spinal cord injury UPDATE

Department of Rehabilitation Medicine

Volume 14, Issue 3 • Fall 2005

INSIDE:

UW Medicine
SCHOOL OF MEDICINE

Disaster & Disability

Emergency preparedness resources

With images of Hurricane Katrina fresh in our minds, this is a good time to make a personal disaster plan. Here are some resources that address the unique concerns of people with disabilities:

Prepare.org, an American Red Cross Web site to help the public prepare for natural and human-caused disasters, with a special emphasis on vulnerable populations. Go to: *www.prepare.org*.

• ***Disaster Preparedness for People with Disabilities*** is a step-by-step guide to getting ready and gathering supplies. Read or download the 48-page booklet at *www.prepare.org/disabilities/disabilitiesprep.htm*, or call your local Red Cross chapter (below) for a copy.

American Red Cross.

- National headquarters, 2025 E Street, NW, Washington, DC 20006; 202-303-4498; *www.redcross.org*.
- King and Kitsap Counties office: 206-323-2345.
- Find a local chapter: *www.redcross.org/where/chapts.asp*.

City of Seattle Emergency Management, helpful information about preparing for and responding to disasters for Seattle residents. 206-233-5076; *sem@seattle.gov; www.cityofseattle.net/emergency_mgt/*.

READYAmerica, U.S. Department of Homeland Security, has information about preparing for all kinds of disasters and publishes a 16-page booklet ***Preparing Makes Sense: Get Ready Now***. Call 800-237-3239 or download from *www.ready.gov*.

SEE "PREPAREDNESS BASICS" ON PAGE 2

UW emergency medical team responds to Katrina

On August 29, Dr. Kathleen Jobe, director of the UW Medical Center emergency medicine department, arrived at the New Orleans airport for a two-week deployment with one of Washington State's disaster medical assistance teams (DMATs). Patients were brought in by helicopter, bus and ambulance. Jobe's team, along with other DMATs, swiftly treated, stabilized and evacuated over 2,700 patients in the first four days of the operation. Most were critically ill due to lack of regular medication or dialysis.

"Many of our patients were disabled, in chairs, or had other mobility problems," Jobe recalled. "My advice (to people with

SEE "RESPONSE TO KATRINA" ON PAGE 2

Dr. Kathleen Jobe sits with patients being transported from the helicopter landing area of the New Orleans airport to the terminal's upper level for triage and treatment by her DMAT (disaster medical assistant team) unit.

Response to Katrina

CONTINUED FROM PAGE 1

disabilities) would be to evacuate early if you have warning. Be sure you have an emergency kit which includes prescription medications, an outline of your medical history and a list of your medications. Also some food and water. Be prepared to be without help for up to 72 hours."

"Military air evacuations cannot take wheelchairs, including nice custom ones," she added. "People were reluctant to leave without their chairs, but eventually had to, via stretcher." But since each chair has a unique number that links it to the owner, all the chairs were returned to their owners by commercial or cargo aircraft. "It's best to evacuate, even without your chair," she advised. "It will find you."

Have food, water and prescription drugs in your disaster kit. Be prepared to be without help for up to 72 hours.

Preparedness basics:

- Create a personal support network of people who will help you in an emergency. Include at least three people at each location where you regularly spend time (home, work, school).
- Make a family and support group communication plan.
- Complete a detailed personal assessment of your capabilities, limitations and assistance requirements.
- Compile a portable, accessible disaster kit that includes a minimum three-day supply of food and water and a two-week supply of medications, personal and medical supplies, and anything related to your disability.
- Prepare an emergency kit for your service animal or pet.

Pressure Ulcer Treatment: *A comprehensive rehabilitation approach*

Most people with SCI are well aware that they have a high lifetime risk of developing pressure ulcers and try to avoid them by following good skin care practices. Yet pressure ulcers continue to be one of the most frequent medical complications of SCI, one which is costly, disabling and potentially life-threatening.

The Model SCI System Statistical Center recently reported that the risk of getting a pressure ulcer is about 15% in the first year after injury and increases steadily thereafter, to about 27% at 25 years postinjury.[1] The recurrence rate is even higher: between 40% and 80% of patients who have had a pressure ulcer develop another one.[2] At any given time, an estimated 17% to 39% of the SCI population suffers from a pressure ulcer.[3]

Pressure ulcers that don't heal with medical (also called conservative) treatment—pressure reduction, wound care, good nutrition—may require myocutaneous flap surgery, which involves covering the wound with a flap of connected skin and muscle tissue and requires a lengthy hospital stay.

What do patients and their providers need to know in order to prevent pressure ulcers, or at least reduce their severity and recurrence?

Pressure ulcer treatment at Harborview

Pressure ulcers do not usually have a single cause but are due to a complex combination of medical, behavioral and lifestyle factors. For this reason, treatment of open ulcers and the prevention of future breakdown should ideally address all these issues. At the University of Washington's Harborview Medical Center, pressure ulcers are managed collaboratively by the departments of rehabilitation medicine and plastic surgery, using a comprehensive rehabilitation protocol that addresses wound healing, seating, mobility, nutrition, exercise, co-morbid conditions, activities of daily living, habits (such as smoking and drinking), and psychosocial issues. If the wound involves underlying bone infection, the infectious disease department may also assist in management.

Harborview receives referrals from all over the region for treatment of pressure ulcers that have not healed with conservative treatment. Many of these referrals are for wounds that could have been avoided or treated earlier with non-surgical methods, according to Dr. Barry Goldstein, UW associate professor of rehabilitation medicine. He and Dr. Loren Engrav, UW professor of plastic surgery, co-direct Harborview's outpatient pressure ulcer treatment program. "There are many misconceptions about the prevention and treatment of pressure ulcers," Goldstein said. "From the referrals that we get, we see that there is a lack of knowledge on the part of many primary care practitioners and surgeons in the community."

"Getting a pressure ulcer can be so life-shattering," said Rosemary Buchmeier, RN, a nurse in Harborview's rehab clinic. "Patients are forced to put their lives on hold for six months to a year while a severe pressure ulcer is healing."

"Often people come to us when they have already failed elsewhere," Buchmeier added. "Typically, the patient had surgery at another facility for a severe wound and was sent home. But the pressure ulcer recurred, and now they're considered too complex for the original facility to handle. So they come to us."

Patients who come to Harborview with a pressure ulcer enter a protocol designed to put them in the best possible condition to heal the ulcer, undergo surgery and help prevent recurrences after they return home. If adherence to the protocol heals the wound without surgery, all the better. Research suggests that a protocol such as this may reduce

CONTINUED ON PAGE 3

Pressure ulcers

CONTINUED FROM PAGE 2

pressure ulcer recurrence following surgery.[2]

"Dr. Engrav wants to change the attitude from 'the patient is coming for flap surgery' to 'the patient needs comprehensive rehabilitation care that might include surgery,'" said Cathy Miller, RN, who works alongside Buchmeier in Harborview's rehab clinic. "We take a functional and realistic look at the life of the person," assessing all the factors (medical, behavioral, equipment, lifestyle) that contribute to pressure ulcers and helping the patient make the necessary changes.

If the wound does not heal after following the protocol, the patient is evaluated and, if appropriate, scheduled for surgery. "Not everyone is an appropriate candidate for surgery," Goldstein explained. "Some patients are

❖ ❖ ❖

If you can't figure out what you're looking at, don't wait. Call a provider.

❖ ❖ ❖

not healthy enough to have surgery and need extra weeks or months to get ready for surgery. Others have problems with alcohol and drug abuse or their living situation. Until such problems are treated, treatments directed at the pressure ulcer (dressings, seating correction, physical therapy, surgery) are ineffectual and a waste of limited resources. This is why patients with severe pressure ulcers are referred to social work and psychology. Other life and mental health problems are evaluated and treated, if related to the pressure ulcer problem."

Following surgery, recovery in the hospital takes several weeks—for the wound to heal, for progressive seating and physical activity, and to reinforce patient education.

Sending the patient home in good health, with proper equipment and healthy behaviors, is extremely important for a successful outcome, as is adherence to the follow-up appointment schedule.

Seek treatment early

"Patients and providers don't call us when the wound is small," Miller noted. "The problem is: it may look small, but it may be bigger underneath."

It's important to know how to recognize the early stages of a pressure ulcer (see "Stages of Pressure Ulcers" sidebar). Damage often begins deep beneath the skin, in the muscle tissue.[4] Therefore, a large deep pressure ulcer may present with a very small blister or opening on the skin surface. Waiting to see if it worsens is the worst possible course of action.

"If you see something red, get off it," Miller warned. "If it comes back or doesn't fade after being off it overnight, see a physician." If the wound is significant, the physician can refer a patient for a seating assessment, including pressure mapping, to correct problems that may be causing the pressure. Other potentially correctable problems are also evaluated. "It might be as simple as solving incontinence," Buchmeier said.

"It's in the very early stages that getting the pressure off makes the most difference," Miller said. "And it's the hardest part because people think it's too small to bother with. But getting off that spot for two weeks may make all the difference."

"It's hard to know if the sore is larger underneath," she continued. "We look for heat, color, a bruised look, hardness, drainage. You need an experienced wound practitioner to look at it."

If the wound is open and draining, "you need to see someone with expertise in wound care, pressure issues, and wheelchair seating," Miller warned. "At that point, you need a comprehensive approach, preferably at a facility that treats people with SCI."

Most primary care doctors have limited experience treating SCI, so patients themselves must have a good grasp of the problem. "I had a client who got her first pressure ulcer after being injured 20 years, went to her primary care doc, and got the wrong advice," Buchmeier said. "The information needs to be inside the head of the injured person."

Stages of Pressure Ulcers

A pressure ulcer (pressure sore, decubitis ulcer, bedsore) is any redness or break in the skin caused by too much pressure on the skin for too long.

Stage I:

• Skin is red or discolored, and this color change does not fade within 30 minutes after removing pressure.

• Take the pressure off and determine the source of the problem. "If you can't figure out what you're looking at, call a provider," said Cathy Miller, RN, rehab clinic nurse at Harborview.

Stage II:

• The topmost layer of the skin is broken, creating a shallow open sore similar to a blister, with or without drainage.

• Take the pressure off and see a physician for evaluation and specific treatment instructions.

Stage III:

• The wound is deeper and extends through skin layers to the fat tissue.

• Requires immediate medical attention.

Stage IV:

• The breakdown extends into the muscle or bone.

• Requires immediate medical attention.

Online illustrations & information:

• Medline Medical Encyclopedia: "Pressure Ulcers" – *www.nlm.nih.gov/medlineplus/ency/article/007071.htm*

• Wound Care Information Network: "Staging Pressure Ulcers" – *www.medicaledu.com/staging.htm*

"Pressure sores are strongly behavioral," Miller said, but it is unclear whether they are always preventable. "The early message used to be: It's your fault if you get a pressure ulcer." For this reason, patients are sometimes too embarrassed to seek help when they see a sore, and simply hope it will go away. The Harborview rehabilitation team encourages patients to detect pressure ulcers early, take pressure off the site immediately, and call at the first sign of a pressure ulcer.

"We don't know all the reasons why people get pressure ulcers," Buchmeier said, "but by doing a comprehensive approach, we put the person in the best possible position for healing and preventing recurrence."

The rehab team recommends scheduling periodic health evaluations with a

CONTINUED ON BACK PAGE.

forum report

The SCI Forum is an evening presentation and discussion series on topics of interest to persons with spinal cord injury and their family members, friends, and caregivers, held monthly at the University of Washington Medical Center during the fall, winter and spring. To learn about upcoming SCI Forums or read reports of past forums, consult our Web site at http://depts.washington.edu/rehab/sci/forum.html. Contact Cynthia Salzman (email: csalzman@u.washington.edu; phone: 206-685-3999) if you wish to be added to the SCI Forum mailing list.

Update on Spinal Cord Injury Research

Presented at the April 12, 2005, SCI Forum by Trent Tredway, MD, assistant professor of neurological surgery, University of Washington

April 12, 2005—The two main goals of SCI recovery research today are to "prevent secondary injury and restore function with partial regeneration," said Trent Tredway, MD, UW assistant professor of neurological surgery. "We know we don't need to effect total cure. Restoring some function will make a huge difference to patients" in terms of independence and quality of life.

A major issue influencing the development of new treatments is "Who's going to pay for this?" he said. The process of moving from animal studies in the lab to human clinical trials and FDA approval is long and expensive. Tredway believes much of the funding will come from the companies that are now developing new treatments.

Already, cost has played a role in focusing research on the acute rather than chronic phase. An experimental procedure such as a stem cell injection can be easily and inexpensively "piggy-backed" onto the stabilization surgery done immediately post-injury (and covered by insurance), whereas a new surgical procedure for the sole purpose of conducting an experimental treatment is harder to justify and fund. Many people with long-time injuries understandably find the focus on acute therapies frustrating. However, Tredway believes that once treatments are developed for acute injuries, "it shouldn't be much of a leap to apply them to more chronic patients."

FDA approval of new treatments involves a three-phase clinical trial process. In phase I, about 20-80 subjects are given the treatment to determine safety, dosage range and side effects. In phase II, the study is expanded to 100-300 subjects to determine if the treatment is effective and safe. Phase III is "the golden phase," Tredway said, and involves 1000-3000 participants at multiple sites, "to confirm effectiveness, monitor side effects, compare it to other standard treatments, and collect information regarding safety."

There are several avenues of research that Tredway believes show promise and may eventually lead to successful treatments for SCI. **Oscillating Field Stimulation (OFS)**,[1,2,3] which uses a small, continuously applied electrical field, has been shown to stimulate growth of frog and mammalian neurons, and results of a phase I trial in humans have been positive.[4] Ten patients whose injuries were classified as complete (ASIA A) 48 hours after injury received OFS one level above and one level below the lesion while undergoing initial stabilization surgery. The OSF continued for 15 weeks. Results showed statistically significant improvement in sensory and motor scores in subjects who had the procedure, compared to patients in a national database who didn't get the procedure. But this was not a "true control," Tredway said, so it's not possible to determine if the improvement was a result of the treatment or would have occurred naturally, since many people improve in the days and weeks following injury. This study showed the procedure is safe, and it is now awaiting phase II clinical trial.

❖ ❖ ❖

We know we don't have to effect total cure. Restoring some function will make a huge difference.

❖ ❖ ❖

Studies using **activated macrophages** to promote spinal cord repair are currently in phase II clinical trials in the U.S.[5] Proneuron Biotechnologies developed this procedure, called ProCord, based on research conducted in Israel by Dr. Michal Schwartz.

Macrophages are a type of white blood cell that helps remove debris and heal tissue after injury. While these cells are active in peripheral nerves, they are limited in the central nervous system (CNS). The ProCord process collects macrophages from the patient's own blood, activates it according to a proprietary process, and injects the cells into the spinal cord at the site of the injury. While Phase I trials in Israel involving ten patients showed some promise, phase II results so far are not as encouraging, and researches are now questioning the reliability of the phase I results.

Embryonic stem cells in spinal cord repair—a highly politicized topic today—are important because "they can give rise to all neural progenitors," Tredway explained, meaning they have the potential to turn into any type of cell under the right conditions (a quality referred to as "pluripotent"). Research by John McDonald, MD, PhD, has demonstrated the potential of these cells in SCI repair using animal models.[6]

Bone marrow stromal cells (BMSCs) also have been shown to have pluripotent capabilities. In rat studies in Japan, BMSCs injected into the cerebrospinal fluid (CSF) improved CNS regeneration.[7]

CONTINUED ON PAGE 5

CONTINUED FROM PAGE 4

In other studies, **gene-modified neural progenitor cells (NPCs)** were genetically altered to develop into a variety of neural cells.[8,9] NPCs intravenously injected into rats 24 hours after injury differentiated into several different neural cells, migrated to the lesion, and survived for at least 56 days after IV injection. "That's a long time to survive," Tredway said. "These results are pretty impressive. IV administration is much simpler and cheaper (than surgery)."

Glial cells—supportive cells that nourish and protect neurons—have been the focus of several studies. **Glial-restricted precursor cells** (a precursor cell population restricted to oligodendrocytes and astrocyte lineages) transplanted into rat spinal cords have been found to alter the growth of CSF axons and may support axonal growth after injury.[10]

Olfactory ensheathing cells from the patient's own olfactory (nasal) mucosa have been shown to reduce scar and cavity formation as well as promote regeneration after SCI. The work was carried out in mice and rats, and there are now efforts to bring this to human trial.[11]

The Geron Corporation in San Francisco, which has several stem cell lines (acquired prior to current restrictions), demonstrated the feasibility and safety of neural transplantation in SCI patients with syringomyelia, although no improvements have been seen yet.[12] Geron is planning Phase I trials of this same procedure in acute ASIA A SCI patients six-to-ten days after injury. Tredway hopes to participate in these trials here at the UW.

Phil Horner, PhD, UW assistant professor of neurological surgery, demonstrated that there are stem cells within the adult spinal cord capable of giving rise to glial cells,[13] suggesting "a higher level of cellular plasticity for the intact spinal cord than previously observed," Tredway said. "This is a very important step" toward finding alternatives to embryonic stem cells.

Tredway believes spinal cord repair will likely involve a combination of several biomedical technologies and modalities. "Technology changes rapidly, and we are just starting to hit our stride in the area of SCI," he said. "The future is now." As long as adequate funding is pumped into stem cell research, "It won't take 20-30 years."

References

1. Borgens RB, Shi R. Uncoupling histogenesis from morphogenesis in the vertebrate embryo by collapse of the transneural tube potential. *Dev Dyn*. 1995 Aug;203(4):456-67.

2. Hinkle L, McCaig CD, Robinson KR. The direction of growth of differentiating neurones and myoblasts from frog embryos in an applied electric field. *J Physiol.* 1981 May;314:121-35.

3. McCaig CD. Spinal neurite reabsorption and regrowth in vitro depend on the polarity of an applied electric field. *Development*. 1987 May;100(1):31-41.

4. Shapiro S, Borgens R, Pascuzzi R, et al. Oscillating field stimulation for complete spinal cord injury in humans: a phase 1 trial. *J Neurosurg Spine*. 2005 Jan;2(1):3-10.

5. Rapalino O. et al. Implantation of stimulated homologous macrophages results in partial recovery of paraplegic rats. *Nat Med.* 1998 Jul;4(7):814-21.

6. McDonald JW, Becker D, Holekamp TF, et al. Repair of the injured spinal cord and the potential of embryonic stem cell transplantation. *J Neurotrauma*. 2004 Apr;21(4):383-93. Review.

7. Ohta M, Suzuki Y, Noda T, et al. Bone marrow stromal cells infused into the cerebrospinal fluid promote functional recovery of the injured rat spinal cord with reduced cavity formation. *Exp Neurol.* 2004 Jun;187(2):266-78.

8. Setoguchi T, Nakashima K, Takizawa T, et al. Treatment of spinal cord injury by transplantation of fetal neural precursor cells engineered to express BMP inhibitor. *Exp Neurol.* 2004 Sep;189(1):33-44.

9. Fujiwara Y, Tanaka N, Ishida O, et al. Intravenously injected neural progenitor cells of transgenic rats can migrate to the injured spinal cord and differentiate into neurons, astrocytes and oligodendrocytes. *Neurosci Lett*. 2004 Aug 19;366(3):287-91.

10. Hill CE, Proschel C, Noble M, et al. Acute transplantation of glial-restricted precursor cells into spinal cord contusion injuries: survival, differentiation, and effects on lesion environment and axonal regeneration. *Exp Neurol.* 2004 Dec;190(2):289-310.

11. Ramer LM, Au E, Richter MW, et al. Peripheral olfactory ensheathing cells reduce scar and cavity formation and promote regeneration after spinal cord injury. *J Comp Neurol.* 2004 May 17;473(1):1-15.

12. Thompson FJ, Reier PJ, Uthman B, et al. Neurophysiological assessment of the feasibility and safety of neural tissue transplantation in patients with syringomyelia. *J Neurotrauma*. 2001 Sep;18(9):931-45.

13. Horner PJ, Power AE, Kempermann G, et al. Proliferation and differentiation of progenitor cells throughout the intact adult rat spinal cord. *J Neurosci*. 2000 Mar 15;20(6):2218-28.

For more information about SCI recovery research:

• **The Reeve-Irvine Research Center**, University of California at Irvine, 2109 Gillespie Neuroscience Research Facility, Irvine, CA, 92697; 949-824-3993; *mhofstad@uci.edu*; *www.reeve.uci.edu/infoabout.html*

• **Spinal Cord Society.** 19051 County Highway 1, Fergus Falls, Minnesota 56537; 218-739-5252 *http://members.aol.com/scsweb/*; **Northwest Chapter**: PO Box 6092, Edmonds, WA 98026; 425-670-2622; *scsnnwgolf@aol.com; www.scsnw.com/*

• **CareCure Community**, W. M. Keck Center for Collaborative Neuroscience, State University of New Jersey, Rutgers. *http://carecure.org*

• **The Miami Project to Cure Paralysis, University** of Miami School of Medicine; 800-STANDUP; *www.miamiproject.miami.edu/*

More SCI Forum reports are archived on our Web site at *http://depts.washington.edu/rehab/sci/forum_reports.html.*

literature review

The articles previewed below were selected from a recent screening of the National Library of Medicine database for articles on spinal cord injury. In the judgment of the editors, they include potentially useful information on the diagnosis or management of spinal cord injury. You may obtain copies of the complete articles through your local medical library or from UW Health Sciences Library Document Delivery Service (call 206-543-3436 for fee schedule).

BONE LOSS

■ Fracture threshold in the femur and tibia of people with spinal cord injury as determined by peripheral quantitative computed tomography.
Peripheral quantitative computed tomography (pQCT) measurements of the tibia and femur were completed and post-SCI fracture history was obtained in 99 subjects with motor complete (ASIA A and B) SCI. Twenty-seven subjects had sustained a fracture of the lower extremities, of which 18 were to either the tibia or fibula. Trabecular (spongy) bone mineral density (BMD) of the femur and tibia distal epiphyses was the most sensitive parameter for distinguishing SCI subjects with and without fractures and can only be determined by pQCT (and not by dual-energy x-ray absorptiometry). Fractures occurred in subjects with trabecular BMD of less than 114 mg/cm 3 and less than 72 mg/cm 3 for the femoral and tibial distal epiphysis, respectively. Approximately 50% of the subjects with chronic SCI (longer than 5 years for femur data and 7 years for tibia) had trabecular BMD values above the fracture threshold in the femur and about one third above the fracture threshold in the tibia. pQCT may help identify subjects who are at risk of fracture through minor trauma.
Eser P, Frotzler A, Zehnder Y, Denoth J.
Arch Phys Med Rehabil. 2005 Mar;86(3):498-504.

■ Trabecular bone is more deteriorated in spinal cord injured versus estrogen-free postmenopausal women.
Osteoporosis is common in postmenopausal women and individuals with SCI. In this study, the effects of estrogen loss (due to menopause) and unloading (non-weight-bearing) on the trabecular (spongy) bone of the knee were assessed in 17 pre- and postmenopausal ambulatory women and in 20 pre- and postmenopausal women with SCI. High-resolution magnetic resonance imaging was used to compare groups on measures of trabecular bone density in the distal femur and proximal tibia, which is the most common fracture site in the SCI population. Trabecular bone of the knee was deteriorated in women with SCI compared to ambulatory women, and premenopausal women with SCI had more bone loss than postmenopausal ambulatory women, suggesting that unloading has a greater influence on bone density than estrogen loss.
Slade JM, Bickel CS, Modlesky CM, et al.
Osteoporos Int. 2005 Mar;16(3):263-72. Epub 2004 Aug 28.

■ Effect of alendronate on bone mineral density in spinal cord injury patients: a pilot study.
Nineteen subjects with SCI longer than 6 months and classified as ASIA A, B or C were randomly assigned to a control or experimental group. Controls received 1000 mg of calcium daily, and the experimental group received 1000 mg of calcium plus 10 mg of alendronate daily. Bone density parameters were analyzed using whole-body dual-energy X-ray absorptiometry at baseline and after 6 months. The experimental group showed improvements in 9 of the 12 bone density parameters measured, although changes were significant in only two parameters. In the control group, an increase in bone density was observed in only one parameter, and the remaining 11 presented either no change or a decrease. Alendronate had a positive effect on bone mineral density in SCI patients and therefore represents a potential tool for prevention and treatment of osteoporosis in this population.
Moran de Brito CM, Battistella LR, et al.
Spinal Cord. 2005 Jun;43(6):341-8.

PAIN

■ Intravenous lidocaine relieves spinal cord injury pain: a randomized controlled trial.
Twenty-four SCI patients with neuropathic pain at or below the level of injury completed a double-blind crossover trial of the sodium channel blocker lidocaine versus placebo infused over 30 minutes. Subjects were assigned to two groups: those with and those without evoked pain (pain due to a normally non-painful stimulus), and then randomized to receive either lidocaine or placebo. Pain was assessed using a visual analog scale and quantitative sensory testing. Lidocaine significantly reduced pain in all patients and in each of the two groups with and without evoked pain. Also, more patients were responders (defined as those who experienced a 33% reduction in pain) to lidocaine (11 subjects) than to placebo (2 subjects). Agents (such as anticonvulsants or antiarrhythmics) with sodium channel-blocking properties may be a treatment option for SCI pain.
Finnerup NB, Biering-Sorensen F, Johannesen IL, et al.
Anesthesiology. 2005 May;102(5):1023-30.

■ Pain following spinal cord injury: the impact on community reintegration.
To examine the relationship between pain and community integration, subjects from 66 consecutive admissions to an inpatient SCI center were followed prospectively. Standardized pain assessments were performed on admission, discharge and 6 months of community living and correlated with assessments of community reintegration using the Reintegration to Normal Living Index. In all, 86% of individuals with SCI reported pain at six months postdischarge, and 27% of these persons had pain that negatively affected activities. Pain intensity alone had the largest single effect on community integration. The results of this study highlight the need to address pain during both the rehabilitation phase of treatment and the early transition into the community.
Donnelly C, Eng JJ.
Spinal Cord. 2005 May;43(5):278-82.

■ Quality of sleep in individuals with spinal cord injury: a comparison between patients with and without pain.
Mailed questionnaires about pain, mood and sleep quality were completed by 191 persons with SCI. Respondents were divided into three groups according to whether they reported: (1) no pain (50 respondents), (2) intermittent pain (42), or (3) continuous pain (99). Patients reporting continuous pain rated pain intensity and unpleasantness significantly higher than those with intermittent or no pain, and had the poorest quality of sleep and the highest ratings of anxiety and depression. Anxiety, together with pain intensity and depression, were the main predictors for poor sleep quality. Melatonin—a neuromodulator involved in regulating anxiety, depression, pain and sleep—is decreased in persons with SCI, and as such is an important avenue for further research.
Norrbrink Budh C, Hultling C, Lundeberg T.
Spinal Cord. 2005 Feb;43(2):85-95.

CONTINUED ON PAGE 7

CONTINUED FROM PAGE 6

OTHER COMPLICATIONS

■ **A longitudinal evaluation of sleep and breathing in the first year after cervical spinal cord injury.**
To examine the incidence of sleep disordered breathing (SDB) after acute tetraplegia and to determine the relation between the Apnea-Hypopnea Index score and selected SDB predictors (benzodiazepine use; measures of respiratory function; neck and abdominal circumference) in tetraplegia, full polysomnography (sleep studies) was performed, and spirometry, maximum inspiratory and expiratory pressures, medication usage, and neck and abdominal girth were assessed in 20 subjects immediately after acute tetraplegia and at 2, 4, 13, 26, and 52 weeks postinjury. Preinjury SDB was estimated using the multivariate apnea prediction equation. Thirteen subjects completed 12 months of follow-up. Three subjects had probable SDB before injury. In the first 48 hours after injury, no subject had SDB. At 2 weeks, 60% had SDB; at 4 weeks, 62%; at 13 weeks, 83%; at 26 weeks, 68%; and at 52 weeks, 62%. While SDB is common in acute SCI and peaks around three months, no relation was found between the SDB predictors and presence of SDB.
Berlowitz DJ, Brown DJ, Campbell DA, Pierce RJ.
Arch Phys Med Rehabil. 2005 Jun;86(6):1193-9.

■ **Evolving risk for thromboembolism in spinal cord injury (SPIRATE Study).**
Two groups of patients admitted to the SCI unit of a rehabilitation hospital during different time periods and tested for venous thromboembolism (VTE) were compared on rates of VTE. The recent group, seen during 1999-2003, consisted of 76 patients, and the previous group, seen during 1992-95, of 243 patients. Analysis variables included level and completeness of SCI, concomitant injuries, surgical procedures, complications, preexisting illnesses, and use of antithrombotic medications. Six (7.9%) subjects in the recent group had VTE, which was a significant decrease from the previous group, in which 21% had VTE. The major differences between the recent and previous patient samples were a decrease in the use of unfractionated heparin (15.8% vs. 56.8%) and an increase in the use of low molecular weight heparin (LMWH) (81.6% vs. 59.7%). This study confirms that the recent switch from unfractionated heparin to LMWH for the prevention of VTE has coincided with a decrease in the frequency of this complication.
Green D, Sullivan S, Simpson J, et al..
Am J Phys Med Rehabil. 2005 Jun;84(6):420-2.

■ **Pressure ulcer prevalence in people with spinal cord injury: age-period-duration effects.**
This was a multicenter cohort study of 3,361 individuals with SCI from 9 Model SCI Systems who were injured between 1986 and 1995 and followed up annually through 2002. At follow-up, 33% were found to have a physician-confirmed pressure ulcer of stage II or greater. Data analysis showed a significant trend toward increasing pressure ulcer prevalence in the recent years (1994-2002 vs 1984-1993) not explained by aging, years since injury, or demographic and clinical factors, but possibly due to shortened length of hospital stay. The risk of pressure ulcers was steady during the first 10 years postinjury and increased after 15 years. Pressure ulcers were more common among the elderly, men, African Americans, singles, subjects with education less than high school, unemployed, subjects with complete injury, and subjects with history of pressure ulcers, rehospitalization, nursing home stay, and certain medical conditions. Injury cause and level had no significant effect.
Chen Y, Devivo MJ, Jackson AB.
Arch Phys Med Rehabil. 2005 Jun;86(6):1208-13.

MISCELLANEOUS

■ **Psychogenic and pharmacologic induction of the let-down reflex can facilitate breast-feeding by tetraplegic women: a report of 3 cases.**
A functional let-down reflex is required to provide adequate milk to a nursing infant. Infant suckling activates tactile receptors in the breast, and this signal is carried via afferent nerves in the T4-6 dorsal roots to the spinal cord and then to neurons in the hypothalamus, which release oxytocin into the bloodstream. Oxytocin triggers milk ejection from the breast (the let-down reflex). Although suckling-induced afferent stimuli are absent in women with SCI above T4 and are reduced if the injury is between T4 and T6, three women in this report (C8, ASIA B; C8, incomplete; C6-7, ASIA A) maintained breast-feeding for 12-54 weeks after delivery using active mental imaging and relaxation techniques or oxytocin nasal spray to induce let-down.
Cowley KC.
Arch Phys Med Rehabil. 2005 Jun;86(6):1261-4.

■ **Measuring energy expenditure using heart rate to assess the effects of wheelchair tire pressure.**
Three women and 11 men with SCI between T4 and L1 participated in four trials of wheelchair propulsion with tires inflated at four different pressures (100, 75, 50 and 25 psi). Each subject wheeled at a constant self-selected velocity for 8 minutes, with 10-minute rests between trials. Oxygen consumption, heart rate and distance traveled were recorded during each trial. Tire pressure was found to affect the amount of energy required for propulsion, but only when tires are substantially deflated (50% or less than maximum recommended value). Tire pressures below 50% inflation add an additional 25% increase in energy expenditure during wheeling. Heart rate correlated well with oxygen consumption in persons at T5 and below and could be used to assess energy expenditure under different conditions of tire pressure when oxygen consumption analysis is not available. Above T5 the relationship between heart rate and oxygen consumption was weaker. Heart rate has limitations and should only be used to measure within-subject differences.
Sawatzky BJ, Miller WC, Denison I.
Clin Rehabil. 2005 Mar;19(2):182-7.

■ **Long-term body-weight-supported treadmill training and subsequent follow-up in persons with chronic SCI: effects on functional walking ability and measures of subjective well-being.**
Thirteen individuals with chronic (mean 7.4 years) incomplete SCI (levels L1 – C4; ASIA B & C) completed thrice-weekly sessions of body-weight-supported treadmill training (BWSTT) for approximately 12 months (144 sessions). At the outset of the study, all were wheelchair-dependent and 11 had no standing or walking capability. All subjects improved in treadmill walking ability and six improved their capacity to walk over ground. Satisfaction with life and with physical function improved significantly. Subjects were assessed again 8 months after completing the 12-month training. There was a slight decline in treadmill walking performance, and over-ground walking scores remained relatively stable. Results suggest that BWSTT has benefits that extend beyond improvements in functional walking and may be appropriate for persons with SCI who do not have residual motor function.
Hicks AL, Adams MM, Martin Ginis K, et al.
Spinal Cord. 2005 May;43(5):291-8.

Spinal Cord Injury Update is supported by grant H133N000003 from the National Institute of Disability and Rehabilitation Research (NIDRR), U.S. Department of Education, Office of Special Education and Rehabilitative Services (OSERS), to the Northwest Regional Spinal Cord Injury System, one of 16 model SCI care systems nationwide. **Project Director:** Diana D. Cardenas, MD, MHA; **Project Co-Director:** Charles Bombardier, PhD.

To add your name to the mailing list, contact the editor, Cynthia Salzman, at the University of Washington Department of Rehabilitation Medicine, Box 356490, Seattle WA 98195-6490; 206-685-3999; *csalzman@u.washington.edu*

Northwest Regional SCI System Web site: *http://depts.washington.edu/rehab/sci*

Pressure ulcers

CONTINUED FROM PAGE 3

rehabilitation medicine physician because these check-ups provide an opportunity to review and correct problems that might increase risk for pressure ulcers, like poor seating, malnutrition, incontinence, and lifestyle issues. Even if a person has gone decades without a problem, aging causes changes that make the skin more vulnerable. And the longer the time since injury, the greater the risk becomes.

Rosemary Buchmeier , RN, and Cathy Miller, RN, welcome calls from physicians or patients who have questions about a skin problem (206-731-2581).

References:

1. *2005 Annual Statistical Report for the Model Spinal Cord Injury Care Systems.* National Spinal Cord Injury Statistical Center, The University of Alabama at Birmingham, Birmingham, Alabama.

2. Kierney PC, Engrav LH, Isik FF, Esselman PC, Cardenas DD, Rand RP. Results of 268 Pressure Sores in 158 Patients Managed Jointly by Plastic Surgery and Rehabilitation Medicine. *Plastic Reconstr Surg.* 102(3):765-772, 1998.

3. Charlifue SL, Lammertse DP, Adkins RH. Aging with spinal cord injury: Changes in selected health indices and life satisfaction. *Arch Phys Med Rehabil* 85(11):1848-53, 2004.

4. *Pressure Ulcer Prevention and Treatment Following Spinal Cord Injury: A Clinical Practice Guideline for Health Care Professionals.* Consortium for Spinal Cord Medicine. Paralyzed Veterans of America. 2000.

MEDCON: 24-hour, toll-free consultation and referral service for health care professionals and persons with SCI.
Seattle: **206-543-5300** Long Distance: **800-326-5300**

Also published online at http://depts.washington.edu/rehab/sci/update.html

Spinal Cord Injury Update
UNIVERSITY OF WASHINGTON
Rehabilitation Medicine, Box 356490
Seattle, Washington 98195–6490

ADDRESS SERVICE REQUESTED

Nonprofit Org.
US Postage
PAID
Seattle WA
Permit 62