24 HIV in the Workplace

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Over 25 years have elapsed since the first cases of what would become known as the *acquired immune deficiency syndrome* (AIDS) were reported in 1981 [1]. The complex interactions between the human immunodeficiency virus (HIV) and the workplace are still being defined and understood. While occupationally acquired infections from HIV are of much individual concern, they are rare events [2]. HIV is neither the most common nor the most infectious bloodborne pathogen. In the United States, seroconversion related to employment has been documented in health care workers and in those who occupationally engage in sexual contact [3–5]. The medical response to HIV exposure has been addressed by various health organizations [6–8]. Because of the currently incurable nature of HIV infection, seroconversion after an exposure to human blood is often the foremost concern for an employee. Prompt treatment following an HIV exposure can decrease the chances of seroconversion. Therefore, evaluation and treatment of potential exposures are urgent medical matters. Occupational medicine providers should be prepared to evaluate and treat or promptly refer employees who present following such an exposure. This chapter will deal primarily with exposure to HIV from nonsexual contact with infectious material. Occupational HIV surveillance, workplace limitations, and employment restrictions, if any, for HIV-positive employees and candidates are also reviewed.

Occupational Exposure to Bloodborne Pathogens: A Brief History

Previous experience with viral hepatitis has provided a framework for response to HIV. Hepatitis B, previously known as *serum hepatitis,* was noted to occur via parenteral infection. Cases arose from blood transfusions, sharing of needles from illicit drug use (e.g., during the Vietnam War), and through immunizations, including 200,000 cases associated with infected lots of yellow fever vaccine among U.S. forces in the 1940s [9]. The identification of the MS-1 (hepatitis A) and MS-2 (hepatitis B) strains by Krugman and colleagues [10] in the Willowbrook studies preceded the detection of the Australia antigen (now referred to as the *hepatitis B surface antigen*) in the late 1960s by Blumberg and colleagues [11]. More complete understanding of hepatitis B pathophysiology was followed by routine serologic testing of blood transfusion units and patients infected with hepatitis and the eventual development of the hepatitis B vaccine. Because of the similar nature of their occupational risks and the timing of a fuller understanding of pathogenesis, the groundwork in the global

*The opinions expressed by the authors do not reflect the policy of the Department of Health and Human Services, the Department of the Navy, or the Department of Defense.

response to occupational risks for hepatitis B has shaped the response to HIV.

The magnitude of the number of health care workers (HCWs) exposed to bloodborne pathogens via occupational needlestick injuries became clearer by the mid-1970s. Preventive strategies for needlestick exposure in HCWs were proposed as early as 1981 [12], the same year as the first case report of AIDS [1]. In 1982, Dienstag and Ryan demonstrated a correlation between the intensity and duration of human blood and body fluid exposure and the likelihood of hepatitis B conversion [13]. The 1983 version of the U.S. Centers for Disease Control and Prevention (CDC) Guidelines for Isolation Precautions in Hospitals was the last to have a specific category for blood and body fluid precautions [14]. The emergence of AIDS cases in the early 1980s and subsequent identification of HIV [15–17] prompted the National Institute for Occupational Safety and Health (NIOSH) and other parts of the CDC to reconsider the utility of having a specific category of blood and body fluid precautions [18,19]. The phrase *universal precautions* has been promoted to raise awareness of potential infectious risks within health care systems. Each patient encounter is a potential risk; therefore, appropriate precautions must be universally applied. The first occupational HIV infection, the result of a needlestick injury, was reported in late 1984 [20]. By 1987, the CDC had reports of six cases of occupationally acquired HIV in the United States. Four of the six had needlestick exposures, whereas the remaining two had extensive blood or body fluid contact from an HIV-infected patient [21]. Use of single-agent antiretroviral postexposure therapy was discussed in a 1989 publication by Henderson and Gerberding [22]. The CDC developed the National Surveillance System for Health Care Workers (NaSH) to track occupationally related disease. In 1991, the U.S. Occupational Safety and Health Administration (OSHA) released legal guidelines to protect employees in an attempt to reduce or mitigate the effects of the estimated 800,000 needlestick injuries that occur in the United States annually [23, 24].

HCWs face a wide range of workplace hazards, including needlestick injuries and mucous membrane exposures. The total number of exposures is unknown but may increase owing to increasing numbers of workers at risk. The health care field in the United States was the largest industry in 2006, employing over 14 million workers. Women represent nearly 80% of the health care workforce in America [25]. Worldwide, there are conservatively estimated to be 35 million HCWs, but this estimate does not include the millions of support staff who also are at risk for occupational exposure [26]. Police, firefighters, sanitation workers, and many others have lower but real occupational risks for accidental exposure to human blood or body fluid [27]. Most research and prevention measures have focused on HCWs, especially in the hospital setting. The results of these efforts have been mixed [28]. A recent anonymous survey of 582 surgical residents at 17 medical centers reported that 99% of surgeons in training experienced a needlestick at some time during their training. Only 51% of the most recent needlestick injuries were reported [29].

The effectiveness of highly active antiretroviral therapy (HAART) for HIV has increased the life expectancy of persons living with HIV/AIDS, thus increasing the number of HIV-positive sources. These therapies have lowered viral load in many of these same individuals, making exposure to their blood or body fluids potentially less likely to result in infection. Table 24-1 shows the CDC statistics for reported occupational HIV infections in HCWs in the United States through 2006 [3]. Whether the result of improved prophylaxis, safety devices, employee education, or underreporting, no occupationally related HIV infections have been reported to the CDC since 2001.

HIV/AIDS Surveillance

The CDC performs HIV/AIDS surveillance for the United States and issues annual reports, most recently providing data through 2005. General trends show a steady increase in the estimated number of persons living with AIDS in the United States—421,873 at the end of 2005. Because it may take a decade or more for an HIV-positive individual to develop AIDS, and because universal testing is not the norm, it is not known precisely how many individuals in the United States are infected. The CDC estimates that over 1 million people in the United States are HIV-positive and that a quarter of them are unaware of their HIV status. The general trends indicate that an increasing number of persons are

Table 24-1
Number of U.S. Health Care Workers with Documented and Possible Occupationally Acquired HIV Infection, Reported through December 2006

Occupation	Documented Transmission	Possible Transmission
Dental worker, including dentist	0	6
Embalmer/morgue technician	1	2
Emergency medical technician/paramedic	0	12
Health aide/attendant	1	15
Housekeeper/maintenance worker	2	13
Laboratory technician, clinical	16	17
Laboratory technician, nonclinical	3	0
Nurse	24	35
Physician, nonsurgical	6	12
Physician, surgical	0	6
Respiratory therapist	1	2
Technician, dialysis	1	3
Technician, surgical	2	2
Technician/therapist, other than those listed above	0	9
Other health care occupations	0	6
Total	57	140

Source: Adapted from Centers for Disease Control and Prevention. *Surveillance of Occupationally Acquired HIV/AIDS in Healthcare Personnel, as of December 2006;* available at *www.cdc.gov/ncidod/dhqp/pdf/bbp/fact_sheet_clearance_revised_090507Dec2006.pdf*.

living with HIV/AIDS owing to both new cases of infection and lengthened life expectancy [30].

Pathophysiology of HIV

HIV is an RNA-based retrovirus of the subfamily Lentivirus, a group of viruses that cause chronic cytopathic infections of the immune system of various vertebrate species. HIV is closely related to the immunodeficiency viruses of nonhuman primates. With the extraordinary growth in the human population in the last half of the twentieth century, human penetration into remote forest habitats, and rapid transit, this virus appears to have made a successful species jump in the relatively recent past. It has been rapidly propagated and amplified by human behavior and susceptibility [31–33].

Humans are the only known reservoir of HIV. Transmission requires intimate contact between susceptible host target cells and free virus or virus contained in inflammatory cells either suspended in human blood or present in other body fluids or tissues. Transmission does not occur via vectors or fomites, intact skin to intact skin contact, the airborne-respiratory route, or the fecal-oral contact. The dominant and characteristic form of ongoing viral infection and pathogenesis occurs with binding of the virus-specific envelope protein complex, gp120/gp 41, to the CD4 molecule expressed on the surface of helper T-lymphocytes as part of T-cell responsiveness to foreign antigens [34]. Binding then initiates the next stages of the viral life cycle: penetration, conversion of the RNA genome to DNA by the unique viral enzyme reverse transcriptase, and integration of viral DNA with host cell DNA. Resting CD4+ T cells may harbor proviral DNA without progressing forward in the viral replication process [35]. CD4+ T cells that are immunologically activated either before or after initial binding of HIV will continue the replication process via expression of viral messenger RNA, formation of viral proteins, followed by virion assembly and budding from the cell membrane. The specific cause of the immunologic activation has not yet been identified and may vary from patient to patient. Figure 24-1 demonstrates a simplified life cycle of HIV. In addition to CD4, coreceptors that play a role in HIV entry into host cells have been identified and are targets for pharmacologic intervention [36].

HIV Treatment

HIV treatment is a rapidly evolving field. Figure 24-1 also lists the classes of HIV medication and indicates their sites of action. The Food and Drug Administration (FDA)–approved medications for the treatment of HIV, as of early 2008, are listed in Table 24-2 [37]. Each drug generally has two to three names, which can complicate the understanding of an HIV source's medication history. Entry and fusion inhibitors work by preventing the processes involved with HIV gaining entry into CD4+ cells. Because of their action early in the process, there is promise for these drugs in acute postexposure treatment [38]. However, their use is not currently recommended without expert consultation [8]. Another recently added category of anti-HIV medication, the integrase inhibitors, acts

Figure 24-1
Simplified life cycle of the HIV virus

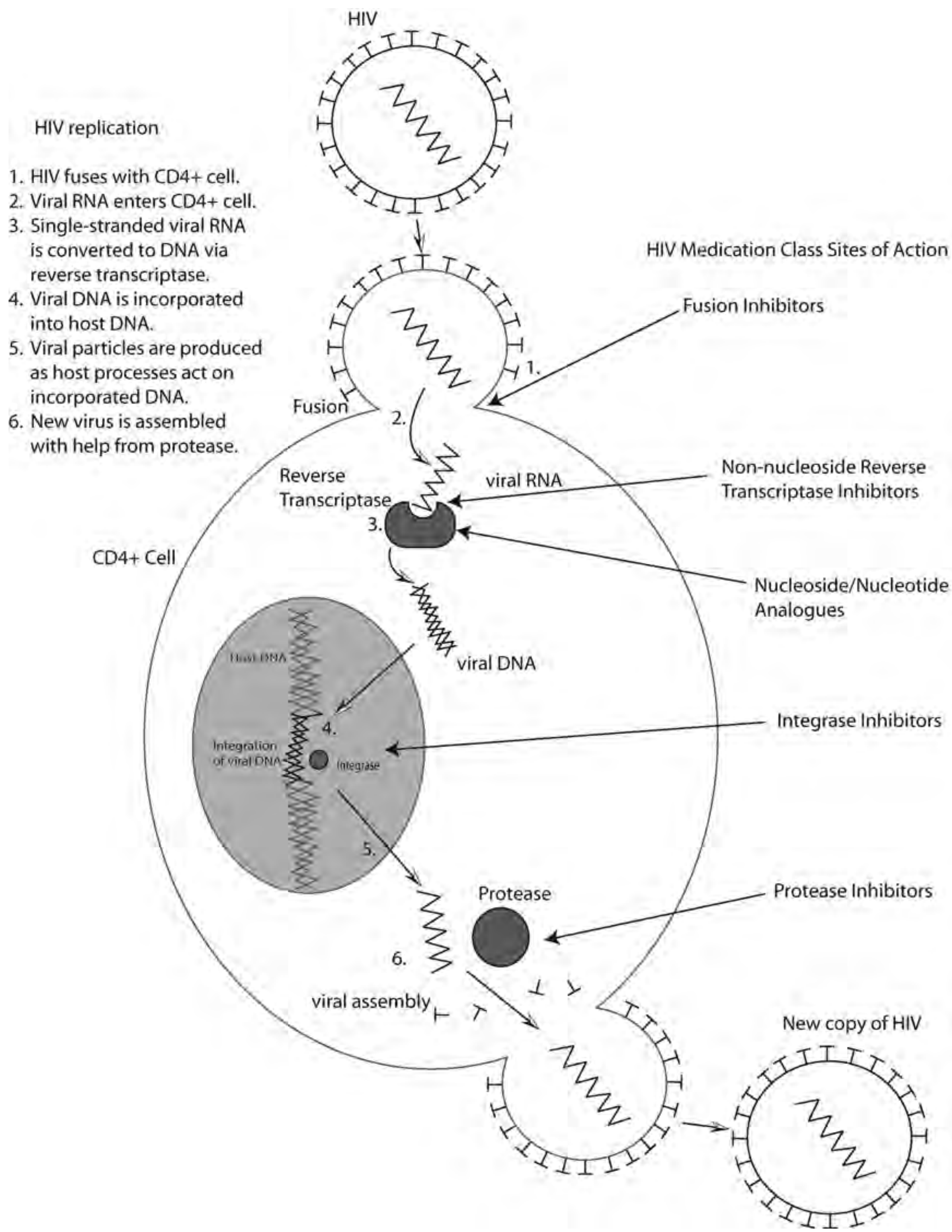

Table 24-2
FDA-Approved HIV Drugs, Early 2008 (Generic Name, Trade Name, Other Identifiers)

Entry/Fusion Inhibitors
Enfuvirtide, Fuzeon, ENF, T-20
Maraviroc, Selzentry, UK-247, 857
Nonnucleoside Reverse-Transcriptase Inhibitors
Delavirdine, Rescriptor, DLV
Efavirnez, Sustiva, EFV
Etravirine, Intelence, TMC125
Nevirapine, Viramune, NVP
Nucleoside/Nucleotide Reverse-Transcriptase Inhibitors
Abacavir, Ziagen, ABC
Didanosine, Videx, dideoxyinosine, ddI
Emtrictabine, Emtriva, FTC
Lamivudine, Epivir, 3TC
Stavudine, Zerit, d4T
Tenofovir DF, Viread, TDF
Zalcitabine, Hivid, dideoxycytidine, ddC
Zidovudine, Retrovir, ZDV, azidothymidine, AZT
Integrase Inhibitors
Raltegravir, Isentress, RGV, MK-0518
Protease Inhibitors
Atazanavir, Reyataz, ATV
Darunavir, Prezista, DRV
Fosamprenavir, Lexiva, FOSAPV, FPV
Indinavir, Crixivan, IDV
Lopinavir LPV
Nelfinavir, Viracept, NFV
Ritonavir, Norvir, RTV
Saquinavir, Invirase, SQV
Tipranavir, Aptivus, TPV
Combination Medications
Atripla: efavirnez, emtricitabine, tenofovir
Combivir: lamivudine, zidovudine
Epzicom: abacavir, lamivudine
Kaletra: lopinavir, ritonavir
Trizivir: abacavir, lamivudine, zidovudine
Truvada: emtricitabine, tenofovir

to prevent the integration of viral DNA into the host cell chromosomes. The role of this agent in postexposure prophylaxis (PEP) is not yet established. The nucleoside/nucleotide analogues and the nonnucleotide reverse-transcriptase inhibitors interfere with reverse-transcriptase conversion of genomic RNA to the proviral DNA. The protease inhibitors interfere with the final steps that produce infective viral units.

Because HIV undergoes essentially continuous replication via an error-prone reverse transcriptase, an individual with chronic infection typically harbors a pool of circulating virus with many different mutations. Clinically, it has been observed that a single drug in an infected individual is likely to select for a preexisting resistant virus. Because monotherapy has demonstrated rapid onset of viral resistance, the institution of highly active antiretroviral therapy (HAART), which combines three or more drugs from different classes, has become the standard of care for individuals being treated for HIV/AIDS [39].

Acute HIV Infection

Clinically, the likelihood of transmission depends on the viral titer of the source and the volume and mode of contact of the infectious material. Virus can be recovered eventually from most body fluids and tissues, but only blood products, sexual fluids, breast milk, and visibly bloody fluids have been associated with transmission [40–43]. Based on these data, the CDC has issued a list of body fluids considered to be infectious (Table 24-3) [8]. Viral titer is determined by the natural stage of infection, treatment, treatment failure, and individual viral

Table 24-3
CDC Definition of Blood or Body Fluid Potential for HIV Infection

Potentially Infectious
Blood
Human tissue
Cerebrospinal fluid
Synovial fluid
Pleural fluid
Peritoneal fluid
Pericardial fluid
Amniotic fluid
Semen
Vaginal secretions
Any visibly bloody body fluid
Not Considered to Be Infectious
Feces
Nasal secretions
Saliva
Sputum
Sweat
Tears
Urine
Vomitus

Source: From Updated U.S. Public Health Service guidelines for the management of occupational exposures to HIV and recommendations for postexposure prophylaxis. *MMWR* 2005;54(RR-9):1–17.

setpoint. In general, it is highest about 3–4 weeks after infection and increases again in late-stage infection or when a particular antiviral regimen is failing [44, 45]. Not surprisingly, clinical and epidemiologic observations document that transmission occurs more often from contact with infected individuals just prior to seroconversion, in late-stage disease, or with a persistent high viral load despite treatment [46, 47].

About 80% of newly antibody-positive individuals recall a brief mononucleosis-like illness, the so-called acute retroviral syndrome, which can occur shortly after transmission and 10–21 days before seroconversion. The clinical manifestations of HIV infection associated with chronic and progressive immunocompromise are not expected for at least many months, more commonly years, and occasionally, a decade or more [44].

Percutaneous exposure is consistently reported to result in seroconversion in about 1 in 300 contacts. However, the risk of actual contacts clearly varies with exposure factors and can be as high as 1 in 20 with penetration of highly infective material into highly vascular tissue [2, 48] (Table 24-4). In the health care setting, blood splashes to oral mucosa and nonintact skin have been rarely associated with transmission [49]. The normal concentration of inflammatory cells in the human mouth could be expected to support receptive transmission, but saliva itself is inhibitory to virus. The rare reports of transmissions owing to bites emphasize both the extent of inflammatory disease and the presence of blood in the mouth of the biter [50]. HIV transmission following a punch to the mouth of a suspect has been reported in a police officer [51]. Isolated HIV transmission has not been associated with ocular mucosal contact alone.

Table 24-4
Factors Predicting Transmission of HIV to Health Care Providers after Percutaneous Exposure

	Adjusted Odds Ratio[a]	**Factor (95% CI)**[b]
Deep (intramuscular) injury	16.1	6.1–44.6
Visible blood on sharp device	5.2	1.8–17.7
Needle used to enter blood vessel	5.1	1.8–14.8
Source patient with terminal AIDS	6.4	2.2–18.9
Zidovudine prophylaxis used	0.2	0.1–0.6

[a]All were significant at $p < 0.01$.
[b]Confidence interval.
Source: Adapted from Centers for Disease Control and Prevention. Case-control study of HIV seroconversion in health care workers after percutaneous exposure to HIV-infected blood—France, United Kingdom, and United States, January 1988–August 1994 *MMWR* 1995;50:931.

Prevention of Blood Exposure for Health Care Workers

Occupational medicine professionals play a significant role in the ongoing implementation of a comprehensive exposure control plan. Their role in defining the plan will vary depending on the size and needs of a given institution. Table 24-5 contains the essential elements of a prevention program for employees who work with or who potentially may be exposed to bloodborne pathogens. OSHA provides a template exposure control plan to be modified to reflect local specifics [52]. A robust surveillance system for bloodborne pathogen incidents evaluates the potential for occupational exposures among all workers in a medical treatment facility. This may involve employees (both full and part time), temporary employees, contract employees (e.g., contract vendors, agency pools, per-diem employees, and individual service providers), volunteers (including adults and students of high school and college age) and medical, nursing, and other professional students.

A workplace program to evaluate potential employee exposures must be available 24 hours a day. This can occur either on-site or at a referral facility. Regardless of where the evaluation is performed, surveillance data detailing the specifics of exposure

Table 24-5
Essential Elements of an Exposure Control Plan

- Determination of employee exposure
- Implementation of various methods of exposure control, including
  - Universal precautions
  - Engineering and work practice controls
  - Personal protective equipment
  - Housekeeping
- Hepatitis B vaccination
- Postexposure evaluation and follow-up
- Communication of hazards to employees and training
- Recordkeeping
- Procedures for evaluating circumstances surrounding exposure incidents

Source: From Model Plans and Programs for the OSHA Bloodborne Pathogens and Hazard Communications Standards, 2003; available at *www.osha.gov/Publications/osha3186.html*.

need to be collected. These data should protect the identity of the employee. In addition to complying with the OSHA sharps log requirement, this ensures timely feedback and identification of worksites where training and procedure review need to be restarted owing to a turnover of personnel or need to be reemphasized to existing employees. An important task is to develop a culture of safety within the entire medical treatment facility and a climate where employees feel that they will be seen promptly and without retribution.

Training

The required elements of bloodborne pathogen (BBP) training are outlined in the OSHA bloodborne pathogen standard [53]. Initial training at the onset of at-risk work and follow-up annual refresher training are required for all personnel at risk for bloodborne pathogen exposure. This may be accomplished with a variety of media, including small or large group presentations, Web-based programs, and written training guides. A written or Web-based copy of the facility specific exposure control plan must be available at the worksite with clear directions on what to do in the case of a potential BBP incident. Preemployment evaluations offer an opportunity to remind future employees of the first aid and response plan to blood or body fluid exposures and to emphasize the benefits of prompt reporting.

Personal Protective Equipment, Engineering Controls, and Work Practices

The most important part of universal precautions is the use of personal protective equipment (PPE) by all employees at all appropriate times. Table 24-6 provides a sample PPE precautions guideline. Engineering controls are the physical objects that isolate or eliminate a BBP hazard. Sharps boxes, self-sheathing needles, and needleless access systems are examples that have been used successfully [54–56]. The revised OSHA standard clarifies the need for employers to select safer needle devices and to involve employees in identifying and choosing those devices. Work practice controls (e.g., prohibiting two-handed recapping of needles) are those institutional changes in the manner a task is performed that decrease the chances of an injury.

Table 24-6
Sample Personal Protective Equipment Precautions

All employees using PPE must observe the following precautions:

- Wash hands immediately or as soon as feasible after removing gloves or other PPE.
- Remove PPE after it becomes contaminated and before leaving the work area.
- Used PPE may be disposed of in [list appropriate containers for storage, laundering, decontamination, or disposal].
- Wear appropriate gloves when it is reasonably anticipated that there may be hand contact with blood or OPIM and when handling or touching contaminated items or surfaces; replace gloves if torn, punctured, or contaminated or if their ability to function as a barrier is compromised.
- Utility gloves may be decontaminated for reuse if their integrity is not compromised; discard utility gloves if they show signs of cracking, peeling, tearing, puncturing, or deterioration.
- Never wash or decontaminate disposable gloves for reuse.
- Wear appropriate face and eye protection when splashes, sprays, spatters, or droplets of blood or OPIM pose a hazard to the eye, nose, or mouth.
- Remove immediately or as soon as feasible any garment contaminated by blood or OPIM in such a way as to avoid contact with the outer surface.

Source: From Model Plans and Programs for the OSHA Bloodborne Pathogens and Hazard Communications Standards, 2003; available at *www.osha.gov/Publications/osha3186.html*.

Proper handling and disposal of sharps and procedures for handling contaminated laundry and clean-up of spills involving blood or other body fluids are important to avoid exposure of coworkers. Although not always clearly considered in the areas at highest risk for BBP incidents, housekeeping and laundry personnel must have appropriate training, including how and where to report an exposure. This training needs to be in an understandable language for the employee.

Institutional commitment to universal precautions, engineering and work practice controls, and PPE can decrease the number of occupational exposures substantially [56]. A clear, well-communicated, and easily accessible plan is crucial to achieve the goal of employees presenting for prompt post-exposure evaluation and potential prophylaxis.

Blood Exposure Evaluation

The evaluation of a worker with a potential exposure to HIV should be considered an urgent medical matter that requires prompt attention. The range of psychological reactions by those exposed