LETTER TO THE EDITOR

Acinetobacter Nares Colonization of Healthy US Soldiers

TO THE EDITOR—Since the onset of hostilities in Iraq and Afghanistan, bacteria of the genus Acinetobacter have become an important cause of infection among trauma patients in military medical treatment facilities.1 These infections are potentially difficult to treat because of the organism's possible broad-spectrum antimicrobial resistance. Little is currently known about the epidemiology of these traumaassociated infections. There are several theories regarding how patients may acquire this organism, one of which is that these patients may have harbored the bacteria on their skin prior to injury. Previous studies have demonstrated that Acinetobacter species can be found colonizing the skin and nares of healthy people.^{2,3,4} At our institution, a large study is underway examining community acquired methicillin-resistant Staphylococcus aureus colonization of the nares among healthy soldiers. We took this opportunity to evaluate whether nares colonization by Acinetobacter species exists in this population.

Study participants were recruited from soldiers undergoing military training at Fort Sam Houston, Texas. This population was geographically diverse (samples were obtained from study participants approximately 3 days after their arrival at Fort Sam Houston from various training bases throughout the US), healthy, and not involved with health care. After informed consent was obtained (Brooke Army Medical Center institutional review board protocol C.2004.163), a BBL CultureSwab with Stuart Medium (Becton Dickinson) was used to swab the anterior nares of each participant. Swabs were then plated onto MacConkey agar (Remel). After incubation at 37°C in ambient air for 24 hours, the plates were examined for growth. Nonfermentative colonies were further identified using the Vitek automated system (BioMérieux Vitek).

Swab specimens from the nares of 293 soldiers were obtained. No soldiers were found to have *Acinetobacter* colonization. Cultures of specimens from 25 soldiers yielded growth of a gram-negative organism, and 1 specimen yielded 2 separate gram-negative organisms. Sixteen of these 26 isolates were not obviously fermentative and were therefore further analyzed with the Vitek automated system. Genera identified included *Proteus* (2 isolates), *Klebsiella* (4 isolates), *Citrobacter* (4 isolates), *Enterobacter* (5 isolates), and *Serratia* (1 isolate).

Our finding that no study participants had *Acinetobacter* nares colonization is in obvious contrast to previous studies. IN previous studies, researchers have examined the prevalence of *Acinetobacter* colonization in healthy people and reported a prevalence of 6%-20% in the anterior nares.^{2,3,4} Several factors may explain the discrepancy between our results and

those of earlier studies. First of all, these prior studies are limited to distinct geographical niches, namely Germany,³ Hong Kong,² North Carolina,⁴ and New Jersey,⁴ and therefore the study populations differed from our military population, which was large and geographically diverse. Secondly, most of the "normal" population described in prior studies is, in fact, drawn from healthcare workers or laboratory workers, thereby potentially biasing the data. Our study population, in contrast, had no extensive exposure to a health care setting.

Some limitations of our study could also explain the discrepancy. *Acinetobacter* colonization has been reported to be of low density.² It is possible that we failed to detect some colonization with our chosen sampling technique. Secondly, it has been suggested that the prevalence of *Acinetobacter* colonization may depend on climatic and seasonal variation.⁴ As our study was a one-time sample, our results may have been affected by these variances.

An important limitation of our study is that only soldiers without a history of deployment provided samples. Soldiers may acquire colonization while in Iraq or Afghanistan, and sampling on their return, or while they are in the country of deployment, may yield different results. Further research into this matter is pending. Because of this, our study population differs somewhat from the population of wounded soldiers in which these infections are occurring.

Regardless, it appears that the rate of *Acinetobacter* species nares colonization in a healthy population is exceedingly low. We can safely conclude that nares colonization prior to deployment is likely of little, if any, importance in the development of subsequent infections in the military population. If nares colonization with *Acinetobacter* exists in wounded soldiers, our study suggests that it was likely acquired while in Iraq or Afghanistan. Clearly, more needs to be learned regarding the epidemiology of *Acinetobacter* infections in military trauma patients; research is ongoing and may shed further light on this matter.

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Nosocomial Postsurgical Meningitis in Children: A 12-Year Survey Comparing Data From 1993-1998 With Data From 1999-2004

TO THE EDITOR—The incidence of nosocomial meningitis after neurosurgery is increasing.¹⁻² In a previous study, a survey of all 8 pediatric and neonatal neurosurgery de-

partments in Slovakia revealed that 101 cases of pediatric bacterial meningitis were recorded during 1993-1998, with an attributable mortality of 15% and sequelae detected in 18% of cases. We conducted a similar survey during 1999-2004, which revealed an additional 57 cases of disease. Here, cases of pediatric nosocomial meningitis during 1999-2004 are compared with those during 1993-1998 with respect to etiology, risk factors, and outcome.

A prospective protocol was used to assess each case of meningitis that occurred during hospitalization in persons aged 18 years or less at any of the 8 pediatric and neonatal neurosurgery departments in Slovakia; the number of such departments has not changed during the past 10 years. Nosocomial meningitis was clinically defined as onset of meningitis after neurosurgery in patients with a positive result of a cerobrospinal fluid culture. Risk factors were assessed, including the etiology of meningitis, the type of neurosurgery, and the presence of underlying disease(s), trauma, abnormal central nervous system findings, central nervous system disease, positive results of blood cultures, malignancy, and hemorrhage. Mortality rates associated with infection and/or to central nervous system sequelae and treatment (ie, surgery and antibiotics therapy) were assessed, and antimicrobial resistance was measured for the pathogens most commonly isolated from the cerebrospinal fluid of study subjects (coagulase-negative staphylococci [CoNS], Pseudomonas aeruginosa, and Acinetobacter baumannii). No major changes in surgical techniques (including shunt insertion and prosthe-

TABLE 1. Comparison of the Etiology of Nosocomial Meningitis During the 2 Study Periods

Pathogen	No. (%) of isolates, by period		
	$ \begin{array}{r} 1993-1998 \\ (n = 115) \end{array} $		P
Gram-positive organisms			
Staphylococcus aureus	10 (8.7)	2 (2.6)	NS
Coagulase-negative staphylococci	, ,		
All species	50 (43)	34 (45)	NS
Other than Staphylococcus epidermidis or Staphylo-			
coccus haemolyticus	0	8 (11)	0.001
Enterococcus faecalis	8 (7.0)	1 (1.3)	NS
Viridans streptococci	1 (0.9)	1 (1.3)	NS
Streptococcus agalactiae	5 (4.3)	0 (0)	
Streptococcus pneumoniae	2 (1.7)	3 (4.0)	NS
Subtotal	76 (66)	49 (65)	NS
Gram-negative organisms			
Enterobacteriaceae	13 (11)	10 (13)	NS
Pseudonymous aeruginosa	6 (5.2)	7 (9.3)	NS
Acinetobacter baumannii	10 (8.7)	10 (13)	0.05
Stenotrophomonas maltophilia, Flavobacterium meningo-			
septicum, or Arcanobacterium haemolyticum	3 (2.6)	0 (0)	NS
Subtotal	29 (25)	26 (35)	0.05
Fungi			
Overall	10 (8.7)	2 (2.7)	NS
Candida albicans	7 (6.1)	2 (2.7)	NS
Aureobasidium mansoni, Clavispora lusitaniae, or	, ,	,	
Rhodotorula rubra	3 (2.6)	0 (0)	NS