

The prevention of hepatitis B transmission in dental practice

Larry I. Lutwick, MD

Abstract

The prevention of the spread of hepatitis B is of utmost importance to the dental profession. The mechanisms of transmission of the virus between practitioner and patient are discussed and preventive options outlined. Despite conscientious history taking, not all virus carriers can be identified without laboratory screening of the entire population. Despite specific precautions high risk exposures to practitioners still occur, putting the dental practitioner at risk for the morbidity and mortality of hepatitis B including the possibility of becoming a virus carrier. Immunoglobulin prophylaxis is available but is expensive, short-lived and is only useful for recognized exposures. The vaccine to prevent hepatitis B should emerge as the primary mode of protection in high-risk groups such as dentists.

Hepatitis B virus (HBV) can exist asymptotically in its human host and may be transmitted from this individual to others, particularly those in contact with the carrier's blood or blood-contaminated body fluids. The use of general or selective screening of individuals for HBV coupled with passive immunoprophylaxis with immune globulin can decrease the transmission of HBV to the health worker but has not eliminated the problem. A vaccine to prevent hepatitis B, now licensed in the United States, should become a major factor in further control of HBV, particularly in health care deliverers. The infectivity and modes of transmission of HBV will be discussed with particular attention to the pediatric dental practitioner. Modes of both passive and active prevention will be outlined.

Hepatitis B

The virus of hepatitis B is a unique human virus which has been reviewed periodically over the last decade.¹ It is the only human virus which is routinely detected by measuring a viral antigen instead of growth in tissue culture or a rising antibody titer. This antigen, hepatitis B surface antigen (HB_sAg), formerly Australia antigen

(AuAg) or hepatitis-associated antigen (HAA), represents excess surface coat protein of HBV and in itself does not connote infectious virus. The presence of HB_sAg in the blood or body fluid or on a dental instrument does not necessarily imply that the infectious form of HBV, the Dane particle, is present. Although Dane particle markers such as DNA polymerase activity and hepatitis B e antigen (HB_eAg) seem to correlate with increased infectivity,^{2,3} the virus can be transmitted with lesser efficiency without these markers.⁴ That the virus can be transmitted easily by the parenteral route, is demonstrated by the fact that serum diluted 100,000,000 times still transmits HBV.⁵ In contrast, HBV seems not to be as easily spread in a nonparenteral setting. Heathcote et al.⁶ and other groups have suggested that only the conjugal partners of the adult index cases of hepatitis B are at major risk for transmission. In the pediatric population, however, transmission from child to child, or child to adult, has been documented without overt close contact.

Saliva may be the major vehicle for transmission of HBV in nonparenteral settings. Although it is clear that HB_sAg can be found in the saliva of individuals who have HB_sAg in their plasma, no substantial data are available to implicate saliva or other body fluids as major vehicles. In fact, two studies have suggested that saliva is not an effective vehicle of transmission in a natural or experimental setting, but may function as a vehicle if inoculated parenterally.^{7,8} It seems most likely that saliva, semen, and other body fluids are infectious by contamination with small amounts of blood and then contact abraded skin or mucous membrane.⁹

Dental Care Transmission of HBV

The dental profession is at substantial risk of infection with HBV. Mosley et al.¹⁰ surveyed 1245 dental practitioners finding 0.9% to be HB_sAg positive (ongoing infection) and 12.7 anti-HB_s positive (previous infection). Not unexpectedly, the study found that evidence of previous infection with HBV increased uniformly with increasing years in the profession. No correlation was noted with any geographic area or size of community and no difference in seropositive rates were found be-

tween those who recognized illicit parenteral drug use in their population and those who did not. Feldman and Schiff¹¹ in a history survey found a significantly higher incidence of history of hepatitis among dentists than lawyers and found that certain subspecialty groups (especially oral surgeons) had a much higher hepatitis attack rate. Although the numbers were small, a higher rate among pedodontists was suggested. Feldman and Schiff also suggested that treating more than 10 narcotic addicts per year was a risk factor, but no difference in risk was found with various methods of sterilization, use of gloves or disposable needles.

It has been demonstrated, although infrequently, that chronic HBV carrier dental care workers may transmit the virus to their patients. As examples, Levin et al.¹² and Rimland et al.¹³ report 12 and 59 individuals contracting HBV disease from individual dental practitioners. A prospective study involving two dentists incubating HBV and 237 contacts revealed no evidence of transmission.¹⁴ The recognition that certain HB_sAg positive health professionals represent a public health hazard is clear—but that the majority of such individuals do not is also clear. If such an individual is identified, the use of precautions such as handwashing before and after patient contact, use of disposable equipment whenever possible, sterilization of reusable instruments between patients and use of masks and particularly gloves are recommended. Only if transmission can be documented despite such measures should any restriction of health care delivery be considered. Tzukert and Sandler¹⁵ have, in addition, found that even in regions where HB_sAg carriage is common and hepatitis B is prevalent, routine dental care could not be identified as an important factor in HBV spread.

The prevention of transmission of HBV from a patient who may or may not be known to be HB_sAg positive to a practitioner is also one of extreme importance. This is particularly so because of the frequency (5-10%) of chronic hepatitis which occurs after overt hepatitis B infection. Chronic hepatitis B may cause substantial problems with morbidity and mortality in addition to the potential problems of transmission to patients, associates and even family members. It is certainly reasonable to attempt to identify high-risk individuals prior to dental treatment being performed. As has been outlined by a British committee,¹⁶ it is not unreasonable to obtain HBV serologies on certain high-risk patients such as those with chronic renal failure, those undergoing dialysis, hemophiliacs, workers or clients from state schools, known parenteral drug addicts, those with a history of recent jaundice and male homosexuals.

In addition, immigrants from areas of the world with high carriage rates (Asia, Africa) should also be screened. The incidence of HB_sAg positivity in Southeast Asian refugees has been reported to be as high as 13%. In contrast, the rate in the overall U.S. population is 0.1-

0.2%. Since it appears that much transmission of HBV in the Third World occurs prior to age two, the pediatric age refugee groups should be considered just as high of a risk. Children from highly endemic areas and those in institutions for the mentally retarded may represent high-risk patients. Both tend to have poor dental health and may require extensive treatment. The Third World children are more likely to be HB_eAg positive and thus are more infectious. Institutionalized children, particularly those with Down syndrome, have a high carriage rate. Institutionalized children may also have periodontal disease which may cause more bleeding. The dental literature suggests that screening for HB_sAg will identify only 20-50% of HBV carriers.¹⁷ This information is incorrect. HB_sAg will only identify a fraction of individuals who will transmit hepatitis of any sort (mostly not hepatitis B) by blood transfusion¹⁸ but will identify essentially 100% of carriers of HBV.

HBV Precautions

If dental treatment is performed on a hepatitis B carrier, certain precautions would be recommended. Ideally, direct providers should be immune to hepatitis B: they should already have had hepatitis B and have naturally formed anti-HB_s or have been vaccinated. To have a population of anti-HB_s positive personnel, it is reasonable for all health care workers in endemic situations to know their anti-HB_s status. Since identification of all HBV carriers is impossible and the identification of anti-HB_s positive health care personnel may be difficult, certain precautions should be taken routinely. Glasses or eyeshields, appropriate disinfectants, gloves, and only sterilizable and/or disposable materials should help decrease HBV transmission. If a known carrier is treated, procedures to decrease aerosolization of potentially infectious material may decrease transmission. Whenever possible, carriers should be treated at the end of the daily treatment schedule.

HBV Exposures

Despite appropriate screening and procedural precautions, exposures still occur. The exposures of high risk are those in which blood or bloody secretions are inoculated through the skin, mucous membrane, or open cut of the practitioner. The degree of risk of oral tissue manipulation without such exposure is low. A recent followup of dental personnel exposed to a patient late in the incubation period of hepatitis B¹⁹ revealed no increase in HBV events as compared to a suitable nonexposed control group. If overt exposure does occur from an HB_sAg positive individual, immunoprophylaxis is recommended if the exposure was to a practitioner who is anti-HB_s negative and therefore susceptible to hepatitis B. Current recommendations from the U.S. Public Health Service²⁰ advise the use of human hyperimmune hepatitis B globulin (HBIG) for such exposures. The available

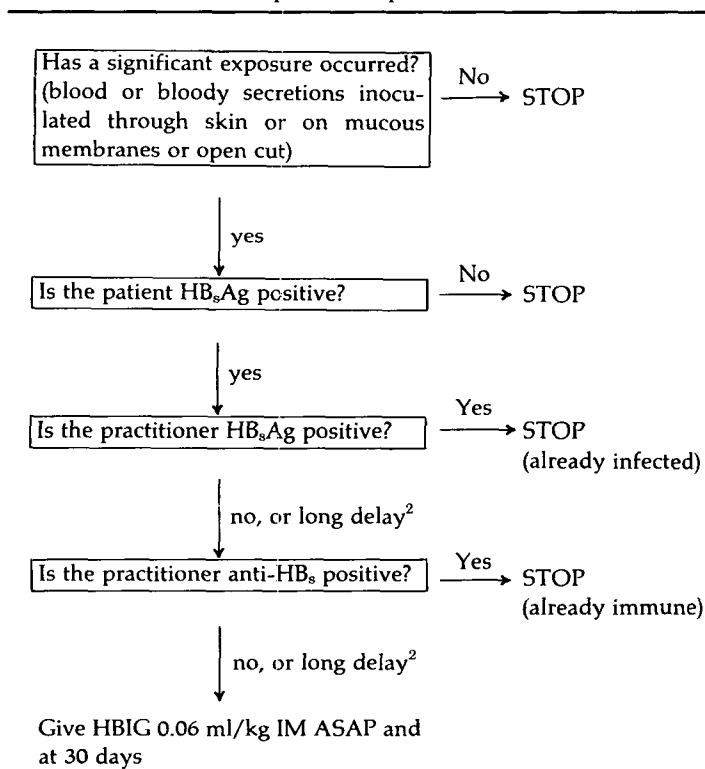
data^{21,22} suggest that passive immunoprophylaxis is useful as compared to placebo. It is unclear whether HBIG is superior to normal immune globulin (IG) which contains lower amounts of protective antibody. The cost of the HBIG regimen is \$300, more than 10 times that of IG. A rational approach to such an exposure is presented in Table 1.

Even under the most ideal circumstances, many significant exposures to HBV are either ignored or unappreciated. This is particularly true because many HB_sAg carriers go unrecognized. A recent report²³ found 15 cases of hepatitis B in hospital workers employed more than two years when only three recalled an exposure. Postexposure HBIG is inadequate to prevent HBV disease because many exposures are unnoticed.

Hepatitis B Vaccine

More studies of active immunoprophylaxis (vaccination) to prevent hepatitis B have been made. Just receiving licensure in the United States, the hepatitis B virus vaccine^a is a totally unique product. Unlike other viral vaccines that are produced in tissue or embryonated egg

Table 1. Algorithm for immunoglobulin prophylaxis in dental hepatitis B exposures.¹



¹ All high risk practitioners should be considered for vaccine when it becomes available.

² The current PHS guidelines¹⁹ recommend the use of HBIG for all blood exposures containing HB_sAg. It does not take into account whether the contact is already immune. If serologic studies can be obtained within 48 hours on the contact, it is reasonable to use IG 0.06 ml/kg initially until results available.

culture, this product is derived directly from the plasma of human chronic HB_sAg carriers. HB_sAg is purified by a rigorous series of precipitation and centrifuging, producing a highly purified product which is inactivated with formalin to prevent any residual infectivity. Prepared for use as an alum precipitate similar to the tetanus toxoid, the vaccine will be recommended for many high-risk groups.

Among the groups which would benefit from the vaccine are health care deliverers including oral surgeons, dentists, and pedodontists, particularly high-risk groups and those involved with risk groups previously mentioned. The vaccine will be recommended for the prevention of all antigenic subtypes of HBV (which cross protect with each other) but not for the prevention of any non-B hepatitis. It will be given as three inoculations at baseline, one and six months. At this time, the cost of the three-dose course is about \$100. This cost is, for the most part, related to the extensive purification process and the extensive safety testing methodology that ensures that each lot of vaccine does not transmit HBV or other pathogens. Theoretical and real potential hazards related to such a vaccine have been discussed in forums^{24,25} and it appears to be a safe product. However, as pointed out by Purcell and Gerin,²⁵ there will probably never be an absolutely safe vaccine. This fact is appreciated by most scientists and physicians, but perhaps not by the lay public, judges, and trial lawyers.

There have been a number of studies of such vaccines in humans utilizing several high-risk settings including hemodialysis patients and staff and young children in a highly endemic area. A reasonably large, controlled, double blind, clinical trial has been performed using male homosexuals who have a remarkably high risk of HBV acquisition (annual incidence of seroconversion to HB_sAg of 7.6% and to anti-HB_s of 11.6%). This study²⁶ revealed a reduction of hepatitis B infection of 92% in the vaccination group as compared to the control group. Ninety-six percent of the vaccinees developed anti-HB_s and none of these responders developed clinical hepatitis B or asymptomatic antigenemia. Interestingly, a decrease in incidence was noted as soon as 75 days following initial vaccination—suggesting that the immunization might be useful if given after exposure. Further followup²⁷ of these individuals revealed a somewhat waning but still protective antibody level. It is unclear at this point when, or if, further boosters are necessary.

In addition to a high degree of efficacy, the vaccine appears to be well tolerated. The Szmuness study²⁶ as well as a French study with hemodialysis staff²⁸ showed no significant difference between side effects in vaccinees and controls. A large part of the total side effects were local soreness, erythema, or induration. Nausea, fatigue, and fever were also noted. No cases of hepatitis B thought to be related to the vaccine have been found.

^a Heptavax-B, Merck, Sharp & Dohne Laboratories, West Point, PA.

The development of a vaccine designed to protect against hepatitis B has become a reality. Because of difficulty in identifying dental personnel who may transmit the virus, and the cost (and only transient protection) of immunoglobulin, the vaccine should emerge as the primary mode of protection against this significant pathogen. All health care practitioners in substantial risk situations should receive vaccination as the biologic product becomes available.

The official recommendations for use of Heptavax-B were published in *Morbidity and Mortality Weekly Report* 31:317-322, 327-328, 1982.

Dr. Lutwick is associate professor of medicine, State University of New York—Downstate Medical School, Division of Infectious Diseases, Department of Internal Medicine, Maimonides Medical Center, 4802 Tenth Ave., Brooklyn, NY 11219. Requests for reprints should be sent to him.

1. Robinson, W.S. and Lutwick, L.I. The virus of hepatitis, type B. *N Engl J Med* 295:1168-1175, 1232-1236, 1976.
2. Alter, H.J., Seeff, C.B., Kaplan, P.M., et al. Type B hepatitis: the infectivity of blood positive for e antigen and DNA polymerase after accidental needlestick exposure. *N Engl J Med* 295:909-913, 1976.
3. Beasley, R.P., Trepo, C., Stevens, C.E., et al. The e antigen and vertical transmission of hepatitis B surface antigen. *Am J Epidemiol* 105:94-98, 1977.
4. Berquist, K.R., Maynard, J.E., and Murphy, B.L. Infectivity of serum containing HB_sAg and antibody to e antigen. *Lancet* 1:1026-1027, 1976.
5. Barker, L.F., Murray, R. Relationship of virus dose to incubation time of clinical hepatitis and time of appearance of hepatitis-associated antigen. *Am J Med Sci* 263:27-33, 1972.
6. Heathcote, J. and Sherlock, S. Spread of acute type-B hepatitis in London. *Lancet* 1:1468-1470, 1973.
7. Bancroft, W.H., Snitban, R., Scott, R.M., et al. Transmission of hepatitis B virus to gibbons by exposure to human saliva containing hepatitis B surface antigen. *J Infect Dis* 135:79-85, 1977.
8. Alter, H.J., Purcell, R.H., Gerin, J.L., et al. Transmission of hepatitis B to chimpanzees by hepatitis B surface antigen-positive saliva and semen. *Infect Immun* 16:928-933, 1977.
9. MacQuarrie, M.B., Forghani, B., and Wolochow, D.A. Hepatitis B transmitted by a human bite. *JAMA* 230:723-724, 1974.
10. Mosley, J.W., Edwards, V.M., Casey, G., et al. Hepatitis B virus infection in dentists. *N Engl J Med* 293:729-734, 1975.
11. Feldman, R.E. and Schiff, E.R. Hepatitis in dental professionals. *JAMA* 232:1228-1230, 1975.
12. Levin, M.L., Maddrey, W.C., Wands, J.R., et al. Hepatitis B transmission by dentists. *JAMA* 228:1139-1140, 1974.
13. Rimland, D., Parkin, W.E., Miller, G.B., et al. Hepatitis B outbreak traced to an oral surgeon. *N Engl J Med* 296:953-958, 1977.
14. Williams, S.V., Pattison, C.P. and Berquist, K.R. Dental infection with hepatitis B. *JAMA* 232:1231-1233, 1975.
15. Tzuket, A. and Sandler, S.G. Dental care and spread of hepatitis B virus infection. *J Clin Microbiol* 8:302-305, 1978.
16. Expert Group on hepatitis in dentistry. Hepatitis in dentistry. *Br Dent J* 146:123-124, 1979.
17. Shields, W.B. Dentistry and the issue of hepatitis B. *JADA* 102:180-182, 1981.
18. Taylor, P.E. Laboratory tests for Australia antigen and antibody. *Br Med Bull* 28:131-141, 1972.
19. SyWassink, J. and Lutwick, L.I. The risk of hepatitis B in dental care deliverers: a contact study. *JADA*, in press.
20. Centers for Disease Control: Immune globulin for protection against viral hepatitis. *Morbidity and Mortality Weekly Report* 30:423-428, 433-435, 1981.
21. Grady, G.F. and Lee, V.A. Hepatitis B immune globulin—prevention of hepatitis from accidental exposure among medical personnel. *N Engl J Med* 293:1067-1070, 1975.
22. Hoofnagle, J.H., Seeff, L.B., Bales, Z.B., et al. Passive-active immunity from hepatitis B immune globulin. Reanalysis of a Veterans Administration Cooperative Study of needlestick hepatitis. *Ann Intern Med* 91:813-818, 1979.
23. Tenney, J. and Kahn, S. Postexposure hyperimmune globulin prophylaxis: an inadequate method for prevention of hepatitis B in hospital personnel. Second International Conference on Nosocomial Infection, Atlanta, Georgia, August 1980.
24. Purcell, R.H. and Gerin, J.L. Hepatitis B vaccines. On the threshold. *Am J Clin Path* 70 (Suppl):159-169, 1978.
25. Gerety, R.J., Tabor, E., Purcell, R.H., et al. Summary of an international workshop on hepatitis B vaccines. *J Infect Dis* 140:642-648, 1979.
26. Szmuness, W., Stevens, C.E., Harley, E.J., et al. Hepatitis B vaccine. Demonstration of efficacy in a controlled clinical trial in a high-risk population in the United States. *N Engl J Med* 303:837-841, 1980.
27. Szmuness, W. Hepatitis B vaccine: a final report of a controlled clinical trial in homosexual men. 1981 International Symposium on Viral Hepatitis, New York, April 1981.
28. Crosnier, J., Jungus, P., Courouze, A.M. et al. Randomized placebo-controlled trial of hepatitis B surface antigen vaccine in French hemodialysis units: I, Medical staff. *Lancet* 1:455-459, 1981.

Information for Authors

All manuscripts must be accompanied by the following written statement, signed by one author: "The undersigned author transfers all copyright ownership of the manuscript entitled (*name of the article*) to the American Academy of Pedodontics should the work be published. The undersigned author warrants that the article is original, is not under consideration by another Journal, and has not been published previously. I sign for and accept responsibility for releasing this material on behalf of any and all coauthors." Authors will be consulted, when possible, regarding republication of their material.