THE NEW YORK STATE
DENTAL Journal
OCTOBER 1997 ■ VOLUME 63 NUMBER 8

Helping Dental Students Find Their Way

Medications as Factor in Oral Disease

The Hazards of Dentistry

OFFICIAL PUBLICATION OF THE DENTAL SOCIETY OF THE STATE OF NEW YORK

Occupational Hazards of Dentistry

A REVIEW OF LITERATURE FROM 1990

Sara Babich, D.D.S.; Ronald P. Burakoff, D.M.D.

■ ABSTRACT ■

A brief survey of the scientific and clinical literature (1990 - present) on occupational hazards in dentistry is presented. The hazards identified are associated with chemical and biological agents. Yet, dentistry is a relatively safe profession. Other adverse health risks arise as new technologies and materials are developed. However, once identified and recognized as a risk, new guidelines, precautions and protocols are rapidly instituted to greatly reduce or even eliminate the occupational hazard.

Every profession has its inherent occupational health risks. Dentistry is no exception. A dentist must be knowledgeable of both the potential health risks and the methodologies, procedures and protocols to reduce risk and afford some degree of protection. At times, a healthier work environment can be established with only minor changes or adaptations.

Occupational hazards are constantly evolving. Once a particular health hazard has been identified, attempts are made to either reduce or eliminate the risk. For example, in the late 1960s and early 1970s, the adverse health effects of waste anesthetic gases were documented; such recognition led to development of adequate scavenging systems.[1] However, as more advanced science and technologies arise, new health risks surface. The use of lasers in dentistry, for example, has allowed dentists to perform some procedures with greater efficacy, but, unfortunately, it has increased their exposure to harmful microbial aerosols. The dentist also risks injury if proper laser safety protocol is not maintained.

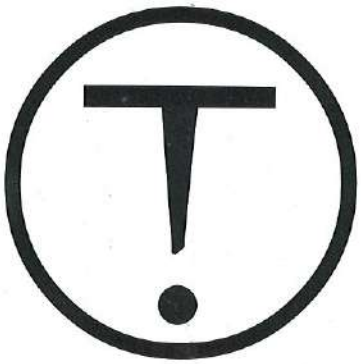
During the course of providing patient care the dentist comes into contact with various biological, chemical and physical agents that could adversely affect health. Thus, it is imperative that the dentist keep abreast of new protocols and technologies for handling chemicals safely, controlling the spread of infectious microbial and viral agents, and avoiding physical risks. Such information is needed not only to maintain the health of the dentist, but also to keep the dental staff and patients safe. This article reviews occupational hazards that are present in the

dental office today, as described in literature published from 1990 to the present.

Infectious Agents

Dental instruments such as the air turbine handpiece (high-speed drill) and the ultrasonic scaler generate dental aerosols, which contain a mixture of potentially infectious agents and antigenic materials. Aerosolized infectious agents and nonliving organic materials originate primarily in the mouth of the patient.[2] Furthermore, splatter from the water spray may contain droplets of the patient's saliva, respiratory secretions and blood.[3] Aerosolized infectious agents include bacteria and viruses; other materials in the aerosol are droplets of lubricating oil from dental instruments and enamel, and dentin particles of respirable size. These latter materials contain both calcium salts (mainly, hydroxyapatite) and an antigenic protein matrix that could evoke an immune response.[2] Percutaneous exposure to patients' blood is another mode of exposure to infectious agents.[4]

The causative agent of tuberculosis is the bacterium, *Mycobacterium tuberculosis*, which is usually spread by the airborne route. The reemergence of tuberculosis, a leading cause of death in humans, is of much concern, especially as multiantibiotic resistant strains have been isolated.[5,6] Although dental care providers are considered to be at low risk of exposure to patients with active tuberculosis, they should be familiar with recent recommendations for preventing transmission of the disease in the health care setting.[7]



On the other hand, it is generally accepted that the dental staff is at greater risk than the general population of acquiring hepatitis B virus (HBV), as a bloodborne infection. Thus, it is the policy of the American Dental Association (ADA) that all dentists be vaccinated against HBV. Of related concern is that the dental staff may be exposed to patients' blood infected with human immunodeficiency virus (HIV). Although there are recorded incidences of accidental percutaneous exposure of dental staff to blood from an HIV-infected patient, there are no reported cases of HIV transmission from the patient to the dentist.[8]

Recently the ADA[9] drew up a series of recommendations for infection control in the dental office and laboratory. These included the wearing of gloves, surgical masks or face shields, and protective clothing; limiting contamination by droplets and splatter through the use of high-volume evacuation; proper patient positioning and rubber dams; proper handling of sharp instruments and needles; adequate hand washing, and suitable disinfection and sterilization procedures for instruments.

The reemergence of tuberculosis, a leading cause of death in humans, is of much concern.

Lasers are a relatively new source of biologically generated aerosols. Carbon dioxide lasers in particular are valuable tools in dental surgery and endodontics. Their advantages over a scalpel or electrocautery in surgical procedures include preciseness in concentrating on a specific tissue area, reduction in trauma to surrounding tissues, and a drier operative site as a result of the coagulating effect of the laser on small blood vessels.[10] The applications of lasers in endodontics include removal of pulp tissue from the root canal, enlargement of the canal space, disinfection of the pulp canal and sealing of the apical foramen.[11]

However, the laser smoke plume may present a health hazard. The energy supplied by the laser beam is so intense that when the tissue is vaporized, the resultant smoke may contain cellular material or intact cells explosively disrupted near the focal point of the beam. Malignant cells (for example, melanoma cells), bacteria and viruses have been detected in laser smoke and may pose a health threat if inhaled.[10,11] Of particular concern is the research by Baggish et al.[12]

in which infectious proviral human immunodeficiency virus (HIV) DNA was noted in laser smoke resulting from laser-treated tissue culture pellets infected with HIV. The use of a mechanical smoke evacuator system near the operative site, in conjunction with respiratory protection, may appreciably reduce the health risk to the dentist and the patient.[10-12]

Allergic Contact Dermatitis

Dental personnel are exposed to many allergens, and work-related skin problems are common. Allergic contact dermatitis (ACD) is a delayed-type of hypersensitivity, with a relatively long period (» 24 hr) required for development of inflammation following exposure. The major causative agents of ACD in dentistry are acrylics, among them methyl methacrylate, ethyleneglycol dimethacrylate and triethyleneglycol dimethacrylate, and bisphenol A, an epoxy resin compound. Dental products that contain acrylic include removable dental prostheses, individual impression trays, orthodontic devices, occlusal splints, fixed crowns and bridges, dental bonding agents and dental composite resins.[13-16]

Occupational ACD appears primarily as pulpitis of the fingertips. Acrylates quickly penetrate practically all surgical latex and polyvinyl chloride (PVC) gloves. The use of 4H-glove fingertips, worn under a disposable latex or PVC glove, has been suggested as a preventive measure.[15]

Latex

Allergic reactions to latex gloves have been noted in nine percent to 12 percent of dental care workers.[17,18] Skin irritation is the most frequent symptom. However, prolonged exposure to latex gloves may develop into immediate (Type I) and delayed (Type IV) hypersensitivity immunologic reactions. Immediate hypersensitivity is characterized by urticaria (pale wheal) and erythema at the site of contact within minutes after exposure. The severity of immediate hypersensitivity ranges from diffuse skin rashes to systemic reactions, including life-threatening bronchospasm, hypotension, anaphylaxis and death. Delayed hypersensitivity, which occurs in the sensitized individual, is characterized by contact dermatitis six to 72 hours after exposure.[17] Alternatives, among them latex-free substitutes, are available.

Delayed hypersensitivity is characterized by contact dermatitis 6 to 72 hours after exposure.

Mercury

Based on weight composition, dental amalgam is 50 percent pure metallic mercury, with lesser amounts of other metals such as silver, copper, zinc and tin. Occupational exposure to metallic mercury, usually in the form of mercury vapor, occurs during the cutting, filling and polishing of amalgam restorations in routine clinical procedures. Additional sources of exposure include the use of squeeze cloth to express the excess mercury, accidental spills, contaminated mechanical amalgamators, high temperature sterilization of mercury-contaminated instruments, heating the amalgam, storing mercury in leaking containers, and amalgam waste discharge from dental vacuum units.[19-22]

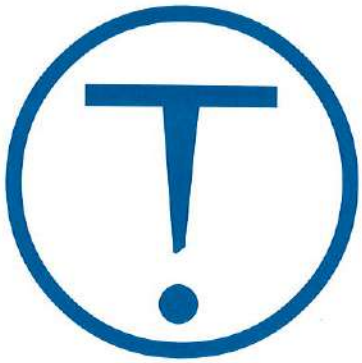
Upon inspiration, about 80 percent of the inhaled mercury vapor diffuses across the alveoli and enters the circulatory system. This elemental mercury (Hg^0) is oxidized to divalent, cationic mercury (Hg^{2+}), which accumulates in tissues, with the kidney being the major site of deposition. There is considerable information on the toxicity of acute exposure to mercury. However, little information is available on the adverse health effects of chronic exposure to low concentrations of mercury. Such exposures are common in the dental office, with dentists averaging twice the mercury concentrations in their blood as nondentists.[23]

Data on adverse health effects associated with occupational exposure to dental amalgam are inconclusive.[24] Studies with laboratory animals noted that mercury vapor and inorganic mercury compounds are potent neuroterato-

genic and reproductive hazards. Yet an epidemiologic study by Brodsky et al.[25] of dentists and dental assistants showed no increased rate of spontaneous abortions or congenital abnormalities in the offspring of men and women exposed to elevated vs. low levels of mercury in dental environments.

Warfvinge[26] reported no toxic effect in a female dentist exposed for one year to elevated levels of mercury vapor from a leaking amalgamator. The dentist became pregnant and a mild bilateral hydronephrosis diagnosed at 20 weeks of gestation was resolved at 32 weeks of gestation. Rowland et al.[27] studied the effect of occupational exposure to mercury vapor on the fertility of female dental assistants. The probability of a conception each menstrual cycle (termed fecundability) was reduced by 37 percent in women who prepared 30 or more amalgams a week and who had poor mercury hygiene work practices. Apparently, further studies are needed to clarify whether mercury is a reproductive hazard to dentists and their staff.

Fung et al.[19] enumerate several measures to reduce exposure to mercury vapor in dental offices. They are: proper storage of mercury-containing materials in unbreakable, tightly sealed containers; proper collection of globular particles of mercury; use of commercial suppressants to clean mercury spills; avoidance of heating mercury or amalgam; and proper ventilation. Furthermore, use of a water spray and high-speed suction during the cutting of amalgam would also



reduce occupational exposures to mercury.[21]

Nitrous Oxide

Since the mid-19th century, nitrous oxide has been used as an anesthetic in dental offices. Because of its analgesic, anxiolytic and psychosedative properties, the use of nitrous oxide, particularly for outpatient sedation, has been increasing; approximately 85 percent of pediatric dentists and 50 percent of general practitioners use nitrous oxide in their practices.[28] Unlike patients in hospital operating rooms, dental patients are not intubated; therefore elevated levels of ambient nitrous oxide from leakage and as unscavenged gas in the dental operatory present a potential hazard to the dentist.[29] The 1991 ADA Survey of Dental Practice found that 58 percent of general and specialty practitioners reported nitrous oxide capability, but one-third lacked scavenging equipment.[30]

Approximately 20 years ago, in their retrospective epidemiological survey of dentists and chairside dental assistants, Cohen et al.[1] provided evidence that nitrous oxide was a reproductive hazard. Spontaneous abortions were increased 2.3-fold for female dental assistants and 1.5-fold for unexposed wives of male dentists. More recently, Rowland et al.[31] reported an elevated risk of spontaneous abortion among female dental assistants who worked with nitrous oxide for three or more hours a week in offices not using scavenging equipment. In an earlier study, Rowland et al.[32] reported a decrease in the fertility of female dental assistants who worked in dental surgeries without scavenging systems. Women who were exposed for more than five hours a week were only 41 percent as likely as unexposed women to conceive during each monthly cycle.

The work by Rowland et al.[31,32] refocused attention on the potential hazards nitrous oxide posed to the reproductive system.[29,33,34] Many of the adverse health effects, including reproductive hazards, resulting from chronic exposure to nitrous oxide have been noted in studies with laboratory animals. These studies have provided a possible biochemical mechanism for the reproductive effects of nitrous oxide.

Apparently, nitrous oxide has a deleterious effect on the metabolism of vitamin B_{12}, which in the reduced state is essential for the functioning of the enzyme, methionine synthetase. This enzyme is responsible for the recycling of the amino acid, methionine, which, in turn, plays a crucial role in the synthesis of thymidine, a nitrogenous base of DNA. Nitrous oxide oxidizes the cobalt ion in reduced vitamin B_{12} (cobalamin); this results in the inability of vitamin B_{12} to function as a cofactor. Exposure to nitrous oxide, therefore, may impair DNA synthesis. Furthermore, a decrease in the activity of methionine syn-

thetase may disrupt folliculogenesis and early development of the conceptus. Lastly, nitrous oxide may interfere with the hypothalamus-pituitary gland-ovary interaction by blocking the action of luteinizing hormone releasing hormone (LHRH), thereby disrupting ovulation.[29,33-36]

Two approaches can minimize the risk of occupational exposure to nitrous oxide. As noted by Donaldson and Meechan,[29] the direct approach is to use a nitrous oxide delivery system that has a mixing dial for proper dosage control, give the patient the minimum effective dose of nitrous oxide, and use scavenging equipment. Ostreicher[37] suggests a biochemical approach; vitamin B_{12} supplements to the dental staff may provide protection against reproductive hazards induced by inhalation of nitrous oxide.

Ethylene Oxide

Ethylene oxide is a gaseous sterilant used in some dental offices to disinfect equipment. A variety of in vitro and in vivo studies have shown ethylene oxide to cause mutations, chromosomal aberrations, fetal abnormalities; it may also be a carcinogen. A few epidemiologic studies have suggested that ethylene oxide is a reproductive hazard for female workers, including hospital sterilizing staff and chemical workers. Rowland et al.[38] noted an increase in spontaneous abortions and preterm and post-term births among female dental assistants exposed to ethylene oxide. The dental offices using ethylene oxide during this study often used a vented tray or bag sterilization system that could have exposed

the dental staff to high levels of ethylene oxide via the respiratory and dermal routes. Individuals handling ethylene oxide, therefore, should wear the required proper protective gear.

Conclusion

Advancements in materials development and in technologies may elicit new occupational hazards in the dental profession. Thus, the dentist should not become lax or complacent. Yet, once a potential adverse health effect is identified, steps are taken to rectify the situation. The field of dentistry is a relatively safe profession and, with proper knowledge and precautions, even those relatively few minor risks can be lessened or even eliminated.❑

References

1. Cohen EN, Brown BW, Wu ML. Occupational disease in dentistry and chronic exposure to trace anesthetic gases. JADA 1980;101:21-31.
2. Allsopp J, Basu MK, Browne RM, Burge PS, Matthews JB. Survey of the use of personal protective equipment and prevalence of work related symptoms among dental staff. J Occu and Environ Med 1997;54:125-134.
3. Ceisel RJ, Osetek EM, Turner DW, Spear PG. Evaluating chemical inactivation of viral agents in handpiece splatter. JADA 1995; 126:197-202.
4. Siew C, Gruninger S, Miaw CL, Neidle SA. Percutaneous injuries in practicing dentists. JADA 1995;126:1227-1234.
5. Brennan, PJ. Tuberculosis: A born again adversary for the dental health care worker. PA Dent J 1993;60:68-71.
6. Mikitka D. Tuberculosis infection in U.S. Air Force dentists. American Journal of Dentistry 1995;8:33-36.
7. Younai FS, Murphy DC. TB and dentistry. NYSDJ 1997; 63:49-53.
8. Gooch BF, Cardo DM, McKibben PS, Cleveland JL, Srivastava PU, Culver DH, Bell DM. Percutaneous exposures to HIV-infected blood. JADA 1995;126:1237-1242.
9. American Dental Association. Infection control recommendations for the dental office and the dental laboratory. JADA 1996; 127:672-679.
10. Nezhat C, Winer WK, Nezhat F, Nezhat C, Forrest D, Reeves WG. Smoke from laser surgery: Is there a health hazard? Lasers in Surg and Med 1987;7:376-382.
11. McKinley B, Ludlow MO. Hazards of laser smoke during endodontic therapy. J Endo 1994;20:558-559.
12. Baggish MS, Polesz BJ, Joret D, Williamson P, Refai A. Presence of human immunodeficiency virus DNA in laser smoke. Lasers in Surg and Med1991;11:197-203.
13. Jolanki R, Kanerva L, Estland T. Occupational allergic contact dermatitis caused by epoxy diacrylate in ultraviolet-light-cured paint, and bisphenol A in dental composite resin. Contact Derm 1995;33:94-99.
14. Kanerva L, Estlander T, Jolanki R, Tarvaine K. Occupational allergic contact dermatitis caused by exposure to acrylates during work with dental prostheses. Contact Derm 1993;28:268-275.
15. Kanerva L, Estlander T, Jolanki R. Occupational skin allergy in the dental profession. Derm Clinics 1994;12:517-532.
16. Kawahara D, Oshima H, Kosugi H, Nakamura M, Sugai T, Tamaki T. Further epidemiologic study of occupational contact dermatitis in the dental clinic. Contact Derm 1993;28:114-115.
17. Safadi GS, Safadi TJ, Terezhalmy GT, Taylor JS, Battisto JR, Melton AL. Latex hypersensitivity: Its prevalence among dental professionals. JADA 1996;127:83-88.
18. Katelaris CH, Widmer RP, Lazarus RM. Prevalence of latex allergy in a dental school. Med J Australia 1996;164:711-714.
19. Fung YK, Molrar MP. Toxicity of mercury from dental environment and from amalgam restorations. Clin Toxicology 1992; 30:49-61.
20. Martin MD, Naleway C, Chou H. Factors contributing to mercury exposure in dentists. JADA 1995;126:1502-1511.
21. Pohl L, Bergman M. The dentist's exposure to elemental mercury vapor during clinical work with amalgam. Acta Odontologica Scandinavica 1995;53:44-48.
22. Rubin PG, Yu M. Mercury vapor in amalgam waste discharged from dental office vacuum units. Archives Environ Health 1996;51:335-337.
23. Goering PL, Galloway WD, Clarkson TW, Lorscheider FL, Berlin M, Rowland AS. Toxicity assessment of mercury vapor from dental amalgams. Fund and Applied Toxicology 1992;19:319-329.
24. Ratcliffe HE, Swanson GM. Human exposure to mercury: A critical assessment of the evidence of adverse health effects. J Toxicology and Environ Health 1996; 49:221-270.

25. Brodsky JB, Cohen EN, Whitcher C, Brown BW, Wu ML. Occupational exposure to mercury in dentistry and pregnancy outcome. JADA 1985;111:779-780.
26. Warfvinge K. Mercury exposure of a female dentist before pregnancy. Brit Dent J 1995;178:149-152.
27. Rowland AS, Baird DD, Weinberg CR, Shore DL, Shy CM, Wilcox AJ. The effect of occupational exposure to mercury vapor on the fertility of female dental assistants. J Occu and Environ Med 1994;51:28-34.
28. Henry RJ. Assessing environmental health concerns associated with nitrous oxide. JADA 1992;123:41-47.
29. Donaldson D, Meechan JG. The hazards of chronic exposure to nitrous oxide: An update. Brit Dent J 1995;178:95-100.
30. Meskin LH. No laughing matter. JADA 1993;124:8-9.
31. Rowland AS, Baird DD, Shore DL, Weinberg CR, Savitz DA, Wilcox AJ. Nitrous oxide and spontaneous abortion in female dental assistants. Amer J Epidemiology 1995;141:531-538.
32. Rowland AS, Baird DD, Weinberg CR, Shore DL, Shy CM, Wilcox AJ. Reduced fertility among women employed as dental assistants exposed to high levels of nitrous oxide. NE J Med 1992;327:993-997.
33. Wynn RL, Nitrous oxide and fertility, Part I. Gen Dent 1993;41:122-123.
34. Wynn RL. Nitrous oxide and fertility, Part II. Gen Dent 1993;41:212-214.
35. Jastak JT, Donaldson D. Nitrous oxide. Anesthesia Progress 1991;38:142-153.
36. Yagiela JA. Health hazards and nitrous oxide: A time for reappraisal. Anesthesia Progress 1991;38:1-11.
37. Ostreicher DS. Vitamin B_{12} supplements as protection against nitrous oxide inhalation. NYSDJ 1994;60:47-49.
38. Rowland, AS, Baird DD, Shore DL, Darden B,Wilcox AJ. Ethylene oxide exposure may increase the risk of spontaneous abortion, preterm birth, and postterm birth. Epidemiology 1996;7:363-368.

Babich

Burakoff

Dr. Babich is a former dental resident at Long Island College Hospital, Brooklyn. Dr. Burakoff is chairman, Department of Dentistry, at the hospital.

www.nycpediatricdentist.com