***Cover:*** *"Filleting the Catch"*, gouache, pen and ink, by Priscilla Cane, a Providence artist. Her work is on display at Providence City Hall, the Shepherd Building, Hasbro Children's Hospital, and Miriam Hospital; and she exhibits at the Bert Gallery.

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## COMMENTARIES



## BLOOD-BORNE PATHOGENS: A CME ISSUE
***Guest Editor: Marguerite Neill, MD***



## COLUMNS



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# COMMENTARIES

## We Can't Afford To See Patients

Many years ago I confidently predicted that the American health care system was so flawed and its economic structure so weak that it would soon reach rock bottom. I opined that a crisis would produce a phoenix to rise from the ashes, a system in which all Americans received reasonable care, doctors received reasonable remuneration, hospitals were reimbursed for services and a trend towards primary care would lead to more efficient use of specialty services. Talk about rose-colored glasses!

I recently spent some time chatting with a neurology department chair at a prestigious institution in New York. Thinking that the salary support I received for teaching students, residents and fellows - 10% - was a bit low, I asked him what percentage of salary the medical school or hospital provided to his faculty. He told me that the secret of his department's success was that only one person received salary support, that every other faculty member was grant, government, industry or foundation supported. The one person who did receive university money was the one faculty member devoted to teaching. "No one objects. He spends all his time going to meetings, talking to students and developing teaching materials. No one else wants to spend their time doing that. There's no reimbursement after all, so they're happy to let him do it and they can use their own time more profitably." "What about clinical work?" I asked. "It costs too much to let anyone spend time seeing patients. We have to limit clinical time to keep afloat."

No, I'm not making this up. Neurology doesn't have a lot of "bells and whistles," that is, money-making procedures. In Rhode Island, the neurology department has vascular, EMG and EEG laboratories. Most neurology clinicians use EMG testing to subsidize their clinical practice, and see many more patients in a day than their academic peers, but the concept of academic departments giving up clinical outpatient medicine wholesale is rather staggering. If one turns George Bernard Shaw's famous quote, "Those who can, do. Those who can't, teach," on its head, we get, in medicine, the new motto, "Those who can, don't. Those who don't, teach." And the more they don't, the more papers they write describing studies on patients they don't see. In my area of expertise, one can see a sub-specialist at a movement disorders center where each of the 15 neurologists sees patients one half day per week and trained with doctors who themselves saw patients at that same rate. All support themselves on grants. None can afford to see many patients. Each one of them could cite a reference reporting that carotid endarterectomy results, like coronary artery bypass grafts, are done best at centers where more are done, performed by surgeons who do them frequently. Why this simple concept that "practice makes perfect" doesn't translate into the practice and teaching of clinical medicine is that money talks and academic departments that are not procedure-heavy are not solvent (compare orthopedics, neurosurgery and ophthalmology to pediatrics and family medicine) The one salaried faculty member cited above who was paid for teaching may or may not see patients of his own. Probably, there's no time. He may, in fact be a superb teacher, and distinguished clinician.

There are large problems here. For one thing, once one acknowledges that the learning process in clinical medicine is an apprenticeship, one can deduce that one learns from experience guided by those more experienced. We don't learn plumbing or carpentry or hairstyling from people who don't practice the craft. The person who designs clothes may not be a good teacher for a would-be tailor. One learns experimental physics from an experimental physicist and not a theoretician.

The system that should have collapsed of its own weight long ago is being stressed even more. Seventy-five million Americans were uninsured at some point over the past three years and over 40 million are uninsured at any time. In Rhode Island, the insurers recently tried to reduce doctor reimbursement. Medicare reimbursement went down and the governor of Rhode Island is attempting to reduce hospital payments for non-reimbursed health care, thus passing on rate increases to the private insurers to avoid having to propose a tax increase. Those of us with health insurance pay higher rates in lieu of higher "taxes."

The fundamental problem is deceit. The government hides the problem. "America has the greatest health care system in the world." Yes, for the majority, and a terribly inadequate, bordering on a barbarous system, for a substantial minority. This deceit, that we are generous and well-meaning when we are not, has consequences for those of us lucky enough not to be affected directly. We teaching doctors see more patients, do a less good job, teach less, study less, do less un-reimbursed research. Our students learn less. They discern that they are often a burden, not a joy, despite spending huge tuitions to the medical school, which goes somewhere to pay someone.

One department at Brown Medical School was recently asked if it would like to submit a proposal to turn a clinical elective into a required rotation. "We'd love to," it said to the dean. "No way can we afford to do this," was the privately voiced opinion of every single faculty member. So a weak proposal was submitted, guaranteeing rejection. "We tried," was the happy response.

Apprenticeship can be a wonderful experience, but the ones who "do" are the ones who should teach. Virtual teachers are only good for virtual patients. In the real world apprentices work with masters. Masters need to be experienced and also need to be paid, just like anyone else.

**– JOSEPH H. FRIEDMAN, MD**

# A Healing Truth in Puns

With the notable exception of hermits and some elected officials, humans rely heavily upon words to communicate with each other. Physicians, more than most professionals, depend upon language to understand the ailments of their patients and then to provide instructions which may diminish or abolish their distress. Words, accurately chosen and unambiguously conveyed, then become the essential currency in the effective patient-physician relationship. It should not come as a surprise, therefore, to encounter amongst the population of physicians those who are gifted with words, who cherish words, and who may even make it their business to study their origins, their subtleties and nuances. And some physicians have ventured one step further in abandoning medicine to take up the thankless task of full-time writing. In truth, not many actually stray from medicine, the rate of truancy being low; yet a small number of these delinquents have achieved notable success as users of words.

Some of the great writers in recent centuries began as physicians. The ranks of physician-writers include Francois Rabelais, Oliver Goldsmith, John Keats, Somerset Maugham, Arthur Conan-Doyle, Anton Chekhov, and Gertrude Stein.

There was, for example, a practicing physician in Manchester, England. His name was Peter Mark Roget [1779-1869], and upon his retirement from medicine, he pursued his hobby of word-collection. He devised a unique kind of dictionary which stored synonyms and antonyms as well as the definitions of English words. He called it a thesaurus, a Greek word meaning treasury. To this day, students, writers and readers keep a copy of Roget's *Thesaurus* readily available.

And then there was Charles Carroll Bombaugh [1828-1906], whose love of English words brought him to devote much of his adult life to the exploration of verbal oddities and curiosities. Bombaugh graduated from Harvard in 1850 and received his MD degree from Philadelphia's Jefferson Medical College in 1853. He practiced medicine in Philadelphia until the Civil War, when he was appointed surgeon to a Philadelphia infantry brigade. Following the civil war he settled in Baltimore; but because of poor health he left the private practice of medicine to work both as an insurance company physician and as an editor of an insurance industry periodical. Bombaugh's lasting contributions to society were his texts on puns, riddles and other forms of word-play.

The pun, regarded by many as an egregious category of humor, has been known and appreciated by writers for millennia. The ancient Greeks delighted in manipulating their words and they even devised a word, *paronomasia*, which sounds much like a terrible psychiatric disorder but means to call something by using a slight name-change, for purposes of irony or humor. The Romans expanded the meaning of their word, *punctum* [a point, as in words such as punctuation or punctual] to signify a point of humor. In Italian, this became *puntiglio*, meaning a fine point, a delightful play on words, which in turn evolved into the English word, pun, a belabored, disingenuous form of low humor based upon the playful use of words with the same sounds but with disparate meanings.

Some years ago the *Wall Street Journal* defined these witticisms as follows:

Paronomasia:
Having fun
Is the measure of pleasure
And so the pun
Is the pleasure I treasure.

Each trade, each profession, has its own collection of cherished puns; and medicine is no exception. While puns are almost never encountered in that stilted, arthritic kind of writing required in the formal medical journals, they do abound in operating rooms, hospital locker-rooms, and wherever physicians meet at the end of tiring and dispirited days. Physicians accept the seriousness of their occupation when encountering words such as bacteria, terminal illness, seizures or colic. And perhaps to lessen the somber gravity of these words, their minds therefore construct absurd definitions: a seizure might be a Roman emperor or a salad; a bacteria becomes the service entrance to a cafeteria; a terminal illness could be sickness in an airport; and colic, or course, is a kind of sheep dog. Surgeons might define the word cauterize as making eye-contact with a pretty woman; post-operative as a letter carrier; and dilate meaning to live longer. Surely these are not examples of enduing humor, but they add a touch of shallow levity to the grave undertakings of medicine. To tolerate puns becomes an individual matter. As one surgeon noted: "Take them or leave them, suture self."

Many puns require a slight variation in the spelling and meaning of the key word. But some words or phrases carry dual meanings and therefore some puns require no spelling revisions. For example, "Does the name Pavlov ring a bell?"

Puns, bad or good, rarely have a single identifiable father. They tend to be born simultaneously in numberless communities since the creation of a pun requires little more than a single person contemplating a word or a phrase, subconsciously examining its sound and idly probing to unearth an alternate, thoroughly absurd meaning. A phrase such as "transcendental meditation" might suggest an alternate phrase such as "transcend dental medication" and then, secondarily, a story is superimposed upon the substituted nonsensical phrase. "There was a philosopher who refused a local anesthetic during root canal therapy because he wanted to transcend dental medication." And thus a pun is autonomously born in a hundred different fertile minds.

Puns can become the critical element to lengthier stories which, until the pun-line appears, seem quite credible. For example, there is the story of the two vultures boarding a commercial airplane. Each carries two dead raccoons. The stewardess says apologetically: "I'm sorry, gentlemen, only one carrion allowed per passenger."

Some plays on words, usually more elaborate than simple puns, are based upon a reversal of words rather than an ambiguity of pronunciation. For example, "Statistics are a major cause of drinking" [a reversal of "Drinking is a major cause of statistics."] Or, "At election time the air is filled with speeches—and vice versa." Silly little puns can sometimes harbor great truths.

Humor, including that mutant species called punning, is often born in stress, pain and doubt. Humor, said James Thurber, is emotional chaos remembered in tranquility. Thus, in a troubled world needful of healing, if a good pun brings a meager smile and then is quickly forgotten, it becomes its own reword.

**– Stanley M. Aronson, MD, MPH**

# Blood-borne Pathogen Education—Once More, with Feeling!

Most days on a busy clinical service, there is a cacophony of "must do's" of various deadlines. Clinical matters usually take precedence for most health care workers, and administrative matters are often *de facto* pushed into the next tier for a variety of very good reasons. Most clinicians have too much to do, regardless of the setting (ER, hospital ward, urgent care center, physician's office) and because of the basic orientation in the health care profession that patients come first, clinical deadlines become the priority. Administrative matters, like the endless rounds of insurance paperwork, credentialing applications (always asking repetitively for the same information) and licensing applications, are pushed into second tier priority – "I'll get to that later".

Blood-borne pathogen education is usually a second tier priority for most physicians. In Rhode Island, it has been tied to medical licensure since the mid-1990s with a requirement for 2 hrs of CME credit in the specific area of blood-borne pathogen education. In July, 2000, *Medicine and Health Rhode Island* published an issue devoted to this topic with CME credits provided. It was one of the most successful issues of the journal, as measured by the number of copies sold and by the number of physicians who utilized it to gain their CME credits for blood-borne pathogen education. Many infection control practitioners have used it for in-services for staff.

One wonders whether these same statements could be made if the education component wasn't a requirement. In all likelihood, probably not. The reasons for this are not glamorous but they reflect reality. In these days of heavy patient loads, chronic understaffing and overflowing in-boxes (both electronic and tangible), we are simply too busy; there's no "down time". Some who perceive they are at risk for BBP transmission have the best of intentions to "take care of this stuff", including their own hepatitis B vaccination but they just never get to it. There are those who don't think they are really at risk, somehow imagining that they can perceive which patients are infected with hepatitis B, hepatitis C and/or HIV. It is worth noting that the rate of new HBV infection in HCWs did not fall until the 1991 OSHA rule was implemented which mandated the provision of access to HBV vaccine to those whose occupations place them at risk of infection. Subsequently the incidence of acute HBV infection in HCWs has fallen to levels below that in the general population.

So in all likelihood, the carrot and stick approach for BBP education works and it seems unlikely that the requirement will be removed in the near future. But is this just a dog-and-pony show, going over the same old material to satisfy a requirement? Where should we be looking to go from here?

I have requested all the authors for the articles for this issue to provide up-to-date material and references (we have included many from 2003). This helps in providing important updates from our prior issue of the journal; examples include the difference in clinical history for HBeAg negative chronic HBV infection, published experience treating acute HCV infection and sources of expert consultation for difficult decisions on HIV post-exposure prophylaxis. Specific articles on non-occupational exposures for HCV and HIV are included, since the evolution of BBP education is to make physicians realize that BBP transmission in non-occupational settings is within the scope of what they need to recognize and either know how to manage, or know where to refer.

What might be some questions for future updates? These are separate from the hardcore science of new vaccines, drugs and immunobiologics. These include whether there are hard outcome data for the effectiveness of various types of BBP education, whether the educational effort results in more accurate exposure recognition especially in the community, how to deal with over-treatment in a pragmatic way, and how to configure access to knowledgeable experts for front-line clinicians.

The population of chronically infected persons with these blood-borne viruses in the US, some of whom are multiply infected, has not markedly changed in recent years. Given that the overall magnitude of transmission risk for patient to provider is much greater than for provider to patient, staying current on BBP-related issues is important for one's personal health as well as one's medical license. The lyrics hopefully have a familiarity to them: make vaccine widely available, prevent exposure when possible, and evaluate rapidly for intervention as needed. Now: once more, with feeling!

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# Hepatitis B: New Options in Diagnosis and Treatment

*Michael Poshkus, MD, and Staci A. Fischer, MD*

Despite the availability of blood product screening and an effective vaccine for twenty years, **hepatitis B (HBV)** remains a significant public health concern in the year 2003. The **Centers for Disease Control and Prevention (CDC)** estimates that there were 78,000 new infections with HBV in 2001,[1] a decrease of 66% from 1981. All U.S. children are now vaccinated against HBV as a part of routine immunization programs and Rhode Island has the highest immunization coverage for hepatitis B among children in the U.S. Despite this accomplishment, there still are 22 to 75 new cases of acute hepatitis B reported each year in Rhode Island, with estimates of 250 to 800 actual new cases each year.[2]. Because there is substantial morbidity and mortality with this bloodborne pathogen, there remains a need for continued vigilance in diagnosis and prevention of transmission of HBV. **Health care workers (HCWs)** are a specific target group for education on hepatitis B prevention. Because they are at occupational risk for acquiring HBV infection, HCWs are recommended for HBV vaccination.[1,3] In addition, HCWs should focus their professional efforts to prevent needlestick injuries, to insure vaccination of at-risk patients and identify candidates for treatment.

## Epidemiology

Infection with HBV remains a significant problem worldwide. An estimated 1/3 of the world's population (2 billion people) have been or are infected; 350 million people are chronically infected with HBV. There are 1 million HBV-related deaths annually;[4] most are due to complications of cirrhosis or **hepatocellular carcinoma (HCC)**.

While nearly all body fluids contain virus in the infected patient, transmission of HBV occurs almost exclusively with percutaneous or mucous membrane exposure to blood or semen.[4] In highly endemic areas such as Southeast Asia and Africa, HBV is usually acquired perinatally or during early childhood. The risk of infection is highest in infants born to HBeAg positive mothers (see Diagnosis), in whom up to 90% will be infected by the age of six months. Children infected early in life are generally asymptomatic, and often reach adulthood without overt manifestations of HBV infection.[4] Rhode Island practitioners are likely to encounter adult immigrants from endemic areas who, as a result of perinatal transmission, may be chronic HBsAg carriers or may progress to development of cirrhosis or HCC.

In areas of the world with a lower prevalence of HBV infection, transmission usually occurs from sexual contact or percutaneous exposure. With such a transmission pattern, the groups at particularly high risk of infection include HCWs, hemodialysis patients, injection drug users, correctional facility inmates and sexual contacts of HBV-infected patients. The risk of HBV transmission with transfusion of blood products in the U.S. is minimal as a consequence of blood donor screening. However, blood product screening is not universal in other parts of the world, and travelers and immigrants may acquire HBV infection from transfusion or injection with nonsterile needles in some overseas medical settings.

With the advent of effective vaccines (see Prevention), the rate of acute HBV in children (ages 1-8) has declined >80%, and that in HCWs, 95%, over the past decade.[5] Approximately 70% of patients with acute hepatitis B are between the ages of 20-39, and 60% are male.[1] Nearly 60% of these patients have been previously treated for a sexually transmitted disease or have been incarcerated prior to diagnosis.[6] Contact with the health care and social services systems represents an opportunity to both prevent and identify HBV infection.

## Clinical Manifestations and Course of Infection

Acute infection with HBV is asymptomatic in 50-70% of adult patients. In those who develop symptoms, nausea, vomiting, anorexia, fatigue, malaise, pruritus, icterus, jaundice and/or right upper quadrant pain develop a mean of 120 days following exposure (range, 45 to 160 days).[4] Up to 20% of affected patients may have a serum-sickness-like reaction to HBV, manifested by rash (erythematous, maculopapular eruption

Table I:

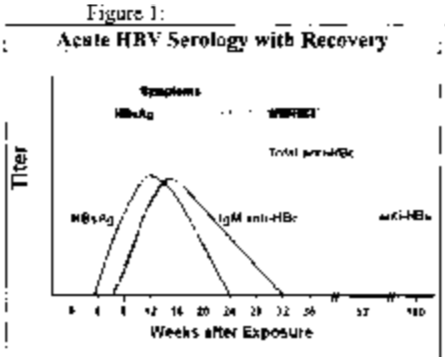
**Interpretation of the Hepatitis B Panel**

| Tests | Results | Interpretation |
|---|---|---|
| HBsAg<br>anti-HBc<br>anti-HBs | negative<br>negative<br>negative | susceptible |
| HBsAg<br>anti-HBc<br>anti-HBs | negative<br>positive<br>positive | immune due to natural infection |
| HBsAg<br>anti-HBc<br>anti-HBs | negative<br>negative<br>positive | immune due to hepatitis B vaccination |
| HBsAg<br>anti-HBc<br>IgM anti-HBc<br>anti-HBs | positive<br>positive<br>positive<br>negative | acutely<br>infected |
| HBsAg<br>anti-HBc<br>IgM anti-HBc<br>anti-HBs | positive<br>positive<br>negative<br>negative | chronically<br>infected |
| HBsAg<br>anti-HBc<br>anti-HBs | negative<br>positive<br>negative | four<br>interpretations<br>possible* |

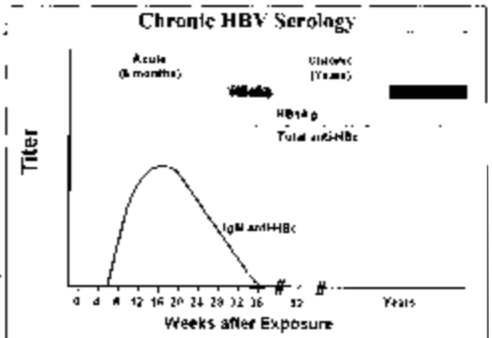
* 1 May be recovering from acute HBV infection
2 May be distantly immune and titer not sensitive enough to detect very low level of anti-HBs in serum
3 May be susceptible with a false positive anti-HBc
4 May be undetectable level of HBsAg present in the serum and the person is actually a carrier

Source: CDC website [1]

Figure 1:




Typical serologic markers and time course for acute Hepatitis B




Typical serology and time course for patients who develop chronic Hepatitis B.

Source: CDC website [reference 1]

or urticaria), arthralgias/arthritis and, on occasion, short-lived fever (which is otherwise an unusual finding with acute viral hepatitis).[7] Other complaints include headache, tobacco intolerance, clay-colored stools and dark amber urine. Physical findings include icterus, jaundice, tender hepatomegaly and splenomegaly. Symptoms usually resolve spontaneously in 4 to 6 weeks. In the acute phase of infection, total bilirubin levels rise, sometimes to 10 mg/dl. Transaminases often rise to levels of 1000 to 2000 IU/ml, with ALT often higher than AST; peak transaminase values do not correlate with the degree of hepatic injury. Alkaline phosphatase levels are usually normal. Total white blood cell counts are usually normal, although mild anemia and atypical lymphocytes (<10%) may be seen. Urobilinogen, bilirubin and protein are often excreted into urine during acute infection.

In a minority (0.1 to 5%) of cases, fulminant hepatitis with hepatic failure develops, causing coagulopathy, encephalopathy and cerebral edema. This complication carries a mortality rate of 63-93%, with death occurring within 3 weeks of symptom onset.[8] In many patients, transaminases fall precipitously as hepatic failure develops. The risk of fulminant disease increases in the presence of coinfection with the delta agent (hepatitis D).[8]

During acute infection, ninety percent or more of affected patients develop protective antibody (HBsAb) and spontaneously clear HBV infection; neonates are an important exception. The remainder develop persistent infection, which may be asymptomatic ("persistent carrier state") or, in up to 40% of chronically infected patients, have progressive disease resulting in cirrhosis and end stage liver disease.[9] Chronic persistent hepatitis results in intermittent (or chronic) jaundice, fatigue and malaise. Some 10 to 20% of chronically infected patients develop immune complex-mediated membranous or membranoproliferative glomerulonephritis , leukocytoclastic vasculitis, cryoglobulinemia or polyarteritis nodosa.[8] Such extrahepatic manifestations of HBV infection may require immunosuppressive therapy; studies of the effect of known antiviral therapies on such processes are ongoing.

*Approximately 70% of patients with acute hepatitis B are between the ages of 20-39…*

## Diagnosis

While elevated transaminases may prompt the discovery that a patient has HBV infection, they are not helpful in following its course. A basic understanding of hepatitis B serology is critical to the diagnosis of acute and chronic HBV infection (Figure 1 and Table 1).

Hepatitis B surface antigen (HBsAg) becomes detectable in serum by 2 months after exposure in most patients and its subsequent clearance from the blood is usually correlated with the development of HBsAb. This loss of circulating HBsAg with appearance of HBsAb usually signifies resolution of infection when serial serologic testing demonstrates that this has occurred in acute HBV infection. This same serologic evolution, if detected in the setting of chronic HBV infection, does not always correlate with resolution, as HBV DNA has been detected by PCR assay in some individuals.[9] The importance of the distinction is that such patients are still infectious and at some risk of cirrhosis and HCC, whereas those with truly resolved infection are not.

Patients with chronic HBV are defined as those with detectable HBsAg for > 6 months.[10] In many chronic carriers, seroconversion with HBeAb corresponds to histologic resolution of inflammation, with remission and regression of fibrosis. These HBeAg negative patients generally have a benign course, with a low risk for cirrhosis or HCC. The presence of HBeAg has been traditionally felt to be a sign of active viral replication and as such, a marker of infectivity. Recently, strains of HBV have been described in the Mediterranean Basin, Middle East and Asia that are unable to express HBeAg, so that viral replication may be occurring despite the absence of HBeAg. These variants tend to be more aggressive, are more likely to result in chronic disease, and are less responsive to available antiviral therapies than HBeAg-positive strains.[11] In patients emigrating from these areas, HBV quantitative PCR testing (HBV DNA) is indicated for definitive diagnosis. Regardless of HBeAg status, all chronic HBV carriers should be advised to avoid hepatotoxins such as alcohol and certain herbal preparations and be vaccinated against hepatitis A to protect hepatocytes from additional injury.

Quantitative PCR testing for HBV viral DNA is now routinely available and is evolving as a diagnostic tool in conjunction with conventional HBV serologic testing. It has been useful in the diagnosis of HBeAg-negative variant disease and in recently described patients who are HBsAb positive, but who have persistent HBsAg, presumably of a variant strain.[4,12] PCR testing is also useful in following the efficacy of treatment with antiviral agents.

Table 2

**Recommended postexposure prophylaxis for exposure to hepatitis B virus**

| Vaccination and antibody response status of exposed workers* | Treatment | | |
|---|---|---|---|
| | Source HBsAg† positive | Source HBsAg† negative | Source unknown or not available for testing |
| Unvaccinated | HBIG§ x 1 and initiate HB vaccine series¶ | Initiate HB vaccine series | Initiate HB vaccine series |
| Previously vaccinated | | | |
| Known responder** | No treatment | No treatment | No treatment |
| Known nonresponder†† | HBIG x 1 and initiate revaccination or HBIG x 2§§ | No treatment | If known high risk source, treat as if source were HBsAg positive |
| Antibody response unknown | Test exposed person for anti-HBs¶¶<br>1. If adequate,** no treatment is necessary<br>2. If inadequate,†† administer HBIG x 1 and vaccine booster | No treatment | Test exposed person for anti-HBs<br>1. If adequate,¶ no treatment is necessary<br>2. If inadequate,¶ administer vaccine booster and recheck titer in 1–2 months |

* Persons who have previously been infected with HBV are immune to reinfection and do not require postexposure prophylaxis.

† Hepatitis B surface antigen.

§ Hepatitis B immune globulin; dose is 0.06 mL/kg intramuscularly

¶ Hepatitis B vaccine

** A responder is a person with adequate levels of serum antibody to HBsAg (i.e., anti-HBs ≥10 milliunits/mL).

†† A nonresponder is a person with inadequate response to vaccination (e.g., serum anti-HBs < 10 milliunits/mL).

§§ The option of giving one dose of HBIG and reinitiating the vaccine series is preferred for nonresponders who have not completed a second 3-dose vaccine series. For persons who previously completed a second vaccine series but failed to respond, two doses of HBIG are preferred.

¶¶ Antibody to HBsAg.

Source: MMWR 2001;50(RR11):1, 42 [22]

## Treatment

Anti-viral therapy is generally reserved for patients with histological evidence of at least moderate disease activity and fibrosis in the setting of persistently elevated ALT and HBV DNA levels. A number of agents have demonstrated promise in the treatment of HBV infection, including alpha-interferon, lamivudine and adefovir. Although the prevention of cirrhosis and HCC is the ultimate goal of treatment, most studies have utilized short-term, serologic endpoints such as loss of HBeAg and HBV DNA levels to evaluate efficacy.[13] As longer term studies are conducted, resistance to antiviral agents is increasingly being described.[14]

Recombinant interferon alpha-2b, administered subcutaneously for 4 to 6 months, has been effective in up to 45% of patients with chronic HBV infection in short-term studies.[15] Its cost and parenteral route of administration may limit its use in certain patients. Lamivudine, an oral nucleoside analogue also used to treat HIV, effectively inhibits its viral replication in many treatment-naïve patients, but resistance develops on therapy in up to 1/3 of cases.[16] Adefovir, a nucleotide analogue, has recently been shown to be effective in treating both HBeAg- positive and -negative patients; emergence of resistance on therapy has not yet been described.[11,17] Further studies of new agents and combinations of agents are ongoing.

## Prevention

The goal of HBV prevention is to minimize the incidence of acute infection thus preventing progression to chronic disease. This is being achieved by the screening of blood, organ and tissue donors, increased public awareness of risk reduction, infection control practices in institutions and vaccination programs.

Infection control practices in the health care setting are geared to prevent transmission of HBV and other blood-borne pathogens from chronic carriers to non-immune hosts.[18] Blood-contaminated objects (e.g., indwelling vascular catheters) and sharp instruments (e.g. needles, surgical instruments) must be handled properly to prevent injury to the HCW themselves or to a colleague. HBV can survive for up to a week on environmental surfaces; disinfection using bleach-containing cleansers is recommended.[19] As the hands of health care workers may provide a means for transmission from a contaminated fomite to a patient, strict contact isolation should be observed in dialysis centers and other settings in which the prevalence of HBV infection may be quite high.

The HBV vaccine is the first vaccine proven to prevent cancer. Significant decreases in the rates of HCC have been seen among children in highly endemic areas after the initiation of HBV vaccination to newborns.[4] Currently in the US, HBV vaccination is recommended for all children between the ages of 0-18 years and to all individuals at higher risk of HBV infection than the general population (HCWs, clients and staff of residential institutions, hemodialysis patients, people with bleeding disorders requiring transfusions, close contacts of people with HBV, IVDAs).

Current vaccine formulations contain recombinant HBsAg and no thimerosol. The vaccine is given as a series of three doses (at 0, 1 and 6 months) intramuscularly in the deltoid area, after which up to 95% of recipients develop protective HBsAb.[20] After receiving only one dose of vaccine, 32-56% of adults achieve protective antibody levels and >70% respond after the second dose.[6] HCWs or others at risk for continued exposure should be tested for HBsAb 1-2 months following the third dose to confirm vaccine response (5-32% of vaccinees are nonresponders).[1,3] If the results are negative, the 3-dose series should be repeated and the patient retested.

Serially following the decline in HBsAb after a documented response to vaccination is controversial, however, some recommend periodic testing of antibody levels and boosters for undetectable levels in those at risk.[3,21] Although HBsAb levels in successfully vaccinated HCWs may decline to unde-

tectable, an anamnestic HBsAb response may still provide protection after exposure.[1,3]

Post-exposure prophylaxis is indicated following percutaneous and perinatal exposure to HBV. (Table 2)[22] HBIG provides passive immunity, but immunization with hepatitis B vaccine is also indicated.

## Conclusion

In the past decade, substantial progress has been made in the control of hepatitis B. There remain areas for improvement, both in vaccination rates of high-risk populations, protection of HCWs, and treatment of acute and chronic disease. Although three drugs are currently approved for HBV therapy (interferon alpha, lamivudine and adefovir), all have shortcomings regarding efficacy, ease of administration, development of resistance, and cost.

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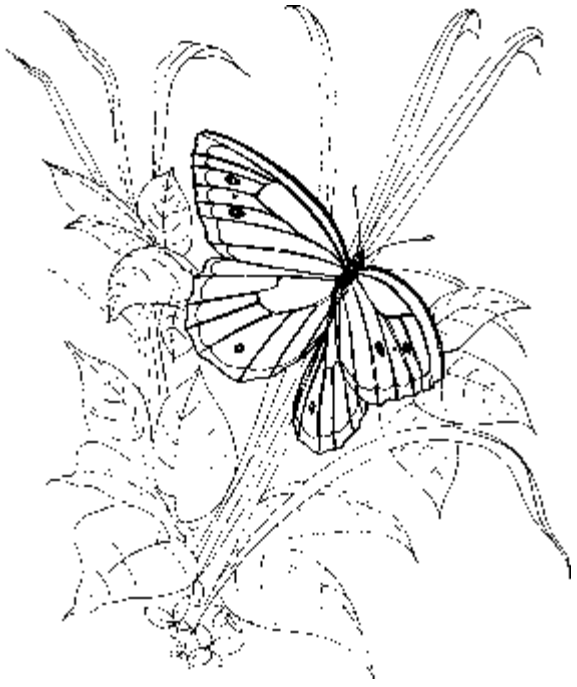

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# Occupational Exposure to Hepatitis C Virus Infection

*Diana Ng, MD, and Edward R. Feller, MD*

**Hepatitis C virus (HCV)** is most commonly spread by contact with infected blood or blood products.[1] Occupational exposure to this blood-borne pathogen is a potentially serious risk to **health care workers (HCW)** for whom an association between increased HCV risk and employment has been noted. An HCV seroprevalence rate of 2.7% among all HCWs has been observed compared to a prevalence of 1.8% in the general US population; however, surgeons, a group deemed at high risk, have a seroprevalence of only 0.8-0.9%.[3]

Any percutaneous contact with blood or blood products is a potential source of HCV infection. HCV seropositivity rates as high as 18% have been reported in emergency room patients at an inner-city hospital.[4] Patients at greatest risk to transmit HCV are IVDUs, trauma victims, individuals known to be positive for other bloodborne pathogens, and patients in high-risk groups for sexually transmitted diseases. Unlike **hepatitis B virus (HBV)**, no vaccine exists against HCV infection. Taken together, the recurrent opportunity for exposure to blood containing HCV and the lack of a protective vaccine underscore the very real need for HCW eduction regarding HCV exposure and infection prevention.

## Diagnostic Difficulties

The average incubation period for HCV after a needlestick injury or other percutaneous exposure is 7-8 weeks. In the health care setting, a diagnosis of hepatitis C may not be established in many patients, including those in high risk groups, thereby blunting overt recognition by a HCW that a blood exposure has occurred to an HCV infected patient. The majority of patients with chronic HCV are undiagnosed and asymptomatic, and may have no abnormalities in routine blood studies including liver transaminases. Detection of infection in a HCW after exposure requires systematic laboratory testing in followup because as many as 75% of persons with acute HCV infection have no symptoms or only mild, nonspecific complaints.[1] Serum antibodies to HCV become detectable 4 to 10 weeks after exposure. Percutaneous injuries may occur at a rate approximating 1 to 3 per 100 operative procedures performed, the frequency varying by the type of surgery, length, and emergent nature.[5,6] A majority of respondents to surveys indicate that they do not routinely report instances of percutaneous exposure to potentially infected blood. Because as many as 70-90% of patients with acute HCV infection will continue to have viremia and chronic HCV infection, identification of occupational exposure has important personal and public health implications.

*Unlike **hepatitis B virus (HBV)**, no vaccine exists against HCV infection.*

## Risk of occupational transmission

HCV transmission poses a serious potential hazard to persons who sustain needlesticks, mucocutaneous blood splashes, or contact of mucous membranes of the eyes and mouth with infectious material. Risk is a function of both prevalence of HCV infection in the patient population and the nature of the exposure; it is influenced by the size of the inoculum, viral titer, type of exposure, and depth of inoculation After needlestick exposure, the HCV seroconversion rate is approximately 1.8%-3%; [3,7] however, one report documented HCV RNA positivity in 7 of 68 HCWs (10%) post-needlestick injury.[8] In comparison, a general estimate of bloodborne virus transmission after needlesticks involving infected blood has been formulated as a rule of threes: HIV is transmitted in 0.3 % of exposures, HCV in 3% and HBV in 30%.[1]

Non-percutaneous exposures rarely result in HCV transmission. Isolated case reports have documented infection following blood splashes to the conjunctivae.[9] No documented HCV transmission has been reported after exposures of intact skin to infected blood. Some studies have reported low levels of HCV RNA in saliva and sweat of seropositive individuals,[10,11] but HCV transmission to health care workers from saliva or other body fluids has not been documented. Compared to HBV, HCV is fragile and rapidly degraded at room temperature; thus, the risk of environmental transmission from infected bloody garments or similar hazards is believed to be minimal.

## Epidemiology of increased risk

The risk of blood contact with HCV is increased in certain health-care environments.[12] Groups at increased risk are those with consistent exposure to blood and the use of sharp instruments, including personnel in the surgical specialties, operating room, intensive care unit, emergency room and dialysis units as well as paramedics. In one study of the risk of percutaneous injuries during surgical procedures,[6] there were ninety-nine percutaneous injuries during 1382 procedures, with 73% occurring during suturing. Not unexpectedly the rate was slightly higher for surgical residents compared with attendings (2.5 and 2.1 per 1000 person-procedures, respectively). Exposure of nurses and technicians was correlated with exchange of sharp instruments such as scalpels and needles during the procedure or during disposal. The highest rates, 9% and 10%, were noted in cardiac and gynecologic surgery, respectively. An Italian

national survey assessing 15,375 general surgical procedures reported 1418 (9.2%) accidental exposures to blood or body fluids. Needlestick injuries were implicated in 36.4% of cases and glove breaching in 33.6%.[13] In one survey,[14] operating room maneuvers associated with injury by sharp instruments were described as holding or retracting tissue with fingers (23%), pulling a needle through tissues with fingers (17%),needle dropped or loose in operative field (15%), and manipulation of a needle in its holder with fingers (6%).

### Post- exposure management

Standard immune globulin is not effective in preventing HCV infection.[2] Currently manufactured immune globulin is produced from plasma from donors screened for antibodies to HCV. Currently neither immune serum globulin nor anti-viral agents are recommended for postexposure prophylaxis. (Table 1) Of considerable interest is a recent study which evaluated the use of alpha-interferon in documented acute HCV infection.[15] Of 44 patients treated with a 24-week course of alpha-interferon, 43 (98%) had a sustained virologic and clinical response. The average time from exposure to initiation of treatment was 89 days. These results from one study with a small sample size suggest that antiviral treatment should be an option offered to individuals with documented acute HCV infection. Evidence is lacking, however, to determine whether anti-viral treatment should be offered only in the course of confirmed occupational transmission or therapy reserved for individuals who remain HCV RNA positive after 6 months.[16]

When a definite or suspected high-risk exposure to HCV has occurred, standard institutionally- established protocols should be initiated. The source of exposure should be tested for serum HCV RNA and the exposed individual should have baseline tests for HCV antibody and serum transaminases. HCV RNA may be present in serum 10–14 days after exposure. Antibodies to HCV may not be detectable until several months later. Because the incubation period of HCV may be as long as 10 weeks, follow-up studies should be performed at 3 and 6 months post-exposure. It is generally recommended that transaminases be the primary tests that are followed; if elevated, then HCV RNA testing should be done.

**Table 1. Management of Occupational Exposure to HCV**

1. **Blood- borne pathogen exposure protocols**
   - Institute institutionally- established policies, begin reporting procedures, consult expert resources , assure confidentiality and informed consent

2. **Test source of exposure for HCV RNA**
   - Exposure to patients without viremia (measured by serum HCV RNA) has minimal risk. HIV and HBV status are also assessed.

3. **Baseline testing of exposed individual**
   - Serum HCV antibody , serum transaminases. Note that HCV antibodies are not detectable until 4-10 weeks after exposure.

4. **Repeat serologic testing for HCV at 3 and 6 months(if source is HCV +)**
   - The majority of acute HCV infections are asymptomatic.

5. **Immunoglobulin prophylaxis is ineffective**
   - HCV antibodies are not protective; no plasma with specific anti-HCV antibodies is available.

6. **Counsel persons who seroconvert to HCV**
   - One study of anti-viral therapy with interferon for acute HCV infection resulted in HCV RNA clearance from serum in 43 of 44 patients.
   - Inform HCW occupationally exposed to HCV that sexual transmission is rare, but can occur.
   - No work restrictions for HCV-positive personnel

### Transmission of HCV from infected personnel or the environment

Nosocomial transmission of HCV is rare, but has been reported due to lapses of appropriate disinfection procedures. Multiple dose vials for injection which became contaminated and decontamination problems with shared dialysis equipment have been implicated in isolated outbreaks.[17] Ineffective decontamination of a colonoscope resulting in patient –to-patient transmission of HCV during colonoscopy has been documented by nucleotide sequencing of HCV isolates.[18]

Rarely, suspected transmissions of HCV from surgeons to patients have been described.[19] Failure of an anesthesiology assistant to wear gloves despite an open hand wound was implicated in HCV transmission from the assistant to 5 patients.[20] Mathematical modeling indicates that if a surgeon is HCV RNA positive, the risk of HCV transmission in a single case is 0.014%.[21] This rate is in the same range as the risk of HCV acquisition by transfusion of blood from first-time donors with blood screened as negative for HCV antibodies. This event likelihood translates to an HCV transmission risk in at least 1 of 5000 surgical procedures performed by a surgeon over 10 years of 0.9%. A sobering report from Spain recounts the story of an HCV positive anesthesiologist who infected 217 of his patients with HCV.[22] The physician, a morphine addict, allegedly gave himself a portion of opioid analgesia intended for patients, then used the same syringe and needle to administer the remainder intravenously to the patient.

In summary, the major risk of HCV in health-care environments is transmission from an infected patient to a HCW. Comprehensive prevention at both an institutional and personal level is vital to decrease the risk for infection. New data supporting the possibility of effective early anti-viral

treatment underscores the importance of post-exposure testing .

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# Sexual Transmission of Hepatitis C: Practical Recommendations

*Patricia Myung, Carol Mallette, MD, Lynn Taylor, MD, Scott Allen, MD, Edward Feller, MD*

Approximately 4 million Americans are infected with the **hepatitis C virus (HCV)**, with a prevalence rate of 4.1% for males and 1.6% for females.[1] This virus is a major cause of chronic liver disease, responsible for approximately 30% of cases of end-stage cirrhosis in the USA. In addition, HCV infection is the most common indication for liver transplantation and is a major risk factor in the development of hepatocellular carcinoma. Most cases of HCV infection in the USA are due to **intravenous drug use (IVDU)**, or receipt of blood products prior to the initiation of routine blood donor screening in the early 1990s.

Although HCV can also be transmitted sexually, this occurs much less frequently than with hepatitis B, another blood-borne viral pathogen. In HBV infection, as many as 40% of non-immune sexual partners may become infected.

We review the epidemiology and risk factors of sexual transmission of HCV to offer informed guidance to clinicians for disease prevention.

## Modes of transmission

As many as 70-90% of individuals acutely infected with HCV do not clear the virus and develop chronic HCV infection marked by the presence of HCV RNA in the blood. HCV antibody is not protective and is present with chronic viremia. Infectiousness is related to ongoing viremia; patients who clear HCV RNA from blood, either spontaneously or through treatment with interferon-based regimens, remain HCV antibody positive, but are not believed to transmit infection.

The most efficient route of HCV acquisition is parenteral. HCV prevalence among **intravenous drug users (IVDUs)** has been reported to be as high as 60-90%. As many as 60-70% of current US cases are believed to be due to IVDU.[2] Transfusion of contaminated blood products accounts for approximately 10-15 % of infections acquired before the introduction, in the early 1990s, of serologic tests for antibodies to HCV. Blood transfusions therefore remain a common cause overall of chronic HCV, but a rare cause of newly acquired infection. An estimate of the current risk of acquiring transfusion-related HCV is as low as 1 in 250,000 transfusions.[2]

While other routes of transmission have been described, all share a common feature of exposure to blood. (Figure 1) These include sharing instruments contaminated with HCV (such as razors for shaving and straws used for intranasal cocaine inhalation), occupational exposure to contaminated blood products, perinatal transmission from an infected mother, and sex with an infected partner. The transmission of blood during tattooing, commercial shaving, or body piercing may confer increased risk, although associated IVDU may be difficult to exclude. Data from medical personnel exposed to HCV indicate a very low risk from mucocutaneous exposure, such as blood splashes to the conjunctivae.[3] Rare nosocomial transmission of HCV has been described, including transmission to patients from an infected surgeon, and transmission to two patients after colonoscopic procedures performed with an instrument contaminated by use on an HCV-infected patient on the same day.[4] No recognized risk factor can be detected in as many as 10-20% of cases. An unknown percentage of these individuals may have an undisclosed history of illicit drug use.

## Pontential contribution of sexual transmission to HCV infection

An increased risk of having sexual partners who are HCV seropositive exists in individuals with HCV, but such data do not prove that transmission occurred through sexual contact. No large studies exist in which a cohort comprised of an HCV- infected case and an uninfected partner is followed prospectively to determine the incidence of subsequent infection over time. In the U.S., approximately 10-12% of individuals with acute HCV infection deny IVDU or a history of blood transfusions, but do report sexual contact with an HCV-positive partner.[5] Other data from the Centers for Disease Control and Prevention (CDC) indicate that 18% of HCV-positive individuals reported sexual contact with an HCV-positive person, or multiple-sexual partners within the previous 6 months as the only HCV risk factor. Women whose sexual partners were HCV-antibody positive were 3.7 times more likely to be HCV-antibody positive than women

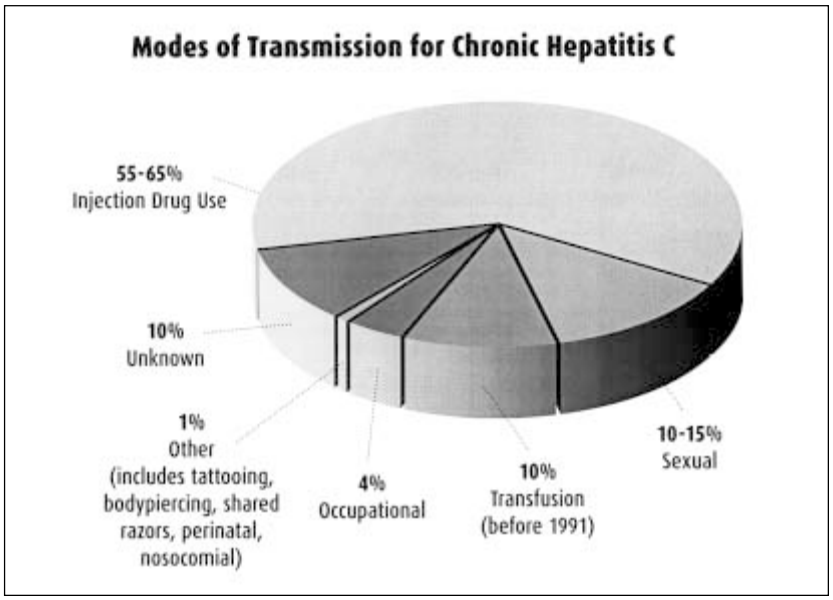

**Table 1: Associations of Risk for HCV Sexual Transmission**

| Clear Association with Increased Risk of HCV Sexual Transmission | No Clear Association with Increased Risk of HCV Sexual Transmission |
|---|---|
| Partners of HCV-infected individuals | HCV viral load |
| Number of lifetime sexual partners | HCV genotype |
| History of sexually transmitted disease | Stage of liver disease |
| Active genital inflammation/infection | Duration of relationship |
| Sexual practices that injure mucosa | Men who have sex with men |
| Non-use of condoms | HIV co-infection |

with HCV-negative partners.[6] Of 311 anti-HCV positive patients, the prevalence of HCV antibody was 10.3%; however, 85% of these reported parenteral exposure (IVDU, transfusions, multiple-use glass syringes for injection). Of 22 tested for HCV RNA, only 8 of 13 of those with detectable virus had the same HCV genotype. These data indicate that non-sexual routes of transmission may be common in couples concordant for HCV seropositivity.

### Evidence for HCV in body fluids

Exchange of infected body fluids across mucosal surfaces is the presumed mechanism of viral sexual transmission. HCV RNA in body fluids other than blood is usually either absent or of very low titer. Some studies of semen, saliva, and vaginal secretions in HCV-positive patients have failed to find HCV RNA.[7,8] Other reports indicate low levels (100 viral copies/ml) in vaginal secretions , semen ,and saliva.[9] One recent study found very low titer HCV RNA in semen ( levels between 20-172 viral copies per ml) in 8 of 24 HCV seropositive men.[10] Manavi and co-workers detected HCV RNA in 8 of 22 cervical smears.[11] Positive findings in this study were confined to cervical lymphocytes, with no HCV found in cervical epithelial cells or granulocytes. It is not clear whether HCV replication is confined to the liver, thus in the absence of suitable target cells for replication in the genital tract, limiting HCV transmission by non-parenteral routes.[12] The exponentially higher titer of HIV and HBV in body secretions of individuals infected with these viruses may explain the greater efficiency of sexual transmission of these infections compared to HCV.

Data from pooled studies of occupational exposure of health professionals with percutaneous exposure to blood from anti-HCV positive patients indicate that an average of 3.5% developed anti-HCV positivity.[13] Rare reports of HCV transmission after mucous membrane exposure (e.g. conjunctivae) exist.

*HCV is not transmitted by coughing, sneezing, or sweating.*

### What is the risk for sexual transmission within monogamous couples?

Seroprevalence data in heterosexual, monogamous partners of HCV-infected individuals suggest a very low transmission rate. Variables most closely correlated with HCV positivity are risks for potential blood-borne exposure ( IVDU with needle sharing , prior blood transfusion).In one study of 94 husbands of women with self-limited or chronic HCV acquired from contaminated immunoglobulin , no partner developed anti-HCV positivity over 10 –15 years.[14] In one prospectively followed cohort of HCV-infected individuals and their seronegative partners, 6 of 449 became infected, an incidence of 12 per 1000 patient-years. Viral homology supported sexual transmission in 4 of 6 cases.[15] A low rate of transmission (3 of 106) was found in female partners of HCV-positive hemophiliacs.[16] Sexual contact is a much less efficient route of transmission of HCV than it is for either HBV or HIV. In female partners of HIV / HCV co-infected men, 13% of women were found to have HIV compared to only 3% positive for HCV.[17]

Common risk factors in couples include shared exposures (IVDU), shared environmental causes or shared behaviors which may account for some proportion of HCV in this setting. Data indicate that the major risk factor for HCV positivity in monogamous heterosexual couples is IVDU. Distinguishing whether transmission has occurred from a sexual partner as opposed to transmission from other exposures requires comparison of HCV genotypes and viral homology. Since genotype 1 is responsible for 90% of HCV in USA, the majority of couples with HCV will be concordant genotypically. In one study in which nucleotide sequencing was used, 12 of 24 couples had the same genotype, but only 3 of 7 of those subsequently analyzed had "highly" homologous viral strains. The difficulty of conclusively documenting sexual transmission is illustrated by the fact that 11 of 12 of these couples infected with the same HCV genotype had at least one additional parenteral risk factor (IVDU) for non-sexual, viral transmission in both spouses.[18]

In one study of 401 monogamous heterosexual couples in whom IVDU in both partners was thought to be excluded, anti-HCV prevalence in both partners was 4.2% while 2.7% had the same HCV genotype.[19] Conflicting data exists to determine whether increasing duration of marriage, a presumed correlate of risk of sexual transmission, is associated with increased risk.[20, 21] Female partners of male hemophiliacs had a 2% prevalence of HCV RNA positivity in one small study. [22]

### What host factors increase the risk of sexual transmission?

The major factor correlating with infectiousness is circulating HCV RNA. Individuals who are anti-HCV positive without circulating HCV RNA (either via spontaneous viral clearance or post-antiviral therapy) have negligible risk of transmitting HCV. Contributing influences may include immune competency of the uninfected partner, presence of disease interfering with mucosal integrity facilitating transmission, sexual behavior

## Table 2. Guidelines to Minimize Sexual Transmission of HCV

**1. Who are likely to be contagious?**
- Only HCV-positive patients with continued viremia (measured by serum HCV RNA) are likely to be contagious.

**2. How common is sexual transmission of HCV?**
- Sexual transmission occurs, but is very uncommon, estimated to occur in approximately 6% of long-term partners of HCV-seropositive individuals.

**3. Recommendations for monogamous couples:**
- Many authorities counsel no change in sexual practices for monogamous couples, but condoms may be used to further lower risk.
- Avoid sharing razors, toothbrushes, nail clippers or other personal items potentially having blood on them.
- No restrictions are needed for less intimate contact (kissing, sweating, sharing cooking utensils)

**4. Additional recommendations for HCV-positive patients with short-term and multiple partners:**
- Condoms are recommended.

**5. Other indications for barrier protection (condoms):**
- The presence of sexually transmitted diseases (STDs) or active genital inflammation.
- Menses.
- Sexual practices that injure the mucosa.

traumatizing mucosal surfaces, potential effect of co-morbid genital infection (trichomonas, chlymadia, etc). Data suggest that male-to-female transmission is a more efficient means of transmission than female-to-male. In one study of patients attending an STD clinic, anti-HCV seropositivity was considerably greater for female contacts of men with anti-HCV than for male contacts of anti-HCV-positive women.[23] In men who have sex with men, HCV prevalence, when controlled for alternate routes of transmission, does not appear to be substantially increased.[23, 24] Large, prospective studies of male-male couples discordant for HCV seropostivity have not been done. No clear association with transmissibility exists for different HCV genotype, HCV RNA viral load, or stage of liver disease. (Table 1)

### Who is at high risk for sexually transmitted HCV?

Patients with increased risk of sexually-acquired HCV are those with multiple sexual partners. An important competing risk in this group is the potential higher incidence of IVDU. Commercial sex workers and patients at **sexually transmitted disease** (**STD**) clinics have been reported to have an increased incidence of HCV infection, but these groups may have an increased incidence of other competing high-risk behaviors (principally IVDU). Median rates of HCV seropositivity are 6% in commercial sex workers in the USA.[19, 25] In a representative study of non-IVDUs at an STD clinic, 7% of men and 4% of women were anti-HCV positive, compared to a prevalence of 1.4% in the general population in the USA.[23] Men who have sex with men have been reported to have a prevalence as high as 4.6% , but risk is strongly correlated with the small percentage who are IVDU, and to a lesser extent, the number of lifetime sexual partners, failure to use condoms, or sexual practices traumatizing mucosa.[24] Although HIV-positive patients have higher serum titers of HCV than HIV-negative individuals, the significance of this finding is unknown.[26] Other data do not document a correlation between level of HCV viremia and infectivity; thus, unknown host or immune factors may be a contributing risk in HIV/HCV co-infected persons. Insufficient data exist to conclude that sexual transmission of HCV is increased or facilitated in men who have sex with men or in the presence of HIV co-infection.

### HCV and other sexually transmitted disease

In a large Veterans Administration study of 34,204 HCV-positive patients compared to HCV-negative controls, HCV was associated with a higher probability for other blood-borne viruses, including HIV (14.1% vs. 3%) as well as other sexually transmitted diseases[27], risks for gonorrhea, syphilis, genital herpes, trichomonas, and viral warts were all increased. Pre-existing genital infection or mucosal inflammation may facilitate infectiousness. Presence of these diseases may identify groups with an increased risk, thereby justifying targeted HCV screening. Because of similar modes of transmission, serologic markers for HBV are increased in HCV-positive individuals, with as many as 67% having evidence of exposure to HBV compared to 3.5% in the general population.[28] Immune status to HBV should be assessed in all HCV-positive individuals and HBV vaccination recommended to all who are not immune.

### What recommendations should physicians give to long-term monogamous partners, one of whom is HCV RNA positive?

Only patients with the continued presence of HCV RNA in serum are likely to be infectious. Sexual transmission is possible, but is a rare event, occurring with a probability between 1 in 10,000 and 1 in 100 per year. Consensus opinion suggests an incidence of 12 per 1000 person-years in partners of HCV infected individuals. These figures translate to a cumulative risk of acquisition of approximately 5% over 20-30 years.[28] Some authorities counsel that the risk is low enough not to require a change in sexual practice for monogamous couples, including men who have sex with men. Condoms may be used to further lower risk. Others advise condom use for all persons at risk of transmission.[29] Barrier protection is, however, recommended for short-term, multiple sexual partners who are also at increased risk for other sexually transmitted diseases. Condoms are recommended in the presence of other sexually transmitted diseases, during menses, and with sexual practices that may injure the ano-genital

mucosa. No restrictions are needed for less intimate contact, including kissing or sharing food. HCV is not transmitted by coughing, sneezing, or sweating. Because HCV is present in blood, an HCV-positive individual should avoid sharing razors, toothbrushes, nail clippers, or other personal items potentially having blood on them. A summary of these recommendations is in Table 2.

All partners of HCV-positive patients should have medical evaluation. Couples must be informed that most individuals with HCV are asymptomatic and may not have liver enzyme elevations in serum. Some suggest routine HCV antibody testing to detect a partner's seroconversion because of the dramatic response of acute HCV infection to interferon.[30] No evidence supports the utility of routine post-sexual exposure prophylaxis with immune globulin or anti-viral agents.

## Conclusions

Concordance for HCV positivity in monogamous sexual relations does not prove sexual transmission as the route of transmission. Other high-risk behavior, especially injection drug use, but also sharing of items prone to be contaminated with blood or unknown common environmental exposures, may be explanations. Although sexual transmission of all these major blood-borne pathogens (HIV,HBV, HCV) has been described, it is much more efficient for HBV and HIV. Although sexual transmission of HCV occurs, it is uncommon and most practical recommendations take this lower risk into account in advising HCV-discordant couples.

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# Prevention of HIV Transmission After Health Care Worker Occupational Exposure

*Karen Tashima, MD, and Margo Weiss*

As of June 2001, occupational exposures to HIV have resulted in 57 documented and over 100 possible cases of HIV infection among healthcare personnel in the United States.[1,2] Although blood or body fluid exposure prevention practices are well established in the healthcare setting, exposures to HIV continue to take place. The **Centers for Disease Control and Prevention (CDC)** have published guidelines on the management of healthcare worker exposures to HIV, including recommendations for antiretroviral medication use to prevent HIV infection.[3]

Providing antiretroviral medications to **healthcare workers (HCWs)** is based on several lines of evidence. In 1993, **zidovudine (ZDV/AZT)** was shown to dramatically reduce mother-to-infant HIV transmission.[3] In 1995, a case control study of HCWs demonstrated that AZT taken after a needlestick exposure reduced the risk of HIV transmission by 79% .[4] Recent studies indicate that HIV infection of dendritic cells is important in establishing HIV infection and that antiretroviral agents block subsequent infection of T cells in *vitro*,[5] thus providing further rationale for PEP after needlestick exposures. Researchers using macaque models found that the timing and duration of antiretroviral agent administration was critical in preventing **simian immunodeficiency virus (SIV)** infection. The highest protection occurred when the macaques were given **postexposure prophylaxis (PEP)** within 24 hours of inoculation with SIV, and when PEP was continued for four weeks.[5]

Henderson advises that clinicians follow four basic principles in managing a possible healthcare worker exposure to HIV.[5] Clinicians or facilities evaluating such exposures should have the initial doses of PEP immediately available to the person exposed. The severity of the exposure should be assessed to determine whether a two or three-drug regimen will be necessary. Clinicians should select a regimen for the health care worker appropriate to the exposure risk. The first doses of medications should be administered within 1-2 hours after the exposure, and continued for 28 days. Finally, workers should be referred for follow-up for side effects and testing with a provider familiar with the CDC guidelines.

Side-effect management is important in assuring the completion of PEP. Medications may require adjustments in some cases. In one registry of HCWs who were prescribed PEP,[6] 54% of those discontinuing PEP or modifying their regimen did so because of side effects. The most frequent side effects were nausea (57%), fatigue or malaise (38%), headache (18%), vomiting (16%), diarrhea (14%) and myalgias or arthralgias (6%). The following are frequently asked questions:

**Table 1. Factors to consider in assessing the need for treatment and follow-up of occupational exposures**

- **Type of exposure**
  - Percutaneous injury
  - Mucous membrane exposure
  - Nonintact skin exposure
  - Bites resulting in blood exposure to either person involved
- **Type and amount of fluid/tissue**
  - Blood
  - Fluids containing blood
  - Potentially infectious fluid or tissue (semen; vaginal secretions; and cerebrospinal, synovial, pleural, peritoneal, pericardial, and amniotic fluids)
  - Direct contact with concentrated virus
- **Infectious status of source**
  - Presence of HBsAg
  - Presence of HCV antibody
  - Presence of HIV antibody
- **Susceptibility of exposed person**
  - Hepatitis B vaccine and vaccine response status
  - HBV, HCV, and HIV immune status

*Source:* Reference 3

**What should a HCW do after an exposure to the blood of a patient?**

Wash needlestick site and cuts with soap and water, flush splashes to the nose, mouth or skin with water, or irrigate the eyes with clean water, saline or sterile irrigants. The exposure should be promptly reported in order to start postexposure treatment as soon as possible.[7]

**What is the risk of infection after an occupational exposure?**

The estimated risk of HIV infection from a needlestick or laceration exposure to HIV- infected blood is 0.3% per contact. The risk is higher for injuries with a hollow bore needle, a needle placed in an artery or vein prior to an exposure, a deep injury, visible blood on the device, or if the source patient had AIDS.[3] The risk of infection from mucous membrane exposure is estimated to be 0.1%, and the risk of exposure to nonintact skin is less than 0.1%.

**Which body fluids other than blood are potentially infectious for HIV and which are not?**

Blood and body fluids containing blood, semen, and vaginal secretions are considered potentially infectious (see Table 1).[3] Other body fluids con-

**Table 1. Basic and Expanded Regimens of Postexposure Prophylaxis against Human Immunodeficiency Virus Infection.***

| Regimen | Doses | Primary Adverse Effects |
|---|---|---|
| **Basic** | | |
| Zidovudine (Retrovir) plus lamivudine (Epivir)† | 600 mg of zidovudine daily in two or three divided doses; 150 mg of lamivudine twice daily | Zidovudine: anemia, neutropenia, nausea, headache, insomnia, muscle pain, weakness; lamivudine: abdominal pain, nausea, diarrhea, rash, pancreatitis |
| Lamivudine plus stavudine (Zerit) | 150 mg of lamivudine twice daily, 40 mg of stavudine (if body weight is ≥60 kg, 30 mg) twice daily | Lamivudine: as above; stavudine: peripheral neuropathy, headache, diarrhea, nausea, insomnia, anorexia, pancreatitis, elevated liver function values, anemia, neutropenia |
| Didanosine, available as a chewable or dispersible buffered tablet (Videx) or as a delayed-release capsule (Videx EC), plus stavudine | 400 mg of didanosine daily taken on an empty stomach if a buffered tablet is used (if body weight is ≥60 kg, 125 mg twice daily if a buffered tablet is used), or 250 mg daily if a delayed-release capsule is used; 40 mg of stavudine twice daily | Didanosine: pancreatitis, lactic acidosis, neuropathy, diarrhea, abdominal pain, nausea, stavudine as above |
| **Expanded (basic regimen plus one of the following)** | | |
| Indinavir (Crixivan)‡ | 800 mg every 8 hr taken on an empty stomach | Nausea, abdominal pain, nephrolithiasis, indirect hyperbilirubinemia |
| Nelfinavir (Viracept) | 750 mg three times daily with a meal or snack, or 1250 mg twice daily with a meal or snack | Diarrhea, nausea, abdominal pain, weakness, rash |
| Efavirenz (Sustiva) | 600 mg daily, at bedtime | Rash (including Stevens–Johnson syndrome), insomnia, somnolence, dizziness, trouble concentrating, abnormal dreaming |
| Abacavir (Ziagen)§ | 300 mg twice daily | Nausea, diarrhea, anorexia, abdominal pain, fatigue, headache, insomnia, hypersensitivity reactions |

*Source:* Reference 8

* The information is adapted from the recommendations issued in 2001 by the Public Health Service.[3]
† A combined formulation is also available (Combivir); the recommended dose is one tablet twice a day.
‡ Abacavir is available as a combined formulation with zidovudine and lamivudine (Trizivir).

sidered potentially infectious are cerebrospinal fluid, synovial fluid, pleural fluid, peritoneal fluid, pericardial fluid and amniotic fluid. Feces, nasal secretions, saliva, sputum, sweat, tears, urine and vomitus are not considered infectious unless they contain blood. Human bites may expose both the person bitten and the person who has inflicted the bite to bloodborne pathogens. Even so, transmission of HBV or HIV infection has only rarely been reported from human bites.[2]

**Which antiretroviral medications for PEP are recommended?**

Two or three drug regimens are recommended based on the severity and type of exposure (Tables 2 and 3). Determining which agents to use or when to alter a PEP regimen is largely empiric. HIV infected patients are more effectively treated with combination therapy, therefore current guidelines recommend the use of at least two anti-HIV medications. AZT and lamivudine (3TC), available as the combination tablet Combivir, is a common medication used in PEP. However, other two nucleoside reverse transcriptase inhibitor combinations, such as stavudine (d4T) and 3TC or d4T and didanosine (ddI) may be used, especially when the exposure source's HIV strain is resistant to AZT or 3TC. A third drug for PEP, a protease inhibitor such as indinavir (Crixivan), nelfinavir (Viracept), or lopinavir/ritonavir (Kaletra), is suggested for more serious exposures. Efavirenz and abacavir are other medications, which can be used in a 3-drug regimen.[8] Nevirapine is not recommended for PEP because of concerns of serious side effects such as liver failure.[3]

**Which PEP medications should not be taken during pregnancy?**

Efavirenz is not considered to be safe for the fetus and should not be taken during pregnancy. In addition, the combination of ddI and d4T has been associated with fatal and nonfatal cases of lactic acidosis in HIV-infected pregnant women, so it should be used with caution with pregnant health care workers who require PEP.[3]

**What if the source patient's HIV status is unknown?**

The source of an occupational exposure should be evaluated for HBV, HCV, and HIV infection. If the source patient's infection status for these viruses is unknown, the source should be informed of the incident and tested for these viruses. Under most circumstances a negative HIV serology in the source patient means that it is safe for the HCW to stop taking PEP.

**What are the symptoms of acute HIV infection and how often should a HCW be tested?**

The symptoms of acute HIV infection usually appear within six weeks of exposure to HIV, and include fever, rash, sore throat, nausea, headache, and lymphadenopathy. Baseline and follow-up HIV antibody tests should be performed at six weeks, three months and six months after an occupational exposure.

Knowledge about the effectiveness of drugs used for PEP is limited. Combination drug regimens are recommended because of increased potency and concerns about drug resistant virus, but any or all drugs for PEP may be declined or stopped by the exposed person. Any exposed person should have six months of follow-up testing, be counseled about the symptoms of an acute HIV infection, and should learn how to prevent the transmission of HIV regardless of whether or not they take PEP. Specifically, the exposed person should practice "safe sex", avoid pregnancy and breastfeeding, and refrain from donating blood or other tissues.[7]

Most health care workers exposed to HIV can be reassured that they are unlikely to seroconvert. Although the risk is small, it important to be properly evaluated by a knowledgeable medical professional. Personnel should be well informed about the most updated PEP guidelines, which may be found in the Centers for Disease Control and Prevention's published guidelines.[3] Other resources include the 24 hour PEPline (National Clinicians'

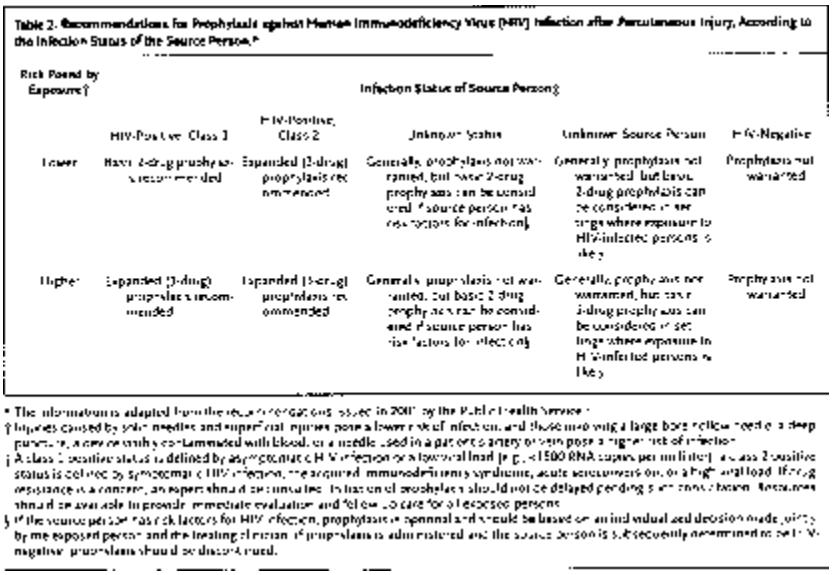

Table 2. Recommendations for Prophylaxis against Human Immunodeficiency Virus (HIV) Infection after Percutaneous Injury, According to the Infection Status of the Source Person.*

| Risk Posed by Exposure† | Infection Status of Source Person‡ | | | | |
|---|---|---|---|---|---|
| | HIV-Positive, Class 1 | HIV-Positive, Class 2 | Unknown Status | Unknown Source Person | HIV-Negative |
| Lower | Have 2-drug prophylaxis recommended | Expanded (3-drug) prophylaxis recommended | Generally, prophylaxis not warranted, but basic 2-drug prophylaxis can be considered if source person has risk factors for infection§ | Generally prophylaxis not warranted, but basic 2-drug prophylaxis can be considered in settings where exposure to HIV-infected persons is likely | Prophylaxis not warranted |
| Higher | Expanded (3-drug) prophylaxis recommended | Expanded (3-drug) prophylaxis recommended | Generally, prophylaxis not warranted, but basic 2-drug prophylaxis can be considered if source person has risk factors for infection§ | Generally, prophylaxis not warranted, but basic 2-drug prophylaxis can be considered in settings where exposure to HIV-infected persons is likely | Prophylaxis not warranted |

* The information is adapted from the recommendations issued in 2001 by the Public Health Service.

† Injuries caused by solid needles and superficial injuries pose a lower risk of infection, and those involving a large bore hollow needle, a deep puncture, a view visibly contaminated with blood, or a needle used in a patient's artery or vein pose a higher risk of infection.

‡ A class 1 positive status is defined by asymptomatic HIV infection or a low viral load (e.g., <1500 RNA copies per milliliter). A class 2 positive status is defined by symptomatic HIV infection, the acquired immunodeficiency syndrome, acute seroconversion, or a high viral load. If drug resistance is a concern, an expert should be consulted. Initiation of prophylaxis should not be delayed pending such consultation. Resources should be available to provide immediate evaluation and follow-up care for all exposed persons.

§ If the source person has risk factors for HIV infection, prophylaxis is optional and should be based on an individual decision made jointly by the exposed person and the treating clinician. If prophylaxis is administered and the source person is subsequently determined not to be HIV-negative, prophylaxis should be discontinued.

*Source:* Reference 8

Post-exposure Prophylaxis Hotline) at 1-888-HIV-4911, or by contacting local HIV providers.

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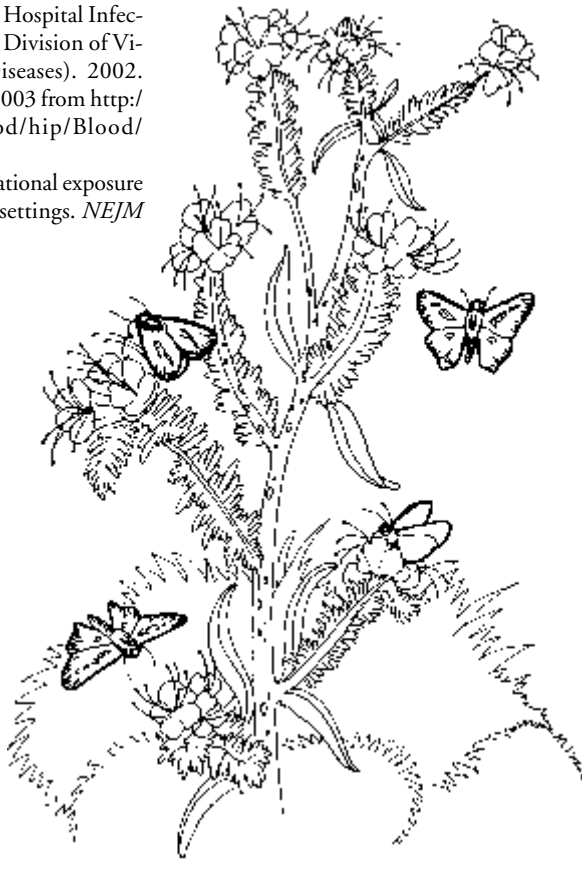

# Case Studies on Nonoccupational Postexposure HIV Prophylaxis

*Brenda Y. Urbina, MD, Roland C. Merchant, MD, Kenneth H. Mayer, MD*

**HIV postexposure prophylaxis (HIV PEP)** concerns the prompt administration of antiretroviral medications after a blood or body fluid exposure in order to prevent an HIV infection. Occupational HIV PEP has been used for work-related HIV exposures most commonly in health care settings but also in other settings where blood exposure occurs on the job (e.g. public safety, prisons, funeral homes). **Nonoccupational HIV PEP (HIV NPEP)** is given to anyone potentially exposed to HIV outside of the healthcare setting or job-related circumstances, such as following sexual exposures or environmental exposures to blood contaminated objects in the community. Occupational and nonoccupational HIV PEP are similar in regards to the type of medications employed, the time frame for using them, and circumstances of some exposures, e.g., needlestick injuries and blood splashes. The **Centers for Disease Control and Prevention (CDC)** recommends administering occupational HIV PEP after certain blood and body fluid exposures in the healthcare setting, and has promulgated guidelines on its provision.[1] These guidelines are largely based on a study of healthcare workers percutaneously exposed to HIV which found that occupational HIV PEP usage decreased the probability of HIV transmission.[2]

Outside the healthcare environment, most public health HIV prevention measures focus on interrupting transmission by encouraging condom use and safer injecting-drug practices. As a means to supplement these primary HIV prevention efforts, several groups have suggested that nonoccupational HIV PEP be employed as a secondary preventive method.[3,4] The need to do so is locally underscored by the number of HIV infection reported to the RI DOH, 122 new cases in 2000 and 147 more in 2001.[5] The United States currently does not have national guidelines for nonoccupational HIV PEP. To bridge this gap, the Brown University AIDS Program and Rhode Island Department of Health composed comprehensive nonoccupational HIV PEP guidelines for the state in 2002. [6]

The Rhode Island nonoccupational HIV PEP guidelines present a hierarchy of recommendations to provide guidance on when nonoccupational HIV PEP might be either recommended, offered, or considered following a possible HIV exposure. The guidelines state that nonoccupational HIV PEP should be **RECOMMENDED** after an exposure to a known HIV-infected source, may be **OFFERED** after a high-risk exposure, and may be **CONSIDERED** after low-risk exposures. (Table 1)

In this article we present vignettes of potential HIV exposures outside the healthcare setting based upon actual cases in Rhode Island with some modification to enhance their educational value and protect patient anonymity. The aim of this article is to broaden physician awareness of what represents an exposure to HIV infection outside the health care setting and to help guide physicians on appropriate use of HIV NPEP.

## CASE #1:

A 10-year-old male is brought to his pediatrician by his mother after he was stuck today by a needle on a syringe he found in a garbage dumpster. This occurred in a park where his mother states that people inject drugs. The needle was discarded and cannot be retrieved. The child has never had an HIV test.

This case illustrates the challenges of determining when to provide HIV NPEP after percutaneous exposures to unknown but possibly high-risk sources. From the information provided, it is clear that a percutaneous exposure did occur from a needle perhaps used to inject recreational drugs. In some communities the prevalence of HIV infection among injecting-drug users is substantial. Although HIV seroconversion overall after a needlestick injury either outside or within the healthcare setting is uncommon, the precise seroconversion risk for this child can not be accurately stated.

Ideally the pediatrician would attempt to quantify the risk by assessing the type of needle (hollow vs. solid bore, small gauge (e.g. diabetic) vs. larger gauge (e.g. drug injection), etc.), the severity of the needlestick (no percutaneous breach vs. superficial vs. deep injury into a blood vessel, etc.), the presence of visible blood on the needle and on the skin, and the volume of blood transferred. Since the needle is not available, the pediatrician could ask the child to describe it, and show him examples of different needles to see if he can identify it. The child could be asked if he recalls seeing blood in or on the needle. However, he may not be able to accurately recall these details. Even if he had retrieved the needle and brought it with him, the needle should not be tested for HIV, since this practice would be of unclear diagnostic utility and could subject laboratory workers to unnecessary risk.

Given that the child is being evaluated on the same day that the exposure occurred, he is within the 72-hour time frame currently suggested for receiving HIV NPEP. A two-drug HIV NPEP regimen would be offered, typically including zidovudine or stavudine with lamivudine. Since the child would be committed to a 28-day regimen, his mother's support for this treatment would be enlisted. HIV testing now, as well as one, three, and six months later, and the child should be referred for follow-up with a pediatric HIV specialist.

## CASE #2:

A 23-year-old female comes to the emergency department after being held prisoner and repeatedly sexually assaulted by her ex-boyfriend during the prior two days. She says she endured several episodes of penile-vaginal intercourse with definite transfer of seminal fluid. Although she is not certain, as far as she knows her ex-boyfriend is not HIV infected. She is sure, because of a recent routine HIV test, that she is not infected. She has no HIV infection risk factors.

This patient was subjected to mul-

TABLE 1

Based upon exposure and source's HIV status
Determine if HIV NPEP* should be recommended, offered, or considered and select an appropriate HIV NPEP regimen

| Exposure | Course of Action |
|---|---|
| Exposure to KNOWN HIV-infected source(s) | **RECOMMEND HIV NPEP**<br>A. Source's medication history is UNKNOWN or the source is known NOT to be on anti-HIV medications<br>zidovudine (AZT) 300 mg or stavudine (d4T) + lamivudine (3TC) (generic) AND nelfinavir (viracept) OR indinavir (crixivan)<br>B. Source's medication history is KNOWN.<br>Under the advisement of a specialist knowledgeable in HIV NPEP and HIV medications, choose a medication regimen that takes into account the source's medication history and drug resistance. If the adverse side effect profile is not available, a regimen that is different from the medications the source currently uses can be used for the initial dose. Choose two nucleoside reverse transcriptase inhibitors, such as:<br>• zidovudine or stavudine + lamivudine AND nelfinavir, indinavir, saquinavir, ritonavir OR lopinavir/ritonavir<br>• stavudine + didanosine AND nelfinavir, indinavir, saquinavir, ritonavir OR lopinavir/ritonavir<br>• didanosine + zalcitabine AND nelfinavir, indinavir, saquinavir, ritonavir OR lopinavir/ritonavir |
| Exposures to UNKNOWN HIV status sources at HIGHER risk of HIV infection | **OFFER HIV NPEP**<br>zidovudine or stavudine + lamivudine<br>A protease inhibitor can be added if the source has multiple high risk factors for HIV infection |
| Exposures to UNKNOWN HIV status sources at LOWER risk of HIV infection | **CONSIDER HIV NPEP**<br>HIV NPEP may be considered on a case-by-case basis. Zidovudine or stavudine + lamivudine may be offered when compelling circumstances exist. |

*NPEP Nonoccupational Postexposure Prophylaxis

tiple high-risk sexual encounters by an assailant whose HIV status is unknown. Studies on consensual sex have estimated that the risk for infection is at least 0.1-0.2% per episode of penile-vaginal intercourse[7]. However, the risk of transmission may be greater from a sexual assault because of the associated trauma and higher likelihood of sexually transmitted diseases in sexual assault survivors and their assailants[8].

The first step would be to attempt to learn more about the HIV risk factors of the assailant. If he has multiple risk factors for HIV infection (e.g., injecting-drug use, male sexual partners, multiple sexual partners, etc.) a three-drug HIV PEP regimen would be offered, usually including zidovudine or stavudine with nelfinavir or indinavir. If she does not know her ex-boyfriend's risk for HIV infection or knows that he is likely not at high risk for an infection, then a two-drug regimen without a protease inhibitor would be offered. Pregnancy testing is recommended. If she is pregnant, medications would still be offered, but accompanied by a discussion of the unknown effects of HIV PEP on her fetus in order to allow her to understand the risks and benefits of HIV PEP in these circumstances. Per CDC recommendations, prophylactic medications against sexually transmitted diseases must be prescribed and emergency contraception should be offered[9]. Other services include involving a sexual assault advocate for her care in the emergency department and for her follow-up, and recommending follow-up with a physician or gynecologist familiar with managing HIV PEP. While the treating physician must always advocate that the assailant undergo HIV testing, obtaining such testing should not delay initiation of the patient's HIV PEP. If the assailant tests negative for HIV, then the patient can be recommended to stop HIV PEP.

## CASE #3:

A 24-year-old male inmate comes to the medical clinic after having consensual, unprotected anal- receptive intercourse with a fellow male inmate last night. His sexual partner is HIV infected. The patient was tested for HIV upon intake to the prison a week earlier and is not infected.

Since the sexual partner who is the exposure source for this patient is known to be HIV infected, the patient is recommended to take HIV NPEP. The current and prior HIV medication history of the source must be obtained, as well as his HIV medication resistance profile. Once this information is gathered, an HIV specialist experienced in prescribing HIV PEP should be consulted and, in conjunction with the patient, an appropriate HIV PEP regimen can be chosen. If either this information or the specialist is unavailable then a three-drug HIV PEP regimen should be promptly initiated, generally with zidovudine or stavudine with lamivudine and nelfinavir or indinavir. If only the source's current HIV medications are known, then the HIV PEP regimen prescribed must include medications that are different from the source's current regimen. The regimen can be adjusted once the source's medication history becomes available. This opportunity can be used as a "teachable moment" to encourage the patient to engage in safer sexual practices, and refer him to further counseling as needed to support his need for improved HIV prevention.

## KEY NOTES FROM THE GUIDELINES:

- Many blood or body fluid exposures need an evaluation for HIV PEP, but most patients do not need HIV PEP
- HIV NPEP is NOT indicated for exposures involving feces, sputum, saliva, urine or vomitus
- HIV NPEP may be ineffective if initiated ≥ 72 hours after initial HIV exposure
- Prophylaxis should be given for 28 days
- Baseline laboratories should include: serologic testing for HIV, and Hepatitis B and C; complete blood count; pregnancy testing; liver enzymes if a protease inhibitor is used
- Test the source whenever possible, but do not delay HIV PEP initiation while awaiting testing

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# Judicial Diagnosis

## Blood-borne Pathogens and HIPAA

*Michael G. Tauber, JD*

*What security measures does HIPAA require that I implement to protect the confidentiality of my medical records pertaining to patients infected with blood borne pathogens such as hepatitis and HIV?*

The HIPAA Privacy Rule does not impose any special requirements for the security of medical records relating to blood-borne pathogens. The same rules apply to these records as do to all other **protected health information** (**PHI**).

Specifically, the Privacy Rule requires that physicians have in place appropriate administrative, physical, and technical safeguards to protect the privacy of their patients' PHI.

Administrative safeguards include policies and procedures designed to restrict access to, and inadvertent or improper disclosure of PHI (for example, requiring that patient records be returned to the file cabinet rather than left out overnight where the cleaning crew might see them). Physical safeguards include such things as locks on file cabinets and record room doors. Technical safeguards include such things as password protection on computer software containing billing or clinical information.

It is important to note that the Privacy Rule does *not* require that you turn your office into Fort Knox. It requires only that *reasonable* safeguards be implemented. What is reasonable in any particular case will depend on the facts and circumstances involved. What is appropriate for a multi-site practice with many physicians and a large office staff may be unnecessary in a small, two-physician office.

In determining what is reasonable, several factors should be considered. Certainly, what other, like-sized, practices are doing is one measure of reasonableness, but it is neither exclusive nor definitive. Some other factors that should be considered in determining how extensive your safeguards must be include cost (both in dollars and staff resources), benefit gained in improving privacy protections, and how widespread or frequent the situation at issue is (i.e., safeguarding against an infrequent occurrence or unlikely event is not as important as protecting against frequent or likely incursions on the privacy of the PHI). For PHI concerning blood borne pathogens, physicians must also consider the particularly sensitive nature of the information. Although the Privacy Rule does not create special protections for PHI containing information about blood borne pathogens, given the potential adverse affects to the patient of an improper or inadvertent disclosure of PHI concerning blood borne pathogens (such as HIV), it would be reasonable to pay special attention to the safeguards in place to protect the privacy of such information.

The Privacy Rule also requires that physicians make reasonable efforts to ensure that only the minimum necessary amount of PHI (including PHI pertaining to blood borne pathogens) is used or disclosed.[1] Although not explicitly a security rule, the minimum necessary requirement affects the security efforts required of physicians. In particular, to meet the requirements of the minimum necessary rule, physicians should establish and enforce policies and procedures that outline who may have access to patients' PHI (including information relating to blood borne pathogens), how much of the PHI may be accessed, and for what purposes.

In addition to the security require-

ments imposed by the HIPAA Privacy Rule (with which compliance was required on April 14, 2003), the new HIPAA Security Rule (which is a *different* set of regulations from the Privacy Rule) will also impose a variety of obligations on physicians. The good news, however, is that there are another two years before the April 20, 2005 compliance date for the Security Rule. Furthermore, the Security Rule only pertains to *electronic* PHI.

You should also be aware that several provisions of state law impose obligations regarding the security of medical records containing information about blood borne pathogens. In particular, R.I.G.L. § 23-6-18 provides that physicians who maintain records containing information on HIV test results are responsible for maintaining full confidentiality of these data, and must take appropriate steps for their protection, including: (1) keeping records secure at all times and establishing adequate confidentiality safeguards for any records electronically stored; (2) establishing and enforcing reasonable rules limiting access to these records; and (3) training persons who handle records in security objectives and technique.

In addition, the Rhode Island Confidentiality of Health Care Communications and Information Act ("Confidentiality Act") requires that *third parties* receiving and retaining a patient's confidential health care information (including information pertaining to blood borne pathogens) establish at least the following security procedures:

(1) Limit authorized access to personally identifiable confidential health care information to persons having a "need to know" that information (other employees or agents may have access to information which does not contain sufficient data to allow the patient to be identified);
(2) Identify an individual or individuals with responsibility for maintaining security procedures for confidential health care information;
(3) Provide a written statement to each employee or agent as to the necessity of maintaining the security and confidentiality of confidential health care information, and of the penalties provided for in the Confidentiality Act for the unauthorized release, use, or disclosure of this information. The receipt of that statement must be acknowledged by the employee or agent, who is to sign and return the statement to his or her employer or principal, who must retain the signed original. The employee or agent must be given a copy of the signed statement; and
(4) Take no disciplinary or punitive action against any employee or agent solely for bringing evidence of violation of the Confidentiality Act to the attention of any person.

Finally, basic negligence principles generally require that a physician act prudently to safeguard medical records.

*What effect does the HIPAA Privacy Rule have on my duty to report infectious, communicable, or occupational diseases to the Department of Health?*

The Privacy Rule explicitly authorizes physicians to make reports concerning infectious, communicable, and occupational diseases without obtaining the patient's consent or authorization. Specifically, 45 C.F.R. § 164.512 (b)(1)(i) provides that, for purposes of public health activities such as public health surveillance and disease control and prevention, a physician may disclose protected health information (including information pertaining to infectious, communicable, and occupational diseases) without authorization or consent to:

"A public health authority that is authorized by law to collect or receive such information for the purpose of preventing or controlling disease, injury, or disability, including, but not limited to, the reporting of disease, injury, vital events such as birth or death, and the conduct of public health surveillance, public health investigations, and public health interventions; or, at the direction of a public health authority, to an official of a foreign government agency that is acting in collaboration with a public health authority."

In Rhode Island, the Department of Health is the public health authority empowered to receive such information. Accordingly, physicians may disclose PHI relating to infectious, communicable, and occupational diseases to the Department of Health without patients' permission.

You should note, however, that if a patient exercises his/her right under the Privacy Rule to ask you for an accounting of disclosures you have made of his/her PHI, you must include in the accounting disclosures of PHI contained in reports of communicable, infectious, or occupational diseases made to the Department of Health. These disclosures of PHI must be included in the accounting even though the reports were required by law.[2]

1. The minimum necessary rule does not apply in a variety of circumstances, including disclosures of PHI to other healthcare providers for treatment purposes.
2. This article is not intended to be legal advice. It presents only a general overview of the subject matter and is not an exhaustive discussion of the topic. Readers are urged to consult an attorney for advice on their particular circumstances.

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# CME Background Information

This CME activity is sponsored by Brown Medical School.

**Target Audience:** This enduring material is designed for physicians licensed in Rhode Island.

**CME Objectives:** At the conclusion of this course, participants should be able to:
* list prevention strategies for HIV transmission after occupational exposure
* know the facts about occupational exposure hepatitis C
* list practical recommendations for sexual transmission of hepatitis C
* know new options in diagnosis and treatment of hepatitis B
* discuss nonoccupational postexposure to HIV prophylaxis
* describe the impact of HIPAA on blood-borne pathogens

**Needs Assessment:** Physicians must have CME credits in blood-borne pathogens for licensure in Rhode Island.

Accreditation Statement: Brown Medical School is accredited by the Accreditation Council for Continuing Medical Education (ACCME) to provide continuing medical education for physicians.

**Credit Designation:** Brown Medical School designates this activity for a maximum of 2 category 1 credits towards the AMA Physician's Recognition Award. It also fulfills the Rhode Island requirement for 2 credits of CME in Blood Borne Pathogens/Universal Precautions.

**Date of Original Release.** This issue was published June 2003. This activity is eligible for CME credit through May 2004.

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**Keep a Copy for Your Files.** Keep a copy of the answers for your files. Compare them with the correct answers, which will be made available upon request, and receipt of submission requirements.

## CME Registration Form

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Please evaluate the effectiveness of the CME activity on a scale of 1 to 5 (1 being poor; 5 being excellent) by circling your choice.

| | |
|---|---|
| 1. Overall quality of this CME activity | 1 2 3 4 5 |
| 2. Content | 1 2 3 4 5 |
| 3. Format | 1 2 3 4 5 |
| 4. Faculty | 1 2 3 4 5 |
| 5. Achievement of educational objectives | |
| * List prevention strategies for HIV transmission after occupational exposure | 1 2 3 4 5 |
| * Know the facts about occupational exposure hepatitis C | 1 2 3 4 5 |
| * List practical recommendations for sexual transmission of hepatitis C | 1 2 3 4 5 |
| * Discuss new options in diagnosis and treatment of hepatitis B | 1 2 3 4 5 |
| * Describe the impact of HIPAA on blood-borne pathogens | 1 2 3 4 5 |
| * Discuss nonoccupational postexposure to HIV prophylaxis | 1 2 3 4 5 |

Please comment on the impact that this CME activity might have on your practice of medicine.

_____

Additional comments and/or suggested topics for future CME activities.

_____

**Blood-Borne Pathogens CME Questions:** *Circle One Response for Each Question.*

1. Blood and body fluids containing visible blood are the only body fluids that can potentially transmit HBV, HCV, and HIV.
   a. True
   b. False

2. What antiretroviral should not be prescribed to a pregnant woman?
   a. Efavirenz
   b. AZT
   c. 3TC

3. The risk of transmission of hepatitis C virus after a needle-stick injury is approximately:
   a. 0.3%
   b. 3.0%
   c. 30 %

4. Acute hepatitis C infection is most commonly :
   a. mild or asymptomatic
   b. characterized by jaundice,anorexia,and fever
   c. associated with hepatic failure

5. Sexual transmission of hepatitis C is:
   a. more common in HCV/HIV co-infected patients than sexual transmission of HIV
   b. very common in men who have sex with men
   c. uncommon in monogamous couples

6. The most common cause of hepatitis C infection in the USA is
   a. transfusion of blood products
   b. intra-venous drug use
   c. sexual transmission
   d. occupational exposure

7. Currently accepted management for needle-stick injuries from a hepatitis C positive patient include :
   a. gamma globulin injection
   b. anti-viral therapy
   c. testing the exposed individual for hepatitis C antibodies

8. Currently approved treatment options for chronic hepatitis B infection include:
   a. Ganciclovir, acyclovir and adefovir
   b. Adefovir, lamivudine and interferon-alpha
   c. Acyclovir, ribavirin and interferon-alpha
   d. Lamivudine, ritonavir and ganciclovir

9. Following needlestick exposure to a patient with chronic hepatitis B infection, the following should be performed on a HCW vaccinated 20 years ago with unknown initial antibody response:
   a. No further evaluation or treatment needed
   b. HBIG + booster vaccination immediately
   c. Check HbsAb and, if <10 mIU/ml, administer HBIG and booster vaccination
   d. Lamivudine 100mg per day for 30 days

10. Regarding hepatitis B vaccination, the following are true:
   a. All hemodialysis patients should be vaccinated and seroconversion confirmed
   b. All students should receive a booster vaccination at the time of college matriculation
   c. HCWs should be revaccinated every 5-6 years
   d. Vaccination should be offered to intravenous drug users, inmates and sexual partners of known hepatitis B carriers
   e. a, b, and c
   f. a, c and d
   g. All of the above

11. A 22-year-old woman presents to the emergency department five days following a sexual assault involving unprotected vaginal intercourse by her ex-husband. She does not know his HIV status, but believes he had a negative HIV test six months ago for a routine physical exam. Which statement most accurately reflects the need of HIV PEP in this situation?
   a. HIV PEP is recommended for this exposure.
   b. HIV PEP should be offered for this exposure.
   c. HIV PEP should be considered for this exposure.
   d. HIV PEP is probably not indicated for this exposure.

12. A 34-year-old woman visits her gynecologist after having consensual sex with her injecting-drug using boyfriend. She requests the "morning-after pill" to prevent pregnancy. Her pregnancy test is positive. Which of the following statements regarding HIV PEP is correct?
   a. Simultaneous provision of emergency contraception and HIV PEP is contraindicated.
   b. Since she is pregnant, she may not receive HIV PEP.
   c. She may be prescribed HIV PEP.
   d. Her boyfriend must be tested for HIV prior to her receiving any medications.

13. Which answer is accurate:
   a. The HIPAA Security Rule pertains only to electronic PHI.
   b. The HIPAA Security Rule pertains only to paper PHI.
   d. The HIPAA Security Rule pertains to both electronic and paper PHI.
   e. The HIPAA Security Rule makes no distinctions between the mode of PHI.

14. The Privacy Rule explicitly authorizes physicians to make reports concerning infectious, communicable, and occupational diseases without obtaining the patient's consent or authorization.
   a. True
   b. False

# IMAGES IN MEDICINE

*Edited by John Pezzullo, MD*

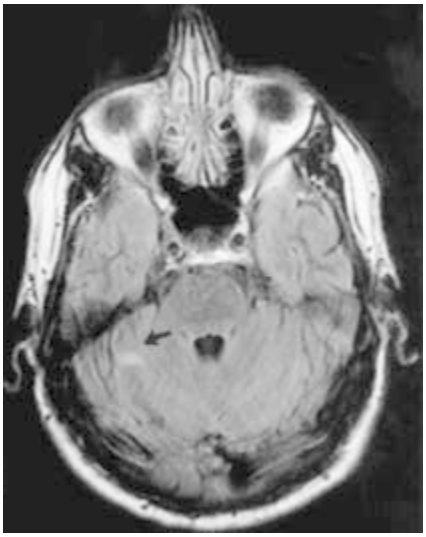

Figure 1a.

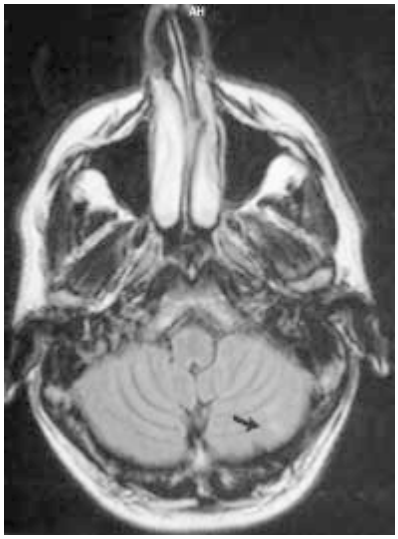

Figure 1b.

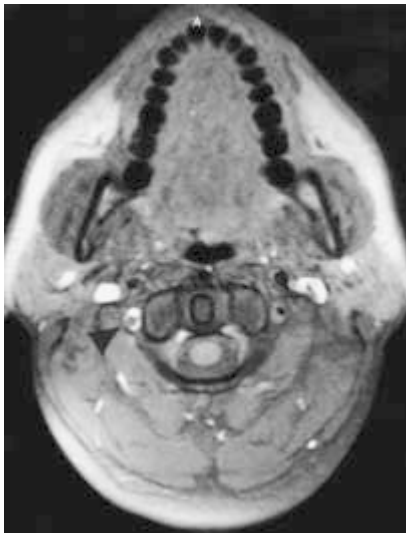

Figure 2.

## *Extracranial Vertebral Artery Dissection*

A 43-year-old man presented to the Emergency Department with acute diplopia, perioral paresthesias, vertigo, and tenderness at the left suboccipital region that began minutes after an errant golf swing at the first tee. Neurological examination revealed conjugate vertical and rotatory nystagmus in primary gaze, bilateral appendicular dysmetria, and a wide-based gait with leftward lateropulsion. Head **computed tomography** (**CT**) was normal. The patient was given intravenous heparin. Brain MRI revealed small acute bilateral cerebellar infarcts, best seen with **fluid attenuation inversion recovery sequence** (**FLAIR**) (arrows, Figures 1a, 1b). T1-weighted fat-suppressed axial images through the neck (arrowhead, Figure 2) demonstrated abnormally increased signal within the wall of the right vertebral artery at the C1 level, diagnostic of vertebral artery dissection with accompanying intramural hematoma. The patient was converted to warfarin therapy and later discharged from the hospital with only residual mild dysmetria.

Dissection of the extracranial carotid and vertebral arteries accounts for >20% of stroke in the young. The vertebral artery is maximally mobile at C1-C2 and is thus susceptible to mechanical injury secondary to cervical rotational forces. As in this case, the signs and symptoms localize most commonly to the medulla or cerebellum. Although some cases are still investigated angiographically, noninvasive T1-weighted fat-suppressed **axial magnetic resonance images** (**MRI**)s through the neck ("dissection protocol") may be more sensitive, demonstrating a pathognomonic bright crescent-shaped intramural hematoma. This protocol has become the test of choice at Rhode Island Hospital for this entity. The increasing use of this MRI technique has uncovered a surprisingly high incidence of cervicocerebral dissection syndromes.

**– LISA A. SHULTZ, MD, AND ANDREW S. BLUM, MD, PHD**

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# Healthcare quality matters to business

By Paul Choquette

*A healthy workforce* is an absolute necessity for any company to stay competitive. As chairman of one of Rhode Island s largest companies, I take great interest in quality-of-care issues.

Health and productivity are interrelated; employees who are out sick, at work but not feeling well, or distracted by the health concerns of a family member are probably not performing at 100 percent.

Also, it is difficult to expect above-and-beyond work performance from people without demonstrating an equal commitment on the company s part to employee well-being. At Gilbane, we pay 90 percent of employee health premiums and contribute to certain other health-related costs, such as gym memberships and Weight Watchers.

Most compelling of all, is the fact that at the end of the day, private industry pays a large portion of the nation s healthcare costs through insurance premiums. We have an interest and an obligation to ensure the dollars we invest in employee health benefits are spent to deliver the right care, at the right time and place, for the right reasons.

For all of these reasons, my company and many others in the Rhode Island business community strongly support the efforts of Quality Partners to increase public reporting of quality data and improve the delivery of care. By helping people take better care of themselves and their families, and by helping providers continuously improve their approach to the delivery of care, quality advocates can make a substantial difference in the health of our workforce.

You often hear executives say, "Our biggest strength is our people." That, in a nutshell, is why healthcare quality matters to business.

My company and many others strongly support the efforts of Quality Partners to make a substantial difference in the health of our workforce.

*Paul Choquette is Chairman and Chief Executive of Gilbane Building Company.*

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Rhode Island Department of Health
Patricia A. Nolan, MD, MPH, Director of Health

*Edited by Jay S. Buechner, PhD*

# Deaths and Hospitalizations Related to Atrial Fibrillation, 1999–2001

*Jay S. Buechner, PhD*

**Atrial fibrillation** (AF) is a common cardiac disorder involving sustained heart rhythm disturbance and affecting an estimated 2.2 million Americans, primarily those ages 65 and older.[1] It is a major risk factor for stroke, the third leading cause of death in the nation and in Rhode Island. Since 1980, it has been recorded with increasing frequency as a contributing cause of death in national data.[2] In this report, statewide death and hospitalization data have been analyzed to describe the burden of AF among Rhode Island residents.

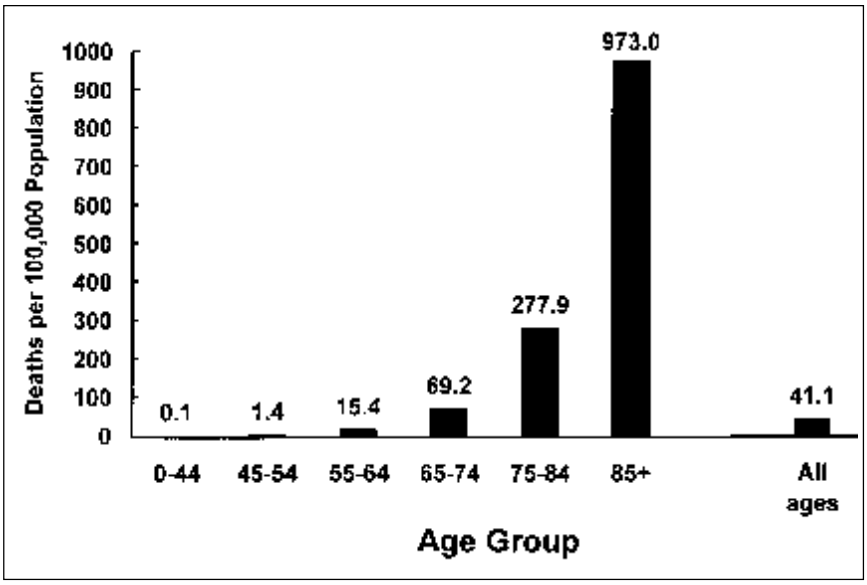


Figure 1. Deaths per 100,000 population (annual average) related to atrial fibrillation, by age group, Rhode Island, 1999-2001.

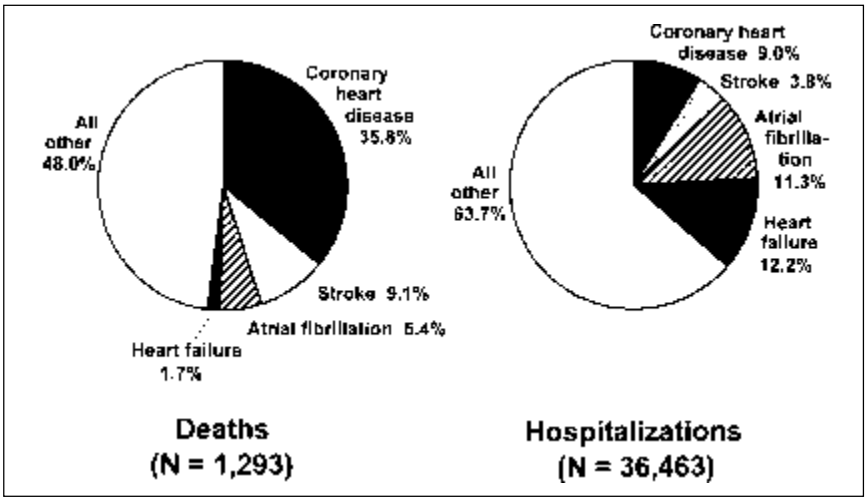


Figure 2. Underlying cause of death for deaths related to atrial fibrillation and principal diagnosis for hospitalizations related to atrial fibrillation, Rhode Island, 1999-2001.

## Methods

Multiple-cause mortality data from the state Office of Vital Records were analyzed for Rhode Island residents for the three-year period 1999-2001. (Data for 2001 are provisional and subject to change.) Deaths related to AF were defined as those with either an underlying or a contributing cause of death code in the range of I40 – I48 in the International Classification of Diseases, 10th revision (ICD-10). For all deaths, underlying cause was grouped as follows, based on ICD-10 codes: Atrial fibrillation (I40-I48), coronary heart disease (I20-I25), stroke (I60-I69), heart failure (I50), and all other causes.

Hospital discharge data from all acute care hospitals in Rhode Island were analyzed for the three-year period 1999-2001. Hospitalizations related to AF were defined as those with any diagnosis code, principal or additional, of 427.3 in the International Classification of Diseases, 9th revision, Clinical Modification (ICD-9-CM). For all discharges, principal diagnosis was grouped as follows, based on ICD-9-CM codes: Atrial fibrillation (427.3), coronary heart disease (410-414, 429.2), stroke (430-434, 436-438), heart failure (428), and all other diagnoses.

Annual average rates of death and hospitalization, including age-

specific, crude, and age-standardized, were calculated for 1999-2001 using 2000 Census data for Rhode Island. Age standardization employed the 2000 US standard population.[3] Age-standardized hospitalization rates were also computed for the population ages 65 and older for comparison to national rates. All national data were extracted from published sources.[1]

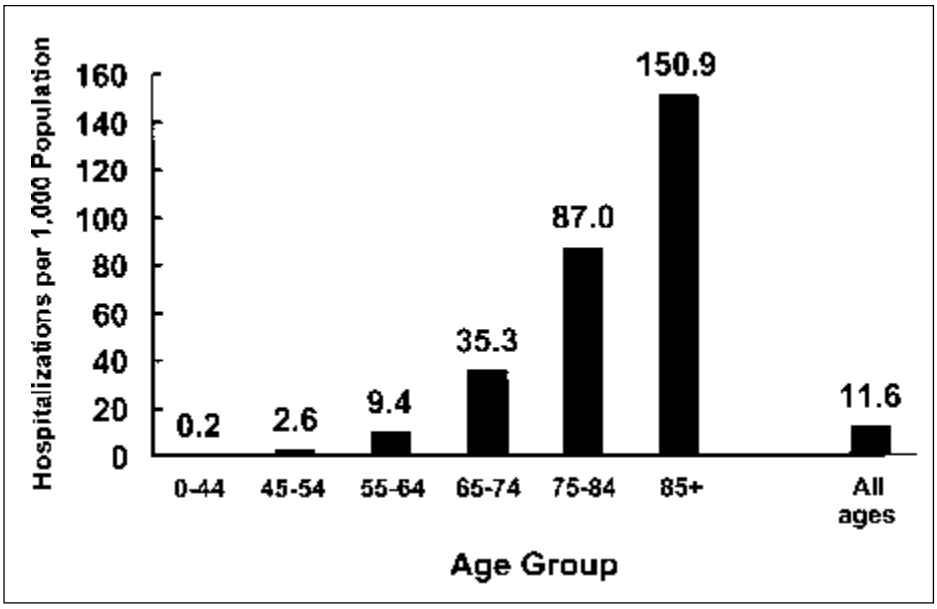


Figure 3. Hospitalizations per 1,000 population (annual average) related to atrial fibrillation, by age group, Rhode Island, 1999-2001.

## Results

Over the period 1999-2001, there were 1,293 deaths related to AF in Rhode Island, representing 4.4% of all deaths. There were 412 AF-related deaths (31.9%) in 1999, 424 (32.8%) in 2000, and 457 (35.3%) in 2001. Of those deaths, 70 (5.4%) had AF as the underlying cause; the other 1,223 (94.6%) had AF as a contributing cause. The crude average annual rate of AF-related deaths was 41.1 deaths per 100,000 population, and the age-standardized rate was 33.7 per 100,000.

Most AF-related deaths were among the elderly; 96.3% were ages 65 and older, 84.5% were ages 75 and older. Age-specific death rates rose sharply and monotonically with age, from near zero for those under age 45 years to 973.0 per 100,000 among those ages 85 and older. (Figure 1) The majority (58.9%) of AF-related deaths occurred among women.

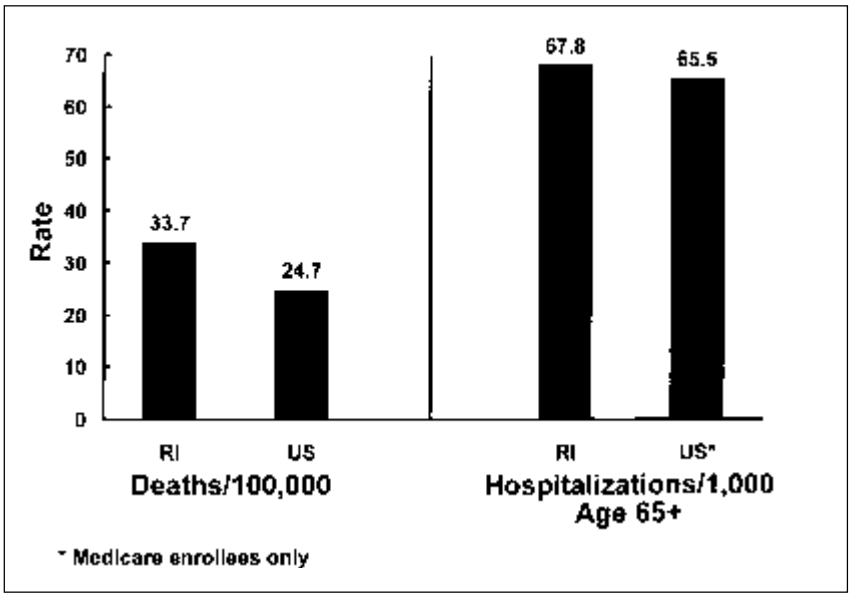


Figure 4. Rates of death and hospitalizations related to atrial fibrillation, Rhode Island (1999-2001 annual average) and United States (1999).

The most common underlying causes of death for AF-related deaths were the **cardiovascular diseases (CVD)**. Other than AF itself (5.4%), other CVD causes included coronary heart disease (35.8% of AF-related deaths), stroke (9.1%), and heart failure (1.7%). (Figure 2)

Over the same period, there were 36,463 hospital inpatient discharges related to AF in Rhode Island hospitals, representing 10.6% of all hospitalizations and over 28 hospitalizations per death. There were 11,999 AF-related hospitalizations (32.9%) in 1999, 12,206 (33.5%) in 2000, and 12,258 (33.6%) in 2001. Of those hospitalizations, 4,114 (11.3%) had AF as the principal diagnosis; the other 32,349 (88.7%) had AF as an additional diagnosis. The crude average annual rate of AF-related hospitalizations was 11.6 hospitalizations per 1,000 population, and the age-standardized rate was 9.9 per 1,000.

As for AF-related deaths, most hospitalizations were among the elderly; 88.7% were ages 65 and older, 67.3% were ages 75 and older. Age-specific hospitalization rates rose monotonically but less sharply with age, from near zero for those under age 45 years to 150.9 per 1,000 among those ages 85 and older. (Figure 3) The slight majority (53.8%) of AF-related hospitalizations were women.

The most common principal diagnoses for AF-related hospitalizations, other than AF itself (11.3%), included heart failure (12.2%), coronary heart disease (9.0%), and stroke (3.8%). (Figure 2)

## Discussion

AF imposes a substantial health burden on Rhode Island's population in terms of deaths and hospitalizations, especially among the elderly. For those ages 85 and older, nearly one percent of the population will die and over 15% will be hospitalized due to AF-related causes every year. AF-related deaths occur substantially more frequently and AF-related hospitalizations occur slightly more frequently here than in the nation generally. (Table 4) These findings are subject to the caveat that Rhode Island's higher rates may be due in part to variations in reporting and coding practices. Nevertheless, AF is a treatable condition and, if identified early, an avoidable risk factor for stroke. Public health prevention efforts involving public and medical education can increase awareness of AF in support of early diagnosis and treatment of this condition.

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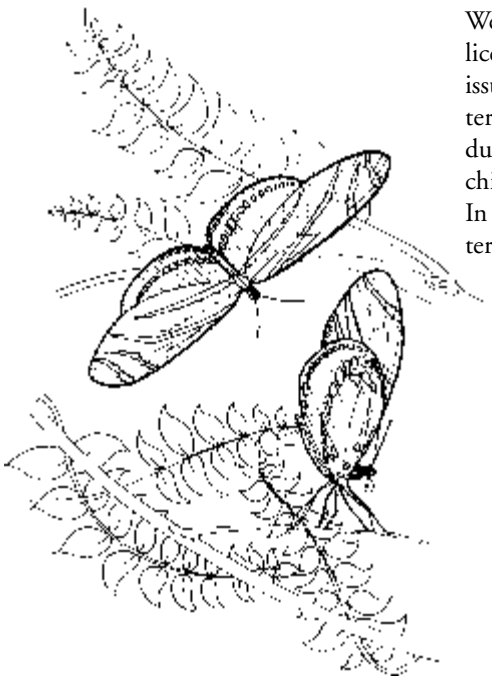
# *Letter to the Editor*

**To the Editor:**

Congratulations to the authors and editor of your CME issue, "The Smallest Patient" (May 2001). My original copy was "borrowed," a sure sign of a great issue, and I thank you for the replacement.

The succinct, well-written article "Rhesus Isoimmunization" prompts a few notes of local historic background.

The basis of the diagnosis, treatment and prevention of hemolytic disease of the newborn comes form the work of Karl Landsteiner (Nobel Prize, 1930) on blood typing and Rh isoimmunization. His son, Ernest Landsteiner, was the great and jovial Chief of Urologic Surgery at Rhode Island Hospital.



The local (Providence Lying In Hospital) adoption of the Liley intrauterine transfusion technique (1963) was due to Clinton B. Potter, MD, who organized and led the hospital transfusion team. I knew he was truly team chief because it was he who drove the car with Dr. William MacDonald, the other obstetrician, the pediatrician Dr. Frank Guinta, the indispensable but modest radiologist Dr. Dick Frates to Boston Lying In Hospital to "see one." We then "did them." The American licensure for Rh immune globulin was issued in 1968 and the need for intrauterine transfusions declined. I think during the time Karlis Adamson was chief of Ob/Gyn at Providence Lying In (1971-1979) there were no intrauterine transfusions.

However, using Adamson's technique of amniotomy and fetal limb extraction, a Rhode Island Hospital team of surgeons, Dr. Frank DeLuca and Dr. Conrad Wesselhoeft, anesthesia-James Robbins, DVM, Dr. Robert Corwin—pediatric cardiologist, and Dr. Richard Frates—radiologist, measured ph. $PO_2$, $CO_2$ in the fetal heart and pulmonary artery. Pulmonary artery flow before and just after first breaths was recorded. With the exception of Bill Wesselhoeft, all of the involved doctors are either dead or retired (there is a difference).

Again, congratulations on "The Smallest Patient."

– Very Truly Yours,
Richard E. Frates, MD
Women & Infants' Hospital,
Rhode Island Hospital, Retired

# – A Physician's Lexicon –

## The Cranial Nerves

Connected nonsense is remembered more readily than unconnected reality. And thus was born the poetic mnemonic, that contrived learning mechanism which employs some nonsensical doggerel to help the learner in recalling some sequence of words. Most physicians will remember how they learned the names, and sequence, of the mammalian cranial nerves long after they've forgotten their precise neural function. The words varied from medical school to medical school, some phraseology being earthier than others, but the mnemonic serves its purpose well since the first letter of each word in the poem was also the first letter of each nerve:

"On old Olympus' towering tops,
A Finn and German viewed some hops."

The first cranial nerve, **Olfactory**, is derived from two Latin words: *olere* [to smell] and *facere* [to make]. The second cranial nerve, **Optic**, comes from the Greek, *optos*, meaning the eye. The Greek root, *op-*, crops up in a number of commonly used words such as Ethiopian, describing those with fiery, burning eyes. It occasionally is written as oph-, as in the word ophthalmic. And **Oculomotor**, the third cranial nerve, is from the Latin, *oculus*, meaning the eye [and not to be confused with the Latin, *occulus*, meaning concealed or mysterious, as in the word occult]. The Latin, *oculus*, crops up in the word inoculate, originally meaning to engraft the eye or bud of a shrub or tree to another.

The fourth cranial nerve, **Trochlear**, is from the Greek, meaning a pulley. **Trigeminal**, the fifth cranial nerve, is from the Latin, *trigeminus*, meaning three born or originating together. Gemini [twins] also described the twin stars Castor and Pollux. **Abducens**, the sixth cranial nerve, is from the Latin,
meaning to draw away from, thus describing its effect upon eyeball movement. The seventh cranial nerve, **Facial**, is from the Latin, *facies*, meaning appearance or face. The eighth cranial nerve, **Auditory**, is from the Latin, *audio*, which is derived from an earlier Greek word meaning sound. **Glossopharyngeal**, the ninth nerve, is derived from two Greek words meaning the tongue [as in glossary] and the windpipe [pharynx].

The tenth cranial nerve, **Vagal**, comes from the Latin, *vagus*, meaning wandering, as in words such as vagrancy and vague. The eleventh, **Spinal Accessory**, is from two Latin words, *spinalis* and *accessus*, the latter meaning to approach or to come near, as in words such as accede or accession. And finally, the twelfth is called **Hypoglossal**, meaning below the tongue.

– STANLEY M. ARONSON, MD, MPH

# Vital Statistics

**Rhode Island Department of Health**
Patricia A. Nolan, MD, MPH, Director of Health

*Edited by Roberta A. Chevoya*

## Rhode Island Monthly Vital Statistics Report

## Provisional Occurrence Data from the Division of Vital Records

| Underlying Cause of Death | Reporting Period | | | |
|---|---|---|---|---|
| | June 2002 | 12 Months Ending with June 2002 | | |
| | Number (a) | Number (a) | Rates (b) | YPLL (c) |
| Diseases of the Heart | 224 | 3,092 | 294.9 | 4,151.5 |
| Malignant Neoplasms | 226 | 2,437 | 232.5 | 7,523.5 |
| Cerebrovascular Diseases | 48 | 536 | 51.1 | 795.0** |
| Injuries (Accident/Suicide/Homicide) | 34 | 406 | 38.7 | 7,370.0*** |
| COPD | 46 | 517 | 49.3 | 512.5 |

| Vital Events | Reporting Period | | |
|---|---|---|---|
| | December 2002 | 12 Months Ending with December 2002 | |
| | Number | Number | Rates |
| Live Births | 1,134 | 13,177 | 12.6 |
| Deaths | 1,049 | 10,430 | 9.9 |
| Infant Deaths | (7) | (91) | 6.9 |
| Neonatal deaths | (4) | (60) | 4.6 |
| Marriages | 478 | 8,271 | 7.9* |
| Divorces | 203 | 3,336 | 3.2 |
| Induced Terminations | 460 | 5,551 | 421.3# |
| Spontaneous Fetal Deaths | 80 | 957 | 72.6 |
| Under 20 weeks gestation | (70) | (887) | 67.3 |
| 20+ weeks gestation | (10) | (70) | 5.3 |

(a) Cause of death statistics were derived from the underlying cause of death reported by physicians on death certificates.

(b) Rates per 100,000 estimated population of 1,048,319

(c) Years of Potential Life Lost (YPLL)

*Note: Totals represent vital events which occurred in Rhode Island for the reporting periods listed above. Monthly provisional totals should be analyzed with caution because the numbers may be small and subject to seasonal variation.*

* Rates per 1,000 estimated population
** Excludes one death of unknown age.
*** Excludes two deaths of unknown age.
# Rates per 1,000 live births

THE RHODE ISLAND MEDICAL JOURNAL

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NUMBER 1

PROVIDENCE, R.I., JANUARY, 1917

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SINGLE COPY, 25 CENTS

## Ninety Years Ago

[June, 1913]

E.D.Clarke, MD, Woonsocket, in "The Proper Management of Obstetrical Work by the General Practitioner," advised on the practical remedies for the "more common causes of death following child-birth." First, he cited sepsis, generally due to the "fingers of the physician or nurse, usually the former." He urged physicians to scrub their hands and arms "as close as for abdominal operations," use sterile rubber gloves and sterile operating gowns. A second culprit was post-partem hemorrhage: he urged physicians to insert hand into the uterus, remove clot, and sweep around the wall of the uterus, also to elevate the foot of the bed. "If the uterus refuses to contract, a piece of ice the size of a hen's egg may be introduced into the uterus." A third culprit was ecclampsia, occurring 1 in 500 cases. If a physician spotted early signs (e.g., albumen, severe headaches, nausea, distorted vision), he should put the patient on a mild diet, encourage her to drink water, and give her a tincture of iron three times a day. If that doesn't work, he should induce labor.

In "Pyosalpinx after Vaginal Hysterectomy – Tubal Pregnancy Co-Incident with Intra-Uteral Pregnancy," Edward S. Brackett, MD, described the case of a 54 year-old married woman, who from ages 20 to 32 gave birth to 4 children and had one miscarriage. Twelve years earlier, she had a vaginal (clamp) hysterectomy. "The clamps were removed after 36 hours and recovery was uneventful." When she felt ill ("a feeling of weight and oppression in the lower abdomen") in 1912, her physician referred her to Dr. Brackett, who initially diagnosed a malignant growth. After surgery, the patient recovered. Dr. Brackett recounts: "After the vaginal hysterectomy, the fimbriated end of the left tube had become adherent to the vaginal vault and for some reason an inflammatory process had developed in the tube 12 years later, either from a new infection or a pinhead opening into the vagina. …the intra-abdominal pressure had gradually inverted the tube through the opening and the tumor which had been mistaken for a malignant growth was the fimbriated end of the left tube."

Fredric J. Farrell, MD, described "Juvenile Tabes Diabetes Case of Inherited Syphilis." This 14 year-old boy, who seemed fine at birth, started school at age 6, but could not read or write. He had a protuberant abdomen, enlarged spleen, double optic atrophy, dilated pupils ("unequal and irregular, of the Argyll-Robertson type,") ataxic gait, and no knee or Achilles' jerks.

## Fifty Years Ago

[June, 1953]

Patrick F. O'Mahony, MD, a resident at Butler Hospital, contributed "Korsakoff Psychosis Showing Unusual Features." This 32 year-old woman "… did not know that she was in a hospital nor did she seem to have any realization that she was ill. When questioned about the identity of the people around her, she claimed to know them all and gave them fictitious names and occupations…Her overall mood was one of euphoria."

Albert H. Jackvony, MD, President of the Medical Society, 1952-53, gave the Presidential Address, "The Medical Society and the Medical Character." "To be a good practitioner of medicine requires more than technical skill. It requires…a broad, sympathetic and tolerant conception of life."

Peter Pineo, MD, Editor, *RI Medical Journal*, reported on the first Western Hemisphere Conference, held in Virginia, of the 6 year-old World Medical Association. He commended the pharmaceutical companies, which financed the meeting: "The big pharmaceutical manufacturing firms are highly to be commended for their relationship with the medical profession."

## Twenty Five Years Ago

[June, 1978]

In "Message from the Dean: Back to the Classroom," Stanley M. Aronson, MD, described the 8th semester option for the class of '79. This semester, "akin to a sabbatical leave," let students take up to 3 campus courses. "There will never be another period of time in their professional lives free of clinical or administrative responsibilities with their attendant energy drain."

Charles E. Millard, MD, in "Application of Continuing Medical Education to Actual Clinical Practice of the Physician,"the Fiske Fund Prize Dissertation, 1977, urged that CME (required by 17 medical societies, 8 specialty societies, and 19 state licensing boards) focus on problems of office practice, not solely hospital cases.

Pierre M. Galletti, MD, PhD, contributed "The Hybrid Artificial Pancreas," which "…offers an exciting new approach to the control of endocrine disorders." He saw the major problem as thrombogenesis.

In "Law: The Magic Mirror," Joseph L. Lennon, OP, Vice President for Community Affairs, Providence College, and Board Member, Blue Shield of Rhode Island, cautioned, "A humane society dedicated to the rights of man must have a government of laws, not men."