Oral health of children with congenital cardiac diseases: a controlled study

SCIENTIFIC ARTICLES

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Abstract

Congenital cardiac disease (CCD) is one of the most common developmental anomalies in children. Affected children require special care in dentistry because of their susceptibility to infective endocarditis from oral infections, yet little information is available on the oral health of children with CCD. The present study, which investigated 39 children with CCD and 33 healthy control siblings, showed that CCD children generally suffered poorer oral health. In patients with primary dentitions, 52% of CCD children had enamel hypoplasia, compared with only 23% in the control group. In addition, CCD children had significantly more teeth with untreated dental decay (mean dmft 4.2 vs. 2.3), and more endodontically treated teeth. Children with CCD also had less than optimal professional and home dental care. Only 31% had professional advice regarding increased preventive dental health behavior, and only 15% used fluoride supplements, although the children resided in a nonfluoridated area. Furthermore, significantly fewer CCD children had parental help with tooth brushing compared to control children. This study shows that children with CCD should be targeted for vigorous preventive dental care. (Pediatr Dent 14:224–30, 1992)

Introduction

Congenital cardiac disease (CCD), one of the most common developmental anomalies in children, occurs in approximately 8–10 of 1000 live births.[1, 2] With better medical care in recent years, most of these children survive well into adulthood. The dental management of children with CCD needs special consideration for several reasons. First, these children are predisposed to develop infective endocarditis from bacteremia induced by dental procedures[3, 4] as well as from chronic poor oral health.[5, 6] Second, severely affected children may have reduced tolerance to the stress induced by dental treatment.[7] Third, complications of CCD such as hematological, respiratory and immunological problems, and drug interaction with chronic medications, must all be considered in planning dental treatment for children with CCD.[8, 9]

In spite of the importance of CCD to dental treatment and the significant numbers of children affected, there is little information available regarding their dental health and treatment requirements. Only one published survey of children with CCD[10] and one recent case report[7] have addressed this problem.

In addition, few studies have addressed the problems of dental management of children with CCD. Although recommendations of prophylactic antibiotic cover for dental treatment of CCD patients have been issued by health authorities in many countries, dental and medical practitioners may differ in their practices of these regimens or may not use prophylactic antibiotics at all.[11–14] Furthermore, many types of dental treatment, such as endodontic treatment for primary teeth, may be controversial for patients with CCD. This is due to the increased risk for infective endocarditis from residual infection in primary tooth roots, which are difficult to debride thoroughly.[7, 15]

Therefore, we examined a group of children with CCD to determine their oral health status, preventive dental health behavior, and the nature of previous dental treatment. Results of this study should be useful for determining risks of dental disease and treatment of children with CCD, as well as for providing guidelines to dental practitioners for their dental management.

Patients and Methods

Patients With Congenital Cardiac Disease

The majority of patients with congenital cardiac disease were children attending the pediatric cardiology clinic at The Prince Charles' Hospital in Brisbane — the major center for treatment of cardiac disorders in the state of Queensland. All pediatric patients attending the clinic residing in and around Brisbane were referred for dental assessment by a cardiologist (DR) who had no knowledge of their oral condition. Thirty-nine of 42 patients' (93%) parents consented to the study. The subjects were a representative sample of the general population of patients with CCD in terms of their cardiac defects.

Control Patients

Healthy siblings of the patients served as control patients. Wherever possible, the siblings selected for comparison were closest in age and matched for gender to the patients with CCD.

Dental and Medical Histories

Oral assessments were performed at the University of Queensland Dental School. The parents and patients were interviewed to obtain medical and dental histories, including information on preventive dental health behavior and fluoride intake as well as prophylactic antibiotic cover for dental treatment. Further details of medical conditions were supplied by the cardiologist after the dental assessment. The dental and medical histories were recorded in comprehensive examination forms.

Diet Histories

A three-day diet history chart was supplied to each patient to fill in for three typical days of the week, including one day of the weekend.

The dietary chart of each patient was analyzed with respect to the mean number of daily sugar frequencies.

Oral Examination

All oral examinations were performed by one author (KBH) who did not have any knowledge of the child's cardiac condition before the dental examination.

The oral soft tissues were examined using a mouth mirror. The periodontal tissues were not probed at the first examination, because bacteremia could have resulted from this procedure.[4]

The teeth were dried and examined using mirror and probe for discolorations, developmental abnormalities (including enamel hypoplasia) and dental caries. Standard WHO Criteria[16] were used for recording dental caries, and the FDI Index of Developmental Defects of Enamel (DDE Index) was used for recording enamel defects.[17]

Briefly, developmental enamel defects were classified as either loss of surface enamel (hypoplasia) or change in enamel translucency (opacity).

The oral hygiene status of the patient was assessed using the modified plaque index of Löe and Silness.[18] In this method, plaque was denoted as either present (score of 1) or absent (score of 0) on each of six key teeth

$$\frac{51/11,\ 64/24,\ 65/26}{71/31,\ 44/84,\ 85/46}.$$

The plaque index was derived as

$$\frac{\text{total plaque score}}{\text{total number of surfaces examined.}}$$

In the permanent dentition, occlusion was assessed using Angle's classification of molar and canine relationships. In the primary dentition, occlusion was assessed using the relationships of the molars as originally suggested by Baume.[19, 20] Thus, the mesial step occlusion was considered normal occlusion and the distal step as mesiocclusion. The flush terminal plane also was considered normal unless the canine relationship was abnormal.

Bite-wing radiographs and orthopantographs were exposed. Teeth congenitally absent and those extracted for caries were noted. Teeth showing pulpal pathology, as well as endodontic treatment, were recorded. Pulp therapy was classified according to radiographic localization of pulp dressings. Thus, a pulp capping refers to a superficial dressing of the pulp, and pulpotomy to amputation of the entire coronal pulp, with dressings localized to the entry of root canals. Proximal caries was detected from bite-wing radiographs.

Statistical Analysis

Student's *t*-test, or Chi-square test, when appropriate, was used for statistical analysis of the data.

Results

Table 1 (next page) shows details of the study patients. Altogether, 39 patients (19 males, 20 females; mean age 7.5 ± 4.0 years, range 2.8–15.0 years) with congenital cardiac disorders and 33 control siblings (22 males, 11 females; mean age 8.6 ± 3.9 years, range 1.0–15.0 years) were available for study. As shown in Table 1, there were no significant differences between the patients with cardiac disorders and the control patients with regard to age, gender, birth weight, and gestational age.

Plaque Indices

Although there was a higher mean plaque score in the CCD group compared with controls (0.65 ± 0.22 vs. 0.58 ± 0.21), the difference was not statistically significant ($P > 0.1$, Table 1).

Dental Caries

Table 1 shows that no significant differences were found in the percentages of caries-free children between the CCD and control groups (30.7% vs. 36.4%, $P > 0.1$).

Table 1 also shows that the mean number of decayed, missing, and filled teeth in the primary dentition (dmft) was significantly higher in the CCD group compared with the control group (4.2 vs. 2.3, $P < 0.01$). This difference is related chiefly to significant differences in the numbers of teeth with untreated decay in the two groups — 79 (15%) in the patients with CCD, compared with 41 (10%) in controls, $P < 0.01$. By contrast, in the permanent dentition, no significant differences were observed in the mean DMFT between the CCD and control groups (Table 1).

Table 1. Demographic data and dental health of cardiac and control patients

Group	Number of patients CCD N = 39	Control N = 33	P value
Age (years)			
(Mean ± SD)	7.5 ± 4.0	8.6 ± 3.9	NS
Range (years)	2.8 – 15.0	1.0 – 15.0	NS
Gender			
Males	19 (49%)	22 (67%)	NS
Females	20 (51%)	11 (33%)	
Birth weight (grams)			
(Mean ± SD)	3155 ± 686	3165 ± 579	NS
Gestational age (weeks)			
(Mean ± SD)	39.4 ± 2.2	39.5 ± 2.2	NS
Plaque index	0.65 ± 0.22	0.58 ± 0.21	NS
Dental caries			
dmft (primary teeth, N = 28)	4.2	2.3	< 0.001
DMFT (permanent teeth, N = 11)	0.9	0.6	NS
No. of caries-free children (%)	12 (30.7)	12 (36.4)	NS
Developmental enamel defects (primary teeth)	16 (52)	7 (23)	< 0.01
Developmental enamel defects (permanent teeth)	8 (38)	7 (28)	NS
Occlusion			
Normal	26 (67)	31 (94)	< 0.001
Mesiocclusion	11 (28)	2 (6)	< 0.001
Distocclusion	2 (5)	0 (0)	NS
Crowding	12 (31)	2 (6)	< 0.001

NS = Not significant.

Developmental Enamel Defects

As shown in Table 1, in the overall CCD group, there were 16 (52%) patients with at least one developmental enamel defect of the primary teeth, compared with only seven patients (21%) in the control group ($P < 0.01$). In contrast, in the permanent dentition, no significant difference in the prevalence of enamel defects was observed between the two groups.

Malocclusion

Thirty-one (94%) of the control patients had normal occlusion, and two (6%) had mesiocclusion (Table 1). By contrast, only 26 (67%) of the overall CCD group had normal occlusion, 11 (28%) had mesiocclusion, and two (5%) had distocclusion. These differences in the prevalence of the three types of malocclusions are statistically significant ($P < 0.01$). Furthermore, there also was a higher prevalence of crowding in the total CCD group, compared with the control group (31 vs. 6% $P < 0.001$, Table 1).

Types of Endodontic Therapy Provided

In the CCD group, there were eight patients (21%) with endodontically treated teeth, compared with three (9%) in the control group ($P < 0.01$), although the total numbers of teeth involved in both groups were small.

Preventive Dental Health Behavior

As this study was done in Brisbane which does not have fluoridated water, it was pertinent to examine fluoride supplementation in the patients. Although the CCD group showed a lower percentage of children currently using fluoride supplements compared with control children (as shown in Table 2, next page), the differences were not statistically significant (15 vs. 21%, $P > 0.1$). Parental attitudes toward fluoride also were fairly similar in the two groups (Table 2).

With regard to oral hygiene, no significant differences in tooth brushing and flossing habits were observed between the CCD and control groups (Table 2). However, only 41% of children with CCD received parental help with tooth brushing, compared with 70% of control children ($P < 0.001$).

Dental Prevention Provided by Professionals

Thirty-one per cent of children with CCD reported that they had been given professional advice regarding the need for improved preventive dental health behavior due to the risk of infective endocarditis from poor dental health. Paradoxically, a smaller percentage received professionally applied topical fluoride therapy compared with control children (36 vs. 48%), although the difference does not appear statistically significant. There were significantly more CCD patients who had received fissure sealants compared with control children (21 vs. 6%, $P < 0.001$, Table 2). Similarly, no differences in recall frequencies were observed in the two groups, although children with CCD tended to have recall frequencies of six months or less (Table 2).

Daily Dietary Sugar Frequencies

Comparison of mean daily sugar frequencies in the diet of CCD and control patients revealed no significant differences (Table 2).

Chronic Medication Intake

Many CCD patients received sweetened medications several times daily, including digoxin syrup containing 30% sucrose, and chlorthiazide and spironolactone containing approximately 20% sucrose. It was therefore of interest to compare the numbers of caries-free children in the groups of children with CCD with and without medication intake, to determine if chronic sweetened medication is associated with increased caries risk. The mean age ± SD, and age ranges of these children were found to be comparable, hence it was pertinent to compare the caries status of these two groups. As shown in Table 3 (next page), in the group of CCD children without medication intake, 38.1% were caries free, compared with only 11.1% in the group who were on chronic medication intake ($P < 0.01$).

Table 2. Preventive dental health behavior of CCD and control patients

	Number of Patients (Percentage of Group)		
	Total CCD N = 39	*Total Control* N = 33	*P value*
Fluoride supplements			
Current	6 (15)	7 (21)	NS
Previous exposure	16 (41)	13 (39)	NS
Topical F therapy	14 (36)	16 (48)	NS
Parental attitude to fluoride			
For	34 (87)	28 (85)	NS
Against	5 (13)	5 (15)	NS
Oral hygiene			
Tooth brushing frequency			
1/day	11 (28)	11 (33)	NS
2/day	26 (67)	21 (64)	NS
> 2/day	2 (5)	1 (3)	NS
Flossing			
No	35 (90)	31 (94)	NS
Yes	4 (10)	2 (6)	NS
Parental help			
No	23 (59)	10 (30)	< 0.001
Yes	16 (41)	23 (70)	< 0.001
Mean no. of daily sugar exposures	3.2	2.9	NS
Professional dental prevention			
Fissure sealants	8 (21)	2 (6)	< 0.001
Recall frequency			
6 months	20 (52)	13 (39)	NS
7–12 months	19 (48)	20 (60)	NS

NS = Not significant.

Types of Antibiotic Prophylaxis Provided to CCD Patients for Dental Treatment

As shown in Table 4 (next page), 15 (38%) patients with CCD had not had previous dental treatment, while five (13%) were not prescribed antibiotics for their dental treatment. The most common antibiotic used was amoxicillin (70%), followed by penicillin V (20%). One patient was prescribed a combination of penicillin V and amoxicillin (10%). The most common route was oral (89%), and the majority of patients (68%) were prescribed the antibiotics in doses recommended by the Australian Dental Association (ADA).[21] Five patients (26%) were given doses of antibiotics above those recommended by the ADA. Seven of the patients (37%) were given the prophylactic antibiotics 1 hr before dental treatment, while three (16%) took it more than a day before their dental treatment.

Discussion

In spite of the significance of CCD in dental management, little previous research has been conducted on the oral health of affected individuals. The present study has shown that children with CCD suffer poorer oral health compared with control siblings. This is particularly evident in the primary dentitions where there were significantly greater numbers of teeth with untreated dental decay. In addition, twice as many children with CCD had developmental enamel defects, compared with controls. Furthermore, CCD children suffered a significantly higher prevalence of mesiocclusion and crowding, compared with unaffected children. In the permanent dentition, these differences are less pronounced, most likely because the number of children in this study with permanent teeth is low.

The results of our study extend those of the study of Berger[10] of more than a decade ago, which also reported that children with CCD had higher levels of dental caries, enamel hypoplasia, and periodontal disease, compared with control children. The presence of enamel hypoplasia and dental neglect also has been shown in a recent case report of a young child with tetralogy of Fallot,[7] and the high prevalence of facial dysmorphism in these patients has been confirmed in a recent study.[22]

Table 3. Number of caries-free children in CCD patients with and without chronic medication intake

	Number of CCD patients	*Mean ± SD*	*Number of caries-free children*
No medication intake	21	7.6 ± 4.0 (range 2.9 – 15.1)	8 (38.1%)
Chronic medication intake (Digoxin 2x/day or digoxin 2x/day plus diuretic 1/day)	18	6.5 ± 3.0 (range 2.9 – 15)	2 (11.1%)

• $P < 0.01$.

The two groups of children are comparable in the mean ages and range of ages.

The poor oral health status of children with CCD may be due to many factors. Increased prevalence of developmental oral defects, such as enamel hypoplasia and malocclusion, is likely to be related to the environmental and hereditary factors associated with the etiology of congenital heart defects.[23–25] It is also possible that enamel hypoplasia in CCD children has resulted from systemic disturbances, such as cardiac failure and surgical complications associated with the cardiac disease during prenatal and neonatal development.[26–27]

Table 4. Types of antibiotic prophylaxis regimens provided to CCD patients for dental treatment

	Number of Patients (Percentage of Group) Total CCD N = 39
No previous dental treatment	15 (38)
No prophylactic antibiotics prescribed for dental treatment	5 (13)
Prophylactic antibiotics prescribed for dental treatment	19 (49)
Antibiotics prescribed	
Amoxicillin	14 (73)
Penicillin V	4 (21)
Combination of Penicillin V and Amoxicillin	1 (5)
Route of administration	
Oral	17 (89)
Parenteral	2 (5)
Dose prescribed	
Australian Dental Association recommendation	13 (68)
< Australian Dental Association recommendation	5 (26)
Unknown	2 (5)
Preoperative time given before treatment	
1 hour before	7 (37)
1 day before	3 (16)
> 1 day before	5 (26)
Unknown	4 (21)
Continuation of antibiotic after treatment	
No continuation	4 (21)
< 3 days after initial dose	9 (47)
> 3 days after initial dose	3 (16)
Unknown	3 (16)

Increased prevalence of dental caries in children with CCD probably results from several predisposing factors, including increased tooth susceptibility from developmental enamel defects. We also have shown that the chronic intake of sweetened medications is associated significantly with increased caries in CCD patients, thus confirming and extending the findings of previous investigators.[28, 29] Although it also is possible that the patients with CCD were indulged more frequently in sweet snack foods due to their chronic illness, analysis of food items from diet histories did not reveal this.

The low levels of oral health in children with CCD compared to controls may have significant implications in the medical care of these patients. Untreated dental decay may develop quickly into pulp infections which are associated with bacteremia, endocarditis, and even brain abscesses in children with

CCD.[3–6, 30] In this study, 21% of children with CCD had untreated pulp pathoses and pulp therapies, indicating that in these children, there are significant possibilities of oral foci of infection which have potential to cause these complications.

The present study has shown that the poor oral health of the children with CCD probably has resulted from inadequate professional and home care. Only 31% of the total group of CCD children indicated that they had been given professional advice regarding the need for increased dental prevention because of their medical condition. In other aspects of preventive dental care, such as topical fluoride therapy and fluoride supplementation, the CCD patients were not treated differently from control children. The overall low levels of fluoride supplement intake in both groups of children reflected previous observations in Brisbane, a town with no fluoridation.[31] Furthermore, significantly fewer CCD children were given parental help during tooth brushing, compared with control children. The reason for this may be that preoccupation with other demanding aspects of health care in these children has led to parents neglecting tooth cleaning.

The present study also has shed light on the antibiotic prophylaxis regimens employed by dentists for preventing infective endocarditis. It is disturbing to note that 13% of CCD patients did not receive prophylactic antibiotics before dental treatment. Although most of those who received prophylaxis were prescribed antibiotics currently recommended by the Australian Dental Association[18] in the correct doses, more than 42% of the CCD group started as early as one day or more before dental treatment. This practice is contradictory to the recommendations of most health authorities[32] which state that the prophylactic antibiotics need to be given only about 1 hr before dental treatment.

In conclusion, this study has shown that children with congenital cardiac disease suffer poor oral health, compared with control children. This is most likely related to abnormal dental formation associated with the cardiac condition, and to dental neglect. Furthermore, a significant number of these children did not receive optimal professional dental care, and only a few were told about the increased need for dental prevention. We hope that the present study has highlighted the oral problems encountered in children with congenital cardiac disease and emphasized the need for vigorous preventive dental care in these special patients.

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1. Nadas AS: Update on congenital heart disease. Pediatr Clin North Am 31:153–64, 1984.
2. Radford DJ: Congenital Heart Disease, in Textbook of Paediatric Practice, YH Thong ed. Sydney: Butterworths, 1989, pp 567–78.
3. Munroe CO, Lazarus TL: Predisposing conditions of infective endocarditis. J Can Dent Assoc 10:483–89, 1976.
4. Cawson RA: Infective endocarditis as a complication of dental treatment. Br Dent J 151:409–14, 1981.
5. Bayliss R, Clarke C, Oakley C, Somerville W, Whitfield AGW: The teeth and infective endocarditis. Br Heart J 50:506–12, 1983.
6. Walker MP: Infective endocarditis. Dental implications, prevention, and prophylaxis failure. Clin Prev Dent 6:17–19, 1984.
7. Rockman RA: Tetralogy of Fallot: characteristics, dental implications and case study. ASDC J Dent Child 56:147–50, 1989.
8. Hunter K, Mac D: Dental aspects of cardiac abnormality. N Z Dent J 70:6–14, 1974.
9. Elliott RH: A dental service for children suffering from cardiac disease. Br Dent J 145:179–80, 1975.
10. Berger EN: Attitudes and preventive dental health behavior in children with congenital cardiac disease. Aust Dent J 23:87–90, 1978.
11. Sadowsky D, Kunzel C: "Usual and customary" practice versus the recommendations of experts: clinician non-compliance in the prevention of bacteria endocarditis. J Am Dent Assoc 118:175–80, 1989.
12. Sadowsky D, Kunzel C: Clinician compliance and the prevention of bacterial endocarditis. J Am Dent Assoc 109:425–28, 1984.
13. Nelson CL, Van Blaricum CC: Physician and dentist compliance with American Heart Association guidelines for prevention of bacterial endocarditis. J Am Dent Assoc 1188:169–73, 1989.
14. Fekete T: Controversies in the prevention of infective endocarditis related to dental procedures. Dent Clin North Am 34:79–90, 1990.
15. Valachovic R, Hargreaves JA: Dental implications of brain abscess in children with congenital heart disease. Oral Surg 48:495–99, 1979.
16. World Health Organization: Oral Health Surveys, 3rd ed, 1987, pp 34–39.
17. Commission on Oral Health, Research and Epidemiology. FDI: An epidemiological index of developmental defects of dental enamel (DDE Index). Int Dent J 32:159–67, 1982.
18. Löe H, Silness P: Periodontal disease in pregnancy. Prevalence and severity. Acta Odont Scand 21:533–38, 1963.
19. Baume LJ: Developmental and diagnostic aspects of the primary dentition. Int Dent J 9:349–58, 1958.
20. Casamassimo P, Christensen J, Fields H: Examination, diagnosis and treatment planning, in Pediatric Dentistry, Infancy Through Adolescence, JR Pinkham ed. Philadelphia: WB Saunders Co, 1988, pp 201–22.
21. Australian Dental Association Inc: Prevention of infective endocarditis associated with dental treatment and disease. Practical guides for successful dentistry, 3rd ed, 1989, pp 1–6.
22. Bell RA, Arensman FW, Flannery DB, Ussery TW, Moss RB: Facial dysmosphologic and skeletal cephalometric findings associated with conotruncal cardiac anomalies. Pediatr Dent 12:152–56, 1990.
23. Radford DJ, Thong YH: Facial and immunological abnormalities associated with tetralogy of Fallot. Int J Cardiol 22:229–36, 1989.
24. Radford DJ, Thong H, Beard LJ, Ferrante A: Immunoglobulin IgG subclass deficiencies in children with congenital heart disease. Pediatr Allergy Immunol 1:41–45, 1990.
25. Jensen S, Jacobsen P, Rotre L, Erk C, Illum F: Oral findings in DiGeorge syndrome. Int J Oral Surg 12:250–54, 1983.

26. Seow WK, Humphrys C, Tudehope DI: Increased prevalence of developmental dental defects in low-birth-weight children: a controlled study. Pediatr Dent 9:221–25, 1987.
27. Seow WK, Masel JP, Weir C, Tudehope DI: Mineral deficiency in the pathogenesis of enamel hypoplasia in prematurely born, very low birthweight children. Pediatr Dent 11:297–302, 1989.
28. Feigal RJ, Gleeson MC, Beckman TM, Greenwood ME: Dental caries related to liquid medication intake in young cardiac patients. ASDC J Dent Child 51:360–62, 1984.
29. Roberts GJ, Roberts IF: Dental disease in chronically sick children. J Dent Child 48:346–51, 1981.
30. Lampe RM, Cheldelin LV, Brown J: Brain abscess following dental extraction in a child with cyanotic congenital heart disease. Pediatrics 61:659–60, 1978.
31. Seow WK, Humphrys C, Powell RN: The use of fluoride supplements in a nonfluoridated city in Australia in 1985. Com Dent Health 4:86–94, 1987.
32. Schulman ST, Amren DP, Bisno AL, Dajani AS, Durack DT, Gerber MA, Kaplan EL, Millard HD, Sanders WE, Schwartz RH, Watanakunakorn C: Prevention of bacterial endocarditis. Circulation 70:1123a–1127a, 1984.

Errata

The March/April 1992 issue of *Pediatric Dentistry* (Volume 14, Number 2) contained two errors in its table of contents.

Anatomy of primary incisor and molar root canals, by Fouad S. Salama, Ronald W. Anderson, Carole McKnight-Hanes, James T. Barenie, and David R. Myers was omitted inadvertently from the table of contents. Their article appeared on pages 117–118.

Enamel defects of the primary dentition and osteopenia of prematurity, by Bernadette K. Drummond, Stephen Ryan, Elizabeth A. O'Sullivan, Peter Congdon, and Martin E.J. Curzon began on page 119 instead of 120.

We apologize to the authors, and regret any inconvenience these errors may have caused. Both articles will be listed correctly in the annual index.