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Nonoccupational Needlestick Injuries and Postexposure HIV Prevention

To the Editor:

We read with interest the article by McCausland et al¹ in the November 2003 issue of *Annals* on nonoccupational post-exposure HIV prevention.

A recently published article reviewed communityacquired (non-health care worker) needlestick injuries presenting to an urban emergency department (ED) in Sydney, Australia, over a 6-year period.² The most common mechanism of injury was exposure to discarded syringes, although there were a number of deliberate assaults.

Table (O'Leary and Green).

The estimated risk of seroconversion after a significant community-acquired needlestick injury from an unknown source.

	Hepatitis B Hepatitis C		
	Virus	Virus	HIV
Risk of seroconversion from a positive source, %	23–62	1.8	IVDU 0.6 HCW 0.3
Seropositive prevalence in the Sydney community (assume IVDU), %	50	50–90	Homosexual IVDU 17 Other IVDU 1
Risk of seroconversion after a CANSI from an unknown source (assume source is IVDU), %	12–31	1.62	0.003–0.05*

IVDU, Intravenous drug user; HCW, health care worker; CANSI, community-acquired needlestick injury.

*Assuming risk of 0.3%.

The authors state that 25% of respondents would recommend postexposure prophylaxis for unintentional needlestick injuries and, as a result, have drawn the conclusion that few recommend drug therapy for high-risk exposures. We would argue that unintentional needlestick exposures are generally low risk and that a 25% rate of postexposure prophylaxis is far too high. The risk of seroconversion from discarded syringes is thought to be extremely low; however, the risk from deliberate assaults may be higher (eg, fresh blood, deeper wound, larger inoculation).

When discussing risk with patients, we must remember that often the source of the blood is unable to be identified. Therefore, while considering the rate of seroconversion, we must also consider the prevalence of disease in the community. For instance, in Sydney the prevalence of HIV in the intravenous drug user population is low at approximately 1%.³ The Table illustrates the calculated risk of seroconversion from a significant community-acquired needlestick injury in Sydney, Australia.

Therefore, the actual risk of HIV seroconversion is quite small and has to be balanced against the real risk of side effects from antiretroviral medications.

We would agree that the provision of postexposure prophylaxis for these injuries is within the scope of ED care. The establishment of protocols based on local disease prevalence data and type of injury will make treatment more uniform.

Fenton Marc O'Leary, MBBS, MRCS (Eng) Timothy Christopher Green, MBBS Royal Prince Alfred Hospital Sydney, Australia

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Screening for Severe Acute Respiratory Syndrome in the Emergency Department

To the Editor:

The articles in the January 2004 issue of *Annals* by Chen et al,^{1,2} Wang et al,³ and Su et al⁴ introduced novel scoring systems for predicting severe acute respiratory syndrome (SARS) in the emergency department (ED). These studies

are indeed very useful first steps in developing diagnostic strategies for this new infectious disease. Nonetheless, I would like to discuss some areas of concern in interpreting and applying the results.

First, it should be noted that in all of these studies, patients were only included if they had documented fever (body temperature >38°C [>100.4°F]). Although Chen et al¹ and Su et al⁴ suggested that their scoring systems could be used in settings where mass screening for SARS is required, application will actually be limited to febrile patients with temperatures greater than 38°C (>100.4°F).

In mass screening, if patients with low-grade fever (37.5°C to 38°C [99.5°F to 100.4°F]) are not included, a substantial number of patients in the early stages of the disease will likely be missed. In a study from our ED-based SARS screening clinic during the outbreak in Hong Kong (which included afebrile patients), it was shown that 19% of SARS patients initially presented without fever, and that radiographic evidence of pneumonia often preceded fever.⁵ Therefore, a more useful tool for mass screening would have included fever or body temperature as a variable, thus allowing the evaluation of afebrile cases.²

Furthermore, the study of Wang et al³ actually enrolled only those cases that met the World Health Organization (WHO) criteria for suspected SARS. Although their scoring system was shown to have a sensitivity of 100%, this perfect figure will apply only specifically to those patients already screened as positive by the WHO criteria or case definition. Unfortunately, the WHO criteria for suspected SARS have been shown to have a sensitivity of only 26%.⁵ In practice, therefore, if 100 SARS patients presented to the ED for screening, only 26 would be correctly triaged to undergo the scoring system evaluation. Thus, relying on this strategy, despite the implementation of a near-perfect decision rule, will still result in an unacceptable number of missed cases.

Finally, the performance of chest radiography was not adequately examined in these studies. We believe that chest radiography will likely be the single most important screen for SARS in the ED setting. It has been shown to be the strongest predictor available in the ED, with an odds ratio of 17.4.⁶ During the outbreak, we performed screening chest radiography on virtually every patient with fever greater than 38°C (>100.4°F), regardless of symptomatology. It remains to be proven whether the scoring systems in these studies, given the limitations addressed by the authors, can significantly outperform chest radiography.

In addition to the multilobar infiltrates evaluated by Wang et al,³ it will be important also to analyze the predictive values for lobar, segmental, or even patchy infiltrative changes. A detailed time sequence of radiographic progression, in relationship to the progression of symptoms, may prove to be useful, as well.

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In reply:

We thank Dr. Chan for his interest in our articles. His comments provide an opportunity for further discussion about triaging potential severe acute respiratory syndrome (SARS) patients.

In regard to his first comment, some patients actually contract SARS-coronavirus and develop clinical symptoms and chest radiographic infiltrates without fever (defined as body temperature >38°C [>100.4°F]). However, such atypical presentations of SARS have mostly been reported in patients with congestive heart failure, compromised immunity, and the elderly.^{1,2} From April 3 to May 12, 2003, there were 2,765 patients (including 602 patients with exposure risk) who presented to our emergency department (ED) for SARS screening without documented fever. Even without fever, they were admitted as long as there were infiltrates on chest radiography. Only 3 of them were finally confirmed to have SARS. All 3 patients were elderly with comorbidities such as heart failure or compromised immunity.

To the contrary, in patients with normal immunity, fever was near universally presented.²⁻⁴ It has been proposed that SARS–coronavirus pneumonia is largely an immunologically mediated process.^{3,5} Patients with normal immunity, in our experience, may have pulmonary infiltrates preceding respiratory symptoms, but rarely before signs of systemic inflammation.⁵ However, there are 2 caveats that should be addressed. First, 40.5% (32/79) of SARS patients documented fever at home but not on arrival to the ED, as