

STATE-OF-THE-ART CLINICAL ARTICLE

Infections in Patients with Spinal Cord Injuries

John Z. Montgomerie

From the Department of Medicine, Rancho Los Amigos Medical Center,
Downey, California

Approximately 8,000 persons survive spinal cord injury (SCI) each year in the United States. About 200,000 Americans have spinal cord injuries, and this number is increasing as life expectancy increases towards normal levels for these persons. Nosocomial infection is common in patients with SCIs because these patients are admitted to the hospital immediately after their injuries occur, and they stay for a considerable time for management of the injuries and for rehabilitation. Because of the prolonged stay, these patients are at greater risk of developing infections with resistant microorganisms including methicillin-resistant *Staphylococcus aureus* (MRSA) and multiresistant gram-negative bacilli. Patients with SCIs frequently have wounds on the neck, chest, or abdomen and often are at additional risk from surgery and the use of blood products. High doses of corticosteroids, given immediately after injury, may predispose these patients to infection as well as to upper gastrointestinal complications [1]. After discharge from the hospital and rehabilitation, the incidence of infection, usually of the urinary tract, respiratory tract, and infected pressure ulcers, continues to be increased for patients with SCIs.

Urinary Tract Infections

Bacteriuria is almost universal in patients with SCIs. This infection may be asymptomatic colonization, but tissue invasion of the urinary tract occurs in most patients initially during hospitalization and rehabilitation and may be a recurrent problem for most of these patients throughout their lives. At one time, these infections were the dominant cause of bacteremia, renal failure, and death among patients with SCIs until the methods of urinary drainage improved; however, serious complications, bacteremia, calculi, pyelonephritis, and renal failure still occur.

An increased volume of residual urine and increased bladder pressure have been the most important factors responsible for

urinary tract infection in patients with SCIs. Performance of intermittent catheterization and bladder-neck manipulation have reduced the morbidity associated with neurogenic bladder. Changes in the bladder associated with the long-term use of indwelling catheters include squamous metaplasia, thickening and fibrosis of the bladder, diverticula, calculi and the presence of multiple organisms, alkaline-encrusting cystitis with urease-producing bacteria, penile and scrotal fistulae, abscesses and epididymitis, and squamous cell carcinoma of the bladder.

Intermittent catheterization is associated with an increased incidence of bacteriuria, which may be influenced by the frequency of catheterization. As the interval between catheterizations increases, the incidence of bacteriuria may increase. Patients who are unable to perform intermittent catheterization themselves are also at greater risk of infection [2].

A wide range of microorganisms has been isolated from patients with SCIs and urinary tract infections. The presence of bacteria that are urease producers raises concerns about calculus formation. Infection due to *Escherichia coli* strains that may be more virulent than other strains has not been well studied. The virulence of some bacterial species, such as *Pseudomonas*, in the urinary tracts of patients with SCIs has been questioned, but there are sufficient data showing that tissue invasion and bacteremia with *Pseudomonas aeruginosa* are not uncommon.

E. coli and species of *Pseudomonas*, *Klebsiella*, and *Enterococcus* have been the predominant microorganisms that cause urinary tract infections in patients with SCIs. A high prevalence of *Proteus* species, which may relate to the more frequent use of indwelling catheters, has been noted at some centers. *Klebsiella*, *Pseudomonas*, and *Proteus* species tend to be more resistant than *E. coli* to commonly used antibiotics, but outbreaks of infection with multiresistant gram-negative bacilli have been described relatively infrequently in SCI units.

A patient's sex and level of injury may affect the microbiology of bacteriuria and colonization. At our institution, the incidence of infection with *Klebsiella* and *Pseudomonas* species among male patients has been high, which may be related to the use of external condom catheters. In one study of female patients with SCIs who underwent intermittent catheterization, *E. coli* and *Enterococcus* species accounted for 71% of infections [3].

Because of loss of sensation, patients with SCIs do not have the common symptoms of urinary tract infection such as fre-

Received 16 July 1997.

Reprints or correspondence: Dr. John Z. Montgomerie, Rancho Los Amigos Medical Center, 7601 East Imperial Highway, 244 HB, Downey, California 90242.

Clinical Infectious Diseases 1997;25:1285-92

© 1997 by The University of Chicago. All rights reserved.
1058-4838/97/2506-0001\$03.00

quency, urgency, and dysuria. The clinical features of UTI may include fever, pyuria, and other "soft" symptoms and signs such as discomfort over the back or abdomen during urination, onset of incontinence, increased spasticity, autonomic hyperreflexia, malaise, lethargy, or observation of cloudy urine with increased odor [4]. It is important to confirm the diagnosis by performing a urine culture.

Blood cultures should be performed if a patient has a high fever. Localization of the infection and exclusion of obstruction and other factors that might influence response to treatment are important. The significance of any level of bacteriuria has been controversial. Small numbers of bacteria may be found and may persist in the bladder urine of patients undergoing intermittent catheterization [5]. A consensus of investigators for the National Institute on Disability and Rehabilitation Research decided that the criteria for quantitative cultures of urine for the diagnosis of bacteriuria include catheter specimens from patients undergoing intermittent catheterization ($\geq 10^2$ cfu/mL), clean-voided specimens from catheter-free males using condom collection devices ($\geq 10^4$ cfu/mL), and specimens from indwelling catheters with any detectable concentration of organisms [4].

The bacteria that cause urinary tract infections have been found colonizing the urethra and perineal skin of male patients with SCIs. A high rate of colonization with *P. aeruginosa* and *Klebsiella pneumoniae*, which reside in the perineum, urethra, and bowel flora and the urine in urine drainage bags, is frequently associated with the use of external urinary catheter systems in men [6].

A wide range of urinary tract pathogens may be found in the environment, and transmission of these organisms on the hands of health care personnel is the most likely means of transmission among patients in hospitals and clinics [7]. The use of antiperspirants or antiseptics to clean the skin or increased bathing has not been shown to significantly alter the rate of perineal skin colonization. The use of medicated soaps may also be counterproductive.

Because of a lack of evidence that treating asymptomatic bacteriuria reduces the incidence of symptomatic bacteriuria or influences the long-term function of the urinary tract or kidneys, the consensus has been that bacteriuria should be treated only when symptoms or signs are present [4]. For symptomatic patients, particularly those with high fevers who may have bacteremia, broad coverage may be necessary until the results of cultures are available because these patients are frequently colonized with resistant bacteria.

In-hospital studies of oral prophylactic antimicrobial therapy to prevent infections or of the use of antibiotic solutions such as neomycin, placed in the bladder at the time of intermittent catheterization, have shown that both these procedures may reduce the number of incidents of significant bacteriuria. This approach has been avoided at most centers because of the risk of the emergence of resistant microorganisms. In addition,

gram-negative bacilli may be replaced with gram-positive organisms such as *Enterococcus* species and *Staphylococcus epidermidis*.

Infected Pressure Ulcers

Pressure ulcers are frequent in patients with SCIs. In a recent study, Richardson and Meyer [8] found that the prevalence of pressure ulcers among recently injured patients with SCIs during the initial hospital stay varied from 13% to 69%, depending on the level of injury and whether the injury was complete or incomplete. Other important factors included the presence of multiple sites of trauma in patients with SCIs and inattention to the skin when patients were receiving care in intensive care units. The sites most frequently involved are the ischial tuberosities, the trochanter, and the sacrum. Infection may result in cellulitis, abscess formation, osteomyelitis of underlying bone, or infection of underlying joints or bursae. Bacteremia is a common complication of neglected pressure ulcers.

Multiple anaerobes and aerobes are found in pressure ulcers [9]. The degree of microbial colonization is determined by the presence of devitalized tissue. Pressure ulcers with tissue necrosis contain aerobes and anaerobes at levels of $>5 \log_{10}$ /g of tissue. The presence of necrotic tissue and a foul smell has been significantly associated with isolation of anaerobes from deep tissue. *Bacteroides* species, *E. coli*, *Proteus* species, *Enterococcus* species, and anaerobic streptococci were found to be the most prominent organisms in pressure ulcers associated with grossly necrotic tissue, and *P. aeruginosa* and *S. aureus* were the most frequently isolated organisms as the ulcers were healing [9]. Significant variability in deep-tissue culture results was observed when specimens from different parts of the same sore were examined. Such observations call into question the value of obtaining repeated cultures of pressure ulcers, but aerobic cultures frequently reveal vancomycin-resistant enterococci or methicillin-resistant staphylococci or multiresistant gram-negative bacilli.

Local signs of infection include cellulitis, abscess formation, drainage, and a foul smell. The diagnosis of osteomyelitis underlying the ulcer is more difficult. The presence of fever and leukocytosis without drainage may indicate joint involvement or infection of a bursa. The development of drainage and exposure of bone suggest that osteomyelitis may be present in the underlying bone at these sites. Findings on radiographs and bone scans may also be misinterpreted as those indicating osteomyelitis because of the frequent presence of heterotopic ossification or periosteal reaction resulting from pressure changes on the underlying bone [10]. Pathological examination of the bone is required for diagnosis, and the performance of quantitative cultures does not help differentiate osteomyelitis from infection or colonization of adjacent tissue [11].

Joint involvement in patients without sensation may be difficult to detect, and the diagnosis may be missed. This is espe-

cially true for patients with trochanteric pressure ulcers in whom infection extends to the hip joint. Aspiration of the joint and injection of radiopaque dyes may determine if the joint capsule is intact. Iliopsoas abscess, an infrequent complication of deep pressure ulcers involving the hip joint, may not be recognized [12].

Exceptional care is necessary to prevent the development of pressure ulcers, especially in patients with high cord lesions. To prevent the development of pressure ulcers, specialized nursing care, care of patients in specialized centers, and prevention of immobilization in the immediate postinjury period (including delays in transportation to the ward, delays in the performance of radiographic procedures, and waiting in emergency departments) are needed [13]. Patients must be trained in skin care before they are discharged from the hospital.

The management of pressure ulcers includes relief of pressure and physical wound debridement. Antibiotic therapy is indicated when there is evidence of bacteremia or local wound infection or at the time of extensive debridement of the wound because bacteremia may occur at that time. Myocutaneous flap surgery may be indicated when the ulceration has progressed to the muscle, bone, bursa, or joint. The aim of this surgery is to restore skin and subcutaneous tissues. Most ulcers are surgically debrided before flap surgery is performed. When an infection extends to the hip joint, it may be refractory to treatment, and resection of the femoral head and neck may be necessary (Girdlestone resection).

The perioperative administration of antibiotics for 5–7 days has been recommended to prevent postoperative flap-wound infection, which is seen after surgery in 2.5%–8% of cases [14]. Anaerobes, gram-negative aerobic bacilli, and staphylococci, including MRSA, have been cultured from postoperative wound infections.

Osteomyelitis associated with pressure ulcers has often been treated with antibiotics for ≤ 6 weeks. Infected bone is usually removed at the time of surgery, and a short course of antibiotics may be adequate. Antibiotic therapy given for >3 weeks did not affect the outcome in one study [10].

Pneumonia

Pulmonary complications including atelectasis (loss of lung volume), pneumonia, chest injury, and pulmonary infarction are the most frequent cause of morbidity and mortality among patients with SCIs. In the immediate postinjury period, pneumonia is the most common pulmonary complication [15], and it has been reported to be the most frequent cause of death [16].

From 5% to 20% of all patients with SCIs develop pneumonia during initial rehabilitation in the hospital. The risk of developing pneumonia is greatest in the early postinjury period and is influenced by factors such as inability to cough effec-

tively, prior anesthesia, and the effectiveness of treatments to clear secretions.

Pneumonia in patients with SCIs is associated with atelectasis, which results from a change in the pattern of respiration and a reduction in coughing. The impairment of the abdominal, intercostal, and diaphragmatic muscles that is associated with progressively higher lesions in the spinal cord results in a higher incidence of pneumonia among quadriplegics than paraplegics.

The preponderance of pneumonias in patients with SCIs occur in the left lower lobe [17]. The anatomy of the mainstem bronchus (the left bronchus branches off at a more acute angle than the right bronchus) probably accounts for the increased incidence of left-sided atelectasis and pneumonia. There are some limitations to the published studies of pneumonia in patients with SCIs because atelectasis and pneumonia have frequently been combined in these reports.

The clinical features of pneumonia in patients with SCIs are fever, leukocytosis, and purulent sputum associated with radiographic changes. Because of the neurological deficits, cough is not a feature.

Radiographic diagnosis is difficult because atelectasis is frequently present and predisposes patients to pneumonia. Atelectasis may be accompanied by fever and/or leukocytosis.

There are no published studies of the microorganisms that cause pneumonia in these patients. It is likely that the flora of the oropharynx, including the gram-negative aerobic bacilli, are involved as they are in most nosocomial pneumonias.

Prevention and control of pneumonia by clearing retained secretions include manual assistance in coughing and postural drainage. Postural drainage requires positions that are sometimes difficult to achieve during the acute postinjury period. Forced vital capacity has been used as a predictor of respiratory problems, and early intubation may be performed to prevent problems.

Following discharge from the hospital, patients with SCIs are at greater risk of developing pneumonia following upper respiratory tract infections. The increased risk of pneumonia in patients with SCIs justifies the use of pneumococcal and influenza vaccines, although I am unaware of studies in this group that have demonstrated the value of these vaccines.

Bacteremia

Bacteremia secondary to urinary tract infections, pneumonia, pressure ulcers, and infections at other sites is not uncommon in patients with SCIs. A feature of bacteremia among these patients has been the low associated mortality [18, 19]. Bacteremia appears to be less life-threatening in patients with SCIs than other patients. The young age of the patients, the source of the infection (the urinary tract), and the lack of underlying disease may contribute to the low mortality. Because of the low mortality, my colleagues and I have also speculated that

patients with recurrent urinary tract infections develop antibodies that may protect them from bacteremia.

Bacteremia in patients with SCIs has usually been the result of bacteriuria associated with bladder manipulation. My colleagues and I have found that infected pressure ulcers that have been neglected are the second most frequent cause of bacteremia [18]. Bacteremia associated with pneumonia has occurred mainly in quadriplegic patients who have usually been ventilator dependent. Central catheter infection, phlebitis, and infected surgical sites have been other sources of bacteremia (table 1).

In those episodes in which the urinary tract was considered to be the source of the bacteremia, enterococci, *E. coli*, and *P. aeruginosa* were the organisms most frequently isolated from the blood. Anaerobic bacteria were most frequently isolated from the blood samples from patients with pressure ulcers, and *S. aureus* was the most commonly isolated aerobic species; only three of 20 patients with pressure ulcers had gram-negative aerobic bacilli isolated from their bloodstreams [18].

MRSA Colonization and Infection

Patients with SCIs have frequently been colonized and infected with MRSA [20, 21]. Patients in SCI units are more frequently colonized than many other patients because they are usually referred from the intensive care units of other institutions, and patients may acquire MRSA during lengthy rehabilitation. MRSA infections occur most frequently in the urinary tract, wounds, lungs, and blood. Patients may remain colonized for months or years. Sites of colonization include the nose, throat, and perineum and urinary tract, but the anterior nares

Table 1. Sources of bacteremia in patients with spinal cord injuries.

Source or site of primary infection	Initial admission (n = 41)	Readmission (n = 62)	Total (n = 103)
Urinary tract infection (no. of deaths)	19	31 (4)	50
Pressure area (no. of deaths)	2	18 (1)	20
Pneumonia	6	4	10
Phlebitis or central catheter (no. of deaths)	1	4 (1)	5
Surgical site	4	0	4
Bowel perforation (no. of deaths)	2 (1)	2 (1)	4
Osteomyelitis of the spine	0	2	2
Not known	6	4	10
Other	2	0	2
Total			107*

NOTE. Data are from [18].

* The same microorganism was cultured from two sources in four patients.

Table 2. Body sites colonized with methicillin-resistant *Staphylococcus aureus* in a spinal cord injury center.

Site	No. positive/no. cultured (%)
Nose	37/67 (55)
Throat	30/67 (45)
Perineum	17/67 (25)
Urine	27/67 (40)
Wounds	58/67 (87)

NOTE. Data are from [21].

and wounds have been the sites most frequently colonized (table 2).

Control measures need to be developed according to the nature of the institution and the extent of the problem. In my institution the use of a protocol introduced in 1978 showed that colonized patients could continue to receive physical and occupational therapy without significant risk of spreading MRSA [20]. The measures included placement of colonized patients in private rooms, surveillance of MRSA isolates, and obtaining specimens for culture from potentially colonized body sites. Gloves and gowns were worn when care givers had direct contact with patients in the ward or at the time of physical or occupational therapy. Precautions were taken until three consecutive cultures, performed weekly, were negative. Patients colonized with MRSA continued to receive intensive physical and occupational therapy. Transfer of MRSA did occur in those areas where more intensive medical and nursing care is provided. Cultures of nasal secretions from hospital personnel were performed if there was a cluster of three or more nosocomial infections.

Asymptomatic bacteriuria is a common problem and may be a risk factor for transmission of MRSA on the hands of care givers who empty drainage bags or handle other urinary drainage systems. Unrecognized colonization of tracheostomy sites and respiratory secretions has also been an important source of spread. Pressure ulcers colonized with MRSA may provide a source of the organism if there is not adequate use of gloves and hand washing.

Other Infections

All patients who undergo surgery may develop infections as a result of the use of blood products. Intravenous access sites may become infected, postoperative pneumonia may occur, and wounds at the site of surgery for the injury may become infected.

There are special problems related to infection for patients with SCIs. Patients with SCIs may have milder chills because shivering may not occur below the level of the injury. The lack of sensation may delay diagnosis of infection in wounds below the level of injury. It is very easy to overlook other infected

sites such as intramuscular injection sites. Bullet wounds in the spine rarely become infected, and meningitis rarely develops, but injuries that penetrate the abdomen and spine may result in anaerobic infection of the vertebrae [22]. Osteomyelitis of the spine below the level of injury is easily missed and may present in association with subcutaneous abscess in the back [22]. For patients with cervical spinal cord injuries, tracheostomy does not increase the risk of infection during subsequent anterior cervical surgery and is therefore not contraindicated [23]. Meningitis may occur after penetrating injuries with spears and knives or as a result of CSF leakage at the time of injury or subsequent to surgery.

Osteomyelitis may occur in spinal cord-injured patients at sites underlying pressure ulcers and in fractures associated with the injury or with the halo orthosis that is often used to stabilize a fractured cervical spine.

In the first 4 weeks after an SCI (particularly those resulting in high-level lesions), acute abdominal emergencies such as gastrointestinal bleeding, peptic ulcer perforation, and pancreatitis are often seen. The diagnosis of these conditions is difficult because of the lack of sensation, and the resultant mortality is high. Blunt trauma to the abdomen is frequently unrecognized in patients with SCIs.

Heterotopic ossification produces erythema and swelling that may result in a misdiagnosis of cellulitis or deep venous thrombosis. This condition occurs around the joints below the level of the injury, most commonly in the hips. If the diagnosis is not clear at the early stages of this disease, ectopic bone formation may not be seen on the radiograph; the process is most readily detected with a technetium bone scan.

Fever

Fever is common in patients with SCIs, and many of these patients have more than one infection accounting for the fever. On the other hand, some patients have unexplained fever. Patients with SCIs may have thermoregulation problems, with lack of sweating below the level of injury. Exposure to high or low environmental temperatures can result in fever or hypothermia. Unexplained fever in quadriplegic patients that occurs soon after injury and persists for weeks has been described [24]. Deep venous thrombosis occurs more frequently than was once recognized and may account for some of the fevers that were unexplained in the past.

Pharmacological Considerations

Physiological changes in patients with SCIs may influence the absorption and elimination of many agents, including antibiotics [25]. It is important to monitor dosing of potentially toxic agents, such as aminoglycosides, if higher doses are being used to treat severe infections.

References

- Berlly MH, Wilmot CB. Acute abdominal emergencies during the first four weeks after spinal cord injury. *Arch Phys Med Rehabil* 1984;65:687-90.
- Cardenas DD, Mayo ME. Bacteriuria with fever after spinal cord injury. *Arch Phys Med Rehabil* 1987;68:291-3.
- Bennett CJ, Young MN, Darrington H. Differences in urinary tract infections in male and female spinal cord injury patients on intermittent catheterization. *Paraplegia* 1995;33:69-72.
- National Institute on Disability and Rehabilitation Research (NIDRR) Consensus Statement. The prevention and management of urinary tract infections among people with spinal cord injuries. *J Am Paraplegia Soc* 1992;15:194-207.
- Gribble MJ, McCallum NM, Schechter MT. Evaluation of diagnostic criteria for bacteriuria in acutely spinal cord injured patients undergoing intermittent catheterization. *Diagn Microbiol Infect Dis* 1988;9:197-206.
- Gilmore DS, Schick DG, Young MN, Montgomerie JZ. Effect of external urinary collection system on colonization and urinary tract infections with *Pseudomonas* and *Klebsiella* in men with spinal cord injury. *J Am Paraplegia Soc* 1992;15:155-7.
- Sanderson PJ, Rawal P. Contamination of the environment of spinal cord injured patients by organisms causing urinary-tract infection. *J Hosp Infect* 1987;10:173-8.
- Richardson RR, Meyer PR Jr. Prevalence and incidence of pressure sores in acute spinal cord injuries. *Paraplegia* 1981;19:235-47.
- Sapico FL, Ginunas VJ, Thornhill-Joyes M, et al. Quantitative microbiology of pressure sores in different stages of healing. *Diagn Microbiol Infect Dis* 1986;5:31-8.
- Thornhill-Joyes M, Gonzales F, Stewart CA, et al. Osteomyelitis associated with pressure ulcers. *Arch Phys Med Rehabil* 1986;67:314-8.
- Darouiche RO, Landon GC, Klima M, Musher DM, Markowski J. Osteomyelitis associated with pressure sores. *Arch Intern Med* 1994;154:753-8.
- Rubayi S, Soma C, Wang A. Diagnosis and treatment of iliopsoas abscess in spinal cord injury patients. *Arch Phys Med Rehabil* 1993;74:1186-91.
- Linares HA, Mawson AR, Suarez E, Biundo JJ. Association between pressure sores and immobilization in the immediate post-injury period. *Orthopedics* 1987;10:571-3.
- Garg M, Rubayi S, Montgomerie JZ. Postoperative wound infections following myocutaneous flap surgery in spinal injury patients. *Paraplegia* 1992;30:734-9.
- Fishburn MJ, Marino RJ, Ditunno JF Jr. Atelectasis and pneumonia in acute spinal cord injury. *Arch Phys Med Rehabil* 1990;71:197-200.
- DeVivo MJ, Kartus PL, Stover SL, Rutt RD, Fine PR. Cause of death for patients with spinal cord injuries. *Arch Intern Med* 1989;149:1761-6.
- Sugarman B. Atelectasis in spinal cord injured people after initial medical stabilization. *J Am Paraplegia Soc* 1985;8:47-50.
- Montgomerie JZ, Chan E, Gilmore DS, Canawati HN, Sapico FL. Low mortality among patients with spinal cord injury and bacteremia. *Rev Infect Dis* 1991;13:867-71.
- Sugarman B, Brown D, Musher D. Fever and infection in spinal cord injury patients. *JAMA* 1982;248:66-70.
- Aeilts GD, Sapico FL, Canawati HN, Malik GM, Montgomerie JZ. Methicillin-resistant *Staphylococcus aureus* colonization and infection in a rehabilitation facility. *J Clin Microbiol* 1982;16:218-23.
- Maeder K, Ginunas VJ, Montgomerie JZ, Canawati HN. Methicillin-resistant *Staphylococcus aureus* (MRSA) colonization in patients with spinal cord injury. *Paraplegia* 1993;31:639-44.
- Malik GM, Sapico FL, Montgomerie JZ. Severe vertebral osteomyelitis in patients with spinal cord injury. *Arch Intern Med* 1982;142:807-8.

23. Northrup BE, Vaccaro AR, Rosen JE, Balderston RA, Cotler JM. Occurrence of infection in anterior cervical fusion for spinal cord injury after tracheostomy. *Spine* **1995**;20:2449–53.
24. Sugarman B. Fever in recently injured quadriplegic persons. *Archives Phys Med Rehabil* **1982**;63:639–40.
25. Segal JL, Brunnemann SR. Clinical pharmacokinetics in patients with spinal cord injuries. *Clin Pharmacokinet* **1989**;17:109–29.

Suggested Additional Reading

- Donovan WH, Carter ER, Bedbrook GM, Young JS, Griffiths ER. Incidence of medical complications in spinal cord injury: patients in specialised, compared with non-specialised centres. *Paraplegia* **1984**;22:282–90.
- Montgomerie JZ, Maeder K. Nosocomial infections in patients with spinal cord injury. In: Mayhall CG, ed. *Hospital epidemiology and infection control*. Baltimore: Williams & Wilkins, **1994**:610–7.

Montgomerie JZ. Treatment of urinary tract infections in persons with spinal cord injury. *Neurorehabilitation* **1994**;4:214–21.

Stover SL, Lloyd LK, Waites KB, Jackson AB. Urinary tract infection in spinal cord injury. *Arch Phys Med Rehabil* **1989**;70:47–54.

The “Conflict-of-Interest Policy” of the Office of Continuing Medical Education, UCLA School of Medicine, requires that faculty participating in a CME activity disclose to the audience any relationship with a pharmaceutical or equipment company which might pose a potential, apparent, or real conflict of interest with regard to their contribution to the program. The author reports no conflict of interest.