge HC. The concentration of fluoride in drinking water to give the point of minimum caries with maximum safety. *J Am Dent Assoc* 1950;**40:**436.
2. Dean HT, Jay P, Arnold FA, Elvove E. Domestic water and dental caries including certain epidemiological aspects of oral *L. acidophilus. Public Health Rep* 1939;**54:**862–88.
3. Beltran-Aguilar Ed, Goldstein JW, Lockwood SA. Fluoride varnishes—a review of their clinical use, cariostatic mechanism, efficacy and safety. *J Am Dent Assoc* 2000;**131:**589–96.
4. Svanberg M, Westergren G. Effect of SNF_2, administered as mouth rinses or topically applied, on *Streptococcus mutans, Streptococcus sanguis* and lactobacilli in dental plaque and saliva. *Scand J Dent Res* 1983;**91:**123.
5. Moudgil A, Srivastava RN, Vasudev A, Bagga A, Gupta A. Fluorosis with crippling skeletal deformities. *Indian Pediatr* 1986;**23:**767–73.
6. Susheela AK. Treatise on fluoride. Project report, sponsored by Task Force on Safe Drinking Water, Government of India. 2003.

Table 9. Causes of dental fluorosis

Direct	Indirect	Distant
• Exposure to high levels of fluorides: >1 ppm of fluoride in drinking water • Airborne fluoride from industrial pollution (aluminium factories, phosphate fertilizers, glass-manufacturing industries, ceramic and brick products) • Fluoride-rich dietary intake—sea food, poultry, grain and cereal products (especially sorghum), tea, rock salt, green leafy vegetables, etc.	• Tropical climate—excess ingestion of water and beverages with a high fluoride content • Presence of kidney diseases affecting the excretion of fluoride • Thyroid and thyrotrophic hormones have a synergistic effect on fluoride toxicity	• Poor nutritional status—deficiency of vitamin D, calcium and phosphates • Decreased bone phosphatase activity is linked to fluoride toxicity

Table 10. Strategies for the prevention of dental fluorosis

Primary prevention	Secondary prevention	Tertiary prevention
• Specific guidelines on the use and appropriate dose levels of fluoride supplements, and use of fluoride toothpaste for young children • In high fluoride areas —provide an alternate supply of drinking water —employ defluoridation techniques at the community or individual level	• Improve the nutritional status, especially of expecting mothers, newborns and children up to the age of 12 years. • Treat other causes of fluoride toxicity such as kidney and thyroid diseases, etc.	Treat the discoloured/disfigured dentition by appropriate aesthetic treatment such as bleaching, micro-abrasion, laminate veneers, etc.

Table 11. Equipment, minimum manpower required and approximate cost for medical interventions for oral and dental diseases

Medical interventions	Equipment/instruments required	Time required	Personnel	Set-up	In dental schools (in Rs)	In private clinics* (in Rs)
Dental check-up	Gloves, face mask, head light, mouth mirror, explorer, tweezers, cotton/ gauze, etc.	5 minutes	Dental surgeon	At all levels	Nil	100–300
ART restorations	All the above + set of 8–10 hand instruments, glass–ionomer cement type IX, vaseline, etc.	15 minutes/ filling	Dental surgeon/ health care workers/ dental hygienist (after adequate training; controversial)	At the PHC and community level	50/- per filling	250–500
Silver filling	Dental clinic set-up with micro-motor/air-rotor and inventory of cutting and filling instruments Cost of clinic set-up, excluding the place, is minimum 2.5 lakh	30 minutes/ filling	Dental surgeon	At the CHC level and upwards	100/- per filling	250–1000 (depending on simple or complex restoration)
Aesthetic fillings	As for silver filling + aesthetic restorative materials (composite resins, compomers, glass–ionomers) + light cure units	30 minutes/ filling	Dental surgeon	At district hospital and upwards	100/- per filling	400–1000
Indirect restorations (full crowns, inlays, veneers, etc.)	Dental clinic supported by well-equipped dental laboratory	Minimum 2 sittings of 30 minutes–1 hour each	Specialist dental surgeon/dental surgeon	At dental colleges, tertiary care hospitals and in private clinics	250/- per restoration	1500–2000
Root canal treatment	Dental clinic as in (3) Instruments for root canal treatment, sealants, gutta-percha, medicaments and irrigants	3–4 sittings of 30 minutes each	Specialist dental surgeon (endodontist)	At district hospital and above	150/-	1500–3000
Scaling and polishing of teeth	Hand scalers/ultrasonic scalers	2–3 sittings of 20 minutes each	Dental hygienist/ dental surgeon	At the CHC level and above	Nil or 50/-	800–1000
Surgical procedures (gingivectomy, flap operation, mucogingival surgery and endodontic surgery)	Dental surgery set-up as in (3) + all surgical instruments and retro-filling materials	45–60 minutes	Dental surgeon/ specialist dental surgeon (periodontist, endodontist, oral surgeon)	At district hospital and above	100/-	1500–5000
Orthodontics—removable	Dental surgery clinic set-up	6–12 months	Orthodontist/ dental surgeon	At district hospital and above	200/- per appliance	2500–3000 per appliance
Orthodontics—fixed appliances	Dental surgery clinic set-up with extraoral radiographic facility and inventory of all orthodontic instruments and supply of brackets, arch wires, elastics, etc.	One year to two-and-a-half years	Orthodontist	At dental schools, tertiary care hospitals and private clinics	2000–3000	15,000–30,000
Complete dentures	Dental surgery clinic set-up supported by a dental laboratory	5–7 sittings at intervals of 2–7 days	Specialist dental surgeon/dental surgeon	At CHC level and upwards	350–500	5000–10,000
Partial denture (removable)	Dental surgery clinic set-up supported by a dental laboratory	3–4 sittings	Dental surgeon	At CHC and upwards	100/- 25/- per additional tooth	300–1000
Partial denture (fixed)	Dental surgery clinic set-up supported by a dental laboratory	3–4 sittings	Specialist dental surgeon (prosthodontist)	At dental schools, tertiary care hospitals and private clinics	250/- per unit	2000 per unit

(Cont.)

Table 11 *(cont.).* Equipment, minimum manpower required and approximate cost for medical interventions for oral and dental diseases

Medical interventions	Equipment/instruments required	Time required	Personnel	Set-up	In dental schools (in Rs)	In private clinics* (in Rs)
Biopsy	Dental surgery set-up	15–30 minutes	Dental surgeon	At the CHC level and upwards	Nil	500–1000
Surgical extraction (impaction)	Dental surgery set-up + all surgical instruments and retro-filling materials	1 hour	Oral surgeon/dental surgeon	At district hospital and above	100	2000–3000
Fracture reduction/ cyst enucleation/ benign growth excision	Dental surgery set-up as for silver filling + all surgical instruments and retro-filling materials	1 hour	Oral surgeon/dental surgeon	At district hospital and above	Nil	5000–8000

PHC: primary health centre; CHC: community health centre; ART: atraumatic restorative treatment
*These rates are common in Delhi; may vary from State to State.

EPIDEMIOLOGY OF ORAL AND DENTAL DISEASES

Oral and dental diseases are widely prevalent in India. Though not life-threatening, these diseases are often very painful, expensive to treat and cause loss of several man-days. On the other hand, they are, to a great extent, preventable. It has now been recognized that oral and general health are closely interlinked. Periodontal (gum) diseases are found to be closely associated with several serious systemic illnesses such as cardiovascular and pulmonary diseases, stroke, low birth-weight babies and preterm labour. Besides, poor oral health affects the functions of mastication and speech, and ultimately the overall well-being of an individual.

The major oral and dental diseases/disorders are (i) dental caries, (ii) periodontal diseases, (iii) dentofacial anomalies and malocclusion, (iv) edentulousness (tooth loss), (v) oral cancer, (vi) maxillofacial and dental injuries, and (vii) fluorosis.

Dental caries

Dental caries is a universal disease affecting all geographic regions, races, both the sexes and all age groups. The prevalence of dental caries is generally estimated at the ages of 5, 12, 15, 35–44 and 65–74 years for global monitoring of trends and international comparisons. The prevalence is expressed in terms of point prevalence (percentage of population affected at any given point in time) as well as DMFT index (number of decayed, missing and filled teeth in an individual and in a population).

As per the WHO Oral Health Surveillance 1992, the DMFT index in 12-year-old Indian is 0.89. In India, different investigators have studied various age groups, which can be broadly classified as below 12 years, above 12 years, above 30 years and above 60 years (Tables 12–15). Based on the analysis of all these tables, the prevalence of dental caries in urban and rural populations at various specified age groups has been calculated (Table 16).

Table 12. Incidence of caries in the age group of less than 12 years

Investigator and year	Index used	State	Place	Sample size	Point prevalence	Mean DMFT
Shourie 1941	Day and Sadwick 1934	Delhi	Delhi (Urban)	69	50.8	2.83
			Delhi (Rural)	54	31.5	1.0
Chopra *et al.* 1985	WHO 1987		Delhi (Urban)	381	34.1	1.14
Gautam *et al.* 2001	WHO 1997		Delhi (Urban)	2366	35.12	1.18
Shourie 1947	Day and Sadwick 1934	Rajasthan	Ajmer (Urban)	178	50.0	2.1
Thapar 1989	Mollers (1966)		Jaipur	?	31.4	0.5
Sehgal 1960		Maharashtra	Bombay	69	39.36	5.9
Anita 1962			Bombay	504		6.64
Tewari *et al.* 1985	WHO 1983		Bombay	220	89.0	5.3
Damle and Patel 1993	WHO 1983		Fishermen community around Mumbai (11–15 years)	431	61.5	1.9

(Cont.)

Table 12 *(cont.)*. Incidence of caries in the age group of less than 12 years

Investigator and year	Index used	State	Place	Sample size	Point prevalence	Mean DMFT
Gaikwad 1993			Aurangabad	1995 (5–14 years)	57.89 (M) 45.2 (F)	0.55
Dutta 1965		West Bengal	Dumdum	180	67.1	2.96
Sarkar and Chowdhary 1992	WHO 1971			40	0.0 (1 year)	
				40	13.2 (3 years)	
				50	25.5 (4 years)	
				50	? (5 years)	
Chowdhary 1967		Uttar Pradesh	Lucknow (Urban)	107	32.7	
Gill and Prasad 1968			Lucknow (Rural)	138	44.0	1.1
Kavita *et al.* 1987	WHO 1983		Dehradun (Urban)		54.7	2.1
			Meerut (Urban)		57.4	1.9
			Lucknow (Urban)		89.0	4.4
			Banaras (Urban)		53.0	1.3
Kavita *et al.* 1987	WHO 1983		Dehradun (Rural)		42.4	1.2
			Meerut (Rural)		50.0	1.4
			Lucknow (Rural)		63.6	1.7
			Banaras (Rural)		54.0	1.5
Mishra and Shee 1979		Orissa	Behrampur		56.6	
Sahoo *et al.* 1986	WHO 1983		Orissa (Urban)	170	58.82	2.52
			Orissa (Rural)	160	57.5	2.66
Damle *et al.* 1982	Mollers 1966	Haryana	Haryana (Rural)	123	74.0	3.3
Gathwala *et al.* 1993	1993		Rohtak	501 (5–13 years)		?
Tiwari 1999	WHO 1987		Haryana	113	36.3 (5 years)	0.87
				157	38.2 (6 years)	0.9
Thapar 1953	?	Punjab	Moga	70	47.7	?
Chopra *et al.* 1983	WHO 1962		Punjab (Urban)	141	61.1	1.72
Chopra *et al.* 1985	WHO 1987		Jalandhar (Urban)	?	46.8	1.5
			Jalandhar (Rural)	151	39.7	1.0
			Abohar (Urban)	145	27.6	0.6
			Abohar (Rural)	150	24.7	0.6
Norboo *et al.* 1998	WHO 1987	Jammu and Kashmir	Leh (Urban)	62	74.6	4.3
			Leh (Rural)	72	63.9	2.3
			Kargil (Urban)	63	70.7	2.9
			Kargil (Rural)	71	63.4	2.2
Tewari *et al.* 1985	WHO 1983	Union Territory	Chandigarh (Urban)	204	59.0	2.26
			Chandigarh (Rural)	197	60.0	2.21
Chawla *et al.* 1993	WHO 1983		Chandigarh (Urban)	?	?	1.2
Goyal *et al.* 1997	WHO 1983		Chandigarh (Rural)	135	1.5 (1 year)	
				144	7.0 (2 years)	2.0
				154	19.4 (3 years)	2.35
				137	28.5 (4 years)	
				95	1.0 (1 year)	
				128	12.0 (2 years)	
				120	23.0 (3 years)	
				11	32.0 (4 years)	
				124	48.0 (5 years)	
Tewari *et al.* 1985	WHO 1983	Bihar	Bihar (Urban)	212	54.0	1.5
			Bihar (Rural)	99	35.0	1.1
Tewari and Mandal 1985	WHO 1983	Madhya Pradesh	Indore (Urban)	147	52.4	2.3

(Cont.)

Table 12 *(cont.).* Incidence of caries in the age group of less than 12 years

Investigator and year	Index used	State	Place	Sample size	Point prevalence	Mean DMFT
Virjee Shankar Aradhya 1987	Johnsen *et al.* 1984	Karnataka	Bangalore (Urban)	673	66.3 (4.5 years)	2.9
			Chickballapur (Rural)	394	58.4 (4.5 years)	2.3
Gupta *et al.* 1987	WHO 1983		Davengere (Rural)	100	25.0	0.6
			Davengere (Urban)	100	53.0	1.68
			Bangalore (Urban)	100	70.0	1.66
Sethi and Tandon 1996	William 1994		Udupi	404	65.5 (3–5years)	
Menon and Indushekhar 1999	WHO 1987		Dharwad	624	2.56	0.03
			Gadag	256	1.17	0.01
Rao *et al.* 1999	WHO 1987		Modbidri	550	75.3	0.2
Sharma *et al.* 1988	WHO 1983	North-east	Shillong	180	88.33	6.36
			Imphal	199	88.44	5.53
			Guwahati	199	80.90	5.35
			Kohima	198	90.40	6.4
Mandal *et al.* 1994	WHO 1983		Sikkim (Urban)	10	61.8	2.50
			Sikkim (Rural)	109	22.02	0.70
Mandal *et al.* 1994	WHO 1983		W. Bengal (Urban)	124	52.42	1.86
			W. Bengal (Rural)	20	48.33	1.48
Gupta *et al.* 1987	WHO 1983	Andhra Pradesh	Hyderabad (Rural)	187	50.8	1.63
Gupta *et al.* 1987	WHO 1983	Kerala	Calicut (Urban)	156	56.41	2.1
			Trivandrum (Urban)	103	51.46	1.81
Kuriarose and Joseph 1999	WHO		Trivandrum	600	57	2.28
Gopinath *et al.* 1999	WHO 1987	Tamil Nadu	Tamil Nadu	97	36.0	36 (M) 17 (F)

DMFT: number of decayed, missing and filled teeth

Table 13. Incidence of dental caries in children above 12 years of age

Investigator and year	Index used	State	Place	Sample size	Point prevalence	Mean DMFT
Shourie 1941	Day and Sadwick 1934	Delhi	Delhi (Urban)	95 (12 years)	54.8	5.7
Shourie 1941	Day and Sadwick 1934		Delhi (Urban)	19	52.7	1.2
			Delhi (Rural)	40 (15 years)	42.5	1.1
Gupta *et al.* 1993	WHO 1983		New Delhi	(12 years)	87.0	0.86
Chopra *et al.* 1995	WHO 1987		Delhi (Urban)	392 (15 years)	20.9	0.42
Shourie 1942	Day and Sadwick 1934	Tamil Nadu	Tamil Nadu (Urban)	42	57	2.0
Gopinath *et al.* 1999	WHO 1987		Tamil Nadu	232 (12 years)	61.2	3.2 (M) 3.7 (F)
Shourie 1947	Day and Sadwick 1934	Rajasthan	Ajmer (Urban)	(15 years)	56.3	
Thapar *et al.* 1989	Moller 1966		Rajasthan (Rural)	(12 years)	31.4	0.5
Chaudhary *et al.* 1957	Own criteria	Uttar Pradesh	Lucknow	368 (12 years)	32.0	1.15
Chaudhary *et al.* 1957	Own criteria		Lucknow	107 (5 years)	32.7	
Gill *et al.* 1968	WHO 1962		Lucknow (Urban)	99 (12 years)	99.0	43.3
Gill *et al.* 1968	WHO 1962		Lucknow (Urban)	23 (15 years)	66.8	0.7
Mehta *et al.* 1987	WHO 1983		Dehradun (Urban)	202 (15 years)	45.0	1.0
			Meerut (Urban)		42.0	1.1
			Lucknow (Urban)		42.6	1.0
			Banaras (Urban)		38.4	1.0
Mehta *et al.* 1987	WHO 1983		Dehradun (Rural)	112 (15 years)	38.2	0.8
			Meerut (Rural)		38.4	0.8
			Lucknow (Rural)		20.5	0.4
			Banaras (Rural)		41.0	0.8
Singh *et al.* 1999	WHO 1987		Faridabad (Rural)	233 (12 years)	33.1	0.79

(Cont.)

Table 13 *(cont.)*. Incidence of dental caries in children above 12 years of age

Investigator and year	Index used	State	Place	Sample size	Point prevalence	Mean DMFT
Singh *et al.* 1999	WHO 1987		Faridabad (Rural)	207 (15 years)	42.5	1.29
Anita 1962		Maharashtra	Bombay	503 (15 years)		2.5
Damle *et al.* 1982	Moller 1966		Naraingarh (Rural)	230 (15 years)	77.2	2.4
Damle and Patel 1984	WHO 1983		Bombay	(15 years)	78.0	3.6
Tiwari *et al.* 1985	WHO 1983		Bombay (Urban)	202 (15 years)	96.0	4.7
Damle and Ghonmode 1993	WHO 1983		Nagpur	(12 years)	82.6	4.0
Damle and Ghonmode 1993	WHO 1983		Nagpur	(15 years)	82.6	4.0
1993?			Nagpur	1811 (12–18 years)	81.3	>3
1994?	WHO 1983		Bombay (Urban)	367 (12 years)	80.0	3.8
Rodrigues and Damle 1998			Mumbai		68.02	
Ali *et al.* 1998	WHO 1987		Akola	508 (5–6 years)	61.4% 2.75+3.98	
Rodrigues and Damle 1998	WHO 1997		Bhiwandi	256 (12 years)	55.5	1.08
Tiwari and Chawla 1977	WHO 1971	Uttaranchal	Chandigarh (Urban)	82 (15 years)	86.6	4.7
Tiwari *et al.* 1983	WHO 1966		Chandigarh			
			(Urban)	217	51.1	1.38
			(Rural)	205 (15 years)	47.5	1.30
Chawla *et al.* 1993	WHO 1983		Chandigarh	(12 years)		1.2
Damle *et al.* 1982	Moller 1966	Haryana	Haryana (Rural)	152 (12 years)	89.5	3.2
Tiwari *et al.* 1985	WHO 1983		Haryana (Urban)	229	50.0	1.35
			Haryana (Rural)	200 (15 years)	47.5	1.30
Sharma *et al.* 1998	WHO		Haryana District (Gurgaon and Mahendragarh)	3031 (12–16 years)	36.7	0.67
Gauba *et al.* 1983	Moller 1966	Punjab	Punjab (Rural)	173 (12 years)	86.1	3.9
Gauba *et al.* 1983	Moller 1966		Ludhiana (Rural)	101 (15 years)	88.1	5.0
Chopra *et al.* 1983	WHO 1962		Punjab (Urban)	255 (12 years)	67.2	1.3
Chopra *et al.* 1995	WHO 1987		Jalandhar (Urban)	150	42.0	0.9
			Jalandhar (Rural)	146	24.7	0.46
			Abohar (Urban)	46	21.0	0.43
			Abohar (Rural)	46	24.0	0.43
Mishra and Shee 1985		Orissa	Orissa	(12 years)	61.1	
Tiwari *et al.* 1985	WHO 1983		Orissa (Rural)	174	63.8	2.1
			Orissa (Urban)	159 (12 years)	63.1	2.1
Sahoo *et al.* 1986	WHO 1983		Orissa Urban)	(12 years)	63.8	2.1
			Orissa (Rural)		67.9	2.0
Sahoo *et al.* 1986	WHO 1983		Orissa (Urban)	175 (15 years)	62.3	2.0
Mandal *et al.* 1994	WHO 1987		Orissa (Rural)	121 (15–16 years)	19.8	0.3
Mandal *et al.* 1994	WHO 1987		Bhubaneshwar (Urban)	120	18.3	0.3
Mandal *et al.* 2001	WHO 1983		Orissa (Urban)	702		
			Orissa (Rural)	351	56.0	
				351	48.7	
Tiwari *et al.* 1985	WHO 1983	Himachal Pradesh	Himachal (Urban)	178	50.0	1.2
			Himachal (Rural)	191 (15 years)	49.0	1.3
Tiwari *et al.* 1985	WHO 1983	Bihar	Bihar (Urban)	160	42.5	1.2
			Bihar (Rural)	202 (15 years)	49.5	1.3
Tiwari and Mandal 1985	WHO 1983	Madhya Pradesh	Indore (Urban)	162 (15 years)	68.0	2.8
Sharma *et al.* 1988	WHO 1983	North-east	Shillong (Urban)	183	60.1	2.1
			Imphal (Urban)	197	63.45	1.76
			Guwahati (Urban)	200	83.5	3.13
			Kohima and Mokokochung (Urban)	195 (15 years)	63.08	2.36
Mandal *et al.* 1994	WHO 1987		Gangtok (Urban)	106 (15 years)	30.2	0.5

(Cont.)

Table 13 *(cont.).* Incidence of dental caries in children above 12 years of age

Investigator and year	Index used	State	Place	Sample size	Point prevalence	Mean DMFT
Mandal *et al.* 1994	WHO 1987		Sikkim (Rural)	106 (15–16 years)	17.9	0.3
Mandal *et al.* 2001	WHO 1983		Sikkim	644		
			(Urban)	323	61.8	
			(Rural)	321	22.0	
Mandal *et al.* 1994	WHO 1987	West Bengal	Calcutta (Urban)	119	21.0	0.3
Mandal *et al.* 1994	WHO 1987		West Bengal (Rural)	118	15.2	0.3
Mandal *et al.* 2001	WHO 1983		West Bengal	720	52.4	5.6
			(Urban)	361	48.3	
			(Rural)	359		
Norboo *et al.* 1998	WHO 1987	Jammu and Kashmir	Leh (Rural)	74	43.2	0.87
			Kargil (Rural)	69 (12 years)	29.0	0.68
Norboo *et al.* 1998	WHO 1987		Leh (Urban)	65	47.7	1.01
			Kargil (Urban)	73 (12 years)	35.8	0.63
Norboo *et al.* 1998	WHO 1987		Leh (Urban)	70	60.0	1.01
			Leh (Rural)	60	45.0	1.15
			Kargil (Urban)	79	47.5	1.2
			Kargil (Rural)	69 (15 years)	39.7	1.0
Nagaraga Rao 1980		Karanataka	Udupi	(2 years)		4.1
Gupta *et al.* 1987	WHO 1983		Davangere (Rural)	98 (15 years)	42.86	1.07
Menon and Indushekhar 1999	WHO 1987		Dharwad	300	31.0	0.78
			Gadag	488 (12 years)	24.6	0.6
Rao *et al.* 1999	WHO 1987		Moodbidri (Urban)	771 (12 years)	67.1	1.29
Menon and Indushekhar 1999	WHO 1987		Dharwad	106	55.7	1.09
			Gadag	127 (15 years)	30.3	0.75
Sogi and Bhasker 2001			Davangere			3.12
Javali and Prasad 2001	WHO		Karnataka	8152 (12–17 years)	43.5 (M) 50.5 (F)	1.57
Kulkarni and Deshpande 2002	WHO 1987		Belgaum	2005 (11–15 years)	45.12%	1.18
Gupta *et al.* 1987	WHO 1983	Andhra Pradesh	Hyderabad	85 (15 years)	34.12	0.96
Retnakumari 2000	WHO 1997		Varkala	119 (12 years)	67.2	2.067
Goel *et al.* 2000	WHO 1987		Puttur	203 (12 years)	59.6	1.87

DMFT: number of decayed, missing and filled teeth

Table 14. Incidence of dental caries in the age group of above 30 years

Investigator and year	Index used	State	Place	Sample size	Point prevalence	Mean DMFT
Barreto *et al.* 1953		Maharashtra	Bombay	331		1.50
Mangi and Jalili 1967		Madhya Pradesh	Madhya Pradesh	331		4.10
Ramachandran *et al.* 1973		Tamil Nadu	Tamil Nadu (Urban)	NA		2.88
			Tamil Nadu (Rural)			2.10
Damle *et al.* 1982	Mollers 1966	Haryana	Haryana (Rural)	667	61	1.70
Tewari *et al.* 1985	WHO 1983		Haryana (Urban)	101	46.5	1.5
			Haryana (Rural)	200	68.0	3.04
Tewari *et al.* 1985	WHO 1983	Uttaranchal	Chandigarh (Urban)	156	81.4	4.38
			Chandigarh (Rural)	196	82.1	4.38
Tewari *et al.* 1985	WHO 1983	Uttar Pradesh	Lucknow (Urban)	199	47.0	1.13
			Lucknow (Rural)	118	45.8	1.22
Tewari *et al.* 1985	WHO 1983	Jammu and Kashmir	J&K (Urban)	NA		4.9
			J&K (Rural)	NA		5.8
Chopra *et al.* 1985	WHO 1987	Punjab	Jalandhar (Urban)	144	34.72	1.08

(Cont.)

Table 14 *(cont.).* Incidence of dental caries in the age group of above 30 years

Investigator and year	Index used	State	Place	Sample size	Point prevalence	Mean DMFT
			Jalandhar (Rural)	145	30.34	0.76
			Abohar (Urban)	140	20.0	0.42
			Abohar (Rural)	149	24.16	0.41
Sharma *et al.* 1985	WHO 1983	North-east	Meghalaya (Urban)	196	54.6	1.18
			Manipur (Urban)	199	63.82	1.86
			Assam (Urban)	244	66.0	1.86
			Nagaland (Urban)	202	62.4	2.13
Mandal *et al.* 1994	WHO 1983		Sikkim (Urban)	107	29.91	0.62
			Sikkim (Rural)	107	24.53	0.60
Tewari and Mandal 1985	WHO 1983	Madhya Pradesh	Indore	66	70.0	3.80
Tewari and Damle 1985	WHO 1983			201	80.0	3.57
Gupta *et al.* 1985	WHO 1983	Kerala	Trivandrum (Urban)	103	79.61	2.21
			Calicut (Urban)	104	78.9	2.16
			Calicut (Rural)	90	47.8	1.2
Gupta *et al.* 1985	WHO 1983	Andhra Pradesh	Hyderabad (Urban)	111	64.86	2.16
			Hyderabad (Rural)	87	44.83	1.16
Gupta *et al.* 1985	WHO 1983	Karnataka	Bangalore (Urban)	98	73.47	2.17
			Davengere (Urban)	102	68.63	2.29
			Davengere (Rural)	102	48.04	1.07
Mandal *et al.* 1994	WHO 1987	Orissa	Orissa (Urban)	5	24.35	0.47
			Orissa (Rural)	114	20.17	0.48
Mandal *et al.* 1994	WHO 1987	West Bengal	West Bengal (Urban)	18	19.49	0.47
			West Bengal (Rural)	20	18.18	0.40
Chopra *et al.* 1995	WHO 1987	Delhi	Delhi (Urban)	388	24.5	0.50
Tewari *et al.* 1995	WHO 1983	Bihar	Bihar (Urban)	149	69	1.75
			Bihar (Rural)	193	63.2	1.85

DMFT: number of decayed, missing and filled teeth; NA: not available

Table 15. Incidence of dental caries in those above 60 years of age

Year	State	Place	Index used	Sample size	Point prevalence	Mean DMFT
1994	Karnataka			300		13.51
2004	Delhi	New Delhi	WHO 1987	1052	72.4	—

DMFT: number of decayed, missing and filled teeth

Table 16. Prevalence of dental caries in different age groups

Age group (years)	Urban	Rural	Average	DMFT
5–6	67.23	46.22	56.72	2.1
12	57.94	36.90	47.39	1.6
15	55.97	43.28	49.59	1.37
30–35	45.21	39.27	42.24	1.39
60–75	79.40	61.90	70.65	—

DMFT: number of decayed, missing and filled teeth

Periodontal diseases

Periodontal diseases affect the supporting structures of teeth, i.e. the gingiva (gums), periodontal ligament, alveolar bone and cementum (covering the roots of the teeth) and are the commonest cause of tooth loss in India. A thin, adherent microbial film on the tooth surfaces, called dental plaque, is the main pathological cause of gingival and periodontal inflammation. Poor oral hygiene, faulty food habits, poor nutrition, presence of metabolic diseases such as diabetes, use of tobacco, etc. are the major contributory factors for periodontal diseases.

Periodontal diseases are common in the adult population, but not very common in children. Several indices are used to measure periodontal diseases, such as plaque index, oral hygiene index, bleeding index, community periodontal index (CPI), etc. A scoring system to score the gradation from mild to severe forms of the disease is also available. Therefore, there is no uniformity in data on the prevalence of periodontal diseases and hence, it is difficult to compare the data. However, it is widely accepted that periodontal

diseases affect over 90% of the Indian population, but the majority of them may have only mild gingivitis and bleeding from the gums, which is reversible with proper oral hygiene measures. More advanced periodontal disease with pocket formation and bone loss, which could ultimately lead to tooth loss if not treated properly, may affect 40%–45% of the population. It is also known that use of tobacco, especially habitual chewing of tobacco, presence of metabolic diseases such as diabetes, nutritional deficiencies, compromised immune status and increasing age are associated with an increase in periodontal diseases.

Table 17 documents only some studies, and highlights totally incoherent data. Moreover, most of the studies have been conducted on the child population, in whom periodontal diseases are not widely prevalent.

Table 17. Periodontal diseases

Investigator and year	State	Place	Index	Sample size	Prevalence
Anuradha *et al.* 2002	Karnataka	Davangere	CPI and plaque index	NA	Decrease with increase in the fluoride content of water
Sogi and Bhasker 2001		Davangere	Oral hygiene index (OHI)	2007 (13–14 years)	NA
Doifode *et al.* 2000	Maharashtra	Nagpur		5061 (all age groups)	Periodontal diseases 34.8% total <15 years 18.4% 15–30 years 36.4% 30–60 years 50.2% 60+ years 54.4%
Rao and Bharambe 1993		Wardha		778 (5–12 years) (Rural) (Urban)	17.8% 22.6% 10.5% 15.0%
Gathwala 1993	Haryana	Rohtak		501 (5–13 years)	36.3% (gingivitis)
Rao and Bharambe 1993	Maharashtra	Wardha		778 (5–12 years)	4.8% (bleeding/abscess)
Shah 2003	Delhi	South Delhi	CPI index	1052 (above 60 years)	100% mild 9.1% moderate 19% severe 71.9%

CPI: community periodontal index

Dentofacial anomalies and malocclusion

The prevalence of malocclusion in India is estimated to be 30% in school-age children (Table 18). Malocclusion may vary from mild to severe, causing aesthetic and functional problems, and may also predispose to dental caries, periodontal diseases as well as increased susceptibility to trauma, especially to excessively proclined teeth. The major dentofacial deformity is cleft lip and palate, which is seen in 1.7/1000 live-births (Table 19).

Table 18. Prevalence of dentofacial anomalies and malocclusion

Author and year	State	Place	Age group (years)	Prevalence (%)
Shourie 1952	Punjab	Punjab	13–16	50
Guaba *et al.* 1998		Ambala	6–15	29.2
Shaik and Desai 1966	Tamil Nadu	Madras	15–25	19.6
Jacob 1969	Kerala	Trivandrum	12–15	44.97
Jose and Joseph 2003		Kerala	12–15	NA
Prasad and Savadi 1971	Karnataka	Bangalore	5–15	51.5
Nagaraja Rao 1980		Udupi	5–15	28.8
Gardiner 1989		South Kanara	10–12	42
Jalili 1989	Madhya Pradesh	Mandu (Tribal area)	6–14	14.4
Kharbanda 1991	Delhi	Delhi	5–13	10–18
Kharbanda 1995		Delhi	10–13	45.7
Goel *et al.* 2000	Andhra Pradesh	Puttur	5–6	1.79
			12–13	36.95

Table 19. Incidence of cleft lip and cleft palate in India (hospital-based studies)

Location of the hospital	Incidence (%)	
	Cleft lip	Cleft lip and palate
Delhi	2.21	0.71
Delhi (AIIMS)	1.40	0.30
Chandigarh	1.0	—
Jaipur	1.12	0.35
Patiala	1.5	—
Lucknow	1.09	—
Ajmer	0.90	—
Mumbai	1.30	0.20
Ahmedabad	1.06	0.24
Chennai	1.60	0.10
Kolkata	0.63	0.16
Hyderabad	1.90	1.90

AIIMS: All India Institute of Medical Sciences

Edentulousness (tooth loss)

Tooth loss results from dental caries, periodontal diseases and trauma. Tooth loss increases with advancing age (Table 20). Loss of the teeth results in decreased masticatory efficiency, causing a shift in dietary practices. This may result in nutritional deficiencies. Tooth loss may also cause problems in speech and affect aesthetics, causing an overall loss of self-esteem and confidence. Very little data are available on tooth loss.

Table 20. Tooth loss (edentulousness)

Age group (years)	Number of missing teeth	Edentulousness (%)
60–64	8.5	11.1
65–74	10.9	19.4
75+	18.1	32.3

Dental fluorosis

In India, a high fluoride content in ground water is endemic in some areas. The states that are most affected are Andhra Pradesh, Gujarat and Rajasthan. Table 21 shows the distribution of fluoride in different states. It has been estimated that about 666.2 lakh people are at risk for fluoride toxicity of which children below the age of 14 years constitute 60 lakh.

Data available from a field survey in Gujarat, Haryana and Delhi are presented in Tables 22, 23 and 24, respectively.

Table 21. Distribution of fluoride analysis of ground water samples from different States of India

States		Number of water samples	Fluoride <1.0 mg/L	Fluoride 1.0–1.5 mg/L	Fluoride >1.5 mg/L	Maximum fluoride value (mg/L)
Uttar Pradesh	No.	502	398	62	42	15.0 (Marksnagar, Unnao district)
	%		79.2	12.4	8.4	
Andhra Pradesh	No.	786	752	19	15	7.90 (Nalgonda district)
	%		95.7	2.4	1.9	
Rajasthan	No.	780	403	114	263	22.0 (Nagaur district)
	%		51.7	14.6	33.7	
Maharashtra	No.	161	156	—	5	5.0 (Chandrapur district)
	%		96.9	—	3.1	
Madhya Pradesh (West)	No.	749	678	51	20	4.5 (Sirohi, Bhind district)
	%		90.5	6.8	2.7	
Karnataka	No.	773	634	91	48	8.3 (Kulgeri, Bijapur district)
	%		82.0	11.8	6.2	
Chandigarh	No.	1	—	1	—	—
	%		—	100	—	
Punjab	No.	332	232	46	54	11.7 (Bathinda district)
	%		69.9	13.9	16.2	
Haryana	No.	306	134	48	124	21.0 (Hissar district)
	%		43.8	15.7	40.5	
Delhi	No.	38	31	4	3	3.25 (Palam)
	%		81.6	10.5	7.9	
Orissa	No.	83	69	5	9	11.0 (Balasore and Bolangir district)
	%		83.1	6.0	10.8	
Bihar	No.	328	313	5	10	4.2
	%		95.4	1.5	3.1	

(Cont.)

Table 21 *(cont.).* Distribution of fluoride analysis of ground water samples from different States of India

States		Number of water samples	Fluoride <1.0 mg/L	Fluoride 1.0–1.5 mg/L	Fluoride >1.5 mg/L	Maximum fluoride value (mg/L)
Tamil Nadu	No.	464	398	53	213	6.8 (Madurai district)
	%		85.8	11.4	2.8	
Gujarat	No.	589	554	15	20	11.0 (Amreli district)
	%		94.1	2.5	3.4	
West Bengal	No.	466	454	—	12	16.0 (Birbhum district)
	%		97.4	—	2.6	
Kerala	No.	676	669	3	4	4.6 (Konnakuzhill district, Trichur)
	%		99.0	0.4	0.6	
Madhya Pradesh (East)	No.	346	340	—	6	—
	%		98.3	—	1.7	
Jammu and Kashmir	No.	117	117	4	1	0.78 (Dablehar)
	%		100	—	—	
Himachal Pradesh	No.	79	74	4	1	9.5 (Dhaulakuwan district)
	%		93.7	5.0	1.3	
States in the North-east	No.	295	295	—	—	0.5 (Darang district)
	%		100	—	—	
Total	No.	7871	6701	521	649	—

Source: Ground Water Authority, India

Table 22. Dental fluoride survey in schoolchildren from 18 districts in Gujarat

District	No. of schools surveyed	No. of students examined in the schools (8 years and above)			No. of students with dental fluorosis	Percentage affected with fluorosed teeth
		Boys	Girls	Total		
Ahmedabad	199	27,947	20,123	48,070	8,537	17.75
Gandhinagar	29	4,436	4,023	8,459	967	11.43
Mehsana	415	62,322	38,912	101,234	25,307	24.90
Banaskantha	367	36,463	20,925	57,388	10,032	17.78
Sabarkantha	278	21,000	18,405	39,405	5,728	14.50
Baroda	240	13,826	11,825	25,651	4,329	16.87
Kheda	210	24,064	19,219	43,283	5,266	12.16
Panchmahal	311	34,603	25,729	60,332	5,207	8.40
Bharuch	42	4,781	4,459	9,240	1,378	14.90
Surat	19	1,697	1,581	3,278	260	7.90
Valsad	14	1,939	1,889	3,828	101	2.60
Junagadh	50	7,075	5,314	12,389	4,097	33.00
Amreli	75	9,159	7,975	17,134	2,855	16.60
Surendranagar	71	7,442	6,010	13,452	2,961	22.00
Jamnagar	28	3,070	2,316	5,386	838	15.50
Bhavnagar	77	10,667	8,472	19,139	2,714	14.10
Rajkot	44	6,065	7,320	13,385	1,971	14.70
Kutch	13	1,599	1,561	3,160	640	20.25
Total	2482	278,155	206,058	484,213	83,188	% range: 2.6–33.0

Source: Gujarat Health Department, 1996–97 (From: Susheela AK. *Treatise on fluoride*. Project report. Sponsored by the Task Force on Safe Drinking Water, Government of India, 2003)

Table 23. Incidence of dental fluorosis in two villages in Haryana

Village	Drinking water fluoride level (mg/L)	Incidence of dental fluorosis (%)
Sotai	1.89–3.83	77
Machgar	0.64	13

Source: MD Thesis of Gajender Singh Meena, AIIMS 1983 (From: Susheela AK. *Treatise on fluoride*. Project report. Sponsored by the Task Force on Safe Drinking Water, Government of India, 2003)

Table 24. Incidence of dental fluorosis in children of 6 schools and status of contamination of drinking water with fluoride in the Palam area of NCTD

School	Total no. of students in the school	No. of students examined for dental fluorosis	Total no. of water samples collected through afflicted students	No. of students afflicted	Percentage afflicted	No. of fluoride contaminated sources (above 1.0 mg/L)	Range of fluoride contamination (mg/L)	No. of safe sources (fluoride below 1.0 mg/L)
1	237	67	44	25	37	17	1.10–2.0	27
2	1017	578	81	98	17	Nil	—	81
3	2100	745	98	119	16	33	1.13–5.01	65
4	1956	1037	86	140	13	4	1.15–2.88	72
5	2000	1290	86	144	11	4	1.62–9.30	82
6	1700	1200	48	55	4.5	20	1.1–12.45	28
Total	9010	4917	443	581	4.5–37	88	1.1–12.45	355

NCTD: National Capital Territory of Delhi
Source: Water Foundation Survey, 2002 (From: Susheela AK. *Treatise on fluoride*. Project report. Sponsored by the Task Force on Safe Drinking Water, Government of India, 2003)

Oral cancer

In India, the incidence of oral cancer is the highest in the world and is preceded by some premalignant lesion. The most important of all premalignant lesions is oral submucous fibrosis. It is characteristically found in people of South-East Asian origin and is associated with the chewing of betel nut. Its prevalence has increased manifold in the past three decades due to increased consumption of *paan masala* and *gutka* by persons of all age groups, including children (Table 25).

The condition has a high malignant potential, 7.5% of the lesions become malignant over a 10-year period and more than one lesion may develop at different sites in the oral cavity.

Data from specialized cancer hospitals across the country over a period of 7 years (1993–2000) are shown in Table 26. The prevalence of oral cancer reported by Population-based Cancer Registries is given in Table 27. A summary of annual incidence of oral cancer of different sites from National Cancer Registries in Mumbai and Chennai for the period 1988–92 is shown in Tables 28 and 29, respectively. It shows the age-standardized incidence rate for different sites. Overall, the incidence per 100,000 population is 29 for males and 14.3 for females, the average for the population being 21.65. When these data are compared with data from other parts of the world (US 4.4, Japan 1.6, UK 2), it is evident that the prevalence in India is much higher. Given the large population of India, the actual number of cases of oral cancer is gigantic.

Table 25. Oral submucous fibrosis in India (1990)

	Incidence 100,000/year	
Sex	North India	South India
Males	5–8	9
Females	2–6	20

Table 26. Number of treated cases in cancer hospitals

ICD 140–149	Number	Data not available from
1993	6209	Bihar, Gujarat and Himachal Pradesh
1994	5961	Bihar, Gujarat, Himachal Pradesh and Maharashtra
1995	6794	Bihar, Gujarat and West Bengal
1996	9444	Bihar, Gujarat, Tripura and West Bengal
1997	9165	Andhra Pradesh, Bihar, Gujarat and West Bengal
Number of hospitals—25		
2000	9430	Bihar, Gujarat and Orissa
Number of hospitals—35		

ICD Code: 140 lip; 141 tongue; 142 salivary gland; 143–145 mouth; 146 oropharynx; 147 nasopharynx; 148 hypopharynx and 149 pharynx
Source: Health Information of India (1993–2000)

Table 27. Population-based Cancer Registry (PBCR) report

			Bangalore		Barshi		Bhopal		Chennai		Delhi		Mumbai	
PBCR	Site of cancer		No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
1990–96	Tongue	M	348	3.4	30	4.7	206	8.1	535	4.7	1242	4.7	1456	5.0
		F	88	0.8	8	1.0	26	1.2	176	1.4	334	1.3	448	1.8
	Oral cavity	M	284	2.9	33	5.2	182	7.2	671	5.9	854	3.3	1601	5.5
		F	726	6.2	11	1.4	104	4.6	610	4.9	427	1.7	919	3.4
	Hypopharynx		560	5.5	68	10.7	166	6.5	550	4.8	555	2.1	1519	5.3
1997–98	Lip		5	3.16	1	3.52	4	3.46	10	3.27	24	3.27	24	3.25
	Tongue		136	3.44	5	2.59	70	8.04	192	5.26	384	4.2	417	4.84
	Oral cavity		101	3.28	13	3.52	82	9.41	190	5.20	333	3.69	480	5.34
	Oropharynx		75	2.45	1	10.36	17	1.95	80	2.19	212	2.35	164	1.90
	Hypopharynx		171	5.55	20	0.00	50	5.74	167	4.57	193	2.14	362	4.20

Table 28. Oral cancer in Mumbai (1988–1992)

Age group		Site of cancer			
(years)	Sex	Lip	Tongue	Salivary gland	Mouth
0–4	M	—	—	0.0	—
	F	0.0	0.0	—	0.0
5–9	M	0.0	—	—	0.0
	F	—	—	—	—
10–14	M	—	—	0.0	—
	F	—	—	0.1	—
15–19	M	—	—	—	0.1
	F	—	—	—	0.0
20–24	M	—	0.1	0.0	0.3
	F	—	0.1	0.0	0.2
25–29	M	—	0.3	0.3	0.4
	F	—	0.4	0.1	0.4
30–34	M	0.1	0.9	0.2	1.4
	F	0.1	0.5	0.2	0.8
35–39	M	0.1	2.4	0.3	3.9
	F	—	1.2	0.7	2.6
40–44	M	0.1	4.8	0.7	6.1
	F	0.1	2.2	0.4	5.1
45–49	M	0.7	9.6	0.6	12.3
	F	0.7	2.9	0.4	6.6
50–54	M	0.7	13.3	1.1	16.7
55–59	F	0.4	5.2	0.8	13.1
	M	1.4	21.5	0.9	22.3
60–64	F	1.5	10.9	—	14.9
	M	1.4	27.5	2.7	23.2
	F	1.7	9.6	1.4	21.3
65–69	M	1.1	38.2	5.7	32.5
	F	2.0	8.3	1.2	18.9
70–74	M	2.4	36.6	4.1	23.0
	F	—	11.6	0.6	23.8
75+	M	3.2	45.0	1.9	30.4
	F	2.0	13.6	2.5	26.2

Note: Annual incidence per 100,000 by age group

Source: Parkin *et al. Cancer incidence in five continents, Vol. VII.* Lyon: IARC Scientific Publications No. 143; 1997

Table 29. Oral cancer in Chennai (1988–1992)

Age group		Site of cancer			
(years)	Sex	Lip	Tongue	Salivary gland	Mouth
0–4	M	—	0.1	—	—
	F	—	—	0.1	0.1
5–9	M	—	—	—	—
	F	—	—	—	0.1
15–19	M	—	0.1	—	—
	F	—	—	0.2	0.2
20–24	M	—	0.1	0.1	0.3
	F	—	0.2	0.1	0.3
25–29	M	—	0.3	0.2	0.2
	F	—	0.3	0.3	0.4
30–34	M	0.3	2.0	0.3	0.4
	F	—	0.2	0.5	2.2
35–39	M	—	2.7	0.5	3.2
	F	—	0.8	—	2.8
40–44	M	0.5	5.1	0.7	4.0
	F	—	1.3	0.2	9.2
45–49	M	1.4	9.0	0.2	11.7
	F	0.5	3.2	1.0	10.3
50–54	M	1.1	15.3	0.5	20.9
	F	0.9	7.6	1.8	30.1
55–59	M	0.7	23.0	1.4	29.3
	F	1.3	7.3	1.3	29.2
60–64	M	2.6	25.9	1.3	34.2
	F	2.6	8.3	—	43.0
65–69	M	2.4	35.6	7.1	47.5
	F	3.0	8.3	0.8	37.5
70–74	M	1.0	23.7	2.1	22.7
	F	3.9	8.9	3.0	29.6
75+	M	—	19.0	1.2	40.4
	F	3.2	6.5	1.1	29.2

Note: Annual incidence per 100,000 by age group

Source: Parkin *et al. Cancer incidence in five continents, Vol. VII.* Lyon: IARC Scientific Publications No. 143; 1997

Tobacco-related cancers

Sites of cancer that have been associated with the use of tobacco (tobacco-related cancers [TRCs]) include the lip, tongue, oral cavity, pharynx (including oropharynx and hypopharynx), oesophagus, larynx, lungs and urinary bladder.

The total proportion of these sites of cancer relative to all sites in males and females is given in Table 30. In males, this proportion varies from 36.1% in Bangalore to 54.6% in Bhopal, whereas in females, Bangalore and Mumbai have the highest proportion of 16.2% and 16.3%, respectively.

Bibliography

1. WHO Oral Health Country/Area Profile Programme. WHO Collaborating Centre, Malmo University, Sweden; 1992.
2. National Cancer Registry Programme, Indian Council of Medical Research. Biennial report 1988–89, Consolidated report of PBCR 1990–96, 1997–98, HBCR 1984–93 and 1994–98. New Delhi: Indian Council of Medical Research.
4. Damle SG. *Pediatric dentistry*. 1st ed. New Delhi: Arya (Medi) Publishing House; 2000.
5. John J. *Textbook of preventive and community dentistry*. New Delhi: CBS Publishers and Distributors; 2003.
6. Singh G (ed). *Textbook of orthodontics*. New Delhi: Jaypee Brothers.
7. Susheela AK. *Treatise on fluoride. Task force on safe drinking water*. Government of India, New Delhi, 2003.

Table 30. Number and relative proportion (%) of specific sites of cancer related to the use of tobacco relative to all sites of cancer

	Bangalore		Barshi		Bhopal		Chennai		Delhi		Mumbai	
Site of cancer	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Males												
Lip	5	0.16	1	0.52	4	0.46	10	0.27	24	0.27	24	0.28
Tongue	106	3.44	5	2.59	70	8.04	192	5.26	384	4.26	417	4.84
Oral cavity	101	3.28	13	0.52	82	9.41	190	5.20	333	3.69	460	5.34
Oropharynx	75	2.43	1	10.36	17	1.95	80	2.19	212	2.35	164	1.90
Hypopharynx	171	5.55	20	0.00	50	5.74	167	4.57	193	2.14	362	4.20
Pharynx, etc.	34	1.10	0	8.81	2	0.23	18	0.49	37	0.41	92	1.07
Oesophagus	221	7.17	17	2.59	70	8.04	295	8.08	393	4.36	564	6.55
Larynx	111	3.60	5	3.63	44	5.05	165	4.52	575	6.37	492	5.71
Lung	218	7.08	7	2.59	104	11.94	370	10.13	897	9.94	783	9.09
Urinary bladder	70	2.27	5	6.74	33	3.79	84	2.30	382	4.23	277	3.21
TRC	1112	36.09	162	38.34	476	54.65	1571	43.02	3430	38.01	3635	42.18
All sites	3081	100.0	193	100.0	871	100.0	3652	100.0	9023	100.0	8617	100.0
Females												
Lip	4	0.11	1	0.47	1	0.13	7	0.17	8	0.09	11	0.13
Tongue	33	0.93	2	0.95	14	1.80	52	1.29	116	1.32	166	1.95
Oral cavity	197	5.54	7	3.32	47	6.04	166	4.12	140	1.59	279	3.28
Oropharynx	13	0.37	0	0.00	1	0.13	16	0.40	46	0.52	27	0.32
Hypopharynx	44	1.24	1	0.47	4	0.51	59	1.47	31	0.35	81	0.95
Pharynx, etc.	9	0.25	0	0.00	0	0.00	3	0.07	10	0.11	29	0.34
Oesophagus	186	5.23	10	4.74	28	3.60	195	4.84	194	2.20	367	4.32
Larynx	13	0.37	2	0.95	5	0.64	20	0.50	75	0.85	75	0.88
Lung	60	1.69	4	1.90	15	1.93	73	1.81	172	1.95	267	3.14
Urinary bladder	21	0.59	2	0.95	5	0.64	37	0.92	93	1.06	72	0.85
TRC	580	16.32	29	13.74	120	15.42	628	15.60	885	10.05	1374	16.16
All sites	3554	100.0	211	100.0	778	100.0	4026	100.0	8805	100.0	8504	100.0

TRC: tobacco-related cancer

Source: National Cancer Registry Programme. *Two-year report of the population-based cancer registries 1997–1998*. New Delhi: Indian Council of Medical Research; 2002

Appendix 1

Baseline and projected scenario for dental health in India, 2000–2015

Based on the prevalence data compiled in this paper, the table below assesses the trends of different oral and dental diseases and gives projection for the next 10 years.

Categories	Prevalence (%)	Age group (years)	Prevalence (in lakh) 2000	2005	2010	2015
Dental caries	50.00	All	5084.7	5484.6	5869.0	6231.8
Periodontal diseases (relatively severe)	45.00	15+	2957.6	3190.2	3413.8	3624.8
Malocclusion	32.50	9–14	401.4	433.0	463.3	491.9
Oral cancer	0.03	35+	NA	0.6	NA	0.8
Fluorosis	5.50	All	559.3	603.3	645.6	685.5
Severe fluorosis	1.0	All	101.7	109.7	117.4	124.6

Note: It is assumed that the prevalence rate will remain unchanged over the period of projections, except for oral cancer and peridontal diseases, due to the rampant use of *paan masala* and *gutka* by persons of all age groups and both the sexes. If minor periodontal diseases are included, the proportion of population above the age of 15 years with this disease could be 80%–90%. The projections may best be viewed as upper bound except for severe periodontal diseases and oral cancers, which are lower bound.
Source: Shah 2004a and 2004b

Acknowledgement

The author gratefully acknowledges the AIIMS authority for allowing to undertake the study. The author also wishes to put on record the contributions made by Dr Ajay Mahal for preparation of the projections table given in the Appendix and Dr Puneet Batra for collection of data during the initial stages.