Managing the care of patients infected with bloodborne diseases

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ABSTRACT

Background. The emergence of the bloodborne pathogens HIV, the cause of AIDS; hepatitis B virus, or HBV; and hepatitis C virus, or HCV, has been a milestone in the history of the dental profession. In the early 1980s, new cases of AIDS increased dramatically, and fear of acquiring this disease compelled clinicians to modify the delivery of medical and dental care to allay fears of transmission on the part of both patients and health care workers. Arguably, the AIDS pandemic has been the most significant factor in the evolution and delivery of modern medical and dental care in the last century.

Overview. To help ally fears and remove barriers to caring for the HIV population, the Centers for Disease Control and Prevention, or CDC, introduced the concept of universal precautions in 1983. This was followed by the Occupational Safety and Health Administration's Bloodborne Pathogens Standard in 1991. Specific to the dental profession was the development of the principles of infection control in dentistry recommended by the CDC (1993); the American Dental Association (1995) and the Organization for Safety & Asepsis Procedures (1997). While initially difficult for some clinicians to acknowledge, these recommendations now are universally accepted throughout the profession, and provision of oral health care to patients infected with bloodborne disease is becoming commonplace. Compliance with recommended infection control practices remains an important component of dental practice. But it must be accompanied by an understanding of infectious and bloodborne diseases and the medical/dental management of the care of infected dental patients.

Conclusions and Practice Implications. The emergence of the bloodborne pathogens and the increasing number of infected patients who seek oral health care compel clinicians to have a thorough knowledge about bloodborne diseases and the medical/dental management of the care of patients presenting with HIV, HBV or HCV infection.

More than 20 years have passed since the first report on June 5, 1981, of an unusual cluster of cases of *Pneumocystis carinii* pneumonia and other opportunistic infections in five homosexual men in Los Angeles that heralded the arrival of AIDS in the United States.[1] This emergence of HIV disease and other bloodborne pathogens has significantly affected the practice of dentistry. Perhaps no other single disease entity has affected the profession as much as has HIV/AIDS. HIV has infected more than 42 million people worldwide[2] and approximately 800,000 to 900,000 Americans are infected with HIV disease, 30 percent of whom are unaware of their infection.[2-4] In the United States alone, an estimated 40,000 people are newly infected each year.[3,4] Some of the important events that occurred in the first 20 years of the AIDS pandemic are listed in Box 1.[1,5-15]

Compliance with recommended infection control practices must be accompanied by an understanding of infectious and bloodborne diseases.

HIV DISEASE

Although a cure for HIV infection remains elusive, sig-

"Practical Science" is prepared each month by the ADA Council on Scientific Affairs and Division of Science, in cooperation with The Journal of the American Dental Association. The mission of "Practical Science" is to spotlight what is known, scientifically, about the issues and challenges facing today's practicing dentists.

nificant advancements in the medical management of HIV disease have markedly reduced the morbidity of this disease in the United States and have improved the overall life expectancy of those infected with HIV (Box 1). The use of highly active antiretroviral therapy, or HAART, has evolved into the standard of care for the management of HIV/AIDS. It has resulted in prolonged suppression of the viral load and significant improvement in immune function, as well as a 60 to 80 percent reduction in new AIDS-defining conditions, hospitalizations and deaths.[7,8,16-18] While highly effective in inhibiting the replication of HIV and reconstituting immune function, many of the drugs in the HAART regimens cause significant adverse reactions[16-18] and have well-documented, multiple, adverse drug reactions and interactions (Table 1).[16-18] These adverse side effects include nausea, vomiting, diarrhea and other gastrointestinal and systemic symptomatology that often can be severe and sometimes can be fatal.[16-18]

Additionally, a class of antiretroviral agents called the protease inhibitors has been implicated in a number of significant side effects that include development of insulin-resistant diabetes, abnormally high glucose tolerance test values, lipodystrophy (blood lipid changes, hypertriglyceridemia and hypercholesterolemia) and fat redistribution (increased abdominal girth, thin extremities, buffalo hump, enlarged breasts).[16-24] The implications of these reactions have not been determined completely, but several studies suggest that elevated levels of lipids, triglycerides and glucose, known

BOX 1

HIV DISEASE: THE FIRST 20 YEARS.*

1981: The first cases of AIDS are reported June 5†

1982: Term "Acquired Immune Deficiency Syndrome" replaces term "Gay-Related Immune Deficiency Syndrome"

1983: Universal precautions are introduced‡; the virus that causes AIDS is identified (Gallo, human T-lymphotropic virus III§; Montagnier, lymphadenopathy-associated virus¶) and is named "human immunodeficiency virus"

1985: First serologic test for HIV is licensed by the U.S. Food and Drug Administration, or FDA; actor Rock Hudson dies of AIDS, becoming the first public figure whose death is openly attributed to AIDS

1986: AIDS drug azidothymidine, or AZT, approved by FDA in a record time of six months#

1989: U.S. AIDS cases reported at 100,000

1991: Estimated number of Americans infected with HIV reaches 1.5 million; basketball star Earvin "Magic" Johnson announces he is HIV-positive

1993: Multiple drugs fail in clinical trials; period of extreme pessimism for those with HIV

1995: FDA approval of first protease inhibitor (saquinivir)**; HIV viral replication rate reported at 10 billion virions/day

1996: HIV viral load testing becomes major method to assess effectiveness of antiretroviral therapy, or ART††; ACTG 076 shows benefit of AZT in reducing perinatal transmission‡‡; initial reports of benefit of highly active antiretroviral therapy, or HAART; ritonavir and indinavir approved by FDA; nevirapine, the first non-nucleoside reverse transcriptase inhibitor, approved; first triple combination HAART study conducted‡‡

1997: 13 percent decrease in AIDS deaths; 60 to 80 percent reduction in new AIDS-defining conditions, hospitalizations and deaths§§¶¶

1999: HIV spread to humans from chimpanzees (occurred in Africa for decades before HIV disease was recognized)

2000: AIDS pandemic raging in Third World—a total of 36.1 million people infected with HIV, 21.8 million deaths, 14,000 to 16,000 new infections per day##

2001: Two distinct epidemics: developed vs. underdeveloped countries

* Modified from Bartlett.[5]
† Centers for Disease Control and Prevention.[1]
‡ Centers for Disease Control and Prevention.[6]
§ Gallo and colleagues.[7]
¶ Barre-Sinoussi and colleagues.[8]
# Volberding and colleagues.[9]
** U.S. Food and Drug Administration.[10]
†† Mellors and colleagues.[11]
‡‡ Sperling and colleagues.[12]
§§ Joint United Nations Programme on HIV/AIDS and World Health Organization.[13]
¶¶ Palella and colleagues.[14]
Mocroft and colleagues.[15]

TABLE 1

DRUGS COMMONLY USED IN DENTISTRY THAT SHOULD NOT BE USED WITH PROTEASE INHIBITORS OR NNRTIs.*†

DRUG USED IN DENTISTRY	PROTEASE INHIBITORS					NNRTIs‡		
	Indinavir	Ritonavir	Saquinavir	Amprenavir	Kaletra	Nelfinavir	Delavirdine	Efavirenz
Analgesics§	None	Meperidine, piroxicam, propoxyphene	None	None	Meperidine, piroxicam, propoxyphene	None	None	None
Antibiotics	Rifampin	None	Rifampin, rifabutin	Rifampin	Rifampin	Rifampin	Rifampin, rifabutin	None
Antihistamines¶	Astemizole, terfenadine	Astemizole, terfenadine	Astemizole, terfenadine	Astemizole, terfenadine	Astemizole, terfenadine	Astemizole, terfenadine	Astemizole, terfenadine	Astemizole, terfenadine
Psychotropic Agents#	Midazolam, triazolam	Midazolam, triazolam	Midazolam, triazolam	Midazolam, triazolam	Midazolam, triazolam	Midazolam, triazolam	Midazolam, triazolam	Midazolam, triazolam
Local Anesthetics	None	None	None	None	None	None	None	None
Herbs	St. John's wort	St. John's wort	St. John's wort	St. John's wort	St. John's wort	St. John's wort	St. John's wort	St. John's wort

* Modified from Bartlett and Gallant.[16]
† NNRTTs: Non-nucleoside reverse transcriptase inhibitors.
‡ Note: no drugs are contraindicated for concurrent use with the NNRTI nevirapine.
§ Alternatives: acetaminophen, acetylsalicylic acid, oxycodone.
¶ Alternatives: Cetirizine, fexofenadine, loratadine.
# Alternatives: Lorazepam, temazepam.

to increase cardiovascular risk in patients without HIV, may predispose patients receiving HAART to increased risk of cardiovascular disease.[16,25]

In the early years of the epidemic, patients usually died of complications from one or more of the opportunistic infections, or OIs, that developed as a result of profound immunosuppression (CD4 count < 200 cells/cubic millimeter).[16,26,27] With HAART and subsequent immune reconstitution, OIs, although still encountered in patients with HIV/AIDS, are seen less frequently.[16,27] However, appropriate prophylaxis for OIs, when indicated, is frequently instituted when the CD4 count falls below 200 cells/mm^3, but this may be safely stopped as long as adequate evidence of immune reconstitution persists.[16,27-30] The most common OIs found in patients with HIV/AIDS and the currently recommended therapy are listed in Table 2.[16] The mouth and pharynx frequently are the site of OIs, and oral health care providers play a critical role in the early diagnosis and treatment of these lesions.

People with HIV infection commonly receive HAART and one or more of the antiviral/antifungal regimens listed in Table 2.[16] A patient's lack of anticipated clinical response to a combined HAART/antiviral/antifungal regimen may suggest that he or she has developed resistance, which is becoming more frequent and problematic.[16] This developing resistance pattern, coupled with multiple adverse drug reactions and interactions—which are common in patients receiving HAART—compel clinicians to be attentive to the drugs each patient is currently taking, any new drugs to be prescribed and the potential interactions that can occur with the introduction of new medication(s) into existing treatment regimens. Therefore, a thorough evaluation of each HIV patient's dental, medical and treatment regimens must be examined and assessed before any dental care is delivered.

HEPATITIS

At least two other significant bloodborne pathogens are frequently encountered in modern dental practice: hepatitis B virus, or HBV, and hepatitis C virus, or HCV.

HBV. HBV is transmitted very efficiently by contact with blood and blood products, by sexual

TABLE 2

MANAGEMENT OF THE MOST COMMON FUNGAL AND VIRAL HIV-RELATED ORAL OPPORTUNISTIC INFECTIONS.

ORGANISM	TREATMENT	ALTERNATIVE	PROPHYLAXIS
Candida Oral, Oropharyngeal	Clotrimazole troche 10 milligrams, five times per day for 10 to 14 days; nystatin vaginal tabs, 100,000 units dissolved by mouth four times per day for 14 days*	Fluconazole 100 mg by mouth per day for five to 10 days*; itraconazole 100 mg per day in oral suspension on empty stomach, swish and swallow	Not recommended
Herpes Simplex, Mild Oral/ Genital, Initial Treatment	Acyclovir 400 mg by mouth three times per day for seven to 10 days; famciclovir 250 mg by mouth three times per day for seven to 10 days; valacyclovir 1 gram by mouth two times per day for seven to 10 days	None	Not recommended
Herpes Simplex, Refractory	Acyclovir 15 mg per kilogram per day intravenously for seven days	Foscarnet 40 mg/kg intravenously every eight hours or 60 mg/kg every 12 hours	Not recommended
Herpes Simplex, Recurrent	Acyclovir 400 or 800 mg by mouth three times per day for five days; famciclovir 125 mg by mouth two times per day for five days; valacyclovir 500 mg by mouth two times per day for five days	None	Acyclovir 400 mg two times per day; famciclovir 125-250 mg two times per day; valacyclovir 1 g per day
Herpes Zoster, Dermatomal (Shingles)	Acyclovir 800 mg by mouth five times per day for at least seven days or until lesions crust; famciclovir 500 mg by mouth three times per day for at least seven days or until lesions crust; valacyclovir 1 g by mouth three times per day for at least seven days or until lesions crust	Acyclovir 30 mg/kg intravenously per day; foscarnet 40 mg/kg intravenously every eight hours or 60 mg/kg every 12 hours	Varicella zoster immune globulin within 96 hours of exposure
Clytomegalovirus	Foscarnet 60 mg/kg intravenously every eight hours for 14 to 21 days; ganciclovir 5 mg/kg intravenously two times per day for 14 to 21 days THEN foscarnet 90 mg/kg per day or ganciclovir 5 mg/kg per day; intraocular ganciclovir device for retinitis and ganciclovir by mouth or intravenously	Alternating or combining foscarnet and ganciclovir	Not recommended

* Modified from Bartlett and Gallant.[16]

contact and, to a lesser extent, perinatally. This bloodborne infection causes approximately 300,000 acute infections annually in the United States. Chronic HBV infection develops in approximately 15,000 to 30,000 (10-15 percent) of these cases, and there are an estimated approximately 1 million HBV carriers in the U.S. population.[31] Fortunately, the infection resolves in 90 to 95 percent of those infected with HBV. For the small minority who develop chronic disease, persistent infection can lead to chronic progressive hepatitis, cirrhosis or hepatocellular carcinoma[32,33] and causes 5,000 to 6,000 deaths per year owing to liver failure.[32,33]

HCV. Infection with HCV also accounts for significant morbidity and mortality in the estimated four million Americans (1.8 percent) who are carriers of the virus. HCV is transmitted efficiently by blood and blood products and is rapidly transmitted by injection drug use, or IDU; however, sexual and perinatal transmission of HCV seems to be infrequent.[34] More selective and specific HCV assays have reduced the incidence of transfusion-transmitted HCV significantly, but

IDU remains a major risk factor in the acquisition of HCV infection.[34,35]

Through multiple mechanisms that have yet to be completely understood, HCV seems to escape immune surveillance. This results in more than 85 percent of those infected being in a chronic carrier state. Symptoms, if present, generally are benign, and most affected patients are relatively asymptomatic for the two decades after infection. Because of the lack of symptoms, the majority of those infected remain unaware of their serostatus.[34] However, as time progresses, so does the disease, leading to necrosis, cirrhosis with associated decreased liver function and an increased incidence of hepatocellular carcinoma.[35] Early diagnosis is critical, and anyone who has ever performed behavior that places him or her at risk of developing HCV should be encouraged to be tested for the virus, as well as other bloodborne diseases.[34-36]

People with HCV infection should be followed closely for evidence of progression of liver disease, because anti-HCV therapy may be indicated.[34,36] New therapies such as interferon-alfa, antiviral therapy (such as ribavirin) and, most recently, pegylated interferon alfa have met with some success.[34,36,37] However, HCV replicates very rapidly and is genetically diverse, resulting in numerous mutations, viral resistance and therapeutic failure.[34-37] Anti-HCV therapy is long (six to 12 months) and arduous, and its side effects (such as profound fatigue, depression, malaise and myalgia) are frequent and pronounced.[34,36,37] Unfortunately, because of the severity of side effects, many patients elect to discontinue anti-hepatitis C therapy and may be unwilling and/or unable to undergo elective dental care.[33] Therefore, the clinician should consider deferring elective dental procedures until the interferon regimen has been completed. However, because of the depression and lassitude that often accompany interferon therapy, oral hygiene may deteriorate, and routine preventive appointments should be encouraged. Despite therapy and the large numbers of liver transplantations being performed on patients with HCV,[34,36] an estimated 8,000 to 10,000 deaths attributable to HCV occur annually.

Coinfection. As HIV, HBV and HCV are transmitted in a similar manner, many patients have coinfection. HIV, HBV and/or HCV coinfection significantly complicates the medical management of the diseases and enhances the probability of the patient's experiencing liver dysfunction.[33] Therefore, clinicians must perform a thorough evaluation of liver function to be knowledgeable about the presence of chronic liver disease, the degree of dysfunction and any potential treatment modifications necessitated by the degree of hepatic disease. Although coinfection with HCV or HBV does not seem to accelerate the progression of HIV disease, the immune decomposition caused by HIV can accelerate the progression of HCV/HBV significantly, lessening the patient's quality of life and reducing his or her overall life expectancy.[16]

DENTAL MANAGEMENT OF THE CARE OF PATIENTS WITH HIV INFECTION

The dental management of the care of patients infected with HIV usually is straightforward; no special facility or equipment is required. Such patients who require the care of a specialist should be appropriately referred, but most treatment can be performed by general practitioners. Specialist referral is indicated using criteria identical to those for referral of patients who do not have HIV infection. Clinicians should comply with the current CDC,[38] OSAP[39] and ADA[40] infection control recommendations with every patient, regardless of the presence or absence of bloodborne disease.

A comprehensive oral health assessment is paramount for the early recognition of and intervention in OIs. Thus, oral health care providers play a significant role in reducing morbidity and improving the quality of life for patients infected with bloodborne diseases. At all times, clinicians must maintain confidentiality for all patients, regardless of HIV serostatus. Proper consent should be obtained before the clinician releases any confidential medical or dental information to other medical or dental care providers.[41-45]

Clinicians should obtain a thorough medical history, including a comprehensive review of systems inclusive of current medications, for all patients. Clinicians should evaluate the patient with HIV infection for susceptibility to infection and bleeding, potential problems with adverse drug reactions and interactions, and the potential overall inability to withstand dental care-related stress and trauma due to HIV-related immunosuppression and systemic manifestations. Clinicians should prescribe antibiotic prophylaxis for patients who are severely neutropenic. Elective dental procedures are contraindicated in patients

with a neutrophil count of less than 500 cells/mm^3 and a platelet count of less than 50,000 cells/mm^3. Patients with profound neutropenia and thrombocytopenia may receive urgent care as indicated, but they may require hospitalization.[41-45]

Patients with HIV infection are living longer and can develop chronic diseases, many secondary to the toxicity of their medications. Abnormalities in lipid metabolism, lipodystrophy and hyperglycemia due to antiretroviral therapy, or ART, possibly increase the patient's risk of experiencing adverse cardiovascular sequelae and the development of diabetes. Thus, clinicians should carefully monitor patients receiving ART for cardiovascular and diabetes-related symptomatology, and they should record the patient's blood pressure at each appointment.[42]

Comorbidities commonly found in conjunction with HIV—such as HBV and HCV—often result in impaired hepatic function. Clinicians must recognize the side effects of, and drug interactions possible with, antiretroviral medications. Clinicians should avoid using acetaminophen in patients with severe liver disease because of its hepatotoxicity. Aspirin and nonsteroidal anti-inflammatory drugs, or NSAIDs, which can decrease coagulation, should not be used in patients with impaired hemostasis.[33] Box 2 outlines further information for clinicians to consider when providing treatment for patients with HIV infection.[16,40-46]

DENTAL TREATMENT CONSIDERATIONS: HEPATITIS

The dental management of the care of patients with hepatitis and subsequent liver disease is similar to the management of the care of patients with HIV infection. Clinicians should comply with the current

BOX 2

FACTORS CLINICIANS SHOULD EVALUATE WHEN PROVIDING TREATMENT FOR PATIENTS WITH HIV INFECTION.*

PATIENT'S COMPLETE BLOOD COUNT AND DIFFERENTIAL (TOTAL WHITE AND RED BLOOD CELL COUNT, HEMATOCRIT AND PLATELET COUNTS)

- Many patients with HIV are neutropenic, thrombocytopenic and anemic
- Values indicate susceptibility to infection and bleeding
- Should be repeated at three- to six-month intervals
- Platelet count should be greater than 50,000 cells per cubic millimeter for elective procedures
- Neutropenia: Severe neutropenia may be associated with increased risk of infection
  - Normal neutrophil count: 4,500 to 10,000 cells/mm^3
  - Mild neutropenia: 2,500 to 4,500 cells/mm^3
  - Severe neutropenia: Below 1,000 cells/mm^3
- White blood cell count less than 500 cells/mm^3 indicates severe neutropenia
  - Prophylaxis indicated
  - Many clinicians use the regimen of a single antibiotic dose one hour before the dental appointment as suggested by the American Heart Association† for the prevention of endocarditis; however, others feel that appropriate antibiotic therapy should continue for as long as open wounds are present in the oral cavity; clinicians should use clinical judgment and evaluate each patient on a case-by-case basis
  - The benefits of dental treatment must be weighed against the risk of infection; elective procedures may be contraindicated
  - Medical consultation is indicated
  - Need for prophylaxis is based on neutrophil count and not on CD4 count

PATIENT'S T-LYMPHOCYTE, OR CD4, LEVELS

- Indicates degree of immunosuppression
- Normal CD4 count 800 to 1,000 cells/mm^3
- In patients with HIV, CD4 levels commonly are below 300 cells/mm^3 (see History of Opportunistic Infections below)
- AIDS diagnosis associated with levels of 200/mm^3 or less
- CD4 count is not an indicator for prophylaxis

PATIENT'S PLASMA VIRAL LOAD

- Indicates degree of viral replication and immunosuppression (destruction of CD4 lymphocytes)
- Measure of therapeutic success or failure of antiviral/antiretroviral therapy
- Prognostic: The higher the viral load, the faster the HIV progression and the poorer the long-term prognosis

MEDICATIONS PATIENT IS TAKING

- Evaluation should include prescription medications, over-the-counter drugs, herbal, naturopathic and homeopathic remedies and treatments and nutritional supplements
- Patients with HIV are frequently taking numerous medications with complex dosing regimens; numerous drug-to-drug interactions have been well-documented (Table 1)
- A complete listing of all medications is essential to minimize potential adverse drug interaction

PATIENT'S HISTORY OF OPPORTUNISTIC INFECTIONS, OR OIs

- Previous viral, fungal or bacterial infections
- Current or previous prophylaxis for OIs
- HIV-associated malignancies

* Based on information from Bartlett and Gallant,[16] American Dental Association Council on Scientific Affairs and Council on Dental Practice,[40] HIVDENT,[41] Pennsylvania Mid-Atlantic AIDS Education and Training Center,[42] Little and colleagues,[43] Patton and colleagues[44] and The Dental Alliance for AIDS/HIV Care.[45]
† Dajani and colleagues.[46]

BOX 3

FACTORS CLINICIANS SHOULD EVALUATE WHEN PROVIDING TREATMENT FOR PATIENTS WITH HEPATITIS.*

ALANINE AMINOTRANSFERASE AND ASPARTATE AMINOTRANSFERASE VALUES

- Nonspecific transaminases
- Often elevated with liver disease
- Marked elevation may indicate decreased liver function
  - Patients may be prone to hemorrhage
  - Drug metabolism may be impaired

PROTHROMBIN TIME/INTERNATIONAL NORMALIZED RATIO

- In patients with suspected liver disease, coagulation test results should be obtained before invasive procedures are performed
- Patient's physician should be consulted to determine the degree of coagulopathy

PATIENT'S COMPLETE BLOOD COUNT AND DIFFERENTIAL (TOTAL WHITE AND RED BLOOD CELL COUNT, HEMATOCRIT AND PLATELET COUNTS)

- Many patients with liver disease are thrombocytopenic and anemic
- Platelet count should be greater than 50,000 for elective procedures

MEDICATIONS PATIENT IS TAKING

- Evaluation should include prescription medications, over-the-counter drugs, herbal, naturopathic and homeopathic remedies and treatments and nutritional supplements
- Patients with liver disease may not be able to metabolize drugs properly
- Modification of normal dosage may be indicated for any drugs metabolized in the liver
  - Local anesthetics
  - Analgesics
  - Antibiotics
- A complete listing of all medications patient is taking is essential to minimize potential adverse drug interactions

* Based on information from Bartlett and Gallant,[16] Little and colleagues[33] and DePaola.[42]

infection control recommendations of the CDC,[38] the Organization for Safety & Asepsis Procedures, or OSAP,[39] and ADA.[40] Referral and confidentiality guidelines are the same as those for patients with HIV disease. Clinicians should evaluate patients with hepatitis for susceptibility to infection and bleeding, potential problems with adverse drug reactions or interactions and the potential overall inability to withstand the stress and trauma of dental care due to hepatitis-related liver dysfunction. As many drugs and medications are metabolized by the liver, it is essential that the clinician perform a thorough medical history with a review of systems, inclusive of medications the patient is taking.[33,42] Clinicians should be aware of the level of liver function as reflected by the alanine aminotransferase test, the aspartate aminotransferase test and other liver function tests. This information will relate to the patient's ability to metabolize drugs.[33,42]

With advanced liver disease, the level of vitamin K—the precursor of the prothrombin-dependent clotting factors that is produced in the liver—can be reduced significantly, resulting in a decrease in clotting factor production. Additionally, in a patient with advanced liver disease, portal vein hypertension can sequester platelets formed in the spleen, resulting in thrombocytopenia and possibly impairing his or her ability to form a clot. Therefore, hemorrhage, which can be excessive, is one of the most common adverse events encountered during treatment of patients with decreased liver function. In light of this, the clinician should perform coagulation tests (prothrombin time/international normalized ratio and platelet count) and evaluate the values before performing any surgical procedures.[33,42] Clinicians should defer elective procedures for patients with abnormal coagulation, and these patients may require hospitalization for urgent dental care. As with patients who have HIV infection, clinicians should avoid using acetaminophen in patients with severe liver disease and avoid using aspirin and NSAIDs in patients with impaired hemostasis.[33,42]

The clinician must consult with the patient's physician before performing invasive dental procedures with any patient in this population.[33,42] Box 3 provides additional information the clinician should consider when providing treatment for patients with hepatitis.[16,33,42]

SUMMARY

Clinicians should follow CDC, OSAP and ADA infection control recommendations for all patients, regardless of their bloodborne serostatus. When treating a patient infected with a bloodborne disease, the clinician should take a thorough medical history, make a record of medications the patient is taking and assess the patient's immunologic and hepatic function to

provide safe and efficacious dental care. ■

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The preparation of this manuscript was supported in part by U.S. Department of Health and Human Services Health Resources and Services Administration, grant 1 H4A HA 00060 01.

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