TABLE
RATES OF NEEDLESTICK SEROCONVERSION AMONG HEALTHCARE WORKERS EXPOSED TO HEPATITIS C VIRUS, WITH A MINIMUM FOLLOW-UP OF 5 MONTHS

| Country | Healthcare Workers | Seroconversion | Percentage | Comments |
|---|---|---|---|---|
| USA' | 50 | 3 | 6.0 | |
| UK[11] | 24 | 0 | | |
| Italy[12] | 123 | 2 | 1.6 | All source patients HIV + |
| Italy[13] | 61 | 0 | | Dialysis source patients |
| Italy[14] | 30 | 0 | | |
| Japan[15] | 88 | 3 | 3.4 | Frozen serum samples collected from 1979 to 1990 |
| Japan[16]* | 91 | 5 | 5.5 | Frozen serum samples collected from 1977 to 1990 |
| Spain[17] | 53 | 1 | 1.9 | 49/53 source patients HIV+ |
| Spain[18] | 31 | 0 | | Most source patients HIV+ |
| Total | 551 | 11 | 2.0 | |

* In this study, among 68 healthcare workers exposed to HCVRNA-positive source patients, seven (10%) showed appearance of HCV RNA by polymerase chain reaction. of whom five (7.3% of the 68) seroconverted by first-generation assays.

14. Stellini R, Calzini AS, Gussago A, Rodella A, Signorini A low prevalence of anti-HCV antibodies in hospital workers. *Eur J Epidemiol* 1993;9:674-675.
15. Sodeyama T, Kiyosawa K, Urushihara A. et al. Detection of hepatitis C virus markers and hepatitis C virus genomic-RNA after needlestick accidents. *Arch Intern Med* 1993;153:1565-1572.
16. Mitsui T; Iwano K, Masuko K, et al. Hepatitis C virus infection in medical personnel after needlestick accident. *Hepatology* 1991;16:1109-1114.
17. Perez-Trallero E, Cilla G, Saenz JR. Occupational transmission of HCV. *Lancet* 1994; 344:548.
18. Hernandez ME, Bruguera M, Puyuelo T Barrera JM, Sanches Tapias JM, Rodes J. Risk of needlestick injuries in the transmission of hepatitis C virus in hospital personnel. *J Hepatol* 1992;16:56-58.

**Vincenzo Puro, MD**
**Nicola Petrosillo, MD**
**Giuseppe Ippolito, MD**
Centro di Riferimento AIDS
Ospedale "L.Spallanzani"
Rome, Italy
**Janine Jagger, MPH, PhD**
Health Care Worker Safety Center
University of Virginia
Charlottesville, Virginia

*This study was supported by the Italian Ministry of Health-AIDS project, grant 9201.04-ISS.*

*The authors reply*

We appreciate the comments and additional citations provided by Puro et al. Specifically, Puro et al are concerned about the "high" rate of HCV transmission found in our study (6% following a needlestick injury) compared with some other studies. Puro et al suggest that the median rate of transmission (1.6%) and the mean rate of transmission (2.0%) of these combined studies indicate that a 6% rate of transmission is too high. In fact, both of these values, and the 2.6% rate that would have been observed if all 117 HCV-positive needlestick injuries were evaluated and no additional infections were found, are within our reported 95% confidence interval (1.3% to 16.6%). The authors are correct in advising caution in the interpretation of the risk of non-A, non-B hepatitis in healthcare workers using active surveillance compared with passive surveillance in the community.

There are a number of potential reasons for the differences in rates of HCV transmission in published reports. For example, there appear to be geographic and time-period differences in published studies that found higher rates of transmission compared with those that found lower rates of transmission[1-8] (Table). It is plausible that the studies cited by Puro et a1,[4-8] which examined exposures that were more recent than the studies showing a higher rate of transmission, and often included healthcare workers from high-risk settings, are more likely to include healthcare workers who were wearing gloves or who reported needlestick injuries that were superficial. The transmission rate of HCV probably is dependent on the depth of injury, the dose or inoculum, and whether the needle first penetrated a latex barrier.[9] Finally, a variety of assays that appear to have different screening characteristics were used in these studies.[10]

We all agree that the solution is a prospective trial involving larger numbers of healthcare workers using a standard assay and PCR. These prospective studies will need to attempt to define things such as the depth of percutaneous injury, the size of the inoculum, and whether the needle first penetrated a latex glove(s). Until then, it is likely that the observed disparity in transmission rates of HCV following an HCV-positive needlestick injury will remain unresolved.

**REFERENCES**

1. Mitsui T Iwano K, Masuko K, et al. Hepatitis C virus infection in medical personnel after needlestick accident. *Hepatology* 1992;16:1109-1114.
2. Sodeyama T, Kiyosawa K, Urushihara A, et al. Detection of hepatitis C virus markers and hepatitis C virus genomic-RNA after needlestick accidents. *Arch Intern Med* 1993;153:1565-1572.
3. Lanphear BP Linnemann CC Jr, Cannon CG, DeRonde MM, Pendy L, Kerly LM. Hepatitis C virus infection in health care workers: risk of exposure and infection. *Infect Control Hosp Epidemiol* 1994;15:745-750.
4. Zuckerman J. Clewley G, Griffiths P, Cockroft A. Prevalence of hepatitis C antibodies in clinical healthcare workers. *Lancet 1994;* 343:1618-1620.

https://doi.org/10.1017/S0195941700007803 Published online by Cambridge University Press

5. Petrosillo N, Puro V, Ippolito G, Italian Study Group on bloodborne occupational risk in dialysis. *Lancet* 1994;344:339-340.
6. Stellini R, Calzini AS, Gussago A, Rodella A, Signorini A. Low prevalence of anti-HCV antibodies in hospital workers. *Eur J Epidemiol* 1993;9:674-675.
7. Perez-Trallero E, Cilla G, Saenz JR. Occupational transmission of HCV. *Lancet* 1994;344:548.
8. Hernandez ME, Bruguera M, Puyuelo T, et al. Risk of needlestick injuries in the transmission of hepatitis C virus in hospital personnel. *J Hepatol* 1992;16:56-58.
9. Lanphear BP Trends and patterns in the transmission of bloodborne pathogens to health care workers. *Epidemiol Rev* 1994;16:437-450.
10. Sugitani M, Inchauspe G, Shindo M, Prince AM. Sensitivity of serological assays to identify blood donors with hepatitis C viraemia. *Lancet* 1992;339:1018-1019.

**Bruce P. Lanphear, MD, MPH**
Departments of Pediatrics and of Community and Preventive Medicine
University of Rochester
School of Medicine and Dentistry,
Rochester, New York
**Calvin C. Linnemann, Jr, MD**
Departments of Internal Medicine and Pathology and Laboratory Medicine
University of Cincinnati
Cincinnati, Ohio

## Wound Infection Surveillance

**To the Editor:**

Postoperative wound infection is an infrequent adverse clinical outcome that properly concerns all surgeons, some epidemiologists, and now a number of the newest players in American healthcare, the "quality improvers." An antecedent requirement for using process variables as adverse outcome surrogates in quality improvement work is to prove their linkage to outcomes of interest. What are needed to demonstrate solidly that postoperative antibiotic orders mark surgical wound infection are aggressive global wound infection surveillance data rigidly obtained with prospective, parallel pharmacy data to rate the putative marker in a pure observational study without surgeons knowing that the probe is underway. To my knowledge, no such data ever have been produced and, even if they appear tomorrow morning in the finest peer-reviewed journals, I foresee lukewarm endorsement of the scheme wistfully suggested by Yokoe and Platt.[1]

I am not surprised that antibiotic use seems a marker for wound infection presence. In play here is a continuing pattern of well-intended, but often unnecessary, clinical practice by lots of colleagues. No competent surgeon would exclude empirical, adjunctive antibiotic use, for example, in a patient with a fresh vascular graft or heart valve and a rip-roaring staphylococcal wound infection! However, in general, antibiotic treatment in a patient with an infected surgical incision is necessary only for spreading infections attacking normal tissues lateral or deep to a wound space or for systemic sepsis. These are uncommon evolutions[2] with all sorts of definable special circumstances, fuzzy semantic areas, and catch-22s that cross every specialty boundary and many operation types. Even when antibiotics *are* indicated as adjuncts in treating wound infection, no scientific data exist to specify, for example, even the duration of the added therapy! *The generic, proper treatment of a surgical wound infection is to open the closed skin incision.*

That patients do well when incision opening is accompanied by antibiotic use unfortunately serves, by the *post hoc ergo propter hoc* fallacy, to reinforce the notion that the antibiotic "might help and can't hurt,"[3] and this reinforcement has been repeated for almost 50 years now -- about eight generations of surgical training. Nobody has ever shown that antibiotics can improve the treatment of the vast majority of opened, suppurated incisions, which are almost impossible to keep from healing by second intention, if only kept clean. Placing a caged canary in almost every such infected patient's hospital room is exactly as effective in an adjunctive sense as ordering antibiotics if the subcutaneous space has been opened for drainage and open wound care. If Yokoe and Platt have contradictory evidence on this point, we need to see it.

The monthly editorial[4] in the same issue as the Yokoe-Platt paper states very early in its second paragraph, that "... comprehensive evaluation of antibiotic use would include every aspect of the process, including *1) the decision to prescribe antibiotics to a patient"* (emphasis mine). Under managed care initiatives, efforts to trim resource waste currently are targeting inappropriate pharmacotherapy. Every dollar counts. Unnecessary antibiotic use, especially by surgeons, will be on every hit list, and the reasons are not concealed: about 25 million operations are performed in this country every year, we use lots of antibiotics, our daily workloads are perused easily in operating room logbooks, and we traditionally have not been exactly penurious in our stewardship of antibiotic dollars. As the reflexive use of antibiotics for "treating wound infections" is exposed and gradually rooted out, the surrogate marker scheme of Yokoe and Platt would ironically be progressively, *silently* disabled by this positive accomplishment, and thereby made completely unreliable.

Modern, computer-equipped hospital pharmacies that resemble air traffic control radar rooms notwithstanding, I remain unconvinced by Yokoe and Platt that the best way to track wound infections is not old-fashioned "shoe leather epidemiology."[5] As we have demonstrated in our continuing journey since 1978,[6] empowering all surgical ward and clinic nurses as primary case-finders for wound infections (with a single wound infection nurse acting as their consultant and our data manager) is an effective approach *because ward and clinic nurses see patients' incisions anyway as* part *of daily care agendas*. This approach meets the near-sacred CQI requirement of involving and bonding multiple team members interdisciplinarily; it generates enough monthly data to satisfy every administrative type who may be worried that we have an "infection problem"; it provides fodder for all sorts of discussions; it does not require chart review or lab work to case-find; and it removes surgeons from a required diagnostic role, thereby defusing bluntly any "fox-watching-the-henhouse" criticisms. The only extra resource outlay is one half the very modest annual salary and benefit costs for one wound infection nurse. That's not much money.

https://doi.org/10.1017/S0195941700007803 Published online by Cambridge University Press