
The Epidemiology of Secondary Conditions Following Spinal Cord Injury

J. Scott Richards, Ken Waites, Yu Ying Chen, Steve Kogos, and M.M. Schmitt

Secondary conditions following spinal cord injury have been well described in the literature and have a documented impact on morbidity and mortality. Many of these conditions are potentially preventable and/or amenable to treatment, with the best outcome in the latter case when treatment is applied early. An epidemiologic approach to secondary conditions attempts to identify factors that can be used to develop risk models for their development, which in turn can be useful to clinicians for monitoring, early identification, and treatment. In this article, we review the literature describing risk factors for five major secondary conditions (chronic pain, respiratory complications, urinary tract infections, pressure sores, and depression) and offer suggestions for clinical monitoring and research. **Key words:** *depression, pain, pressure sores, respiratory complications, spinal cord injury, urinary tract infection*

Secondary conditions by definition develop after onset of a disease or following trauma. It is hard to conceive of a disease or neurological disorder with more potential secondary conditions with such significant consequences as spinal cord injury (SCI). Given that SCI is a chronic condition, exposure to the risk of concomitant secondary conditions remains high throughout the lifespan and, in many instances, actually increases over time.

It would be impossible to review in detail the incidence, prevalence, and risk factors for all secondary conditions known to accompany SCI. Thirty such conditions were cited, for example, by an outpatient sample of persons with SCI as potentially posing a significant negative impact on function and quality of life.¹ It is important to understand what risk factors might be associated with the development of secondary conditions so that surveillance and early treatment intervention efforts can be more informed. Such risk fac-

tors need to be readily identifiable and therefore easily tracked so that such surveillance can be practically instituted.

There have been a significant number of

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published investigations of risk factors for the development of a wide variety of secondary conditions. For the purposes of this review, we have chosen to report on five of the most common and most significant secondary conditions in terms of their prevalence and impact, and we review what is known about the risk factors for their development. Commonalities across secondary conditions will be noted and suggestions made with regard to research and clinical implications. These five conditions are chronic pain, respiratory complications, urinary tract complications, pressure sores, and depression.

Chronic Pain

Incidence/prevalence

Chronic pain is, unfortunately, a frequent complication of SCI; in a sense it represents “insult added to injury.” Current thinking is that there are several subtypes of chronic pain. There have been numerous proposals for SCI pain classification schemes.² Recently, the International Association for the Study of Pain proposed a scheme³ that, it is hoped, all clinicians and researchers will adopt so a common nomenclature will be used. The major subtypes of SCI pain, according to most researchers, are musculoskeletal, neuropathic, and visceral. However, there are no large-scale studies that investigate the incidence and prevalence of SCI pain using these subcategories (although it is generally stated that musculoskeletal and neuropathic pain are common, and visceral pain is less common).

There have been many estimates of the frequency of chronic pain in this population. Frequency is used here, rather than incidence or prevalence, as most estimates are derived from cross-sectional convenience samples

and do not represent true population estimates. Estimates of the frequency with which pain is reported by persons with SCI range from 18% to 96%.⁴ Such disparate estimates reflect differing methodologies, such as how pain was defined in the questionnaire, when people were asked, and the like. In general, chronic pain seems to be present in the majority of persons with SCI, and severe pain is reported in a smaller proportion, perhaps 25%.⁵ In one attempt to define prevalence in a population-based sample of persons with SCI, Rintala and colleagues reported a prevalence figure of 75%, with 20% of their sample having to quit work because of pain and 40%–60% reporting sleep difficulties because of pain.⁶ By virtually any definition, chronic pain seems to be a frequent complication for persons with SCI, and it represents a severe problem for roughly a quarter of the population.

Impact of chronic pain

Data on the cost of treating chronic pain are lacking, so cost estimates have not been offered in the literature. In fact, until recently, there was not a great deal of attention paid to treating chronic pain in this population because it was generally believed that “nothing worked.” However there have been some hopeful developments in this regard with at least one surgical approach⁷ and systemic medications,⁸ particularly newer anti-seizure medications, showing some efficacy, primarily for neuropathic pain. Wear and tear on joints over the years secondary to transfers, pressure relief maneuvers, pushing manual chairs, and so on tends to produce musculoskeletal pain, which is usually easier to manage medically than neuropathic pain.⁹ More research work is needed, however, in terms of wheelchair design as well as the

financial, social, and psychological barriers to acceptance of power-assisted mobility as opposed to manual mobility to move the research closer to prevention rather than treatment of musculoskeletal pain.

The impact of chronic pain has been better demonstrated in terms of quality of life than costs. In a study at our center using data from the National Spinal Cord Injury Statistical Center, we were able to identify two groups of persons with SCI with and without pain who were closely matched on demographic and neurologic variables that might influence pain report. In this comparison, persons with pain reported significantly lower subjective ratings of quality of life, physical health, mobility, and social integration and higher levels of distress.⁴ Pain is frequently rated by persons with SCI as one of the most troublesome secondary conditions in terms of its impact on their lives.¹⁰

Trends in incidence/prevalence

Because methods for defining and asking about pain have varied so much over the years, as well as across researchers, it is difficult to determine whether there have been changes over time in the reported incidence and prevalence of this complication. However, it is interesting to speculate about changing etiology and pain. Although not consistent across researchers,⁴ some have reported that SCI caused by gunshot is associated with more neuropathic pain than other etiologies. There has been a relative increase in gunshot-caused SCI in the national SCI database¹¹ over the last 20 years, and, therefore, it is possible that we are seeing more persons with SCI presenting with this complication than was the case 20 years ago. Longitudinal studies, preferably population-based and with consistent methodology over

time, would be needed to determine whether this trend is verifiable. One trend that was recently noted in a cross-sectional sample of persons in the national SCI statistical database was decreasing interference of pain over time despite ratings of pain severity that remained constant over the same time frame.¹² This is encouraging in that it suggests that persons with SCI learn to tolerate pain better over time so that it has less of an impact.

Risk factors

Putzke and Richards⁴ recently completed a review of factors reported to be related to the report of pain in persons with SCI. Age (77%) and lesion level (61%) were found by different researchers to be associated with the presence of pain; but gender, completeness of lesion, etiology, and injury duration were not found to be consistently related to the report of pain. Again, differing definitions of pain make it difficult to generalize across studies. Injury duration may require a different examination. If one looks at the development of subtypes of SCI pain over time, some trends are emerging. There may be an initial “bolus” of musculoskeletal pain secondary to the initial injury itself: fractures, spinal instability, postsurgical pain, and the like. Much of this resolves over the first year. Musculoskeletal pain may increase over subsequent years secondary to the overuse syndrome described previously. Neuropathic pain may increase over time.¹³ More research is needed to understand the natural history of the development of SCI pain subtypes. If there are predictable patterns that portend when these subtypes of pain develop, more emphasis can be placed on clinical surveillance, early treatment, and possibly prevention.

Respiratory Complications

Incidence/prevalence

Secondary complications of the respiratory tract occur in 50% to 100% of persons during the acute postinjury period, and they rival urinary tract dysfunction in terms of severity and frequency, especially among persons with high cervical and/or complete injuries with tetraplegia and among the elderly.¹⁴ Conditions associated with SCI that contribute to increased respiratory dysfunction leading to specific complications such as pneumonia and respiratory failure involve mechanical problems resulting from loss of neural control mechanisms, respiratory muscle paralysis, impaired cough, decreased inspiratory capacity, hypoventilation, less effective mucociliary clearance of secretions, aspiration, apnea, exaggerated bronchospasm, atelectasis, and skeletal deformities of the spine and chest.¹⁴⁻²¹ Jackson and Groomes¹⁹ reported that 67% of persons hospitalized with recent SCI experienced 544 respiratory complications, with atelectasis being the most common (36.4%), followed by pneumonia (31.4%) and ventilatory failure (22.6%). Another recent study of persons hospitalized for acute traumatic cervical SCI showed that respiratory complications, specifically the requirement for mechanical ventilation, surgery, occurrence of pneumonia, or use of tracheostomy, were more important in determining length of hospitalization and hospital costs than the level of injury.

Impact of respiratory complications

Owing to advances in management of neurogenic bladder dysfunction, respiratory complications, most notably pneumonias, are now the most common cause of death in persons with SCI during both the acute and

chronic phases of SCI and in all age groups.^{14,22-24} Persons with SCI were 37 times more likely to die of pneumonia or influenza than comparable individuals in the general population, according to one study.²² The incidence of death in the SCI population from septicemia and pneumonia has also been shown to be substantially higher than in the general population,²⁵ and bacteremias that occur in persons with longstanding SCI are more likely to originate from the respiratory tract than from any other organ system.²⁶

Although pneumonias have been implicated as the most common cause of death following SCI, the frequency of respiratory failure not related directly to infection and its significance during the acute postinjury period cannot be overlooked because of its relatively long duration and associated costs, particularly when mechanical ventilation is required. Data from the National Spinal Cord Injury Statistical Center indicate that 3% of persons with SCI are ventilator dependent at time of hospital discharge and an additional 3% may require short-term ventilation post discharge.²⁷

Risk factors

Aggressive pulmonary management aimed at prevention of aspiration, hypoxemia, and atelectasis; maintenance of adequate alveolar ventilation; and clearing of secretions to circumvent deterioration into respiratory failure and development of bacterial pneumonia can improve pulmonary-related outcomes.¹⁴ Such interventions include aggressive pulmonary toileting, manually assisted coughs, and postural drainage supplemented with bronchodilators, mucolytics, and implementation of intubation and mechanically assisted ventilation as necessary.¹⁴ Most persons with injuries

above C3 will likely require long-term ventilatory assistance; in these individuals, insertion of a tracheostomy tube may be beneficial to reduce complications of nasotracheal intubation such as subglottic stenosis and sinusitis.¹⁴ A major dilemma faced by caregivers of persons with SCI who require intubation and mechanical ventilation is how to balance the need for maintenance of adequate alveolar ventilation and removal of secretions with the increased risk of nosocomial pneumonia coinciding with bypassing the innate mucociliary defense mechanisms of the lower respiratory tract.²⁷ Various noninvasive respiratory support systems such as electrophrenic diaphragmatic pacing, nasal apparatus, or mouthpieces to deliver positive airway pressure are now gaining increased attention as alternatives to traditional methods.^{14,27}

Thoracic trauma may occur simultaneously with SCI; under these circumstances, individuals may be at risk for other types of respiratory complications that include pneumothorax, hemothorax, and pleural effusion. Each of these conditions can further complicate the acute postinjury period and, depending on severity, can impact subsequent rehabilitation programs.

Another potentially serious and sometimes fatal respiratory complication resulting from immobility following SCI is pulmonary thromboembolism. Although the true incidence is unknown, studies from the 1960s indicated that pulmonary thromboembolism occurred in 5% to 13.2% of SCI patients, was a significant cause of death during the first 3 months following injury, and often occurred in the absence of recognizable manifestations of deep vein thromboses.²⁸⁻³¹ More recently, data from the National SCI Statistical Center showed that

pulmonary embolism occurred in 2.6% to 3.8% of patients during acute care or rehabilitation.³² This apparent reduction in occurrence over time may be due to more widespread use of low-molecular weight heparin for thromboprophylaxis, external compression stockings, and external compression devices to reduce the occurrence of deep vein thromboses.²⁷ It is important to note that 81% of persons with SCI who did not receive anti-thrombotic prophylaxis developed deep vein thromboses, which placed them at risk for pulmonary embolism, according to one study.³³ Clinical factors associated with the development of thromboembolism following SCI include a number of non-SCI variables such as advanced age, obesity, congestive heart failure, trauma to lower extremities, and prior history of thromboembolism that are operative in other populations.³¹ Treatment and prevention of thromboembolism after SCI has recently been reviewed in depth by Chen.³¹

Due to advances in intensive care and multidisciplinary team management, the occurrence of respiratory complications during the acute period following SCI has diminished somewhat over the past 25 years.²⁷ However, in aggregate, respiratory complications in various forms still contribute significantly to morbidity, mortality, and economic burden beyond the immediate postinjury period and extending throughout the lives of individuals with SCI because the pathophysiological states that favor their occurrence are mostly permanent.^{14,17-19,27,34} The progressive compromise of the respiratory system and its ability to counteract infections as part of the normal aging process, independent of SCI, are also reasons for concern as more persons with SCI have longer life expectancies.

Given the importance of respiratory complications as secondary disabilities following SCI, preventive strategies to reduce the occurrence of various conditions that can be due to infections with bacteria or viruses should be considered. Immunization with the 23-valent pneumococcal vaccine will elicit an antibody response in persons with SCI comparable to that of able-bodied persons, but it is not known precisely how long protective antibody concentrations will be maintained or whether there is a benefit from revaccination.^{35,36} Influenza vaccination has also been shown to be beneficial in preventing morbidity and mortality due to influenza and secondary bacterial pneumonias in high-risk populations such as the elderly as well as in otherwise healthy adults, yet both of these vaccines are underutilized in the SCI population.³⁵⁻³⁷ Patient education during acute hospitalization and rehabilitation that stresses the need for annual influenza vaccination and administration of the pneumococcal vaccine prior to discharge should be included in all care plans with the eventual goal of reducing the frequency of these common, potentially serious, and even deadly illnesses.

Urinary Tract Infection

Neurogenic bladder dysfunction associated with urinary stasis and use of various forms of catheter for bladder drainage predisposes persons with SCI to the development of urinary tract infection (UTI). Renal failure is no longer the leading cause of death,³⁸ but UTI remains one of the most common secondary complications in persons with SCI.³⁹⁻⁴¹

An understanding of the epidemiology of UTI in the SCI population is hampered by the inconsistent definition of bacteriuria and

UTI in the medical literature. A consensus conference sponsored by the National Institute on Disability and Rehabilitation Research (NIDRR) in 1992 defines significant bacteriuria in persons with SCI as (a) $\geq 10^2$ colony forming units (cfu) of uropathogens per milliliter (mL) of urine in catheter specimens, (b) $\geq 10^4$ cfu/mL in clean-void specimens, and (c) any detectable concentration of uropathogens in indwelling catheter or suprapubic aspirate specimens.⁴² Most published studies have used colony count $\geq 10^5$ cfu/mL as the criterion for significant bacteriuria. Others simply obtained data from patients' self-reports.

The diagnosis of UTI in persons with SCI is complicated by the poor sensitivity and specificity of clinical symptoms and signs.^{43,44} Dysuria, frequency, and urgency that are usually observed in able-bodied patients with UTI are often absent. Common manifestations of UTI in persons with SCI include fever, increasing spasticity, autonomic dysreflexia, urinary leakage, suprapubic and flank pain or tenderness, cloudy and odorous urine, and malaise or lethargy. Pyuria, leukocytes in the urine, is an excellent indicator of UTI in able-bodied patients but has low specificity in persons with SCI because of the irritative effect of the catheter on the bladder wall.^{44,45}

Several articles recently provided a systematic review on pathogenesis, risk factors, prevention, and treatment of UTI in persons with SCI.⁴³⁻⁴⁹ We summarize epidemiologic aspects of these articles and review important findings from the recent literature.

Incidence and prevalence

For community-residing persons with SCI managed with either intermittent or condom catheterization, Waites et al. demonstrated

that the incidence of significant bacteriuria, defined as $\geq 10^5$ cfu/mL, was 18.4 episodes per person-year whereas more severe bacteriuria associated with fever was 1.8 episodes per person-year.⁵⁰ During initial hospital care, the overall asymptomatic bacteriuria rate was reported to be 2.7 episodes per 100 patient-days (about 10 episodes per patient-year) and symptomatic UTI was 0.68 per 100 patient-days (about 2.5 episodes per patient-year).⁵¹ The overall prevalence of bacteriuria/UTI at any given time point ranges from 20% to 57%.^{39–41,50}

Risk factors

As expected, the risk of bacteriuria/UTI after SCI is increased by structural and physiological abnormality, including vesicoureteral reflux, high intravesicle pressure, increased postvoid residual urine, urinary stones, and detrusor-sphincter dyssynergia.^{45,48} The association of sociodemographic and injury characteristics with the presence of bacteriuria/UTI is somewhat inconsistent. In general, the bacteriuria/UTI risk appears to be greater for women, African Americans, the elderly, and persons with cervical injuries, neurologically complete lesions, and poor personal hygiene.^{41,50–52} Personal income, education level, and years since injury seem not to be important. The role of other psychosocial factors on UTI such as self-esteem, adjustment to disability, social support, and access to services is less well understood.⁴⁸

The most important risk factor for UTI among both inpatients and outpatients with SCI is the method of bladder drainage.^{45,50,51} The indwelling urethral catheter presents the greatest risk of bacteriuria/UTI. The risk of the development of bacteriuria/UTI is similar for intermittent and condom catheteriza-

tion, which is higher than for suprapubic catheters. Daily changing of the condom catheter is shown to decrease the incidence of bacteriuria/UTI in community-residing persons with SCI.

Microbiological characteristics

Unlike persons with a functionally normal urinary tract, UTI associated with neurogenic bladder dysfunction is caused by a much wider variety of organisms and is often polymicrobial and resistant to multiple antimicrobial agents.⁵³ The types of bacteria present in urine vary by a person's gender, age, and method of bladder drainage.^{53,54} Waites et al. demonstrated that *Escherichia coli* was the most prevalent organism isolated from women, whereas *E. coli*, *Pseudomonas aeruginosa*, *Klebsiella pneumoniae*, and *Proteus mirabilis* were the most frequently isolated bacteria, with no single organism predominant, from men. *E. coli* was dominant in intermittent catheter and catheter-free groups, whereas *E. coli* and *P. aeruginosa* share similar frequency in indwelling and condom catheter groups.⁵³

Polymicrobial bacteriuria is more common in condom and indwelling catheter groups than in intermittent catheter and catheter-free groups.⁵³ Men present a greater risk of polymicrobial bacteriuria than women, which is well explained by the method of urinary drainage.⁵³ The occurrence of multi-drug-resistance bacteria is higher for men, the elderly, and users of condom and indwelling catheters.⁵³

Morbidity

UTI has a well-documented significant negative impact on morbidity and associated quality of life. UTI is the most common source of blood stream infection in SCI out-

patients who require rehospitalization.⁵⁴ UTI can lead to secondary urologic complications including bladder and kidney stones, vesicoureteral reflux, and renal or perinephric abscess.⁴³ Liguori et al.⁵⁵ indicated that fewer UTIs were related to more hours of work per week and a higher level of functioning. Self-reported UTI occurrence has also been associated with lower outdoor activity participation.⁵⁶

Impact

Despite the improvement in medical care, bladder management, catheter materials, and patient education, UTI remains a major source of morbidity and mortality in persons with SCI. Given the potential adverse impact of UTI on personal health and societal costs, research into various strategies for primary and secondary prevention needs to continue. In particular, the field would benefit from knowledge as to which subgroup of persons with SCI would benefit from a particular intervention (gender differences, upper vs. lower motor neuron bladder, etc.) so that clinicians can be more targeted in their assessments and interventions.

Pressure Sores

Incidence/prevalence

A pressure ulcer or pressure sore can be defined as a lesion on any skin surface that results from pressure, shear, or friction.⁵⁷ Pressure sores are an underestimated health care problem for individuals with SCI and often necessitate extended hospitalization with accompanying high costs.^{58,59} McKinley and colleagues⁶⁰ found that pressure sores are the most frequent secondary medical complication in all years of follow-up for persons with SCI. Estimates of the

incidence and prevalence of pressure sores vary greatly. However, most studies suggest an incidence rate of at least 30% for persons with SCI within the first 5 years of injury.^{58,59,61,62} Byrne and Salzberg⁵⁸ estimated an annual incidence of pressure sores of the 200,000 SCI patients in the United States to be 23%, or 46,000 individuals. The prevalence of pressure sores seems to be related to level of injury and neurologic impairment, with 18.8% of patients with incomplete paraplegia reporting pressure sores during their acute care and 52% of patients with complete tetraplegia reporting pressure sores.⁶³ Pressure sores may be graded as one of four grades, with four the most severe and one the least destructive in terms of tissue damage.⁶⁰ Almost one half of pressure sores during rehabilitation are grade one, and grades three and four account for only 8.6% of all ulcers.⁶³

Unfortunately, the percentage of individuals with SCI who develop pressure sores seems to increase with postinjury years; 15% of 1-year postinjury individuals report an existing pressure sore and almost 30% of 25-years postinjury individuals report a current pressure sore.⁶³ Stover et al.⁶⁴ suggested that despite advances in patient care for SCI and attempts to minimize pressure sores the rate of pressure sore development remained constant across the then 20-year history of SCI Model Systems data collection for those admitted within 24 hours of injury. For example, the rate during years 1976–1979 was 34.7%, while the rate during 1989–1992 was 30.0%.

Impact of pressure sores

The impact of pressure sores on the spinal cord-injured individual, family, and the community cannot be overestimated. The

impact can be measured in health-related, financial, and emotional consequences. Each year in the United States, pressure ulcer-related complications related to any medical condition cause 60,000 deaths.⁵⁸ Furthermore, among individuals with SCIs, an estimated 7%–8% will die of these complications. Many individuals with SCI will never endure a life-threatening pressure sore, but “less severe” pressure sores remain a significant threat to their overall well-being. Jones et al.⁶² suggest that skin-related complications are the leading cause of failure to achieve educational, vocational, and social goals. Other authors agree and propose that pressure sores can impair an individual’s functional ability and impede employment and educational pursuits.⁶⁰ The financial cost of treating a highly preventable condition such as pressure ulcers is a significant burden to persons with SCI and to the community. Pressure sores account for approximately 25% of the overall cost of treating people with an SCI.⁶¹ Estimates of the total annual cost to the US health care system of treating pressure sores in SCI patients have ranged from \$1.2 billion^{61,62} to between \$2 and \$5 billion.⁵⁸ At least one author hypothesizes that prevention may cost less than one tenth of that figure.⁶² Although cost estimates vary, there is no doubt that the prevention of pressure sores is a far less expensive endeavor than the treatment.

Risk factors

Numerous risk factors have been identified in the development of pressure sores, but the usefulness of this information may be limited by several factors. First, in one of the more comprehensive reviews of risk factors for pressure sore development, Byrne and Salzberg⁵⁸ found that more than 200 risk

factors have been identified and described in the published literature, which makes it an overwhelming, if not impossible, task for most health care professionals to effectively evaluate and incorporate this knowledge into clinical practice. Second, these authors suggest that the usefulness of research on persons with SCI is frequently limited by an inadequate sample size. For an excellent review of these risk factors, the reader is encouraged to refer to Byrne and Salzberg’s work⁵⁸; however, these factors will be summarized briefly. Pressure ulcer risk factors can be classified into one of several broad categories, including (a) severity of injury, (b) preexisting conditions, (c) psychological factors, (d) medication factors, (e) movement factors, (f) physical factors, and (g) nutritional factors. There is a considerable literature within each of these categories; some of the most significant of these findings are reported in the following discussion.

In one study, Salzberg et al.⁵⁷ looked at comorbidities of pressure sores and found that diabetes and renal disease were twice as prevalent in the pressure sore group compared with a group of persons with SCI without pressure sores. Other risk factors identified in this study included smoking, pulmonary disease, and malnutrition. Another study sought to develop a profile of the person who develops a pressure sore and concluded that those with a high risk of developing a severe pressure sore (a) would be male in 75% of the cases; (b) would have a lower level of education; (c) would not practice standing; (d) would have no spasticity; (e) would be older than 40 years; and (f) would present with other pathologies, such as alcoholism, psychological disorders, and malnutrition.⁵⁹ McKinley et al.⁶⁰ propose that time post injury is a better predictor for

pressure sore development than the age of the individual. These authors also found comorbidities with pressure sores at the first annual exam that included pneumonia, deep vein thrombosis, and autonomic dysreflexia. In addition, they concluded that the percentage of males presenting with one or more pressure sores was significantly greater than that of females at annual years 1 and 2, and there was a trend in all years for men to report more pressure sores in their annual exams. Last, these authors suggest from clinical experience that individuals who sustained SCI from an act of violence generally developed pressure sores more frequently than did individuals with different etiologies of their injury.

Preventing/treating pressure sores

As with most medical problems that have largely behavioral antecedents, “an ounce of prevention is worth a pound of cure” with pressure sores. Unfortunately, even the best efforts of the individual with SCI and their family members will, at times, fail to prevent pressure sores. Preventive strategies are well known. Proper bed positioning and frequent turning are essential.⁶⁴ Properly fitted wheelchair cushions are required. Those who are physically able should do a routine “push-up” in which they relieve pressure on the sacral area by hoisting themselves for several seconds from the surface on which they are sitting or lying. Family members are instructed to help with these maneuvers if needed.

One challenge in the prevention of pressure sores is that of compliance. For many persons with SCI, pressure sores are not physically painful, and there may be no financial or other consequences inherent in

treating them.⁶² Thus, there is little incentive for the patient to engage in recommended preventive strategies. A behavioral approach that has yielded increased compliance rates (and, consequently, lower rates of pressure sores) is an intervention that includes a health plan, clinic visits, and financial rewards.⁶² In this study, the authors were able to achieve a reduction in the severity of pressure sores with individuals who had a history of high recidivism rates for pressure sores and a history of behavioral and psychological factors that had previously interfered with their ability to manage their skin care.

Depression

Incidence/prevalence

Estimates of the prevalence and incidence rates of depression in individuals with SCI vary greatly, with estimates ranging from 15% to 40%. It is often difficult to accurately compare the results across studies because of differences in the methods that have been used to identify depression and depressive behavior. Some researchers elect to investigate depressive behavior as assessed by self-report symptom checklists, whereas others have used interviews to diagnose major depressive disorder (MDD). A recent investigation using data collected through the SCI Model Systems found that 22% of individuals had probable MDD 1 year after injury (J.S. Richards, personal communication).

Research has shown that depressed behavior in persons with SCI is associated with poor self-ratings of health status⁶⁵ and increased physical secondary conditions such as pressure ulcers and UTI.⁶⁶ In addition, self-reported depression has been associated with higher medical care and personal assistance costs.⁶⁷

Impact

The mortality rate for individuals with SCI who are depressed is five times greater than that for persons of the same age and gender in the general US population.⁶⁸ The risk of suicide appears to be associated with time since injury, with individuals who are 1 to 5 years post injury displaying the highest suicide rates. Suicide risk in individuals with SCI may be higher than that of the general population; individual characteristics that place a person who is depressed at risk for suicide, such as male gender, lower levels of social support, and history of substance abuse, are overrepresented in the SCI population.

For many years, it was thought that there were universal reactions to SCI. Adjustment to disability was believed to be a progression through a predictable sequence of stages. Depression was expected to occur as a person passed through the stages of adjustment to SCI. The lack of a “depressed” stage was viewed as an indication that the person had not truly accepted the nature and finality of their injury, whereas the presence of a “depressed” stage was viewed as an indicator of healthy adjustment to SCI.^{69,70} These stage models of adjustment following SCI are not supported by research, and depression following SCI does not predict healthy adjustment to SCI.⁷¹

Risk factors

Injury characteristics do not reliably predict the incidence of MDD in persons with SCI. Rather, individual differences and personality variables have been found to predict adjustment to SCI better than any characteristic of the injury itself. Lower levels of distress have been reported by individuals with an internal locus of control compared to

those with external locus of control.⁷² In addition, rehabilitation patients who display problem-focused coping styles and seek social support to cope with problems report less depression and more positive impressions of rehabilitation progress than patients who display emotion-focused coping styles.^{73–75} A history of depression, chronic pain, lack of social support, and life stressors are all risk factors for depression following SCI.^{65,75}

Discussion

It is interesting to compare across secondary conditions for common risk factors. It would appear, for example, that increasing age is associated with increasing frequency of pressure sores, UTIs, respiratory complications, and chronic pain and that persons with tetraplegia, as opposed to those with lower level lesions, are more likely to incur those same complications. Time post injury predicts the development of both pressure sores and chronic pain. Gender, level of education, and race are not strong risk factors across secondary conditions; although for specific conditions, clear relationships exist (males are more likely to develop pressure sores and females are more likely to develop UTIs). For other conditions such as depression and to some extent chronic pain, the literature is either silent or inconsistent with regard to risk factors. Patients with tetraplegia and patients of advancing age seem to be at generally higher risk for the development of secondary conditions either because of prolonged exposure to the conditions that predispose to the development of those conditions or because of compromises in physiology that can accompany aging. Increased vigilance is, therefore, in order for patients with tetraplegia, particularly as they age,

because it appears that they are at greater risk for these debilitating complications.

The variables used as risk factors by many researchers, and those summarized here, tend to be the most objective and readily achieved: demographics, neurologic characteristics, and time measures. It is a just criticism that these variables may be several steps removed from any causal relationship to secondary conditions and really serve as "marker" variables. For example, in the case of depression, if increasing time post injury were found to be related to an increased frequency of depression, there is no information inherent in the variable of time to help explain why depression risk might increase, much less to provide implications for prevention or treatment. Risk factors that might be much more closely associated with the development of secondary conditions would include availability of social support over time, changes in ability to pursue pleasurable activities, and the like. However, it is much more difficult and costly to gather information about those factors. If such studies were done, however, they would likely produce risk models that could better inform surveillance and treatment decisions.

The prevention of secondary conditions remains the most difficult problem of all. We

do, in fact, have a great deal of information about prevention; persons with SCI and their caregivers are given that information in myriad ways at all rehabilitation centers. For some, if not most, that information is sufficient to ensure compliance and health. For others, information alone is not sufficient. It may be impossible to prevent all secondary conditions all the time, even with the best self-care, but it is frustrating to all clinicians and ultimately demoralizing if not devastating to the person with SCI that they are not more successful (e.g., in the case of pressure sores). With shrinking inpatient rehabilitation stays, it will become more and more difficult to provide all the information, much less make the behavior changes necessary, for optimal self-care and minimal development of secondary conditions. Therefore, we as clinicians and researchers will need to develop ways to reach beyond the hospital to institute novel programs like that of Jones and colleagues⁶² to help our clients maintain optimal health post injury.

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REFERENCES

1. Vines CL. *Identifying Secondary Conditions in Arkansas with Spinal Cord Injuries*. Little Rock, AR: Arkansas Spinal Cord Commission; 1996.
2. Bryce TN, Ragnarsson KT. Epidemiology and classification of pain after spinal cord injury. *Top Spinal Cord Inj Rehabil*. 2001;7(2):1-17.
3. Siddall PJ, Yezierski RP, Loeser JD. Pain following spinal cord injury: clinical features, prevalence and taxonomy. *IASP Newsletter*. 2000;3:3-7.
4. Putzke JD, Richards JS. Pain after spinal cord injury: important predictors, outcomes, and future directions in cost-effectiveness analysis. *Top Spinal Cord Inj Rehabil*. 2001;7(2):84-92.
5. Nepomuceno C, Fine PR, Richards JS. Pain in patients with spinal cord injury. *Arch Phys*

- Med Rehabil.* 1979;60:605–609.
6. Rintala DH, Loubser PG, Castro J, Hart KA, Fuhrer MJ. Chronic pain in a community-based sample of men with spinal cord injury: prevalence, severity, and relationship with impairment, disability, handicap, and subjective well-being. *Arch Phys Med Rehabil.* 1998;79:604–614.
 7. Lammertse DP, Falci SP. Surgical management of central pain in persons with spinal cord injury: the Dorsal Root Entry Zone (DREZ) procedure. *Top Spinal Cord Inj Rehabil.* 2001;7(2):41–50.
 8. Kezar LB, Ness TJ. Systemic medications. *Top Spinal Cord Inj Rehabil.* 2001;7(2):57–72.
 9. Apple, D. Pain above the injury level. *Top Spinal Cord Inj Rehabil.* 2001;7(2):18–29.
 10. Jackson AJ, Richards JS, Mott P. *Secondary Conditions of Spinal Cord Injury: The State of the Science [proceedings]*. Birmingham, AL: Rehabilitation Research Training Center, University of Alabama at Birmingham; 2003.
 11. McKinley WO, Jackson AJ, Cardenas DD, DeVivo MJ. Long-term complications after traumatic spinal cord injury: a regional Model Systems analysis. *Arch Phys Med Rehabil.* 1999;80:1402–1410.
 12. Cardenas DD, Bryce T, Shem K, Elhefni H, Richards JS. Gender/minority differences in the pain experience of persons with spinal cord injury. Submitted for publication.
 13. Siddall PJ, Taylor DA, McClelland JM, Rutkowski SB, Cousins MJ. Pain report and the relationship of pain to physical factors in the first 6 months following spinal cord injury. *Pain.* 1999;81:187–197.
 14. Lanig S, Peterson W. The respiratory system in spinal cord injury. *Phys Med Rehabil Clin North Am.* 2000;11:29–43.
 15. Bellamy R, Pitts FW, et al. Respiratory complications in traumatic quadriplegia. *J Neurosurg.* 1973;39:596–600.
 16. Niederman MS. Strategies for the prevention of pneumonia. *Clin Chest Med.* 1987;21:193–196.
 17. Reimes HD, Harris RC. Pulmonary complications of acute spinal cord injuries. *Neurosurgery.* 1987;21:193–196.
 18. Fishburn MJ, Marino RJ, et al. Atelectasis and pneumonia in acute spinal cord injury. *Arch Phys Med Rehabil.* 1990;71:197–200.
 19. Jackson AB, Groomes TE. Incidence of respiratory complications following spinal cord injury. *Arch Phys Med Rehabil.* 1994;75(3):270–275.
 20. Viroslav J, Rothblatt R, et al. Respiratory management, survival, and quality of life for high-level traumatic tetraplegics. *Respir Care Clin North Am.* 1996;2:313–322.
 21. Lucke KT. Pulmonary management following acute SCI. *J Neurosci Nurs.* 1998;30:91–104.
 22. DeVivo M, Black K, et al. Causes of death during the first 12 years after spinal cord injury. *Arch Phys Med Rehabil.* 1993;74:248–254.
 23. Krause J, Sternberg M, et al. Mortality after spinal cord injury: a 11-year prospective study. *Arch Phys Med Rehabil.* 1997;78:815–821.
 24. DeVivo M, Krause J, et al. Recent trends in mortality and cause of death among persons with spinal cord injury. *Arch Phys Med Rehabil.* 1999;80:1.
 25. Soden RJ, Walsh J, et al. Causes of death after spinal cord injury. *Spinal Cord.* 2000;38:604–610.
 26. Waites KB, Canupp KC, et al. Bacteremia after spinal cord injury in initial versus subsequent hospitalizations. *J Spinal Cord Med.* 2001;24(2):96–100.
 27. Winslow C, Rozovsky J. Effect of spinal cord injury on the respiratory system. *Am J Phys Med Rehabil.* 2003;82(10):803–814.
 28. Tribe CR. Causes of death in early and late stages in paraplegia. *Paraplegia.* 1963;1:19–47.
 29. Walsh JJ, Tribe CR. Phlebo-thrombosis and pulmonary embolism in paraplegia. *Paraplegia.* 1965;3:209–213.
 30. Watson N. Anticoagulation therapy in prevention of venous thrombosis and pulmonary embolism in spinal cord injury. *Paraplegia.* 1968;6:113–121.
 31. Chen D. Treatment and prevention of thromboembolism after spinal cord injury. *Top Spinal Cord Inj Rehabil.* 2003;9:14–25.
 32. Chen D, Apple DF Jr, et al. Medical complications during acute rehabilitation following spinal cord injury—current experience of the Model Systems. *Arch Phys Med Rehabil.* 1999;80(11):1397–1401.
 33. Geerts WH, Code KI, et al. A prospective study of venous thromboembolism after major trauma. *N Engl J Med.* 1994;331(24):1601–1606.
 34. Winslow C, Bode RK, et al. Impact of respiratory complications on length of stay and hospital costs in acute cervical spine injury. *Chest.* 2002;121(5):1548–1554.
 35. Darouiche RO, Groover J, et al. Pneumococcal vaccination for patients with spinal cord

- injury. *Arch Phys Med Rehabil.* 1993;74(12):1354–1357.
36. Waites KB, Canupp KC, et al. Immunogenicity of pneumococcal vaccine in persons with spinal cord injury. *Arch Phys Med Rehabil.* 1998;79(12):1504–1509.
 37. Evans CT, Legro MW, et al. Influenza vaccination among veterans with spinal cord injury: part I. A survey of attitudes and behavior. *J Spinal Cord Med.* 2003;26:204–209.
 38. DeVivo MJ, Krause JS, Lammertse DP. Recent trends in mortality and causes of death among persons with spinal cord injury. *Arch Phys Med Rehabil.* 1999;80:1411–1419.
 39. Anson CA, Shepherd C. Incidence of secondary complications in spinal cord injury. *Int J Rehabil Res.* 1996;19:55–66.
 40. Whiteneck GG, Charlifue SW, Frankel HL, et al. Mortality, morbidity, and psychosocial outcomes of persons spinal cord injured more than 20 years ago. *Paraplegia.* 1992;30:617–630.
 41. Noreau L, Proulx P, Gagnon L, Drolet M, Laramee MT. Secondary impairments after spinal cord injury: a population-based study. *Am J Phys Med Rehabil.* 2000;79:526–535.
 42. The prevention and management of urinary tract infections among people with spinal cord injuries: National Institute on Disability and Rehabilitation Research Consensus Statement: January 27–29, 1992. *J Am Paraplegia Soc.* 1992;15:194–204.
 43. Stover SL, Lloyd LK, Waites KB, Jackson AB. Urinary tract infection in spinal cord injury. *Arch Phys Med Rehabil.* 1989;70:47–54.
 44. Cardenas DD, Hooton TM. Urinary tract infection in persons with spinal cord injury. *Arch Phys Med Rehabil.* 1995;76:272–280.
 45. Siroky MB. Pathogenesis of bacteriuria and infection in the spinal cord injured patient. *Am J Med.* 2002;113(1A):67S–79S.
 46. Trautner BW, Darouiche RO. Prevention of urinary tract infection in patients with spinal cord injury. *J Spinal Cord Med.* 2002;25:277–283.
 47. Morton SC, Shekelle PG, Adams JL, et al. Antimicrobial prophylaxis for urinary tract infection in persons with spinal cord dysfunction. *Arch Phys Med Rehabil.* 2002;83:129–138.
 48. Shekelle PG, Morton SC, Adams JL, Clark KA, Pathak M, Vickrey BG. Systematic review of risk factors for urinary tract infection in adults with spinal cord dysfunction. *J Spinal Cord Med.* 1999;22:258–272.
 49. Galloway A. Prevention of urinary tract infection in patients with spinal cord injury – a microbiological review. *Spinal Cord.* 1997;35:198–204.
 50. Waites KB, Canupp KC, DeVivo MJ. Epidemiology and risk factors for urinary tract infection following spinal cord injury. *Arch Phys Med Rehabil.* 1993;74:691–695.
 51. Esclarin De Ruz A, Garcia Leoni E, Herruzo Cabrera R. Epidemiology and risk factors for urinary tract infection in patients with spinal cord injury. *J Urol.* 2000;164:1285–1289.
 52. 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.
 53. Waites K, Chen Y, DeVivo MJ, Canupp KC, Moser SA. Antimicrobial resistance in gram-negative bacteria isolated from the urinary tract in community-residing persons with spinal cord injury. *Arch Phys Med Rehabil.* 2000;81:764–769.
 54. Waites KB, Canupp KC, Chen Y, DeVivo MJ, Moser SA. Bacteremia after spinal cord injury in initial versus subsequent hospitalizations. *J Spinal Cord Med.* 2001;24:96–100.
 55. Liguori PA, Cardenas DD, Ullrich P. Social and functional variables associated with urinary tract infections in persons with spinal cord injury. *Arch Phys Med Rehabil.* 1997;78:156–160.
 56. Sapountzi-Krepia D, Soumilas A, Papadakis N, et al. Post traumatic paraplegics living in Athens: the impact of pressure sores and UTIs on everyday life activities. *Spinal Cord.* 1998;36:432–437.
 57. Salzberg CA, Byrne DW, Cayten CG, van Niewerburgh P, Murphy JG, Viehbeck M. A new pressure ulcer risk assessment scale for individuals with spinal cord injury. *Am J Phys Med Rehabil.* 1996;75:96–104.
 58. Byrne DW, Salzberg CA. Major risk factors for pressure ulcers in the spinal cord disabled: a literature review. *Spinal Cord.* 1996;34:255–263.
 59. Vidal J, Sarrias M. An analysis of the diverse factors concerned with the development of pressure sores in spinal cord injured patients. *Paraplegia.* 1991;29:261–267.
 60. McKinley WO, Jackson AB, Cardenas DD, DeVivo MJ. Long-term medical complications after traumatic spinal cord injury: a regional

- model systems analysis. *Arch Phys Med Rehabil.* 1999;80:1402–1410.
61. Krause JS, Vines CL, Farley TL, Sniezek J, Coker J. An exploratory study of pressure ulcers after spinal cord injury: relationship to protective behaviors and risk factors. *Arch Phys Med Rehabil.* 2001;82:107–113.
 62. Jones ML, Mathewson CS, Adkins VK, Ayllon T. Use of behavioral contingencies to promote prevention of recurrent pressure ulcers. *Arch Phys Med Rehabil.* 2003;84:1–7.
 63. National Spinal Cord Injury Statistical Center. *Annual Report for the Model Spinal Cord Injury Care Systems.* Birmingham, AL: Author; 2003:86–91.
 64. Stover SL, DeLisa JA, Whiteneck GG. *Spinal Cord Injury: Clinical Outcomes from the Model Systems.* Gaithersburg, MD: Aspen Publishers; 1995.
 65. Schulz R, Decker S. Long-term adjustment to physical disability: the role of social support, perceived control, and self-blame. *J Personality Social Psychol.* 1985;48:1162–1172.
 66. Herrick SM, Elliott TR, Crow F. Social support and the prediction of health complications among persons with spinal cord injuries. *Rehabil Psychol.* 1994;39:231–250.
 67. Tate D, Forchheimer M, Maynard F, Dijkers M. Predicting depression and psychological distress in persons with spinal cord injury based on indicators of handicap. *Am J Phys Med Rehabil.* 1994;73:175–183.
 68. DeVivo MJ, Black KJ, Richards S, Stover SL. Suicide following spinal cord injury. *Paraplegia.* 1991;29:620–627.
 69. Frank RG, Elliott T, Corcoran J, Wonderlich SA. Depression after spinal cord injury: Is it necessary? *Clin Psychol Rev.* 1987;7:611–630.
 70. Frank RG, Van Valin P, Elliott T. Adjustment to spinal cord injury: a review of empirical and nonempirical studies. *J Rehabil.* 1987;53:43–48.
 71. Elliott TR, Frank RG. Depression following spinal cord injury. *Arch Phys Med Rehabil.* 1996;77:816–823.
 72. Frank RG, Umlauf RL, Wonderlich SA, Ashkanzi G, Buckelew SA, Elliott T. Coping differences among persons with spinal cord injury: a cluster analytic approach. *J Consult Clin Psychol.* 1987;55:727–731.
 73. Moore AD, Bombardier CH, Brown PB, Patterson DR. Coping and emotional attributions following spinal cord injury. *Int J Rehabil Res.* 1994;17:39–48.
 74. Elliott TR. Social problem-solving abilities and adjustment to recent-onset spinal cord injury. *Rehabil Psychol.* 1999;44:315–332.
 75. Li L, Moore D. Acceptance of disability and its correlates. *J Social Psychol.* 1998;138:13–25.