Scientific Article

Dental disease, caries related microflora and salivary IgA of children with severe congenital cardiac disease: an epidemiological and oral microbial survey

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Abstract

The objectives were to determine levels of dental caries, plaque accumulation, gingival inflammation, knowledge of dental health practices, and oral bacterial loading of S. mutans, Lactobacillus sp., Candida sp., and salivary IgA in the mouths of children afflicted with severe congenital heart disease. A total of 60 children from the cardiac units of the Hospital for Sick Children and Guys Hospital Paediatric Department were compared with 60 case-matched control children from the Department of Orthodontics and Paediatric Dentistry, UMDS (Guys Dental School), London. Using the methodology of the World Health Organization, the decayed, missing and filled surfaces and teeth of primary (dmft) and permanent (DMFT) were compared. There were similar levels of caries in the cardiac (dmft 3.9 and DMFT 2.7) and the control (dmft 3.7 and DMFT 2.0). A significant difference was the proportion of untreated carious lesions in the cardiac group (52%) compared to the control group (32%; $P < 0.001$). Standard oral microbiological techniques were used to isolate S. mutans, Lactobacillus sp., Candida sp., and conventional methods for estimating salivary IgA. There was no difference between the cardiac and the control group.

Children with severe congenital cardiac disease have moderately high levels of dental caries with a significantly greater amount of untreated disease. The high bacterial loading associated with high levels of bacterial dental plaque and gingivitis may put cardiac patients at unnecessary risk of developing bacterial endocarditis. (Pediatr Dent 18:228–35, 1996)

Dental care of the chronically sick child has been neglected.[1] A preoccupation with the principal medical condition often results in neglect of other facets of the child's health.[2] Such children often present with a dental emergency that—because of the associated medical problems—can be difficult to manage both medically and dentally.[2] Cardiovascular disease in children complicates dental care for three reasons: the risk of a dental bacteremia that may lead to infective endocarditis, the increased risks associated with general anesthesia in a child who has usually had several surgical procedures, and the risk of prolonged bleeding in children taking warfarin.[3] In addition there is the additional morbidity, often considerable, associated with pain and infection in the mouths of children afflicted with recurrent problems associated with their underlying cardiac disorder.

A detailed knowledge of the dental and oral condition of such children is essential if preventive care is to be directed effectively.[4]

There are no data available on the levels of dental disease in children with serious cardiac disorders although two recent studies provide extensive data on children with heart defects.[5,6] The Great Ormond Street Hospital for Children in London is a tertiary referral center receiving patients from all over the United Kingdom, Europe, and the Middle East. The Paediatric Cardiac Unit at Guys Hospital London, is a regional center receiving patients from district general hospitals for specialist pediatric cardiac care. These two hospitals provide treatment for only the most severe cases of congenital heart disease (such patients are usually untreatable in other centers).

The purpose of our study was to investigate the dental and gingival health of these children with severe congenital cardiac defects, the health awareness of the parents, the dental health practices of the children in the study, and the presence and distribution of caries related micro-organisms and salivary IgA.[7–9]

Patients and methods

Sample and examination

Children aged 2 to 16 years with severe congenital cardiac disease attending The Great Ormond Street Hospital for Children (GOS) or Guys Hospital (Guys), both large hospitals in London, UK, formed the study

group. The severe nature of the congenital disease can be judged from the list of conditions that usually required surgical treatment (Table 1). The control group comprised siblings and other children attending for routine recall at the Children's Department of Guys Dental Hospital. Each member of the study group was matched with a patient in the control group by age, gender, ethnicity and social class.[10]

TABLE 1. MAJOR DIAGNOSTIC CATEGORIES FOR CARDIAC PATIENTS

Diagnosis	*Number of Cases*
Transposition of the great arteries	4
Aortic stenosis	3
Aortic stenosis, Williams syndrome	1
Aortic stenosis, coarctation of the aorta	3
Vascular heart and lung disease	1
Pulmonary stenosis	4
Pulmonary stenosis, ventriculoseptal defect	2
Tetralogy of Fallot	4
Tetralogy of Fallot, ventriculo- & atrioseptal defects	2
Congenital valvular defect	1
Atrial septal defect	6
Ventriculoseptal defect	2
Coarctation of the aorta	2
Coarctation of the aorta, atrioseptal defect	2
Coarctation of the aorta, ventriculoseptal defect	2
Ventriculoseptal defect, pansystolic heart murmur	1
Aortic and pulmonary stenosis	1
Pulmonary atresia	6
Pulmonary atresia, valve replacement	1
Pulmonary atresia, multiple ventriculoseptal defects	3
Pulmonary atresia, atrioseptal defect	1
Pulmonary atresia, ventriculo- & atrioseptal defects	2
UHLS syndrome, Ebstein's anomaly	1
Severe conduit obstruction	1
Atrioseptal and ventriculoseptal defects	3
Marfans syndrome, large atrioseptal defect	1
Total	60

All patients in the control group were healthy children.

The study children were examined on the cardiac ward when admitted for investigation or surgery and the control children examined in a dental surgery. In both groups the teeth were examined visually with a fiber-optic light. A sickle-shaped-ball-ended probe was used to remove any gross debris from the teeth. The teeth were not further cleaned or dried prior to the examination. The methods for clinical examination were those of the World Health Organization's guidelines for basic oral health surveys.[11] The methods of the British Association of Community Dentistry are similar.[12] Each surface was charted for caries and developmental defects of enamel.[13] Plaque deposits, gingival inflammation, and gingival bleeding were assessed using a simplification of the index of O'Leary by estimating plaque as present or absent on the mesiobuccal, distobuccal, distolingual, and mesiolingual quadrisections of each tooth surface.[14] This method was used because it provides a better estimate of oral bacterial loading than other methods since it uses data from every tooth surface adjacent to the gingival margin. No radiographs were exposed, as this was not possible on the cardiac wards. At the same time as the dental examination, the parent completed a questionnaire to allow assessment of parental knowledge and current practice of dental health procedures, a diet history detailing frequency of consumption of food and drink, and also a three-day diet diary to be completed at home and returned in a stamped addressed envelope.

Microbiology

Collection of bacteriological samples

A sample of plaque was obtained from each patient by gently flossing between the molar teeth in the primary and/or secondary dentitions in all four quadrants (until a visible amount of plaque was obtained). When this was not possible, plaque was obtained with a small ball-ended probe from as close to the approximal region as possible. The sample was placed in 1 ml of reduced transport fluid (RTF). Three milliliters of unstimulated saliva was collected by asking the child to spit gently into a universal tube. For very young children, or children unable to spit due to postoperative weakness, saliva was collected using a syringe without a needle.

The plaque/RTF and saliva samples were diluted to 10^{-1}, 10^{-2}, and 10^{-3} and plated onto selective media within 4 hr of collection.[15] Three types of agar plates were used and were prepared on a weekly basis. These were Rogosa agar (Oxoid Unipath, Oxford, UK); Sabouraud's dextrose agar (Oxoid Unipath, Oxford, UK); and TYC Agar (LabM, Amersham, UK) with 20% w/v sucrose added and 0.1 units/ml of Bacitracin™. Control organisms from the Guy's Dental School reference set were similarly plated out, and processed with every batch of samples for visual comparison. Reproducibility was examined by replating 10% of plaque and saliva samples from both cardiac and control patients.

The TYCSB and control plates were incubated anaerobically in an atmosphere of 10% CO_2 in H_2 at 37°C for 5 days. The Rogosa and control plates were incubated in 5–10% CO_2 in air. The Sabouraud's dextrose and control plates were incubated aerobically at 37°C for 2 days. Cultures from the plaque and saliva samples were compared visually with those from the control organisms. The total number of colony forming units in saliva were calculated as cfu/ml of saliva. The counts for plaque refer to the 1 ml of RTF because the original volume of plaque could not be reliably measured.

Mutans streptococci

All colonies were gram stained and examined under the light microscope to verify the presence of chains of gram-positive cocci. Presumptive *mutans Streptococcus* colonies were selected at random and tested for purity by plating onto Columbia™ blood agar and incubating the inoculated plates anaerobically at 37°C for 24 hr. The growth on the purity plates was examined, and if deemed pure, the organisms were subjected to rapid carbohydrate fermentation activity consisting of five sugars: glucose, mannitol, sorbitol, arginine, and esculin.[16]

Lactobacillus

For the first six subjects, colonies were gram stained and examined under the light microscope to verify the presence of gram-positive rods. Thereafter, 10% were also checked in the same way.

Candida

Ten percent of colonies were gram stained and examined under the light microscope to verify the presence of gram-positive yeast cells. Fifty percent of these were further speciated by the Microring YT Identification System (Medical Wire & Equipment Co, New Jersey). This compares the susceptibility pattern of a yeast isolate to a variety of antimicrobial agents and dyes, with a table of susceptibility profiles that is used to enable identification.

Salivary IgA

Approximately 1 ml of saliva was collected in a universal tube and frozen and stored at -20°C until all samples were collected. The method of assaying the IgA was that previously used in the immunology laboratory of the dental school of Guys and St Thomas Hospital Trust.[17,18]

Salivary IgA concentrations were calculated by reference to a standard preparation of purified human colostral IgA at a starting concentration of 160 ng/ml. Standards were diluted in six doubling serial dilutions in duplicate on each microtiter plate, leaving four wells as blanks. All sample values falling within the range of the standard curve were calculated and averaged to provide a mean value for the sample in μg/ml.

Reproducibility

Cohen's kappa was used to assess the intraexaminer agreement for caries at the beginning, middle and end of the study. Duplicate examinations on 10% of the sample were carried out. It was not possible to recall patients for the re-examination and, therefore, 10% of the patients were re-examined shortly after the first examination. Ten percent of samples were plated in duplicate to enable repeatability to be assessed by the technique of Bland and Altman.[19] For the IgA analyses, 10 saliva samples taken from consecutive patients attending the children's department at Guy's were divided and each was run in duplicate.

Statistical analysis

The data for dental caries, plaque accumulation, gingival inflammation, gingival bleeding, and developmental defects of enamel and diet were processed in SPSS™ for Windows™ Release 6[20] and analyzed using the Mann Whitney two-sample statistic. The parents' attitudes and beliefs toward dental health were analyzed using the chi-square test and Fisher's exact probability test. Further exploration of the data was carried out using simple correlations between selected variables using Spearman's rank correlation. An attempt at multiple regression proved fruitless so is not reported.

Results

A total of 120 children were recruited into the study, equally divided between the control and study groups. They were evenly matched for age (Table 2), social class, gender, and ethnicity. Of the cardiac group, 48 were from GOS and 12 from Guys.

There was no significant difference between the control and cardiac groups for caries of primary teeth or caries of permanent teeth (Table 2).

The patients exhibited a full range of defects from white opacities to frank hypoplasia. There were no statistically significant differences between the control and cardiac groups although when white opacities were considered in isolation there were more in the control group than in the cardiac group.

There were no differences in the number of sites covered with plaque between the control and cardiac groups (Table 2). In both groups there was a significant difference between the primary teeth and permanent teeth, with primary teeth having almost twice as many surfaces covered with plaque ($z = 3.06$, $P = 0.0022$). This is unrelated to the number of primary or permanent teeth present in the mouth.

The control and cardiac groups had similar levels of gingival inflammation related to both primary and permanent teeth (Table 2). The gingivae related to primary teeth were significantly less inflamed than those related to permanent teeth ($z = -3.07$, $P = 0.021$).

The primary teeth had less than 6% of plaque-covered surfaces with gingival inflammation whereas permanent teeth had 38% (chi square = 75.01, df = 1, $P < 0.001$).

There was significantly more bleeding from gingivae associated with permanent teeth in the control group than in the cardiac group. In addition, gingivae associated with permanent teeth in both groups showed significantly more bleeding than gingivae associated with primary teeth ($z = -2.55$, $P = 0.0107$).

The frequency of food intake in total or as sugar alone was no different. The attempt to statistically weight the sugars consumed by giving greater importance to sticky or retentive items, including sweetened medicines, gave a value of 15.4 in the control and 18.1 in the cardiac children ($z = -1.46$, $P = 0.1447$). These data included the consumption of syrupy medicines.

The levels of treatment of primary teeth were simi-

TABLE 2. DENTAL CARIES EXPERIENCE, ORAL MICROFLORA, SALIVARY IgA AND HEALTH BEHAVIOR IN CONTROL AND CARDIAC CHILDREN

		Control Group				*Cardiac Group*			
Age (all subjects) N = 120		N = 60	8.9	3.9		N = 60	8.8	4.0	*P values* = 0.847
Caries Age Group (yrs)	*N*	*Variable*	*Mean*	*(sd)*	*N*	*Variable*	*Mean*	*(sd)*	*Significance*
2–10	39	age	6.5	2.4	40	age	6.5	2.4	= 0.829
(primary teeth)		dmft	3.9	3.2		dmft	3.7	3.2	ns
		dmfs	8.3	8.3		dmfs	7.3	9.6	ns
		plaque	30.2	20.4		plaque	28.5	21.6	ns
		gingivitis	1.8	2.5		gingivitis	3.4	9.6	ns
		bleeding	0.8	1.7		bleeding	0.4	0.8	ns
5 - 16	44	Age	10.6	2.9	45	age	10.5	3.2	ns
(permanent teeth)		DMFT	2.0	2.9		DMFT	2.7	3.4	ns
		DMFS	3.8	6.6		DMFS	3.6	4.8	ns
		Plaque	16.2	12.0		Plaque	14.9	15.8	ns
		Gingivitis	6.3	6.7		Gingivitis	4.0	1.1	ns
		Bleeding	3.2	4.3		Bleeding	0.8	2.0	< 0.005 sig
Evidence of caries i.e. d,m or f (% age)	60	83.3			60	78.3			ns
Permanent teeth with untreated caries (%age)	60	32			60	52			<0.001 sig
Frequency of sugar intake	60	number/day	4.5	3.4			5.5	4.1	= 0.2592 ns
Statistically weighted sugar intake (incl medicines)	60	statistically weighted/day	15.4		60	18.1			=0.01447ns
Oral microflora									
S. mutans	49	Saliva	269,181	630,844	48	Saliva	309,079	396,564	ns
	49	Plaque	140,610	203,478	48	Plaque	125,740	200,470	ns
Lactobacillus sp.	49	Saliva	9,263	35,521	48	Saliva	8,942	20,112	ns
	49	Plaque	3,963	15,657	48	Plaque	9,408	43,218	ns
Candida sp.	49	Saliva	53	244	47	Saliva	236	1,000	ns
	49	Plaque	94	162	47	Plaque	162	590	ns
Salivary IgA (µg)	50		268	143	50		281	124	ns
White Opacities (% age)	60	45			60	32			< 0.001 sig
General health problems a serious risk (% age)									
Contracting flu	60	15			60	50			< 0.0001
Broken arm	60	38			60	60			< 0.01
Bleeding gums	60	71			60	55			= 0.0582
Infection	60	43			60	61			< 0.05
Dental health care (% age)									
Brush twice/day	60	73			60	21			< 0.01
Fluoride beneficial	60	35			60	65			< 0.005
Never visited dentist	60	5			60	19			< 0.02

lar in both groups, being approximately 49% untreated surfaces, 29% extracted surfaces, 17% of surfaces restored, and only 5% of surfaces with recurrent caries. The permanent teeth showed different levels of treatment in the control and cardiac groups. (Table 2) These data show a significantly higher proportion of untreated surfaces in the cardiac group than in the control group.

The parents of the cardiac children were more aware of other potentially harmful health problems. They believed that contracting the flu (chi square 19.7, df = 4, $P < 0.001$), having a broken arm (chi square 10.844, df = 4, $P < 0.02$), bleeding gums (chi square 14.216, df = 4, $P < 0.001$), or an infection (chi square 9.801, df = 4, $P < 0.05$) were serious problems for children with heart disease. Both groups thought that congenital heart disease was a serious problem, although there was no difference between the groups (Fisher's exact test, $P = 0.145$, ns). The parents of both groups of children did not think that having a dental extraction was potentially serious.

The parents of both groups of children were well versed in their knowledge of good preventive practices such as the undesirability of eating sweets between meals, the beneficial effect of toothbrushing to gingival health, and the positive benefits of fluoride supplements and fissure sealants.

More control children (73%) regularly brushed their teeth twice a day, whereas 21% of the children with cardiac disorders never or hardly ever brushed twice a day (chi square 14.46, df = 4, $P < 0.01$). Compared to the control group, more of the parents of cardiac children believed fluoride tablets were beneficial (chi square 10.8, df = 4, $P > 0.005$), but 65% of them did not know if fluoride tablets were of any benefit. Eleven children in the cardiac group (19%) had never visited the dentist compared to only three (5%) in the control group (chi square 7.708, df = 2, $P < 0.02$).

The distribution in saliva and plaque of *mutans streptococci, Lactobacillus sp., Candida sp.*, is shown in Table 2. Overall, there were no significant differences between the control and cardiac children. Salivary *Streptococcus mutans* counts were higher than plaque counts for both groups. A large proportion of children had no detectable *Lactobacillus sp.* in saliva (40.6%) and plaque (68.8%). The yeasts detected were *Candida albicans.*

The levels of immunoglobulin in saliva are shown in Table 2. Apart from one subject who was a negative reactor, all subjects had IgA levels from 250 µg/ml to 802 µg/ml.

All tests on reproducibility for caries, plaque, gingivitis, microbiological assays, and salivary IgA were within acceptable limits. For example, the reproducibility of caries before, during, and after the study using Cohen's Kappa was 0.8287, 0.8284, and 0.8915 respectively, which are very high levels of agreement.

Discussion

No significant differences were established in our study between the caries scores for children with severe congenital heart disease and the closely matched healthy controls. This result is similar to the findings of two previous reports from Leeds, UK, and Brisbane, Australia.[5,6] In comparing this study carried out in London with one carried out in Leeds, England,[5] the overall figures for dmft and DMFT show higher levels of dental caries in both the primary and permanent dentitions for our study. With regard to the permanent teeth, using the principal of meta-analysis,[21] there was a statistically significant difference between the Leeds cardiac cases and the London with DMFT Leeds = 1.2, SD = 1.59, N = 34; DMFT London = 2.7, SD = 3.4, N = 45, 77df, $P < 0.0198$. The Australian data obtained in Brisbane[6]also show lower figures for the permanent dentition, but because the standard deviation is not published, it is impossible to estimate the importance of the difference using meta-analysis. The dental caries experience reported in the Great Britain surveys coordinated by the British Association of Community Dentistry[12] show lower levels of caries for both the primary dentition (GB survey dmf range 1.16–2.82, this study dmf = 3.7) and the permanent dentition (GB survey DMF range 1.12–2.1, this study, DMF = 2.7). Further comparison with data from local dental surveys show dmft values at age 5 years of 1.16 for Surrey,[22] an affluent county in southeast England, with a dmft of 3.7 for this study. The dmft levels at 5 years in Greenwich,[23] an inner city borough of London, are 2.4. It is apparent that these caries levels, which conform to our expectations of caries in prosperous and less prosperous areas in and around Lon-

TABLE 3. SPEARMAN RANK CORRELATIONS IN POOLED DATA OF CONTROL AND CARDIAC CHILDREN

Variable 1		*Variable 2*	*Correlation*	*P*
Age	v	Carious surfaces	0.2551	0.005†
Age	v	Carious teeth	0.2726	0.003†
Age	v	Plaque	-0.1732	0.059
Age	v	Gingivitis	0.4188	0.0001
Age	v	Gingival bleeding	0.3390	0.0001
Carious surfaces	v	Plaque	0.1649	0.072
Carious teeth	v	Plaque	0.2120	0.02
Carious surfaces	v	Gingivitis	0.1879	0.04
Carious teeth	v	Gingivitis	0.2488	0.006
Carious surfaces	v	Frequency sugar intake	-0.1223	0.81
Carious teeth	v	Frequency sugar intake	-0.0642	0.6
Carious surfaces	v	Plaque	0.1649	0.072
Carious teeth	v	Plaque	0.2120	0.02
Carious surfaces	v	Gingivitis	0.1879	0.04
Carious teeth	v	Gingivitis	0.2488	0.006
Carious surfaces	v	Salivary IgA	0.1963	0.05
Carious teeth	v	Salivary IgA	0.2255	0.024
Carious surfaces	v	Salivary *S. mutans*	0.1784	0.80
Carious teeth	v	Salivary *S. mutans*	0.2130	0.036
Carious surfaces	v	Plaque *S. mutans*	-0.0364	0.072
Carious teeth	v	Plaque *S. mutans*	0.0141	0.890
Carious surfaces	v	Salivary *Lactobacilli sp*	0.3163	0.002
Carious teeth	v	Salivary *Lactobacilli sp*	0.2782	0.006
Carious surfaces	v	Plaque *Lactobacilli sp*	0.1863	0.066
Carious teeth	v	Plaque *Lactobacilli sp*	0.2127	0.035
Carious surfaces	v	Salivary *Candida sp*	0.0622	0.547
Carious teeth	v	Salivary *Candida sp*	0.0954	0.355
Carious surfaces	v	Plaque *Candida sp*	0.1787	0.078
Carious teeth	v	Plaque *Candida sp*	0.2268	0.025
Plaque	v	Gingivitis	0.2199	0.016
Plaque	v	Gingival bleeding	0.0932	0.312
Gingivitis	v	Gingival bleeding	0.7312	0.0001

don, are markedly less than for the subjects in this study. Clearly there are difficulties of interpretation when comparing studies in different parts of the country and in different countries. Notwithstanding this, it is possible to cautiously conclude that the cardiac children attending GOS and Guys have higher levels of dental caries than cardiac children in Leeds and Brisbane. In addition, it appears that the cardiac children in London also have higher levels of dental caries than healthy children in the Great Britain survey,[12] and in two health districts in and near London.

The levels of developmental dental anomalies are consistent with those found in other epidemiological studies on cardiac patients and suggest that illness associated with congenital cardiac disease and associated open heart surgery are not potential factors in the etiology of developmental dental anomalies.[24]

The levels of plaque were similar in both primary and permanent dentitions, although the levels in the cardiac group are slightly lower. The possible reason for this is that the examiner knew whether a child was in the cardiac or control group and was reluctant to disturb plaque in case to avoid a bacteremia being precipitated. The levels of gingival inflammation in the cardiac group represent a significant amount of damaged and ulcerated tissue, which may allow ingress of oral bacteria leading to the cardiovascular system. This is the major justification for using the modified scoring system in our study, which provides a reliable estimate of the number of tooth surfaces in total that are covered with plaque or affected by gingivitis. This is important as it indicates the level of bacterial loading present in the mouths of children with congenital cardiac anomalies associated with the levels of diseased and ulcerated gingival tissue that directly indicates the potential for the development of odontogenic bacteremias.

Although there were no differences between control and cardiac children in our study, both groups have evidence of significant amounts of dental disease that are higher than in the population at large. In children with cardiac disorders, this indicates potential morbidity and stress while carrying out dental treatment in addition to that from the underlying heart condition. This may be a subconscious worry for the parents and could account for the significantly increased levels of untreated disease observed in the children with heart disease. The explanation may be more prosaic as parents of cardiac children delay or avoid seeking dental treatment because of their preoccupation with the underlying medical problem. Anecdotal evidence suggests that missing school or not wanting the child to go through more than is deemed absolutely necessary are factors that lead to the parents' unwitting neglect of their child's dental health.

Significantly increased levels of gingival bleeding occurred in the control group not, as might be expected, in the cardiac group. This finding is in contrast to earlier studies that showed that children with cardiac disease had worse gingival health.[25, 26] There is no clear explanation for this, but it is possible that the examiner (CPS) was subconsciously trying to avoid causing any gingival bleeding in the cardiac group and examined the teeth and gingivae very gently. An alternative explanation might be that the majority of the cardiac children were inpatients and because their routine was disturbed, had forgotten to brush their teeth so the mildly traumatic effect of toothbrushing was not apparent. This would cause some gingival bleeding in the control group that would be apparent to the dentist examining the gingivae.

The number of tooth surfaces covered with plaque is significantly greater in the primary dentition and probably reflects the poor toothbrushing dexterity usually seen in children younger than age 8 years.[27] The significantly increased proportion of plaque-covered permanent tooth surfaces that exhibited gingivitis has not been reported before but is to be expected, as the very rapid turnover of cells in the rapidly growing periodontal structures outstrip the possible cytotoxic effects of bacterial dental plaque. The increased proportion of inflamed gingival surfaces seen in the permanent dentition is a matter of concern as the potential for bacteremia from the chronically inflamed gingivae is a possible factor in subacute bacterial endocarditis. Although the mean levels of plaque accumulation and gingival inflammation were relatively small, the mean figure disguises the very wide range of tooth quadrisections associated with plaque and gingivitis. The average length of a quadrisection of a tooth at the gingival margin is 4.5 mm. Using the raw data in this study, it is clear that in some children the length of gingival margin with gingivitis was, at a conservative estimate, 396 mm. If we assume that the gingival inflammation extends for 1.5 mm in a gingivoapical direction (also a conservative estimate) then the total area of inflamed gingiva in some mouths is on the order of 594 mm^2. This is a sizeable area of inflamed and ulcerated soft tissue and, as has been shown in this study, to be invariably associated with significant deposits of bacterial dental plaque. Coupled with the intermittent trauma of chewing food and toothbrushing, it is not surprising that such everyday activities cause significant odontogenic bacteremia.[28] The increased levels of gingivitis associated with the permanent dentition may be a contributory factor in the increased incidence of older children contracting bacterial endocarditis, as this condition is extremely rare in children under 10 years of age.[29]

The lack of any difference in the number of dietary sugars and sugary medicines was surprising because at the beginning of the survey we thought that children with cardiac disease may take many sweetened medicines, have a poor appetite, eat many sugary snacks, be over-indulged by their parents, and often be given sweets in an attempt to compensate for their medical

problems. As the data show, this was not the case and this is reflected in the similar levels of caries, plaque scores, and number of inflamed gingival surfaces.

The increased levels of untreated disease in the cardiac children compared with the controls referred to above have been reported previously.[30–32] It appears that despite advances in preventive care, improved anesthetic services, and increased awareness, children most at risk from serious complications of dental disease are not receiving a satisfactory standard of dental care. It is of considerable importance to the children with congenital heart disease because the open carious lesions, extensive deposits of plaque, and widespread areas of gingival inflammation represent an enormous bacterial loading of Viridans streptococci within the mouth. This is important in further development of dental disease and the potentially life threatening condition of bacterial endocarditis from Viridans streptococci.

The parents of children with congenital heart disease were generally more aware of the potential harm to the children of debilitating illnesses such as influenza, trauma, or a systemic infection. Of specific interest is the belief among significantly more parents of cardiac children that bleeding gums are potentially harmful to children with congenital disease. This aspect of their knowledge may be an important springboard for developing increased awareness of the need for comprehensive dental care.

The knowledge of dental health practices was good among both groups but was not universally practiced, especially among children from the cardiac group. Particularly disappointing was that a large number of children brushed their teeth only once or no times per day. Paradoxically, this is because the parents and children may be aware that bleeding gums are potentially harmful and are unaware that if gingivae bleed on brushing, it is necessary to brush more to encourage resolution of the gingival inflammation. A further cause of concern is the large number of parents who did not use fluoride tablets and, perhaps more disturbing, did not even know if fluoride tablets were of any benefit.

The most disappointing and potentially the most injurious to the children was the fact that 19% of the children with congenital heart disease had never visited a dentist. As a consequence, these children had not received the benefit of individual preventive dental advice and, where appropriate, dental treatment. It is difficult to assess the importance of this in terms of a serious health hazard. Nevertheless, the pain and infection present in this group of chronically sick children is an unacceptable state of affairs. Furthermore, recent evidence shows that dental bacteremia occurs from a wide variety of dental operative procedures as well as home care procedures such as toothbrushing in the presence of inflamed gingivae.[28] Current practice is to recommend that children with heart disease seek out and attend a dentist, the advice usually being accompanied by the presentation of a "Heart Card" detailing antibiotic prophylaxis regimens if extractions are required. We believe that the data presented here show that this strategy has failed. Parents need more active encouragement to ensure that their children receive adequate preventive advice about dental health and the availability of dental treatment. This could be achieved by the addition of community or hospital dental officers to pediatric cardiac units. We believe that this is the only way the dental health of children with congenital heart disease can be improved to acceptable levels.

It is not possible to compare the data on salivary IgA with other studies on children with congenital disease as this is the first to estimate the salivary levels of this important immunoglobulin. Nevertheless, the levels found do compare with those found in other studies.[33] The importance of salivary IgA in bathing the teeth, gingivae, and mucosa is well established.[34] It is clear that the presence of congenital heart defects, even of a serious nature, has no effect on the levels of salivary IgA. The importance of salivary IgA appears to be in protecting against the adherence of mutans streptococci.[35] The similar levels of dental caries and salivary IgA in both control and cardiac groups are consistent with a correlation of caries and immunoglobulin levels, which is demonstrated in our study.

Conclusion

In conclusion, children with severe congenital heart disease suffer levels of caries, plaque, and gingivitis higher than the general population. In addition, the cardiac group have a higher level of untreated caries. These children are exposed to open infection. Children with severe congenital cardiac disease need to be targeted by dental health care professionals. An effective way of achieving this would be for dental surgeons to attend cardiac assessment clinics to ensure that each child has the benefit of a full dental diagnosis and access to primary preventive care.

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