APPLIED AND ENVIRONMENTAL MICROBIOLOGY, Aug. 1995, p. 3165–3168 Vol. 61, No. 8
0099-2240/95/$04.00+0
Copyright © 1995, American Society for Microbiology

Quantitative Analysis of Bacterial Aerosols in Two Different Dental Clinic Environments

DANIEL GRENIER

Groupe de Recherche en Ecologie Buccale, Faculté de Médecine Dentaire, Université Laval, Sainte-Foy, Québec, Canada G1K 7P4

Received 31 January 1995/Accepted 18 May 1995

Microbial aerosols are generated during dental treatments and may represent an important source of infection. This study was designed to quantify bacterial air contamination during dental treatments in both a closed dental operatory and a multichair dental clinic. Air was sampled by using a slit type of biological air sampler. Following air sampling, blood-supplemented Trypticase soy agar plates were incubated at 37°C under anaerobic conditions for 7 days. The maximum levels of air contamination in the closed dental operatory were observed while dental treatments were being performed (four trials; 216 ± 75 CFU/m^3 for ultrasonic scaling treatments and 75 ± 22 CFU/m^3 for operative treatments). At 2 h after completion of the treatments, the bacterial counts were about the same as the pretreatment levels (12 to 14 CFU/m^3). In the second part of the study, a multichair dental clinic was divided into four areas, and air contamination was monitored at each site. Three sites were located in active dental treatment areas, whereas no dental treatments were performed within an 11-m radius of the fourth site. At 3 h after the beginning of dental treatments, the highest bacterial counts were obtained in the three active dental treatment areas (76 to 114 CFU/m^3). However, there was noticeable contamination in the inactive dental treatment area (42 CFU/m^3). Thus, bacterial aerosols were able to spread into areas where there was no dental activity. My data show that dental treatments significantly increased the levels of bacterial air contamination in both a closed dental operatory and a multichair dental clinic. Whether such levels of contamination have any influence on infection rates is not known.

Infection control has long been considered one of the main concerns of the dental community. Indeed, infectious agents may be transmitted to patients and dental staff via several vectors, including instruments and air (6, 9, 11, 13). Numerous studies concerning the importance of airborne transmission of pathogens in hospitals have been described previously (3, 15). Dental operatories are usually small rooms where the air becomes stagnant because of increased humidity. In addition, many procedures in dental clinics are associated with the generation of potentially hazardous aerosols. This suggests that cross-contamination via the air may also occur in dental environments.

The propelling force of a high-speed dental drill and the cavitation effect of an ultrasonic scaler, both combined with a water spray, can generate numerous airborne particles derived from blood, saliva, tooth debris, dental plaque, calculus, and restorative materials. Microorganisms can be suspended in and carried by these very small particles into the surrounding air of an operatory. Thus, infectious agents responsible for pneumonitis, influenza, and hepatitis, as well as skin and eye infections, may be transmitted during dental procedures. Several factors, including humidity, temperature, particle size, and ventilation, could influence the spread and infectious potential of microbial aerosols inside an operatory (2, 7, 15). Most dental aerosol droplets have a diameter of 5 μm or less and are concentrated within 2 ft (ca. 61 cm) of the patient's mouth (10). The aerosols may penetrate into the respiratory tract and directly invade the alveoli of the lungs. However, the ability of these aerosols to produce infections is related to the quantity and pathogenicity of the invading microorganisms, as well as the immune capacity of the patient.

In previous studies, investigators have described increases in bacterial air contamination following dental treatments in closed dental operatories (5, 8, 10, 12, 14). However, in most of these studies the researchers used procedures or conditions that were not ideal for accurate quantification of bacterial aerosols arising from oral cavities. In addition, there are no previous data concerning bacterial contamination of the air in multichair dental clinics, such as those found in dental schools. The aim of this investigation was to use a slit type of air sampler to quantify bacterial aerosols generated during dental treatments. This study was conducted to observe variations before, during, and after dental treatments in two different clinical environments, a closed dental operatory and a multichair dental clinic.

Air sampling in the closed dental operatory. Air contamination was monitored in the closed dental operatory (volume, 55 m^3) by using a Slit-to-Agar biological air sampler (model STA 101; New Brunswick Scientific Co., Inc., Edison, N.J.). This sampler drew air at a high speed through a narrow slit and blew it over a solid culture plate. The plate rotated at a uniform speed under the slit, and a complete rotation of the plate took 60 min. In each case the air sampler was located approximately 4 ft (ca. 122 cm) from the patient's mouth (height, 3 ft [ca. 91 cm]) and was operated at an airflow rate of 20 liters/min. Air samples were collected for a 30-min period before the treatment was begun and for 30 min once the treatment had started. At the end of this period, the patient was transferred to another dental operatory to complete the treatment, and sampling was continued for 30 min in the original room to determine the time that dental aerosols remained in suspension. Air sampling was also performed for 30 min at 2 and 4 h posttreatment. After each sampling period, the culture plate was immediately processed to determine bacterial growth. Microbial air contamination was evaluated for the following two types of dental treatments: (i) prophylactic treatment in which an ultrasonic scaler was used and (ii) operative dental treatment in which a high-speed dental drill was used (the patient wore a rubber dam). During the dental treatments, which were performed by different dentists, the high-speed drill and the

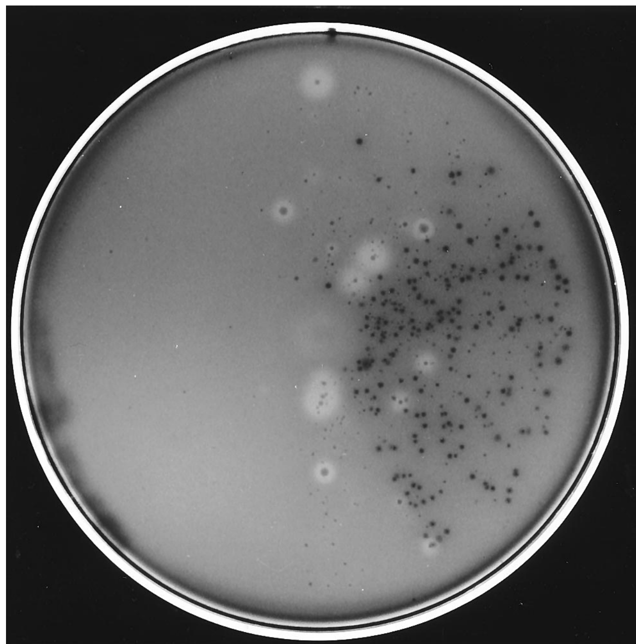

FIG. 1. Blood-supplemented Trypticase soy agar plate after collection of dental aerosols and incubation under anaerobic conditions for 7 days. Samples were collected before dental treatment (left side) and during ultrasonic scaling treatment (right side).

ultrasonic scaler were used for approximately 8 and 15 min, respectively. The patients were healthy adults with adequate dental hygiene. Bacterial counts were determined on Trypticase soy agar plates (BBL Microbiology Systems, Cockeysville, Md.) (65 ml in 150-mm plastic dishes) supplemented with 5% (vol/vol) human blood, 10 μg of hemin per ml, and 1 μg of vitamin K per ml. Immediately after sampling, the plates were placed in an anaerobic chamber containing N_2, H_2, and CO_2 (80:10:10) and incubated for 7 days at 37°C. These growth conditions were used because most bacteria that originate from oral cavities are anaerobes or facultative anaerobes. The total numbers of CFU were determined, and the data were expressed as the number of CFU per cubic meter of air sampled. Four trials were performed for each dental treatment, and the mean ± standard error was calculated.

The levels of bacterial air contamination generated during dental treatments were easily and accurately quantified by using the slit type of biological air sampler. Figure 1 shows an example of the levels of bacterial air contamination before (left side of the plate) and during (right side of the plate) an ultrasonic scaling treatment in the closed dental operatory (trial 3). Microbial air contamination data for four different trials obtained before, during, and after ultrasonic scaling treatment are shown in Table 1. As expected, the bacterial counts before the dental procedure were low (12 ± 4 CFU/m^3). Once the scaling treatment started, the levels of air contamination increased substantially (7- to 34-fold; 216 ± 75 CFU/m^3). Immediately after the treatments ended, the levels of bacterial contamination of the air decreased by approximately 80% (to 44 ± 14 CFU/m^3), which suggests that bacterial aerosols settle rapidly. At 2 and 4 h after the treatments ended, the counts were about the same as they were before the dental treatments began. As the use of a water spray with a turbine handpiece increased the relative humidity in the room, it is unlikely that the drastic decreases in the number of CFU were associated with a loss of bacterial viability because of the airborne state of the material (dehydration). These decreases also were not related to the susceptibility of bacteria to oxygen in the atmosphere. After the aerosols were collected, the plates could be kept under aerobic conditions for 24 h prior to culturing under anaerobic conditions without any decrease in the number of CFU (data not shown).

TABLE 1. Bacterial air contamination before, during, and after ultrasonic scaling treatments in a closed dental operatory

Trial	Level of contamination (CFU/m^3)				
	Before treatment	During treatment	At the end of treatment	2 h after treatment	4 h after treatment
1	15	175	58	13	3
2	5	88	18	10	10
3	22	433	75	7	3
4	5	168	25	8	8
Mean ± SE	12 ± 4	216 ± 75	44 ± 14	10 ± 1	6 ± 2

Table 2 shows that the bacterial contamination generated during the operative dental treatments was less than the contamination generated during the ultrasonic scaling treatments. This finding may be related to the fact that patients wore a rubber dam while being treated. However, a clear increase in the level of air contamination (75 ± 22 CFU/m^3) was associated with the use of the high-speed drill. At 2 h after the treatments ended, the counts reached base levels. Although different clinical settings and sampling procedures make data difficult to compare, Larato et al. (8) observed a similar air microbial contamination pattern (before, during, and after an operative treatment) in a closed dental operatory.

Air sampling in the multichair dental clinic. The multichair dental clinic (36 m by 18 m by 3 m; 80 dental chairs) was used by 35 dental students during the summer months. Air samples were collected at four sites over 30-min periods. As Fig. 2 shows, three sites (sites 1, 2, and 4) were located in active dental treatment areas, whereas no dental treatments were performed within an 11-m radius of the fourth site (site 3). A wide variety of dental procedures, including ultrasonic scaling and high-speed drilling, were performed by the students. Each site was sampled (i) 30 min before dental treatments began, (ii) 3 h after the dental treatments began, (iii) at the end of dental treatments (i.e., after 6 h of dental treatments), and (iv) 7 h after the dental treatments ended. Air samples were collected on Mondays for five consecutive weeks, and bacteria were grown as described above.

The mean values obtained for each site are shown in Table

TABLE 2. Bacterial air contamination before, during, and after operative treatments in a closed dental operatory

Trial	Level of contamination (CFU/m^3)				
	Before treatment	During treatment	At the end of treatment	2 h after treatment	4 h after treatment
1	20	102	113	ND[a]	ND
2	13	121	48	8	5
3	3	33	18	8	<3[b]
4	20	42	25	20	13
Mean ± SE	14 ± 4	75 ± 22	51 ± 22	12 ± 4	9 ± 4

[a] ND, not determined.
[b] Less than the detection limit (3 CFU/m^3).

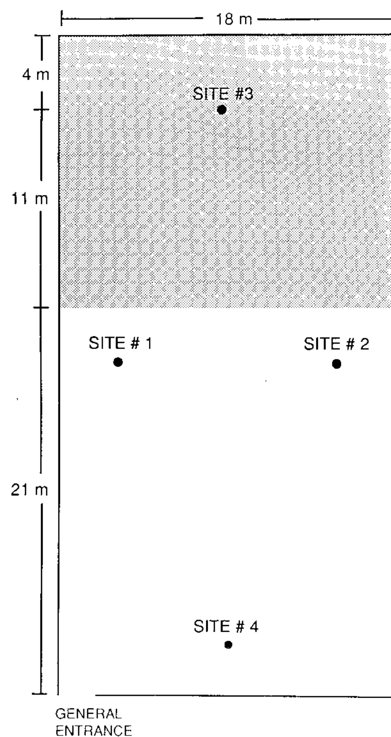

FIG. 2. Schematic diagram of the multichair dental clinic. The shaded area is the area where no dental activity occurred.

3. The levels of air contamination were obviously low at all sites before the treatments began (mean levels of contamination, 10 to 16 CFU/m^3). At 3 h after the treatments started, the areas where dental treatments were performed experienced clear increases in levels of air contamination (mean levels of contamination, 76 to 114 CFU/m^3). Interestingly, site 3, where no dental treatments were carried out within an 11-m radius, also experienced an increase in the level of bacterial air contamination (mean level of contamination, 42 CFU/m^3). The levels of bacterial contamination of the air were only slightly higher at the end of dental treatments (i.e., after 6 h of activity). At 7 h after all treatments ended, the microbial counts were comparable to the counts obtained before the treatments began. The data obtained in the multichair dental clinic show that dental aerosols have the capacity to spread quite rapidly, even into areas where there is no dental activity. Air-handling systems and human activity may have accounted for the dissemination of bacterial aerosols, as previously demonstrated in other environments (2, 15). As both immunosuppressed and infected patients may be treated in a multichair dental clinic, my data raise the question of whether all patients should be treated in closed operatories rather than in the wide-open clinics commonly found in dental schools.

TABLE 3. Bacterial air contamination before, during, and after dental treatments in a multichair dental clinic

Site	Level of contamination (CFU/m^3; mean ± SE)[a]			
	Before treatments	3 h after treatments began	At the end of treatments	7 h after treatments ended
1	13 ± 2	114 ± 15	116 ± 25	7 ± 1
2	10 ± 2	76 ± 19	94 ± 24	12 ± 5
3	15 ± 4	42 ± 11	48 ± 9	8 ± 2
4	16 ± 3	99 ± 21	133 ± 37	9 ± 4

[a] Air samples were collected on five consecutive Mondays.

My investigation differed from previous investigations (5, 8, 10, 12, 14) in that the numbers of microbes in dental aerosols were determined by using a slit type of biological air sampler and culture plates were incubated in an anaerobic chamber. Anaerobic conditions were used because this study was designed to quantify oral bacteria. It is likely that the actual microbial contents of air in dental clinics are much higher than the contents reported in this paper. Indeed, the culture medium and growth conditions which I used did not allow me to recover all types of microorganisms, including more fastidious bacteria, viruses, and mycetes. Furthermore, aerobic bacteria, such as *Pseudomonas* spp. that are found in high numbers in dental unit waterlines (1, 16) and are likely to be present in dental aerosols, cannot grow under the anaerobic conditions which I used. The location of the air sampler also strongly influences the recovery of bacteria. Much higher bacterial counts would have been obtained if air nearer the patients' mouths had been sampled.

Control of airborne transmission of infectious diseases associated with indoor environments is especially important in medical environments. In dental clinics, several infectious agents could be acquired by dental staff and patients by airborne transmission. In addition, dental aerosols containing opportunistic pathogens should also be considered hazardous for immunosuppressed patients, who could develop serious infections. In this study I confirmed a potential transmission route for infectious agents, and my data support the importance of protecting against cross-infectious agents contained in dental aerosols. As suggested in the infection control guidelines of the American Dental Association (4), operators and dental assistants should always wear masks, gloves, and eyeglasses with lateral protective shields. Because of the high risk of cross-contamination in dental clinics, research should be directed toward developing effective means for controlling and removing dental aerosols. In this vein, Fine et al. (5) recently demonstrated that preprocedural rinsing with an antiseptic mouthwash can significantly reduce the microbial contents of aerosols generated during ultrasonic scaling. Laminar unidirectional airflow, air ventilation, and air filtration could also be beneficial in dental environments and should be considered.

I thank R. McCabe (Université de Montréal) and A. Prévost (Université de Montréal) for their contributions.

REFERENCES

1. **Bourassa, N., D. Grenier, D. Mayrand, J. Morin, and L. Dufour.** 1995. Bacterial analysis of dental unit waterlines. J. Dent. Res. **74:**153.
2. **Burge, H. A.** 1985. Indoor sources for airborne microbes, p. 139–148. *In* R. B. Gammage and S. V. Kaye (ed.), Indoor air and human health. Lewis Publishers, Inc., Chelsea, Mich.
3. **Burge, H. A.** 1990. Risks associated with indoor infectious aerosols. Toxicol. Ind. Health **6:**263–274.
4. **Council on Dental Materials, Instruments and Equipment, Council on Dental Practice, and Council on Dental Therapeutics.** 1988. Infection control recommendations for the dental office and the dental laboratory. J. Am. Dent. Assoc. **116:**241–248.
5. **Fine, D. H., C. Mendieta, M. L. Barnett, D. Furgang, R. Meyers, A. Olshan, and J. Vincent.** 1992. Efficacy of preprocedural rinsing with an antiseptic in reducing viable bacteria in dental aerosols. J. Periodontol. **63:**821–824.
6. **Jakush, J.** 1986. Infection control in the dental office: a realistic approach. J. Am. Dent. Assoc. **112:**458–468.
7. **Kraidman, G.** 1975. The microbiology of airborne contamination and air sampling. Drug Cosmetic Ind. J. **3:**40–43.
8. **Larato, D. C., P. F. Ruskin, A. Martin, and R. Delanko.** 1966. Effect of a dental air turbine drill on the bacterial counts in air. J. Prosthet. Dent. **16:**758–765.

9. **Lu, D. P., and R. F. Zambito.** 1981. Aerosols and cross-infection in dental practice—a historic view. Gen. Dent. **2:**136–143.
10. **Micik, R. E., R. L. Miller, M. A. Mazzarella, and G. Ryge.** 1969. Studies on dental aerobiology. I. Bacterial aerosols generated during dental procedures. J. Dent. Res. **48:**49–56.
11. **Molinari, J. A., V. A. Merchant, and M. J. Gleason.** 1990. Controversies in infection control. Dent. Clin. N. Am. **34:**55–69.
12. **Pagniano, R. P., R. C. Scheid, S. Rosen, and F. M. Beck.** 1985. Airborne microorganisms collected in a preclinical dental laboratory. J. Dent. Educ. **49:**653–655.
13. **Runnells, R. R.** 1988. An overview of infection control in dental practice. J. Prosthet. Dent. **59:**625–629.
14. **Travaglini, E. A., D. Larato, and A. Martin.** 1966. Dissemination of organism-bearing droplets by high-speed dental drills. J. Prosthet. Dent. **16:**132–139.
15. **Walter, C. W.** 1980. Prevention and control of airborne infection in hospitals. Ann. N.Y. Acad. Sci. **353:**312–330.
16. **Williams, J. F., A. M. Johnston, B. Johnson, M. K. Huntington, and C. D. Mackenzie.** 1993. Microbial contamination of dental unit waterlines: prevalence, intensity and microbiological characteristics. J. Am. Dent. Assoc. **124:**59–65.