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Hepatitis: a new crisis in pedodontics

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Abstract

Pedodontists by and large have not been concerned with viral hepatitis as a health hazard in practice. However, based upon the high incidence of viral hepatitis in children and adolescents from lower socioeconomic areas, of recent immigration from Africa, Asia, or other tropical countries, with certain handicapping conditions, or receiving certain pharmacodynamic therapy, abusing drugs, with male homosexual precocious sexual activity, and from other environments conducive to viral hepatitis many pedodontic practices are at risk for disease transmission. Exposure to viral hepatitis often necessitates direct immune globulin prophylaxis specific to the type of viral hepatitis. All pedodontic practices should review and update their disease control protocol.

Introduction

Pedodontists in the past have demonstrated an alarming lack of concern for the health hazards of viral hepatitis. Two epidemiological factors may have contributed to this paucity of concern. First, pedodontists as a specialty group have had the lowest incidence of viral hepatitis exposure of any specialist or generalist groups in most serological studies of dental professionals. Second, serological evidence indicating exposure to viral hepatitis increases throughout life with children generally exhibiting lower exposure rates than adults. These two factors, although still credible in the 1980's, must be analyzed more cautiously in view of present disease morbidity data. Children from certain socioeconomic areas, immigrant children from certain African, Asian, and tropical countries, and certain handicapped children have now been shown to have a high incidence of exposure to viral hepatitis. In view of these findings it appears that viral hepatitis may become a new crisis in pedodontics.

Viral Hepatitis

Viral hepatitis may be differentiated into three clinically similar, but immunologically and epidemiologically different types of disease (Table 1). Hepatitis A viral disease (HAV), previously referred to as infectious hepatitis, is sporadic, epidemic within families, has an incubation period of between two and six weeks, and is not dependent upon the route of inoculation. A viremia occurs during the incubational and acute phases of HAV. Rarely does HAV produce a carrier state after the clinical phase. Morbidity is age-related with asymptomatic infection and anicteric illness predominating in children. Mortality is usually less than 1%.

Hepatitis B viral disease (HBV), previously referred to as serum hepatitis, has an incubation period of between six and 26 weeks depending upon the route of inoculation with the oral route usually resulting in the longer incubation times. Again, a viremia occurs during the incubational and acute phases and, occasionally, post-disease patients may be chronic carriers. Morbidity and mortality are variable depending upon severity of viremia and age of patient.

Hepatitis Non-A, Non-B is the third and most recently discovered type. It is thought to be a major

Table 1. Viral hepatitis.

	Type A	Type B	Type Non-A, Non-B
Agents	Hepatitis A Virus	Hepatitis B Virus	Non-A, Non-B Hepatitis Viruses
Incubation	2-6 weeks	6-26 weeks	6-26 weeks
Occurrence	Epidemic	Point source	Post-transfusion
Carrier State	Rare	Yes	Yes

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cause of transfusion related hepatitis and is probably the result of more than one of the causative viruses that have been implicated. The incubation and clinical course are similar to HBV, but serological and immunological studies are exclusive for HBV. Cases of sporadic, clinically obvious hepatitis in urban centers are approximately 25% HAV, 50% HBV, and 25% Non-A, Non-B.[1]

Clinically, all types appear undifferentiated with respect to liver pathology with histological evidence of tissue inflammation and cell necrosis. The major complications and high incidence of mortality can be attributed to massive hepato-cellular necrosis. The clinical course of the disease is characterized by three periods (Table 2).

Table 2. Clinical phases of viral hepatitis.

I	*Anicteric* (non-jaundiced) prodromal period with symptoms of loss of appetite, abdominal pain, nausea, general weakness, and malaise (flu-like symptoms).
II.	*Icteric* (jaundiced) period with classic signs of jaundice.
III.	*Recovery* period with no symptoms but with complete clinical and biochemical recovery.

The first clinical phase is usually the anicteric (non-jaundiced) prodromal period characterized by loss of appetite, abdominal pain, nausea, general weakness, and malaise. The second clinical phase is the icteric (jaundiced) period characterized by the classic signs and symptoms of jaundice. The final clinical phase is the recovery period which is usually three to six weeks in duration where the patient usually has no symptoms. Modern therapeutic regimens include diet management and reduced physical activity. Complete clinical and biochemical recovery often takes from three to four months with periodic evaluation of liver function and serological tests necessary. Hepatitis has been detected in asymptomatic patients by the use of liver function and serological tests.[2-4] It is possible for subclinical cases to exist mimicking a flu-like syndrome.

Since 1966, with the discovery of the Blumberg "Australian" antigen, now known as the Hepatitis B surface antigen (HBsAg), it became possible to recognize through serological testing the hepatitis B viral disease (HBV). It further became possible to differentiate B from other Non-B hepatitis diseases and to identify chronic carriers of the B virus.[5-7] Typically, the HBsAg develops 20 to 100 days after exposure, is detectable in the serum for one to 120 days after development, and then disappears within four months. The serum becomes negative for the HBsAg and, in most instances, antibodies specific to HBsAg (anti-HBs) develop and are detectable in the serum for many years thereafter. Immunity is evidenced in a person by the presence of these anti-HBs which are serum markers for previous HBV infection.[8,9]

The Dane particle is a 42 nm particle with HBsAg on its surface and hepatitis B core antigen (HBcAg) in its core. It is the intact virus (virion) — a double-shelled particle enclosing a unique circular double-stranded DNA and a specific DNA polymerase. These antigenic determinants are shared by numerous smaller spherical and tubular particles which presumably represent HBV surface antigen produced in gross excess by infected hepatocytes. The Dane particle is the only viral structure known to contain nucleic acid with DNA sequences capable of integrating into chromosomal CNA of HBV-infected liver.

A circulating protein antigen exclusively seen in HBV infection termed the Hepatitis B "e" antigen (NBeAg) is probably a structural antigen of HBV linking surface to core antigen layers, and is viewed as a serum marker of active virus replication and thus infectivity. A carrier of HBV is thought to be a better transmitter, or more infectious, if the carrier also has a positive test for HBeAg.

Only about .01% of HBV particles are intact infectious viruses, but since HBV particles circulate in enormous titers of approximately 10^{12} HBV particles/ml of blood, the observation of approximately 10^6 Dane particles/ml of blood is significant. Therefore, the amount of infected blood that can transmit HBG is relatively small (10^5 to 10^6 ml).[10,11]

Sensitive serological tests have now demonstrated that hepatitis can be attributed to hepatitis A virus, hepatitis B virus, and Non-A, Non-B viruses. Non-A, Non-B hepatitis is a viral hepatitis-like syndrome in the absence of serological evidence for hepatitis A virus (HAV), hepatitis B virus (HBV), cytomegalovirus (CMV), or Epstein-Barr virus (EBV). No serological test to date is available on humans for Non-A, Non-B viruses. Evidence, however, does exist indicating that more than one virus is responsible for Non-A, Non-B hepatitis.[12-16]

Carrier states exist for hepatitis B and Non-A, Non-B hepatitis, but are extremely rare for hepatitis A. Approximately 5-10% of persons infected with hepatitis B will circulate HBsAg for up to two years or for as long as 25 years before seroconversion to antibodies.[4,8,17,18] In general, however, HBsAg rarely persists for more than four months. Studies report the incidence of prolonged or chronic active liver disease to be 20-40% for Non-A, Non-B hepatitis, which appears higher than for hepatitis B and may indicate a greater ability in perpetuating a chronic carrier state.[19] Some carriers have been reported as healthy in the absence of progressive liver disease, but may still be infec-

tious. A defective autoimmune response may prevent carriers from seroconverting to antibodies. It is the propensity to produce chronic hepatitis and chronic carrier states that makes control of hepatitis B and Non-A, Non-B hepatitis extremely frustrating.

The transmission factors are likewise specific according to the type of hepatitis (Table 3). Hepatitis A is essentially transmitted by fecal-oral contact with infectivity decreasing greatly by the time overt clinical hepatitis develops. Therefore, the maximum infectivity time occurs prior to acute illness during incubation. Serological IgM-anti-HAV testing is often performed to detect acute HAV infection. Parenteral transmission of HAV is rare. Hepatitis B and Non-A, Non-B hepatitis are transmitted by both parenteral and non-parenteral contact. Although hepatitis B and Non-A, Non-B can be transmitted within families, true epidemic hepatitis is usually hepatitis A.

Among the body fluids not apparently blood contaminated that can transmit HBV and probably Non-A, Non-B agents are: saliva, nasopharyngeal secretions, tears, breast milk, vaginal secretions, semen, urine and feces. Many of these fluids, however, contain very low levels of the antigen. Saliva was positive for HBsAg in 76% of patients with acute hepatitis B and 80% of the chronic carriers of HBV.[20,21] Gingivitis with accompanying micro-leakage of blood into the oral cavity has been cited as a factor also.[22-25] It appears that the non-parenteral routes, however, are not as effective in transmission as the parenteral routes. More serological research is necessary before the various non-parenteral routes for Non-A, Non-B agents can be studied and confirmed.

Table 3. Transmission Factors.

	Parenteral	Non-Parenteral
HAV	Rare	Very common
HBV	Common	Common, less effective
HNANB	Common	Common, less effective

Hepatitis B Exposure Studies in Dental Professionals

It is estimated that approximately 3.5% persons in the United States have had hepatitis B with a carrier rate of approximately 0.5-1.0%. Surveys have reported a 6.3% to 20.8% (average 13.6%) range for previous exposure to hepatitis B in general dentists with a carrier rate of approximately 1% to 2%.[8,26-38] It is estimated that in health care workers the incidence of the HBsAg carrier rate is approximately 1% to 2%.[17] Surgical dental specialty groups have a reported increase in this exposure rate with oral surgeons having a 27% total exposure rate and a 2% to 3% carrier rate and endodontists having a 9% total exposure rate.[8,37,38]

Periodontists in unpublished data are reported to have an exposure rate in this same range.[39] Orthodontists in unpublished data are reported to have an exposure rate of 8.9%.[39] Pedodontists as a group, however, have never been tested but usually have the lowest incidence of dentists tested. Dental auxillaries appear to have a low exposure incidence.[39,40] Physicians and nurses have an average exposure rate of 15% to 13% respectively.[41] Physicians specializing in family practice, internal medicine, pediatrics, and obstetrics-gynecology have exposure rates of approximately 18%.[42] Other health personnel at high risk, however, are kidney dialysis staff personnel at 28% and clinical laboratory personnel at 19%.[41] It now appears that exposure trends develop during professional education as evidenced by the increasing incidence in dental school classes[43,44] (Table 4).

Table 4. Hepatitis B Exposure Rate in health-professionals.

Health Group	Approximate HBV Exposure Rate
General Physician	~ 15%
Surgical Physician	~ 27%
Pathologist	~ 27%
Pediatricians	~ 18%
Administrative Physician	~ 4%
Kidney Dialysis Staff	~ 28%
Clinical Lab Staff	~ 19%
General Dentists	~ 14%
Oral Surgeons	~ 27%
Endodontists	~ 9%
Periodontists	~ 9%
Orthodontists	~ 9%
Dental Auxillaries	~ 7%
Dental Students	~ 5%
General Population	~ 3.5%

Viral Hepatitis in Children and Adolescents

Hepatitis A accounts for more than 31,000 cases (15 per 100,000) in the United States yearly.[45] Sero-epidemiological studies have reported that over half the U.S. population has evidence of past infection by mid-adult life.[46] Serological surveys in New York have demonstrated that anti-HA is present in 30% to 75% of adults, depending largely on socioeconomic factors.[47] Because of the high proportion of asymptomatic, anicteric, undiagnosed hepatitis in children, age-specific incidence rate for hepatitis A does not accurately reflect this problem in children and adolescents.

Accurate reporting in all age categories is probably no better than one out of every five cases. In children, this is further exaggerated by the low frequency of icteric infection which is 1:3 to 1:10.[48] Anti-HA inci-

dence has been reported to be between approximately 10% to 30% in lower socioeconomic children and only 2% in upper class children.[49-51] This may indicate that hepatitis A occurs in all ages rather than primarily children and that anti-HA increases with increasing age. It appears that socioeconomic factors play a major role in this incidence.

Hepatitis B has increased ten times in incidence since 1966 with approximately 17,000 cases (eight per 100,000) reported yearly.[45,52] Surveys of first time blood donors from the general U. S. population indicate that approximately 3.5% have anti-HBs indicating previous HBV infection. The carrier rate is estimated at between 0.5% and 1.0% of the population seropositive for HBsAg.[46] This is consistent in children and adolescents with a reported 4.1% incidence of anti-HB in children 0-19 years of age in middle class Staten Island, New York.[53]

However, a recent study of children from diverse ethnic and socioeconomic groups in New York City below age 10 reported incidences of anti-HBs in Caucasians at 5.2% in Hispanics at 16.4%, and in blacks at 21.7%.[54] Anicteric cases of hepatitis B are again exaggerated in children with a low frequency of 1:20.[47] It appears that in hepatitis B socioeconomic factors also play a major role in epidemiology.

Hepatitis B may be contracted by young children in several situations. Newborn children may have obtained a hepatitis B infection from a hepatitis B carrier mother by transplacental transmission, direct transmission during birth process, and transmission by mother's milk.[55-56] This vertical transmission process is now under close scrutinizing review. It appears that intrauterine infection is not an important mode of maternal-fetal transmission. Since most children develop antigenemia three to five months after birth, the time of infection is most likely during labor and delivery.[61] Breastfeeding has definitely been implicated because of the possibility of the virus or antigen being present in human milk and the possible ingestion of infectious serum that commonly exudes from cracked nipples.[65]

Areas of the world with high endemic rates of chronic carrier states have reported high rates of maternal-infant transmission.[67] Although it has been previously reported that infants became persistent carriers, more frequent serological testing of infants may indicate a lower rate of carrier state development.[67] Frequent occurrence of carrier states in infants in certain countries may be due to an inadequate immune response related not to immaturity of the immune system, but rather to environmental or genetic factors of a polygenic nature.[68] Viral hepatitis B has the potential, then, to be a severe disease in young children and could very well account for the large numbers of chronically infected adolescents and adults in our general population.[60] Serological evidence again documents for hepatitis B that exposure increases throughout life. Further studies are necessary to establish population characteristics that influence frequency at various ages.

Nonparenteral transmission of hepatitis B in normal family units has been suggested.[69] This has been rare in the U.S., possibly due to the low incidence of carrier-state children, yet common in African, Asian, and tropical countries. Introduction of a carrier-state child in a family unit with subsequent infection of other family members has been reported.[70] Studies of family members of individuals with HBsAg have revealed that spouses and sexual partners develop serological evidence of infection more frequently than other household contacts.[71]

Currently the U.S. age group most vulnerable to viral hepatitis is young adults (20-24 years) followed by 15-19 year olds and 25-29 year olds. For hepatitis A there is a preceding but smaller peak of incidence in the five to nine year olds. Although there was a lack of hepatitis B cases in persons less than 15 years old prior to 1977, this is no longer true.[46] Substantial geographic differences in incidence have always been reported, with the Pacific areas continuing to demonstrate the highest incidence in the U.S.[45] Seasonal variation for viral hepatitis has, however, diminished remarkably.[46]

Non-A, Non-B hepatitis accounts for approximately 20% of adult sporadic viral hepatitis. However, it accounts for 80% to 90% of all transfusion related hepatitis with the remaining being hepatitis B.[14,72] The blood products necessary for replacement therapy are clearly recognized as the source of infection with increased risk from commercial donor blood and blood concentrates derived from large donor pools.[47,73] Until specific serological testing on humans is available, epidemiological interpretations regarding incidence and age specificity are invalid.

Viral Hepatitis in Specific Patient Populations

Based upon the precarious asymptomatic and anicteric nature of viral hepatitis, it is not surprising that many people have no knowledge of their previous infection or present carrier state. Those stating a history of hepatitis usually do not know what type they had. Some people who have previously been refused health care because of admitting to a past episode of hepatitis often withhold this information when seeking health care again.

Studies specific for dental patients have confirmed these facts.[74-77] There are, however, certain specific

patient populations noted to be at higher risk for hepatitis exposure (Table 5).

In 1972 approximately 70% of hepatitis B infection was precuteaneous drug associated with the largest incidence in the 15-29 year age group.[78] Drug abusers may have a carrier rate approximating 50%.[18] Carrier states appear not to be maintained if drug abuse is eliminated.[79] Up to 85% of drug abusers show serologial evidence for HBV infection.

Numerous studies have confirmed that patients of institutions for the mentally retarded or severely disabled have a significant increase in the incidence of hepatitis A and hepatitis B.[51,80-83] A range of 10% to 30% for prevalance of HBsAg in institutional populations has been reported, with patients having Down's Syndrome approximating 50%.[80,82] Patients undergoing hemodialysis (renal dialysis) have greater risk of hepatitis A and hepatitis B exposure.[84,85] Serological evidence of HBV infection occurred in 50% of the patients and 34% of the staff at 15 national dialysis centers. The carrier rate in the patients, however, was 16%.[85] Kidney and other organ transplant patients and other immunosuppressed patients (leukemia and any immunodeficient disease) have now been shown to be at greater risk for viral hepatitis.[86] Dialysis and transplant carriers now account for approximately 33% of all accidental HBV infection in needlestick episodes.

Patients receiving transfusion, especially those receiving multiple transfusion and/or concentrate transfusion for coagulation disorders, are at great risk for transfusion related hepatitis.[87] Transfusion related hepatitis is approximately 10%-20% hepatitis B and 80%-90% Non-A, Non-B hepatitis.[14,72] Anti-HBs have been demonstrated in 90% of hemophilia patients treated with these blood products.[14,88] Post-transfusion hepatitis B has been drastically reduced since initiation of routine blood bank screening of blood donors. Episodes, however, are still reported from blood donor units due to low-level infectious chronic HBsAg carriers in whom the HBsAg titers fluctuate and can escape the radioimmunoassay detection prior to blood donation.[89]

An epidemiological investigation of post-transfusion hepatitis indicates that the frequency of both hepatitis B and Non-A, Non-B hepatitis can be reduced by reducing commercial blood donors in relation to volunteer blood donors (34.6% vs. 6.7).[14] With the advent of more sophisticated serological tests it might soon be possible to prescreen blood donors for identification and detection of low level titers of HBsAg in blood donors. Male homosexuals who have frequent intimate contact with different partners are at great risk for viral hepatitis. Some studies indicate that over 50% have some serological marker for HBV infection. Male homosexuals currently represent about 16% of the total HBsAg carriers in the U.S.[90]

Table 5. Viral Hepatitis in specific patient population.

Drug abusers (precutaneous)
Institutionalized mentally retarded patients, especially those with Down's Syndrome
Hemodialysis patients
Immunosuppresed patients
Post-transfusion patients, especially hemophilia patients
Male homosexuals
Refugees or immigrants from Africa, Asian, and other tropical countries

Other patient categories with higher exposure rates for viral hepatitis include patients with a history of familial association with the disease, chemotherapy or radiation therapy which suppress immunological defenses, tattooing, ear-piercing, acupuncture and other needlesticking procedures from non-professionals, and other liver diseases.[91,92]

The Refugee Crisis

Epidemiological studies in Africa, Asia, and other tropical countries demonstrate that 5%-10% of the general population in these countries have chronic HBV infection with maternal-infant vertical transmission being the dominant mechanism maintaining this high prevalence.[93] Most of these carriers are asymptomatic with no knowledge of previous exposure. There is a preferential persistence in the young males. Since the African or Asian carrier will usually circulate virus titers higher than North American HBsAg carriers, they are likely to be more infectious.

The recent influx of Southeast Asian refugees has dramatized not only the plight of these people but also their special health problems. With approximately 14,000 Indochinese refugees arriving in the United States monthly, an awareness of their specificity for viral hepatitis is mandatory. Over 150,000 Vietnamese refugees have now been resettled in the United States since 1975.

Current U.S. immigration and naturalization laws list the following conditions that do not exclude a refugee from entry if they are adequately treated: tuberculosis, leprosy, chronic alcoholism and drug addiction. By law, health screening of refugees prior to entry for tuberculosis, leprosy, and venereal disease is necessary.[94] By law, viral hepatitis screening is not necessary. The U.S. Department of Health, Education and Welfare, however, is making a concerted effort to have these screenings performed upon entry into the U.S. Concern has been expressed regarding the trans-

mission of tuberculosis and hepatitis to the population of the U.S. by these refugees.

Screening data on Indochinese refugees entering North America indicate that 12%-13% of them are positive for HBsAg with most individuals being asymptomatic and unaware of their chronic carrier state.[95,96] This addition to the U.S. carriers represents only a 4% increase in total carriers for the U.S. and poses only a small risk to the general population.[96] Since the refugees are often screened and identified as chronic carriers, the risk they represent may be less than that from unrecognized carriers in the general population and other high risk patient groups. Testing for HBsAg and entering the results in the refugee's medical record is recommended by the Center for Disease Control of each Indochinese refugee after arrival into the U.S.[97] Since many of these refugees have significant dental disease, the dental professional may be the first health care person requesting this information.[98] To assist with problems in language differentials, the U.S. Department of Health, Education and Welfare has established a toll free number (800-424-0212) for use by health care professionals.[99]

Among adopted Vietnamese children studied, HBsAg and anti-HBs incidence was 27.3% and 30.3% respectively, with other family members having HBsAg and anti-HBs incidence of 0% and 13.5% respectively, with 23.1% of the children being positive for anti-HBs.[70] Transmission of both hepatitis A and hepatitis B has been documented in family units with HBsAg-positive adopted children.[70] Therefore, other family members, especially siblings of adopted refugees, should be considered a high-risk patient group.

Disease Transmission in Dental Practice

In practice, dental personnel are continually exposed to the blood of patients through minor hand abrasions or accidental inoculation by needle or instrument stick. Although not as effective as blood, saliva does appear to be the most efficient transmission vector of the non-parenteral body fluids. Since this fluid contaminates not only dental personnel, but most dental instruments and equipment, there is a need for genuine concern in dental practice regarding hepatitis transmission. Transmission in the dental office has been well documented.[100-103] Therefore, the dental personnel should be keenly aware of aseptic technique and proper disease transmission safeguards. Numerous articles have been presented in the literature concerning the prevention of transmissable disease. A review of these articles should be paramount for dental personnel.[8,23,91,104-115] This article is not meant to diminish their importance, but rather to emphasize that the subject of prevention of all transmissible diseases is in itself an entire area for review.

Exposure Protocol for Viral Hepatitis

Exposure to viral hepatitis may involve both parenteral and non-parenteral exposure. The importance of differentiating this exposure for an episode is often critical to exposure protocol. When in doubt regarding the episode of exposure for a particular type of viral hepatitis, assume the exposure regardless of parenteral versus non-parenteral transmission-caused-disease inoculation until proven otherwise.

Immume globulins are sterile, intramuscular solutions containing antibodies derived from human donor blood. One type of immune globulin (ISG) contains varying amounts of antibodies for hepatitis A and hepatitis B depending upon preparation after 1972. Titer levels of antibody for hepatitis B can attain 1:5000 by PHA in some preparations but must be greater than 1:64. The cost is approximately $15-$20 for one dose of this ISG. Neither hepatitis A nor hepatitis B has been transmitted by immune globulin. Immune globulin with high titers of anti-HBs (greater than 1:100,000 by PHA), prepared from donor pools preselected for these antibodies, are now designated as hepatitis B immune globulin (HBIG). The cost is approximately $150 for one dose of this HBIG. Neither hepatitis A nor hepatitis B has been transmitted by HBIG. Prophylactic administration of immune globulins has been both pre-exposure and post-exposure. Immune globulin preparations rarely cause adverse reactions and are not contraindicated during pregnancy.[46]

Although exposure to hepatitis A usually involves non-parenteral, fecal-oral contamination, experimental animal studies have demonstrated parenteral transmission. The importance of this to health care professionals is unknown.[116] Viral excretion of HAV will peak during late incubation and early illness, and drop rapidly with the onset of jaundice. Maximum infectivity will occur usually two weeks, then, before the onset of jaundice. When administered before, or within, one to two weeks after exposure to hepatitis A in appropriate dose, ISG prevents illness in 80-90% of those exposed. The use of ISG more than two weeks after exposure or after onset of clinical illness is not indicated. Pre-exposure prophylaxis may be indicated for exposure to non-human primates and for travel to some foreign countries. Post-exposure prophylaxis is recommended only in cases of close personal contact, institutional contact, and common source contact.[46]

Exposure to hepatitis B is perplexed by the infectivity of its chronic carrier state. Furthermore, the value of HBIG versus the newer preparations of ISG has been debated over the past few years. There is evidence that HBIG, compared with low titer anti-HBs ISG, merely prolongs the incubation period and leads to reduced incidence of passive-active immunity.[117]

Currently the U.S. Public Health Service Advisory Committee on Immunization Practices unequivocably recommends HBIG only for known exposure to blood containing hepatitis B virus by accidental needle-stick or by contact with mucous membranes.[46] One dose of HBIG must be administered within a one-week period after this exposure, and a second identical dose administered one month after the first.

A single dose has been further recommended for infants within seven days of birth if born to HBsAg positive mothers with acute hepatitis occurring in the third trimester.[46] It has been further recommended that all cases of percutaneous, oral, mucosal, sexual, or neonatal exposure to patients with acute hepatitis B, or in cases of time-limted exposure to chronic carriers having markers denoting infectivity, be administered the HBIG regimen.[117]

Pre-exposure prophylaxis has been, at times, recommended for patients and staff of hemodialysis units and custodial staffs of institutions for the mentally retarded.[46] It should be noted, however, that only 6% of the persons who are accidentally contaminated with needle-stick blood from an HBsAg positive donor develop hepatitis B.[116] If HBIG is not available for use in the above-mentioned episodes, ISG can be administered similarly. The theoretical advantage of HBIG over ISG remains controversial. A vaccine utilizing purified HBsAg is now being developed and may be replacing HBIG in the next few years.

Exposure to Non-A, Non-B hepatitis is also perplexed by lack of serological identification and existence of a chronic carrier-state that is infective. Although not scientifically based, any exposure dictates the administration of ISG within one week. It appears from limited studies that ISG has been of benefit in reducing the total incidence of hepatitis, the icteric cases, and the chronic sequelae.[116, 118] (Table 6).

Assessment of Pedodontic Practice

It would behoove each pedodontist to assess his or her practice for risk determination. Those pedodontists and their staff that deliver dental health care to children and adolescents from lower socioeconomic areas, of recent immigration from Africa, Asian, or other tropical countries, with certain handicapping conditions, or receiving certain pharmaco/dynamic treatment, abusing drugs; male homosexual children and adolescents involved in precocious sexual activity, and children and adolescents from any other environment conducive to viral hepatitis must be considered at risk.

Pedodontists and their staff that are at risk should have serological tests to determine their past hepatitis exposure and/or present immunity. These tests should be performed on personnel at risk at least every two years and optimally every year. All practices, not just those at risk, should routinely use advanced disease control aseptic techniques. Pedodontists and their entire office staff should review and update these disease control methods on a periodic basis.

Dental practice should commence with a thorough patient background analysis including specific health history data and personal information that might detect a patient in one of the risk categories. As comprehensive as this may be, not every suspected risk patient can or will be detected. Because of this, routine prevention for disease transmission is mandatory in the pedodontic practice.

Table 6. Exposure protocol for viral hepatitis.

Type	Immunoglobulin
Hepatitis A	ISG within 1-2 weeks after exposure.
Hepatitis B	HBIG within 1 week after exposure and again in 1 month. If HBIG is unavailable, ISG may be substituted.
Hepatitis/Non A, Non B	ISG within 1 week after exposure.

Management of Children in the High Risk Groups

Management of Children and adolescents in the high risk groups may require a specific office/facility protocol dependent upon the level of sophistication of disease transmission prevention. Optimally, all high risk patients should have serological identification and be afforded the highest level of disease transmission prevention. This is often not possible or practical in all circumstances. If serological identification is unknown or uncertain for a given patient in the high risk group, it is advisable to consider this patient seropositive and able to transmit viral disease. The highest level of prevention of disease transmission should therefore be adopted.

Approximately half of seropositive patients may have progressive chronic liver disease with continued hepatocellular necrosis. This is determined by evaluation of liver enzyme function. If oral surgical procedures are to be done on seropositive patients, not only should the highest level of preventive disease transmission procedures be utilized, but liver function should be ascertained to prevent possible problems in bleeding and drug metabolism and/or detoxification. If drug therapy requiring metabolism and/or detoxification in the liver is anticipated on a seropositive pa-

tient, liver function should likewise be ascertained prior to therapy.

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References

1. Dienstag, J. L., et al: "Etiology of sporadic hepatitis B surface antigen negative hepatitis," *Ann Int Med,* 87:1-6, 1977.
2. Krugman, S.: "Hepatitis: current studies of etiology and prevention," *Hosp Prac* 10(11):39-46, 1975.
3. Krugman, S., Giles, J. P., and Hammond, J.: "Infectious hepatitis: evidence of two distinctive clinical, epidemiological, and immunological types of infection," *JAMA,* 200:365-372, 1967.
4. Redeker, A. G.: "Viral hepatitis: clinical aspects," *Am J Med Sci,* 270:9-16, 1975.
5. Blumberg, B. S., Sutnick, A. I., and London, W. T.: "Hepatitis and leukemia: their relation to Australian antigen," *Bul, N Y Acad Med,* 44:1566-1586, 1968.
6. Prince, A. M.: "An antigen detected in the blood during the incubation period of serum hepatitis," *Proc Nat Acad Sci U S A,* 60:814-821, 1968.
7. Prince, A. M., et al: "The serum hepatitis virus specific antigen (SH): a status report," in *Perspectives in Virology from Molecules to Man,* ed. Pollard, M., New York: Academic Press, 1971, Vol. 7, p 241.
8. Council on Dental Therapeutics: "Type B (serum) hepatitis and dental practice," *JADA,* 92:153-159, 1976.
9. Brunell, P. A.: "Hepatitis," in *Nelson Textbook of Pediatrics,* Vaughan, V. C., McKay, R. J., and Behrman, R. E., 11th ed., Philadelphia: W. B. Saunders Co., 1979, pp 907-914.
10. Robinson, W. S., and Lutwick, L. J.: "The virus of hepatitis, type B," *N Engl J Med,* 295:1168-1174, 1232-1236, 1976.
11. Magnius, L. O., and Espmark, J. A.: "New specificities in Australia antigen positive sera distinct from LeBouvier determinants," *J Immunol,* 109:1017-1021, 1972.
12. Mosley, J. W., et al: "Multiple hepatitis viruses in multiple attacks of acute hepatitis," *N Engl J Med,* 296:75-78, 1977.
13. Hruby, M. A., and Schauf, V.: "Transfusion related short incubation hepatitis in hemophilic patients," *JAMA,* 240:1384-1385, 1978.
14. Feinstone, S. M., and Purcell, R. A.: "Non-A, Non-B hepatitis — editorials," *JAMA,* 240:1384-1385, 1978.
15. Alter, H. J., et al: "Transmissible agent in Non-A, Non-B hepatitis," *Lancet,* 1(1):459-463, 1978.
16. Tabor, E., et al: "Transmission of Non-A, Non-B hepatitis from man to chimpanzee," *Lancet* 1(1):463-466, 1978.
17. Chambers, T. C., and Alter, H. J.: "Management of the asymptomatic carrier of the hepatitis-associated (Australia) antigen. Tentative considerations of the clinical and public health aspects," *N Engl J Med,* 285:613-617, 1971.
18. Ling, C. M., and Overby, L. R.: "Prevalence of hepatitis B virus antigen as revealed by direct radioimmune assay with 125 I-antibody." *J Immunol,* 109:834-841, 1972.
19. Knodell, R. G., Conrad, M. E., and Ishak, K. G.: "Development of chronic liver disease after acute Non-A, Non-B hepatitis post-transfusion hepatitis," *Gastroenterology,* 72:902-909, 1977.
20. Heathcote, J., Gateau, P., and Sherlock, S.: "Role of hepatitis B antigen carriers in non-parenteral transmission of the hepatitis B virus," *Lancet,* 2(2):370-371, 1974.
21. Villarejos, V. M., et al: "Role of saliva, urine, and feces in the transmission of type B hepatitis," *N Engl J Med,* 291:1375-1378, 1974.
22. Gilbert, R. I., et al: "Hepatitis B antigen in saliva, urine, and stool," *Infection and Immunity,* 11:142-145, 1975.
23. Crawford, J. J.: "New light on transmissibility of viral hepatitis in dental practice and the control," *JADA,* 91:829-835, 1975.
24. Ward, R., et al: "Hepatitis B antigen in saliva and mouth washings," *Lancet,* 2:726, 1972.
25. MacQuarrie, M. B., Forghani, B., and Wolochow, D. A.: "Hepatitis B transmitted by human bite," *JAMA,* 230:723-724, 1974.
26. Jones, D. M., Tobin, T. O. H., and Turner, E. P.: "Australia antigen and antibodies to Epstein Barr virus, cytomegalovirus, and rubella virus in dental personnel," *Br Dent J,* 132:489-491, 1972.
27. Glenwright, H. D., et al: "Serum hepatitis in dental surgeons," *Br Dent J,* 136:409-413, 1974.
28. Berris, B., et al: "Letters: frequency of hepatitis in dentists in Ontario," *Ann Int Med,* 81:699-700, 1974.
29. Berris, B., et al: "Frequency of hepatitis in dentists in Ontario," *Ont Dent,* 52:16-17, 1975.
30. Feldman, R. E., and Schiff, E. R.: "Hepatitis in dental professionals," *JAMA,* 232:1228-1230, 1975.
31. Mosley, J. W., et al: "Hepatitis B virus infection in dentists," *N Engl J Med,* 293:729-734, 1975.
32. Mosley, J. W., and White, E.: "Viral hepatitis as an occupational hazard of dentists," *JADA,* 90:992-997, 1975.
33. McGinnis, J. P., and Mincer, H. H.: "Occurrence of hepatitis B serum antigen and antibody in faculty of the University of Tennessee College of Dentistry," *J Tenn Dent Assoc,* 56:100-103, 1976.
34. Smith, J. L., et al: "Comparative risk of hepatitis B among physicians and dentists," *J Inf Dis,* 133:705-706, 1976.
35. Weil, R. B.: "Preliminary report of hepatitis B serological survey," *N Y S Dent J,* 42:531-537, 1976.
36. Spearman, J., and Bader, M.: "Letters to the editor: dentistry and hepatitis," *JADA,* 94:25, 1977.
37. Goldstein, J., Morse, K., and Gullen, W.: "Epidemiology of hepatitis B among endodontists," *J Endodont,* 4:336-339, 1978.
38. Goldstein, J.: "Results of the AAE hepatitis survey in Las Vegas," *J Endodont,* 4:340, 1978.
39. Rathbun, G.: Personal communication, 1980.
40. James, S. P., and Sampliner, R. E.: "Hepatitis B in the dental setting: dental hygienists," *J Md Dent Assoc,* 21:26-30, 1978.
41. American Dental Association: "GP's risk 3 times greater than population," *ADA News,* Nov. 26, 1979, p 1.
42. Denes, A. E., et al: "Hepatitis B infections in physicians," *JAMA,* 239:210-212, 1978.
43. Hollinger, F. B., et al: "Hepatitis B prevalence within a dental student population," *JADA,* 94:521-527, 1977.
44. Goebel, W. M., and Gitnick, G. L.: "Hepatitis B virus infection in dental students — A two year evaluation," *J Oral Med,* 34:33-36, 1979.
45. Center for Disease Control, U. S. Dept. of Health, Education and Welfare: "Morbidity trends for viral hepatitis — United States 1977," *Morbidity and Mortality Weekly Report,* 28:106-107, 1979.

46. Center for Disease Control, U. S. Dept. of Health, Education and Welfare: "Immune globulins for protection against viral hepatitis," *Morbidity and Mortality Weekly Report,* 26:425-442, 1977.
47. Aach, R. D.: "Viral hepatitis — A to E," *Med Clin North Am,* 62:59-70, 1978.
48. Silverberg, M: "Acute viral hepatitis: modern knowledge," *Ped Ann,* 6:298-310, 1977.
49. Szmuness, W., et al: "Distribution of antibody to hepatitis A antigen in urban adult populations," *N Engl J Med,* 295:755, 1976
50. Maynard, J. E., et al: "Preliminary serologic studies of antibody to hepatitis A virus in populations in the United States," *J Infect Dis,* 134:528-530, 1976.
51. Stevens, C. E., et al: "Antibody to hepatitis A antigen in children," *J Ped,* 91:436-438, 1977.
52. Center for Disease Control, U. S. Dept. of Health, Education and Welfare: "Reported cases of specified notifiable diseases, United States 1968-1977," *Morbidity and Mortality Weekly Report,* 26:2, 1978.
53. Cherubin, C. E., et al: "Acquisition of antibody to hepatitis B antigen in three socioeconomically different medical populations," *Lancet,* 2:149-151, 1972.
54. Cherubin, C. E., et al: "Evidence of hepatitis B infection in hospitalized children in New York City," *J Pediatr,* 88:893-907, 1976.
55. Aziz, M. A., et al: "Transplacental and postnatal transmission of the hepatitis-associated antigen," *J Infect Dis,* 127: 110-112, 1973.
56. Schweitzer, I. L., et al: "Viral hepatitis B in neonates and infants," *Amer J Med,* 55:762-771, 1973.
57. Papaevangelou, G., Hoofnagle, J., and Kremastinou, J.: "Transplacental transmission of hepatitis B virus by symptom-free chronic carrier mothers," *Lancet,* 2:746-748, 1974.
58. Stevens, C. E., et al: "Vertical transmission of hepatitis B antigen in Taiwan," *N Engl J Med,* 292:771-774, 1975.
59. Okada, K., et al: "Hepatitis B surface antigen in the serum of infants after delivery from asymptomatic carrier mothers," *J Pediatr,* 87:360-363, 1975.
60. Gerety, R. J. and Schweitzer, I. L.: "Viral hepatitis type B during pregnancy, the neonatal period, and infancy," *J Pediatr,* 90:368-374, 1977.
61. Lee, A. K. Y, Ip, H. M. H., and Wong, V. C. W.: "Mechanisms of maternal-fetal transmission of hepatitis B virus," *J Infect Dis,* 138:668-671, 1978.
62. Beasley, R. P.: "Transmission of hepatitis by breast feeding," *N Engl J Med,* 292:1354, 1975.
63. Smith, J. L., and Hindman, S. A.: "Transmission of hepatitis by breast feeding," *N Engl J Med,* 292:1354, 1975.
64. Beasley, R. P., et al: "Evidence against breast feeding as a mechanism for vertical transmission of hepatitis B," *Lancet,* 2:740-741, 1975.
65. Krugman, S.: "Vertical transmission of hepatitis B and breast feeding," *Lancet,* 2:916, 1975.
66. Boxall, E. H.: "Breast feeding and hepatitis B," *Lancet,* 2:979, 1975.
67. Dupuy, J. M., et al: "Hepatitis B in children, Part II: study of children born to chronic HBsAg carrier mothers," *J Pediatr,* 92:200-204, 1978.
68. Dupuy, J. M., Kostewicz, E., and Alagille, D.: "Hepatitis B in children, Part I: Analysis of 80 cases of acute and chronic hepatitis B," *J Pediatr,* 92:17-20, 1978.
69. Lander, J. J., Alter, H. J., and Purcell, R. H.: "Frequency of antibody to hepatitis-associated antigen as measured by a new radio-immunoassay technique," *J Immunol,* 106:1166-1171, 1971.
70. Vernon, T. M., et al: "Hepatitis A and B in the family unit — nonparenteral transmission by asymptomatic children," *JAMA,* 235: 2829-2831, 1976.
71. Perrillo, R. P. et al: "Hepatitis B antigen, DNA polymerase activity and infection of household contacts with hepatitis B virus," *Gastroenterology,* 76:1319-1325, 1979.
72. Tabor, E. T., et al: "Acute Non-A, Non-B hepatitis — prolonged presence of the infectious agent in blood," *Gastroenterology,* 76:680-684, 1979.
73. Hoofnagle, J. H., et al: "The prevalence of hepatitis B surface antigen in commercially prepared plasma products," *J Lab Clin Med,* 88: 102-113, 1976.
74. Tullman, M. J., and Boozer, C. H.: "Past infection with hepatitis B virus in patients in a dental school," *JADA,* 97:477-478, 1978.
75. Tullman, M. J., et al: "Prevalence of hepatitis B surface antigen in a dental school patient population," *J Public Health Dent,* 38:4-9, 1978.
76. Goebel, W. H.: "Reliability of the medical history in identifying patients likely to place dentists at an increased hepatitis risk," *JADA,* 98:907-913, 1979.
77. Tullman, M. J.: "The treatment of hepatitis B from dental school patients," *Oral Surg,* 49:214-216, 1980.
78. Seeff, L. B.: "Hepatitis in the drug abuser," *Med Clin North Am,* 59:843-848, 1974.
79. Stimmel, B., Salvatore, V., and Schaffner, F.: "Hepatitis B surface antigen and antibody — a prospective study in asymptomatic drug abusers," *JAMA,* 234:1135-1138, 1975.
80. Chance, E. R.: "Prevlence of hepatitis associate antigen (HAA) in an institution for the mentally retarded," *Am J Ment Def,* 77:1-5, 1972.
81. Dietzman, D. E., et al: "The occurrence of epidemic infectious hepatitis in chronic carriers of Australia antigen," *J Pediatr,* 80:577, 1972.
82. Madden, D. L., et al: "Epidemiology of hepatitis B virus in an institution for mentally retarded persons," *Am J Ment Def,* 80:369-375, 1976.
83. Tiku, M. L., et al: "Hepatitis B_e antigen and antibody activity in hepatitis B virus infection," *J Pediatr,* 91:540-544, 1977.
84. Szmuness, W., et al: "Hepatitis type A and hemodialysis," *Ann Int Med,* 87:8-12, 1977.
85. Schiff, E. R.: "Epidemiology of viral B hepatitis," *Med Clin North Amer,* 59:835-842, 1974.
86. Donaldson, D.: "Homologous serum hepatitis and the dental treatment of renal dialysis and kidney transplant patients," *Br Dent J,* 132: 391-393, 1972.
87. Sanger, R. G.: "Transfusion related hepatitis in hemophiliacs," in *Recent Advances in Dental Care for the Hemophiliac,* ed. Powell, D., Los Angeles: Dental Concepts, 1979, pp 51-54.
88. Hasiba, U. W., Spero, J. A., and Lewis, J. H.: "Chronic liver dysfunction in multitransfused hemophiliacs," *Transfusion,* 17:490-494, 1977.
89. Hoofnagle, J. H., et al: "Antibody to hepatitis B core antigen — a sensitive indicator of hepatitis B virus replication," *N Engl J Med,* 290:1336-1340, 1974.
90. Szmuness, W., et al: "On the role of sexual behavior in the spread of hepatitis B infection," *Ann Int Med,* 83:489-495, 1975.
91. Dental Health Committee of the British Dental Association: "Prevention of transmission of serum hepatitis in dentistry," *Br Dent J,* 137:28-30, 1974.
92. Zuckerman, A. J.: "The occupational hazard of viral hepatitis," *Proc Royal Soc Med,* 64:1211-1212, 1971.

93. Szmuness, W., Pierce, A. M., and Dubolt, G.: "The epidemiology of hepatitis B infections in Africa," *Am J Epidemiol,* 98:104-110, 1973.
94. Center for Disease Control, U. S. Dept. of Health, Education and Welfare: "Health status of Indochinese refugees," *Morbidity and Mortality Weekly Report,* 28:385-399, 1979.
95. Laboratory Center for Disease Control: "Screening of Indochinese refugees — Ottawa, Ontario," *Can Dis Weekly Report,* 5:157-162, 1979.
96. Center for Disease Control, U. S. Dept. of Health, Education and Welfare: "Viral hepatitis, tuberculosis, and dental care of Indochinese refugees," *Morbidity and Mortality Weekly Report,* 29:1-3, 1980.
97. Center for Disease Control, U. S. Dept. of Health, Education and Welfare: "Health status of Indochinese refugees: malaria and hepatitis B," *Morbidity and Mortality Weekly Report,* 28:463-470, 1979.
98. Center for Disease Control, U. S. Dept. of Health, Education and Welfare: "Health screening of resettled Indochinese refugees — Washington, D.C., Utah," *Morbidity and Mortality Weekly Report,* 29:4-11, 1980.
99. American Dental Association: "Refugee care," *ADA News,* April 28, 1980, p 8.
100. Levin, M. L., et al: "Hepatitis B transmission by dentists," *JAMA,* 228:1139-1140, 1974.
101. Williams, S. V., Pattison, C. P., and Berquist, K. R.: "Dental infection with hepatitis B," *JAMA,* 232:1231-1233, 1975.
102. California Morbidity Weekly Report: "An oral surgeon related hepatitis B outbreak," No. 14, 1976.
103. Rimland, O., et al: "Hepatitis B outbreak traced to an oral surgeon," *N Engl J Med,* 296:953-958, 1977.
104. Miller, W. V., Walsh, J. H., and Paskin, S.: "Ethylene oxide sterilization to prevent post-transfusion hepatitis," *N Engl J Med,* 280:386, 1969.
105. Bryman, J. A.: "Hypochlorite solutions and viral hepatitis," *JAMA,* 230:9, 1974.
106. Trevelyan, M. R.: "The prosthetic treatment of hepatitis B antigen positive patients," *Br Dent J,* 137:63-65, 1974.
107. Watkins, B. J.: "Viral hepatitis B: a special problem in prevention," *JASPD,* 6(3):8-13, 1976.
108. Parker, R. B.: "Dental office procedures for breaking an infection chain," *JASPD,* 6(3):18-21, 1976.
109. Miller, R. L.: "Generation of airborne infection by high speed dental equipment," *JASPD,* 6(3):14-17, 1976.
110. Grady, G. F.: "Hepatitis B from medical professionals: How rare? How preventable?," *N Engl J Med,* 296:995-996, 1977.
111. Sanger, R. G., Bradford, B.A., and Delaney, J. M.: "An inquiry into the sterilization of dental handpieces related to transmission of hepatitis B virus," *JADA,* 96:621-624, 1978.
112. Council on Dental Materials and Devices and Council on Dental Therapeutics: "Infection control in the dental office," *JADA,* 97:673-677, 1978.
113. Peterson, N. J., Bond, W. W., and Favero, M. S.: "Air sampling for hepatitis B surface antigen in a dental operatory," *JADA,* 99:465-467, 1979.
114. Whitacre, R. J., et al: *Dental Asepsis,* Seattle: Stoma Press, 1979, p 112.
115. Autio, K. L., et al: "Studies on cross-contamination in the dental clinic," *JADA,* 100:358-361, 1980.
116. Schenker, S.: "Hepatitis prevention after accidental needlestick exposure," *JAMA,* 240:2325, 1978.
117. Prince, A. M.: "Use of hepatitis B immune globulin: Reassessment needed," *N Engl J Med,* 299:198-199, 1978.
118. Mosley, J. W.: "Chronic hepatitis B virus infection," *JAMA,* 240: 2325-2326, 1978.