SPINAL CORD MEDICINE

CLINICAL PRACTICE GUIDELINE: AUTONOMIC DYSREFLEXIA

Acute Management of Autonomic Dysreflexia:

Adults with Spinal Cord Injury Presenting to Health-Care Facilities

consortium for SPINAL CORD MEDICINE
CLINICAL PRACTICE GUIDELINES

Administrative and financial support provided by Paralyzed Veterans of America

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Foreword

Autonomic dysreflexia (AD), with its sudden and severe rise in blood pressure, is a potentially life-threatening condition that can occur in anyone with a spinal cord injury (SCI) at or above the level of the sixth thoracic vertebra (T6). The resolution of AD requires quick and decisive treatment. Health-care providers who work with SCI are very familiar with the diagnosis and treatment of AD. However, because the onset of AD is so rapid and the symptoms are so severe, individuals with this condition are often rushed to the nearest health-care facility, which may be staffed by health-care providers who have little or no experience in the treatment of AD. The purpose of this booklet is to make accessible to health-care providers a clinical practice guideline that can be used when an adult with SCI presents to their facility with the signs and symptoms of AD.

The AD guideline panel, composed of physicians, nurses, and a pharmacist, each of whom has many years of experience working with individuals with SCI, developed the recommendations in this guideline under the auspices of the Paralyzed Veterans of America (PVA). A number of resources were used in this endeavor. The groundwork was laid by the Eastern Paralyzed Veterans Association, which submitted an initial document to the AD panel for consideration. A methodologist worked closely with the panel, assisting with extensive literature searches and reviews of pertinent articles and creating a database. AD panel meetings and numerous teleconferences were coordinated by PVA. Finally, a draft of the AD guideline was circulated for peer review among 17 Spinal Cord Medicine Consortium organizations, and further revisions were recommended by the panel, based on the reviewers' comments.

This is the first edition of this clinical practice guideline—*Acute Management of Autonomic Dysreflexia: Adults With Spinal Cord Injury Presenting to Health Care Facilities.* I hope that the guideline will play an important role in the evaluation and management of individuals presenting with signs and symptoms of AD. I also hope that the guideline will stimulate further clinical studies in this important area.

Todd Linsenmeyer, MD
Chairman
Autonomic Dysreflexia
Guideline Development Panel

Preface

Guidelines Development and Use: A Message From the Consortium Steering Committee Chairman

Health care and rehabilitation services for persons with spinal cord injury are costly. Unfortunately, they also are threatened by the recent emphasis on cost reduction—both in the managed care environment and in the Department of Veterans Affairs (VA) Medical Centers. The challenge facing providers of these services is how to achieve optimal outcomes in the face of shrinking resources. In September 1994 the Paralyzed Veterans of America responded to this challenge by assuming a leadership role in the development of clinical practice guidelines (CPGs) for the spinal cord injured.

In an attempt to involve providers across the continuum of services for spinal cord injury, PVA organized a consortium of groups and organizations to develop and disseminate CPGs. This is a unique role for an organization that represents the needs of consumers. PVA recruited relevant organizations of physicians, therapists, nurses, psychologists, and social workers and asked each organization to designate a representative to participate in consortium activities on a steering committee. Two organizations with a payer perspective—the Department of Veterans Affairs Spinal Cord Injury Program and the Insurance Rehabilitation Study Group—also participated in the Consortium Steering Committee. Each representative has participated in the development, review, and revision of each CPG draft. Now, consortium member organizations are being given an opportunity to endorse the CPGs and to assist in their dissemination to the field.

We on the Consortium Steering Committee invite and encourage providers who implement the clinical practice guidelines to measure how outcomes are affected. Are costly complications avoided? Do functional gains occur more quickly? Are lengths of stay shorter as a result of implementation of the CPGs?

Future consortium activities will be in two areas:

- Development of additional clinical practice guidelines.
- Development of a cycle of review and revision of previously published CPGs.

It is our intent to periodically update each set of guidelines as new research and treatment modalities become available.

I want to extend my appreciation to all of the panel members and to their chairman, Todd Linsenmeyer, for their excellent work in developing this CPG. I also want to thank members of the Consortium Steering Committee and to extend special thanks to Frank A. Morrone, John L. Carswell, and Dawn M. Sexton in PVA's Health Policy Department for their leadership and support in this effort. To my partner in this endeavor, J. Paul Thomas, I owe particular praise for the energy and commitment he devoted to this project.

Kenneth C. Parsons, MD
Chairman
Spinal Cord Medicine CPG Consortium
Steering Committee

Acknowledgments

The chairman and members of the panel wish to express special appreciation to the individuals, to the professional organizations that were involved in the Spinal Cord Medicine Consortium, to the expert health care providers who reviewed the draft documents, and to the consumers, advocacy organizations, and the staffs of the numerous medical facilities and spinal cord injury rehabilitation centers who contributed their time and expertise to the development of these guidelines.

Kit N. Simpson, Andrea K. Biddle, and their fine staff in the Health Policy and Administration Department at the University of North Carolina at Chapel Hill masterfully conducted the initial and secondary-level literature searches, evaluated the quality and strength of evidence of the scientific investigations, constructed evidence tables, and performed meta-analyses of the benefits and effects of the various preventive and therapeutic modalities and interventions, as warranted.

Members of the Consortium Steering Committee, representing 17 professional organizations, were joined by 34 expert reviewers in providing outstanding scientific and clinical analysis. Through their valuable comments, they helped to refine the recommendations and to identify additional supporting evidence from the scientific literature. The quality of the technical assistance from these dedicated reviewers contributed significantly to the professional consensus building that is hopefully achieved through the guideline development process.

The AD guideline development panel is grateful for the many technical support services provided by the various departments of the Paralyzed Veterans of America. In particular, the panel recognizes the organizational and managerial skills of J. Paul Thomas and Dawn M. Sexton in the Health Policy Department; the guidance in writing, formatting, and art work provided by James A. Angelo and Patricia E. Scully in the Communications Department; the excellent technical review of both the clinical practice guidelines and the consumer guide provided by medical writers Joellen Talbot and Barbara Shapiro; and the intensive efforts of both PVA staff and consultants who developed the glossary, standardized the nomenclature, and indexed the guidelines. Appreciation is expressed for the steadfast commitment and enthusiastic advocacy of PVA's senior officers, including Immediate Past President Richard Grant, National President Kenneth C. Huber, Executive Director Gordon H. Mansfield, Deputy Executive Director John C. Bollinger, and the entire PVA Board of Directors. Their generous financial support has made the CPG consortium and guidelines development process a successful venture.

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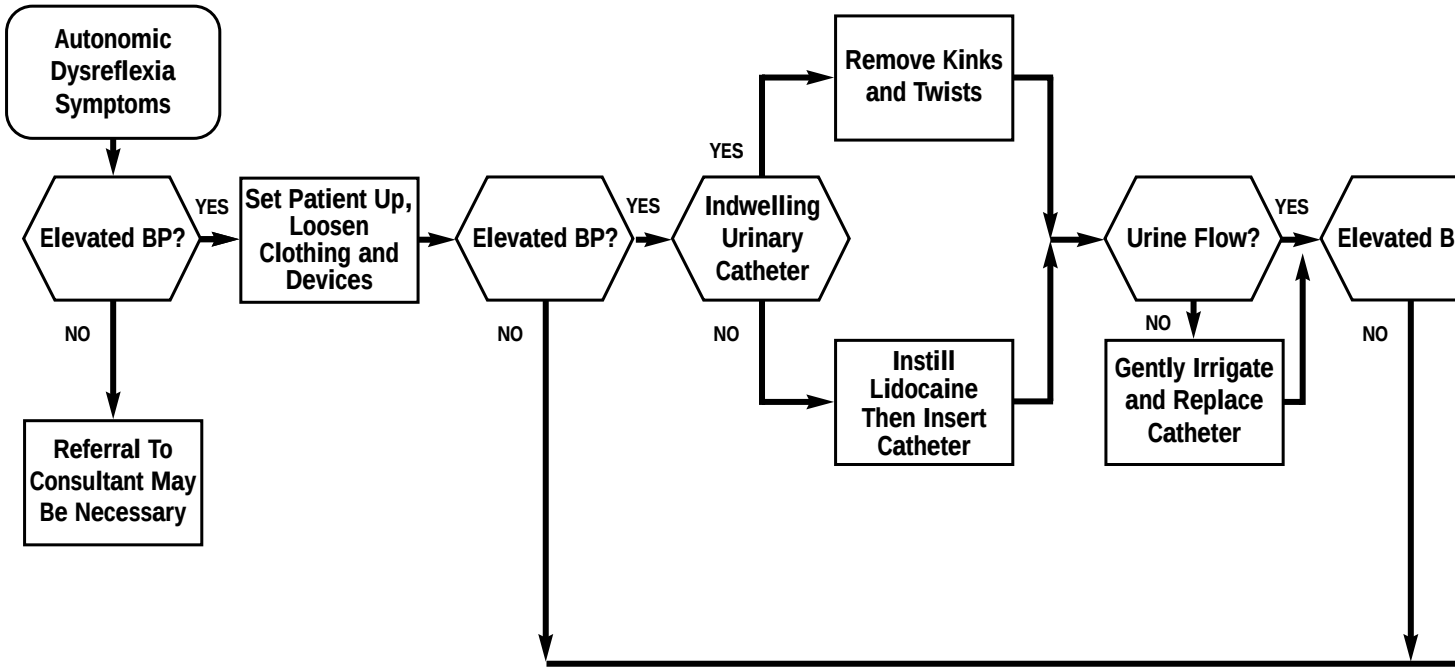
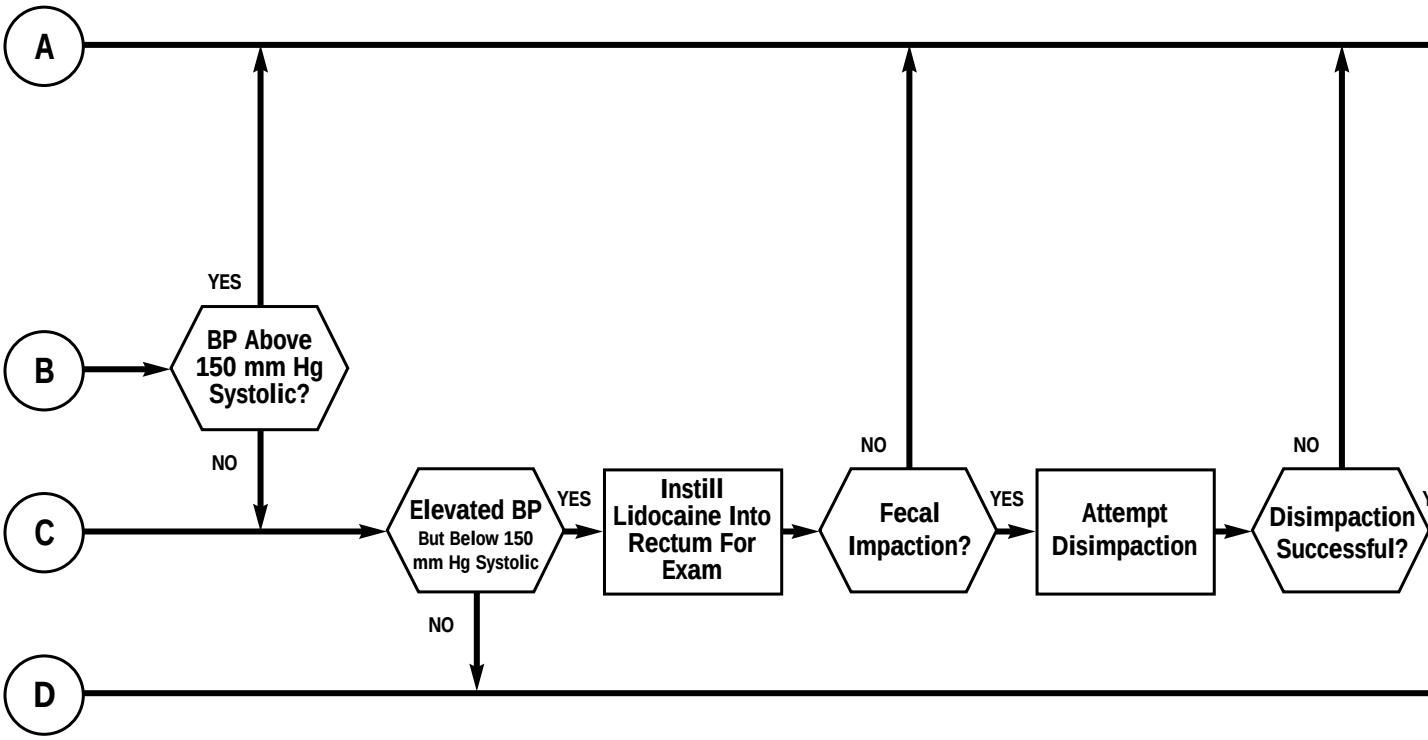
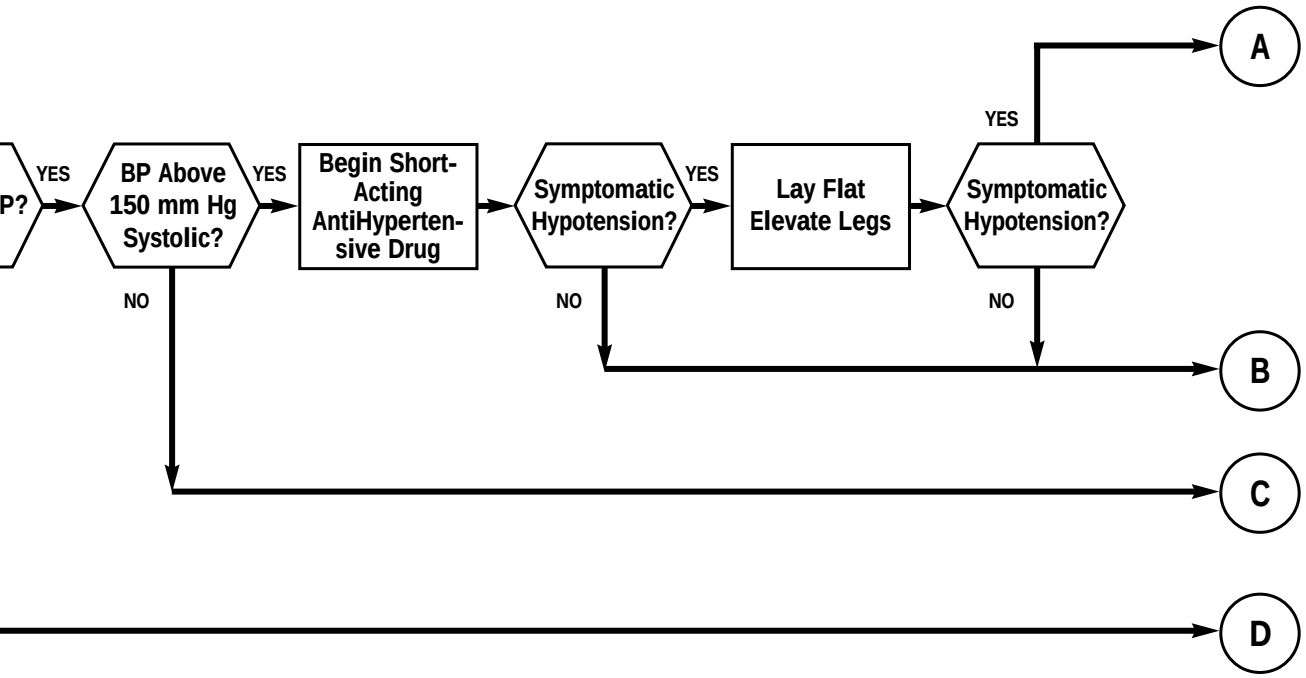
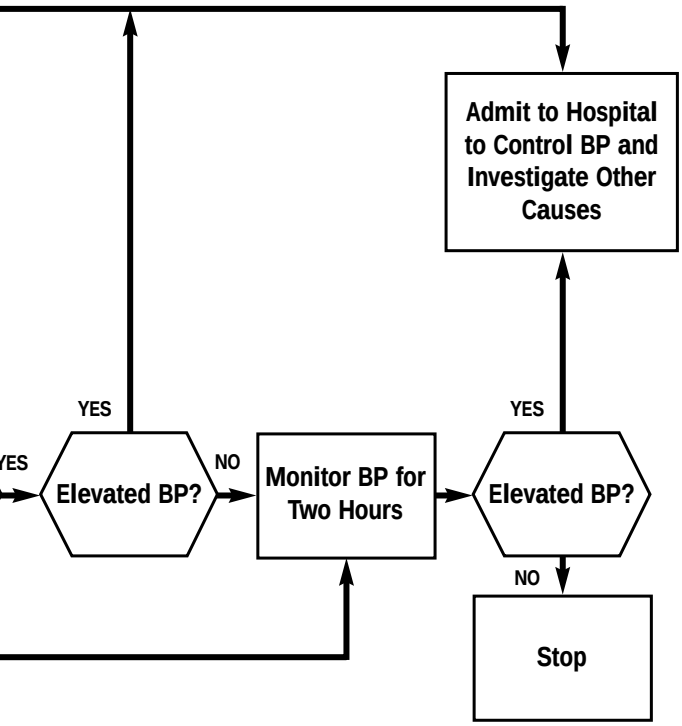
Algorithm—
Summary of Treatment Recommendations






YES
BP Above 150 mm Hg Systolic?
YES
Begin Short-Acting AntiHypertensive Drug
Symptomatic Hypotension?
YES
Lay Flat Elevate Legs
Symptomatic Hypotension?
YES
NO
NO
NO
A
B
C
D


Admit to Hospital to Control BP and Investigate Other Causes
YES
YES
Elevated BP?
NO
Monitor BP for Two Hours
Elevated BP?
NO
Stop

Summary of Treatment Recommendations

NOTE: Pregnant women should be referred to an appropriate consultant.

1. Check the individual's blood pressure.
2. If the blood pressure is not elevated, refer the individual to a consultant, if necessary.
3. If the blood pressure is elevated and the individual is supine, immediately sit the person up.
4. Loosen any clothing or constrictive devices.
5. Monitor the blood pressure and pulse frequently.
6. Quickly survey the individual for the instigating causes, beginning with the urinary system.
7. If an indwelling urinary catheter is not in place, catheterize the individual.
8. Prior to inserting the catheter, instill 2 percent lidocaine jelly (if readily available) into the urethra and wait several minutes.
9. If the individual has an indwelling urinary catheter, check the system along its entire length for kinks, folds, constrictions, or obstructions and for correct placement of the indwelling catheter. If a problem is found, correct it immediately.
10. If the catheter appears to be blocked, gently irrigate the bladder with a small amount of fluid, such as normal saline at body temperature. Avoid manually compressing or tapping on the bladder.
11. If the catheter is draining and the blood pressure remains elevated, proceed with step 16.
12. If the catheter is not draining and the blood pressure remains elevated, remove and replace the catheter.
13. Prior to replacing the catheter, instill 2 percent lidocaine jelly (if readily available) into the urethra and wait several minutes.
14. If the catheter cannot be replaced, consider attempting to pass a coude catheter, or consult a urologist.
15. Monitor the individual's blood pressure during bladder drainage.
16. If acute symptoms of autonomic dysreflexia persist, including a sustained elevated blood pressure, suspect fecal impaction.
17. If the elevated blood pressure is at or above 150 mm Hg systolic, consider pharmacologic management to reduce the systolic blood pressure without causing hypotension prior to checking for fecal impaction. If the blood pressure remains elevated but is less than 150 mm Hg systolic, proceed to step 20.
18. Use an antihypertensive agent with rapid onset and short duration while the causes of autonomic dysreflexia are being investigated.
19. Monitor the individual for symptomatic hypotension.
20. If fecal impaction is suspected, check the rectum for stool, using the following procedure: With a gloved hand, instill a topical anesthetic agent such as 2 percent lidocaine jelly generously into the rectum. Wait approximately 5 minutes for sensation in the area to decrease. Then, with a gloved hand, insert a lubricated finger into the rectum and check for the presence of stool. If present, gently remove, if possible. If autonomic dysreflexia becomes worse, stop the manual evacuation. Instill additional topical anesthetic and recheck the rectum for the presence of stool after approximately 20 minutes.
21. Monitor the individual's symptoms and blood pressure for at least 2 hours after resolution of the autonomic dysreflexia episode to make sure that it does not recur.
22. If there is poor response to the treatment specified above and/or if the cause of the dysreflexia has not been identified, strongly consider admitting the individual to the hospital to be monitored, to maintain pharmacologic control of the blood pressure, and to investigate other causes of the dysreflexia.
23. Document the episode in the individual's medical record. This record should include the presenting signs and symptoms and their course, treatment instituted, recordings of blood pressure and pulse, and response to treatment. The effectiveness of the treatment may be evaluated according to the level of outcome criteria reached:
    - The cause of the autonomic dysreflexia episode has been identified.
    - The blood pressure has been restored to normal limits for the individual (usually 90 to 110 systolic mm Hg for a tetraplegic person in the sitting position).
    - The pulse rate has been restored to normal limits.
    - The individual is comfortable, with no signs or symptoms of autonomic dysreflexia, of increased intracranial pressure, or of heart failure.

24. Once the individual with spinal cord injury has been stabilized, review the precipitating cause with the individual, members of the individual's family, significant others, and care givers. This process entails adjusting the treatment plan to ensure that future episodes are recognized and treated to prevent a medical crisis or, ideally, are avoided altogether. The process also entails discussion of autonomic dysreflexia in the spinal cord injury individual's education program, so that he or she will be able to recognize early onset and obtain help as quickly as possible. It is recommeneded that an individual with a spinal cord injury be given a written description of treatment for autonomic dysreflexia at the time of discharge that can be referred to in an emergency.

Overview of Consortium Guidelines Development

Over 5,000 years ago, the Edwin Smith surgical papyrus described two cases of complete tetraplegia as "an ailment not to be treated." The field of spinal cord injury (SCI) medicine has come a long way! But recent health-care reforms and managed-care strategies have brought about a new model of health care, one that demands innovative approaches to ensure provider accountability, optimal program performance, and purposeful outcome measurement.

The development of clinical practice guidelines (CPGs) is a significant new trend in the U.S. health provider community. CPGs are based on evidence derived from the scientific literature and enhanced by the collective opinions of physicians, academic experts, and researchers on how best to treat specific clinical conditions. CPGs are important tools that physicians, nurses, therapists, and consumers can use in making health-care decisions.

In its continuing examination of the nation's health-care system, the Paralyzed Veterans of America came to recognize clinical practice guidelines as a major mechanism that could enhance program performance, facilitate accountability, and improve the quality of care for people with spinal cord injury and disease—both veterans and nonveterans. During an extensive study of the processes used to develop clinical practice guidelines, PVA learned three important lessons. First, to be credible, PVA's guidelines would have to be based on scientific evidence; second, expert consensus was critical to the process; and third, all parties involved with SCI health-care clinical decisionmaking would have to be involved.

The Spinal Cord Medicine Consortium

Seventeen organizations, including PVA, joined in a consortium to develop clinical practice guidelines in spinal cord injury medicine. A steering committee was established to lead the guidelines development process, identify topics, and select panels of experts for each topic. The steering committee is composed of one representative with clinical practice guidelines experience from each consortium member organization. The committee chairman is Kenneth C. Parsons, a noted spinal cord injury physician at the Institute for Rehabilitation and Research in Houston, Texas, and president-elect of the American Spinal Injury Association. PVA provides financial resources, administrative support, and programmatic coordination of consortium activities.

To provide all of the organizational representatives with a standard frame of reference, PVA conducted an orientation workshop featuring experts from leading professional organizations with extensive guidelines development experience. The workshop faculty included senior staff members from the Academy of Family Practice, the Academy of Pediatrics, the American Academy of Otolaryngology—Head and Neck Surgery, the American College of Cardiology, the Agency for Health Care Policy and Research (AHCPR), and the American Medical Association.

After studying the processes used to develop other guidelines, the consortium steering committee unanimously agreed on a new, modified, scientific evidence-based model derived from AHCPR. The model is:

- Interdisciplinary, to reflect the multiple information needs of the spinal cord medicine practice community.
- Responsive, with a time line of 12 months for the completion of the guidelines.
- Reality-based, to make the best use of the time and energy of the busy clinicians who serve as panel members and field reviewers.

The consortium's approach to the development of evidence-based clinical practice guidelines is both innovative and cost-efficient. The process recognizes the specialized needs of the national spinal cord injury medicine community, encourages the participation of both payer representatives and disabled consumers with spinal cord injury and disease, and emphasizes utilization of the evidence available in the international scientific literature.

The Spinal Cord Medicine Consortium is unique to the clinical practice guidelines field in that it employs highly effective management strategies based on the availability of resources in the health care community, it is coordinated by a recognized national consumer organization with a reputation for providing effective service and advocacy for people with spinal cord injury and disease, and it includes third-party and reinsurance payer organizations at every level of the development and dissemination process. The consortium expects to initiate work on four or more CPG topics per year, with evaluation and revision of previously completed guidelines as new research demands.

Guideline Development Process

The guideline development process adopted by the Spinal Cord Medicine Consortium consists of 12 steps, leading to panel consensus and organizational endorsement. After the steering committee chooses a topic and selects a panel of experts, consultant methodologists from the University of North Carolina's Department of Health Policy and Administration review the international literature, prepare evidence tables of research data, and conduct statistical meta-analyses, as warranted. The panel chairman then assigns specific topics to the expert panel members, and writing begins on each component, based on the references and other research materials furnished by the methodologist.

The panel members complete their drafts, and a draft guideline document is generated during the first meeting of the panel. The CPG panel incorporates new literature citations or other evidence-based information not previously available. At this point, charts, graphs, algorithms, and other visual aids, as well as a complete bibliography, are added, and the full document is sent to legal counsel for review.

After legal analysis to consider antitrust and other restraint-of-trade matters, the draft CPG document is reviewed by clinician experts from each of the consortium organizations plus select clinical experts and consumers. The review comments are assembled and the document is revised to reflect the reviewers' comments. Following a second legal review, the CPG document is distributed to all consortium organization governing boards. Final technical details are negotiated among the panel chair, members of the organizations' boards, and expert panelists. If substantive changes are required, the draft will receive a final legal review. The document is then ready for editing, formatting, and preparation for publication.

The benefits of clinical practice guidelines for the spinal cord injury medicine field are numerous. Among the more significant applications and results are the following:

- Clinical practice options and care standards.
- Medical and health professional education and training.
- Evaluation studies of CPG use and outcomes.
- Building blocks for pathways and algorithms.
- Research gap identification.
- Cost and policy studies for improved quantification.
- Primary source for consumer information and public education.
- Knowledge base for improved professional communication.

History of AD Guideline Development

Four years ago, the Eastern Paralyzed Veterans Association developed a guideline for the treatment of autonomic dysreflexia (AD) for the Castle Point VA Medical Center. The guideline was refined by the Clinical Practice Committee of the American Paraplegia Society (APS) and was about to be approved and distributed by APS. Once APS learned of the Spinal Cord Medicine Consortium, however, the organization decided to turn the AD guideline over to the consortium for further development and methodological and legal review.

AD Guideline Methodology

The methodology team's strategy for finding evidence relating to issues about autonomic dysreflexia in SCI patients closely resembles the methods recommended by AHCPR and by the National Academy of Sciences Institute of Medicine. First, an initial search of the MEDLINE database from 1966 to the present was conducted, the main issues included in autonomic dysreflexia were identified, and the volume of literature available on the subject was estimated. Then, a limited number of selected overviews and review articles was retrieved and used to further identify relevant issues. The main areas of interest were epidemiology, pathophysiology, prophylaxis, and management of acute and chronic autonomic dysreflexia.

Data extraction forms were developed to standardize the data used for evaluation. This form included sections on study population, demographics, inclusion and exclusion criteria, study design, parameters for identifying autonomic dysreflexia, subject attrition, management or prophylactic therapies, diagnostic measurements, results, and conclusions. The forms were then pilot-tested by three experienced abstractors who evaluated three articles from the initial search. The data extraction form was then revised to more closely reflect the abstractors' evaluation experiences.

A primary search strategy was identified with the panel chair and PVA staff, and inclusion and exclusion criteria were established. Articles involving nontraumatic paralysis were excluded, as were articles focusing on pediatric patients. Articles discussing diseases that considered differential diagnoses without mention of autonomic dysreflexia were excluded. All articles written in English, Dutch, French, German, Italian, Spanish, and Swedish were included. Animal studies were included because of the uncertainty of the disease pathophysiology. Case studies also were included since the literature is lacking in nonanecdotal studies. Review articles and articles studying other diseases were included if autonomic dysreflexia was one of the outcomes.

A primary search of the MEDLINE database was performed on key words in this order: 1990 through the present, 1984 to 1990, 1976 to 1984, 1966 to 1975. Because no MeSH subheadings exist for autonomic dysreflexia, text word searches were performed using the following words: autonomic dysreflexia, autonomic hyperreflexia, paroxysmal hypertension, paroxysmal neurogenic hypertension, autonomic spasticity, sympathetic hyperreflex, mass reflex, neurovegetative syndrome, and vegetative dysregulation.

All of the abstracts were printed and reviewed, using the inclusion and exclusion criteria listed earlier. All of the articles that met those standards were copied. If an article did not have an abstract or if it was not clear that the article met the standards, the article was copied for further evaluation. Rare and foreign language articles were ordered from national sources, including the National Library of Medicine at the National Institutes of Health.

The data extraction forms were used to evaluate the approximately 300 articles found in the primary search. Secondary search topics, including eclampsia/preeclampsia in SCI women, were evaluated, and articles not included in the primary search were extracted and reviewed. Extracted information was compiled into evidence tables according to the main objective of the study. One table covered review articles, case studies, and studies that mentioned autonomic dysreflexia. Other tables included epidemiology and diagnosis, pathophysiology, prophylaxis, and management.

The methodologists, panel chair, and PVA staff identified a core subject of key papers (approximately 25) that covered the major issues. These articles were sent to panel members for study and consideration. During the subsequent period, the methodologists responded to queries from the panel chair and panel members and created a data set (from the Maryland Hospital Discharge database) of hospital admissions by people with SCI to address cost questions that might arise.

Strength of Evidence for the Recommendations

Several approaches are available for evaluating the quality of research studies and the evidence derived from them (Feinstein et al., 1985; Sackett et al., 1985). Most reviews use a hierarchy of evidence that places more weight on certain designs than on others. In studies of treatment outcomes, the greatest weight is usually placed on randomized, controlled trials, followed by observational studies, uncontrolled case series, and, finally, case reports. Two methods were used to evaluate and indicate the quality of the evidence on which each of the AD guideline recommendations was based.

The methodologists began by employing the hierarchy first discussed by Sackett (1989) and later enhanced by Cook et al. (1992) and the U.S. Preventive Health Services Task Force (1996). The levels of evidence are presented in table 1. Additionally, each study was evaluated for internal and external validity. Factors affecting internal validity (i.e., the extent to which the study provided valid information about the patients and the conditions studied) included sample size and statistical power; selection bias and inclusion criteria; selection of control groups, if any; randomization methods and comparability of groups; definition of interventions and/or exposures; definition of outcome measures; attrition rates; confounding variables; data collection methods and observation bias; and methods of statistical analysis. External validity—the extent to which the study findings are generalizable to conditions other than the setting of the study—was evaluated through an examination of the characteristics of the study population, the clinical setting and environment, and the investigators and providers of care. The resulting rankings were provided to the panel members during the deliberation process.

TABLE 1
Hierarchy of the Levels of Scientific Evidence

Level	Description
I	Large randomized trials with clear-cut results (low risk of error)
II	Small randomized trials with uncertain results (moderate to high risk of error)
III	Nonrandomized trials with concurrent or con temporaneous controls
IV	Nonrandomized trials with historical controls
V	Case series with no control

Next, each of the guideline recommendations was classified, depending upon the level of scientific evidence used in the development of the specific recommendation. The scheme used by the panel is shown in table 2 (Sackett, 1989; U.S. Preventive Health Services Task Force, 1989). It should be emphasized that these ratings, like those just described, represent the strength of the supporting evidence, not the strength of the recommendation itself. The strength of the recommendation is indicated by the language describing the rationale and is based on the discussion that occurred during the panel and expert reviewer deliberations.

TABLE 2.
Categories of the Strength of Evidence Associated

With the Recommendations

Category	Description
A	The guideline recommendation is supported by one or more level I studies
B	The guideline recommendation is supported by one or more level II studies
C	The guideline recommendation is supported only by level III, IV, or V studies

Sources: Sackett, D.L., Rules of evidence and clinical recommendations on the use of antithrombotic agents, *Chest* 95 (2 Supp) (1989): 2S–4S; and U.S. Preventive Health Services Task Force, Guide to clinical preventive services: *An assessment of the effectiveness of 169 interventions* (Baltimore: Williams and Wilkins, 1989).

Category A requires that the guideline recommendation be supported by scientific evidence from at least one properly designed and implemented randomized, controlled trial, providing statistical results that consistently support the guideline recommendation. Category B requires that the guideline recommendation be supported by scientific evidence from at least one small randomized trial with uncertain results; this category also may include small randomized trials with certain results where statistical power is low. Category C recommendations are supported either by nonrandomized, controlled trials or by trials in which no controls are used.

If a guideline recommendation is supported by literature that crosses two categories, both categories are reported (e.g., a guideline recommendation that is supported by both level II and III studies would be classified as categories B/C).

In situations where no published literature exists, consensus of the panel members and outside expert reviewers was used to develop the guideline recommendation and is indicated as “Expert consensus.”

Treatment Overview

Individuals with a spinal cord injury at the thoracic level T6 or above are generally at risk of developing autonomic dysreflexia, although cases involving injuries as low as T8 have been reported (Kurnick, 1956; Erickson, 1980). This condition must be properly assessed and treated quickly and efficiently at the earliest signs or symptoms to prevent a potentially life-threatening crisis. **Of most concern is the significant and potentially dangerous elevation in blood pressure (BP).**

Autonomic dysreflexia results from various noxious stimuli, which in turn trigger sympathetic hyperactivity. The two most common terms for this syndrome are autonomic hyperreflexia and autonomic dysreflexia. It also has been referred to as paroxysmal hypertension (Thompson and Witham, 1948), paroxysmal neurogenic hypertension (Mathias et al., 1976), autonomic spasticity (McGuire and Kumar, 1986), sympathetic hyperreflexia (Young, 1963), mass reflex (Head and Riddoch, 1917), and neurovegetative syndrome (Ascoli, 1971).

The spinal cord injured individual, family members, significant others, physicians, and nursing staff must understand both the underlying causes and the plans for corrective action to prevent autonomic dysreflexia from occurring or progressing.

Purpose of the Recommendations

These recommendations have been developed to aid in the care of individuals with a spinal cord injury who present to health care facilities. The purpose is to promote awareness of acute autonomic dysreflexia and its management and to assist health-care providers, particularly those who are not experienced with the care of individuals who have an SCI.

Pathophysiology of Autonomic Dysreflexia

Autonomic dysreflexia occurs after the phase of spinal shock in which reflexes return. Individuals with injuries above the major splanchnic outflow have the potential of developing autonomic dysreflexia.

The major splanchnic outflow is T6 through L2 (the level of the second lumbar vertebra). Intact sensory nerves below the level of the injury transmit impulses to the spinal cord, which ascend in the spinothalamic and posterior columns. Sympathetic neurons in the intermediolateral gray matter are stimulated by these ascending impulses. Sympathetic inhibitory impulses that originate above T6 are blocked due to the injury. Therefore, below the injury, there is a relatively unopposed sympathetic outflow (T6 through L2) with a release of norepinephrine, dopamine-beta-hydroxylase, and dopamine.

The release of these chemicals may cause piloerection, skin pallor, and severe vasoconstriction in the arterial vasculature, which can cause a sudden elevation in blood pressure. The elevated blood pressure may cause a headache. Intact carotid and aortic baroreceptors detect the hypertension.

Normally two vasomotor brainstem reflexes occur in an attempt to lower the blood pressure. (Parasympathetic activity originating from the dorsal motor nucleus of the vagus nerve—cranial nerve X—continues following a spinal cord injury.) The first compensatory mechanism is to increase parasympathetic stimulation to the heart via the vagus nerve to cause bradycardia. However, this bradycardia cannot compensate for the severe vasoconstriction. According to Poiseuille's formula, pressure in a tube is affected to the fourth power by change in radius (vasoconstriction) and only linearly by change in the flow rate (bradycardia). The second compensatory reflex is an increase in sympathetic inhibitory outflow from vasomotor centers above the spinal cord injury. However, the inhibitory impulses are unable to pass below the injury, and above the level of injury there may be profuse sweating and vasodilation with skin flushing (Kurnick, 1956; Erickson, 1980).

Signs and Symptoms

An individual may have one or more of the following signs or symptoms when he or she is having an episode of autonomic dysreflexia. Symptoms may be minimal or even absent, despite an elevated blood pressure. Some of the more common symptoms are:

- A sudden and significant increase in both the systolic and diastolic blood pressure above their usual levels, usually associated with bradycardia. **A individual with SCI above T6 often has a normal systolic blood pressure in the 90–110 mm Hg range.** Therefore, a blood pressure of 20 mm to 40 mm Hg above baseline may be a sign of autonomic dysreflexia (Guttman et al., 1965).
- Pounding headache.
- Profuse sweating above the level of the lesion, especially in the face, neck, and shoulders, or possibly below the level of the lesion.
- Goose bumps above or possibly below the level of the lesion.
- Flushing of the skin above the level of the lesion, especially in the face, neck, and shoulders, or possibly below the level of lesion.
- Blurred vision.

- Appearance of spots in the patient's visual fields.
- Nasal congestion.
- Feelings of apprehension or anxiety over an impending physical problem.
- Minimal or no symptoms, despite an elevated blood pressure.
- Cardiac arrhythmias, atrial fibrillation, ventricular contractions, and atrioventricular conduction abnormalities.

Causes

Autonomic dysreflexia has many potential causes. It is essential that the specific cause be identified and treated in order to resolve an episode of autonomic dysreflexia. Following are some of the more common causes (Kuric and Hixon, 1996):

- Bladder distention.
- Urinary tract infection.
- Bladder or kidney stones.
- Cystoscopy, urodynamics, or detrusor sphincter dysinergia.
- Epididymitis or scrotal compression.
- Bowel distention.
- Bowel impaction.
- Gallstones.
- Gastric ulcers or gastritis.
- Invasive testing.
- Hemorrhoids.
- Gastrocolic irritation.
- Appendicitis or another abdominal pathology or trauma.
- Menstruation.
- Pregnancy, especially labor and delivery.
- Vaginitis.
- Sexual intercourse.
- Ejaculation.
- Deep vein thrombosis.
- Pulmonary emboli.
- Pressure ulcers.
- Ingrown toenail.
- Burns or sunburn.
- Blisters.
- Insect bites.
- Contact with hard or sharp objects.
- Constrictive clothing, shoes, or appliances.
- Heterotopic bone.
- Fractures or other trauma.
- Surgical or diagnostic procedures.
- Pain.
- Temperature fluctuations.
- Any painful or irritating stimuli below the level of injury.

Treatment Recommendations

An individual with an SCI at or above T6 presents with an acute onset of signs and symptoms of autonomic dysreflexia.

NOTE: Pregnant women should be referred to an appropriate consultant.

1. **Check the individual's blood pressure.** (Scientific evidence—One level III study and one level V study; grade of recommendation—C.)

   Elevated blood pressures can be life-threatening and need immediate investigation and treatment (Guttman et al., 1965).

2. **If the blood pressure is not elevated, refer the individual to a consultant, if necessary.** (Expert consensus.)

   There may be other medical problems that are causing the signs and symptoms of autonomic dysreflexia.

3. **If the blood pressure is elevated and the individual is supine, immediately sit the person up.** (Scientific evidence—One level III study and one level V study; grade of recommendation—C.)

   Performing this maneuver may allow a pooling of blood in the lower extremities and may reduce the blood pressure (Guttman et al., 1965; Cole et al., 1967).

4. **Loosen any clothing or constrictive devices.** (Scientific evidence—One level III study and one level V study; grade of recommendation—C.)

   Performing this maneuver may allow a pooling of blood in the abdomen and lower extremities and may reduce the blood pressure (Guttman et al., 1965; Cole et al., 1967).

5. **Monitor the blood pressure and pulse frequently.** (Scientific evidence—Two level III studies and three level V studies; grade of recommendation—C.)

   Blood pressures have the potential of fluctuating quickly during an AD episode. Therefore, pressures need to be monitored every few minutes (every 2 to 5 minutes is commonly cited), until the individual is stabilized. Individuals with spinal cord injury usually have impaired autonomic regulation, and therefore blood pressures can rapidly fluctuate (Pollock et al., 1951; Kurnick, 1956; Guttman et al., 1965; Cole et al., 1967; Erickson, 1980; Kewalramani, 1980; Colachis, 1992; Kuric and Hixon, 1996).

6. **Quickly survey the individual for the instigating causes, beginning with the urinary system.** (Scientific evidence—Two level III studies and three level V studies; grade of recommendation—C.)

   The most common cause of autonomic dysreflexia is bladder distension (Guttmann and Whitteridge, 1947; Arieff et al., 1962; Wurster and Randall, 1975; Lindan et al., 1980; Kewalramani, 1980; Trop and Bennett, 1991; Colachis, 1992; Lee et al., 1995).

7. **If an indwelling urinary catheter is not in place, catheterize the individual.** (Expert consensus.)

   The most common cause of autonomic dysreflexia is bladder distension (Guttmann and Whitteridge, 1947; Arieff et al., 1962; Wurster and Randall, 1975; Lindan et al., 1980; Kewalramani, 1980; Trop and Bennett, 1991; Colachis, 1992; Lee et al., 1995).

8. **Prior to inserting the catheter, instill 2 percent lidocaine jelly (if readily available) into the urethra and wait several minutes.** (Expert consensus.)

   Catheterization can exacerbate autonomic dysreflexia. The lidocaine jelly may decrease the sensory input and relax the sphincter to facilitate catheterization.

9. **If the individual has an indwelling urinary catheter, check the system along its entire length for kinks, folds, constrictions, or obstructions and for correct placement of the indwelling catheter. If a problem is found, correct it immediately.** (Expert consensus.)

10. **If the catheter appears to be blocked, gently irrigate the bladder with a small amount of fluid, such as normal saline at body temperature. Avoid manually compressing or tapping on the bladder.** (Expert consensus.)

    Use of a larger volume or of a cold solution might irritate the bladder and exacerbate autonomic dysreflexia. If a lidocaine solution is readily available, irrigation with it may be beneficial by decreasing sensory input from the bladder. Bladder pressure or tapping may also increase sensory input and exacerbate autonomic dysreflexia.

11. **If the catheter is draining and the blood pressure remains elevated, proceed with step 16.** (Expert consensus.)

12. **If the catheter is not draining and the blood pressure remains elevated, remove and replace the catheter.** (Expert consensus.)

    Irrigating and changing the catheter should be done as quickly as possible. Pharmacologic management may become necessary if the blood pressure remains elevated and/or if catheter replacement is difficult.

13. **Prior to replacing the catheter, instill 2 percent lidocaine jelly (if readily available) into the urethra and wait several minutes.** (Expert consensus.)

    Catheterization can exacerbate autonomic dysreflexia. The lidocaine jelly may decrease the sensory input and relax the sphincter to facilitate catheterization.

14. **If the catheter cannot be replaced, consider attempting to pass a coude catheter, or consult a urologist.** (Expert consensus.)

    A coude catheter may be useful if there is an associated bladder neck obstruction.

15. **Monitor the individual's blood pressure during bladder drainage.** (Expert consensus.)

    Sudden decompression of a large volume of urine might produce hypotension. (See step 19.)

16. **If acute symptoms of autonomic dysreflexia persist, including a sustained elevated blood pressure, suspect fecal impaction.** (Scientific evidence—One level II study and two level V studies; grade of recommendation—B/C.)

    Fecal impaction is the second most common cause of autonomic dysreflexia (Colachis, 1992; Lee et al., 1995).

17. **If the elevated blood pressure is at or above 150 mm Hg systolic, consider pharmacologic management to reduce the systolic blood pressure without causing hypotension prior to checking for fecal impaction.** (Expert consensus.) ***If the blood pressure remains elevated but is less than 150 mm Hg systolic, proceed to step 20.*** (Scientific evidence—One level V study; grade of recommendation—C.)

    Reviewer opinion varied on whether the next step should be investigating other causes (e.g., fecal impaction) or initiating pharmacologic management. The control of hypertension may need to be addressed prior to digital stimulation or other diagnostic maneuvers, which may exacerbate autonomic dysreflexia.

    There are no studies showing the exact pressure at which the blood pressure becomes dangerous. For this guideline, the panel decided to adopt 150 mm Hg systolic BP as the value at which pharmacological treatment should be considered, based on Guttman et al. (1965). A person with an injury at or above T6 would be expected to have a baseline BP between 90 and 110 mm Hg. Guttman et al. (1965) described an AD episode as occurring when the systolic BP reached 20 to 40 mm Hg above baseline.

18. **Use an antihypertensive agent with rapid onset and short duration while the causes of autonomic dysreflexia are being investigated.** (Scientific evidence—One level V study; grade of recommendation—C.)

    Nifedipine and nitrates are the most commonly used agents (Dykstra et al., 1987; Braddom and Rocco, 1991; Thyberg et al., 1994). If nifedipine is used, it should be in the immediate release form. Bite-and-swallow is the preferred method of administration. Sublingual nifedipine administration may lead to erratic absorption. Nifedipine should be used with extreme caution in elderly people or in people with coronary artery disease. In individuals without spinal cord injury, immediate release nifedipine has been reported to cause shunting of the blood away from the heart and reflex tachycardia, and to result in an uncontrollable fall in blood pressure (Grossman et al., 1996).

    A review of the literature through October 1996 reveals that there have been no reported adverse effects from the use of nifedipine when used to treat autonomic dysreflexia. Nifedipine has been discussed in the literature as a prophylactic treatment for autonomic dysreflexia. Other drugs that have been used to treat autonomic dysreflexia with severe symptoms include hydralazine, mecamylamine, diazoxide, and phenoxybenzamine (Braddom and Rocco, 1991). In an appropriately monitored setting, the panel supports the use of an intravenous drip of sodium nitroprusside for rapid titration of blood pressure. If 2 percent nitroglycerin ointment is used, 1 inch may be applied to the skin, above the level of spinal cord injury. There are no studies reporting on the best agent to use.

19. **Monitor the individual for symptomatic hypotension.** (Expert consensus.)

    Treat severe (symptomatic) hypotension by laying down the individual and elevating the legs. Additional corrective measures are not usually required. However, if indicated, consider intravenous fluids and adrenergic agonists (i.e., in a monitored setting, intravenous norepinephrine for reversal of severe hypotensive events).

20. **If fecal impaction is suspected, check the rectum for stool, using the following procedure. With a gloved hand, instill a topical anesthetic agent such as 2 percent lidocaine jelly generously into the rectum. Wait approximately 5 minutes for sensation in the area to decrease. Then, with a gloved hand, insert a lubricated finger into the rectum and check for the presence of stool. If present, gently remove, if possible. If autonomic dysreflexia becomes worse, stop the manual evacuation. Instill additional topical anesthetic and recheck the rectum for the presence of stool after approximately 20 minutes.** (Scientific evidence—One level II study, two level V studies; grade of recommendation—B/C.)

    A rectal examination may exacerbate autonomic dysreflexia (Watkins, 1938; Bors and French, 1952). Instillation of a local anesthetic agent may

decrease the occurrence of autonomic dysreflexia during the exam.

21. **Monitor the individual's symptoms and blood pressure for at least 2 hours after resolution of the autonomic dysreflexia episode *to make sure that it does not recur.*** (Expert consensus.)

    The hypertension and symptoms may have resolved because of the medication rather than the treatment of the cause. Symptoms managed by pharmacologic treatment may begin to reverse themselves within this time frame.

22. ***If there is poor response to the treatment specified above and/or if the cause of the autonomic dysreflexia has not been identified, strongly consider admitting the individual to the hospital to be monitored, to maintain pharmacologic control of the blood pressure, and to investigate other causes of the autonomic dysreflexia.*** (Scientific evidence—One level V study; grade of recommendation—C.)

    Because of the loss of sensation, individuals with spinal cord injury can have significant pathology with minimal symptoms. These may include problems such as acute abdominal pathology, long bone fractures, and ingrown toenails (Braddom and Rocco, 1991). Individuals with spinal cord injury frequently may have a positive urine culture. However, this may not be the precipitating cause for autonomic dysreflexia, and therefore other causes of autonomic dysreflexia also should be investigated.

23. **Document the episode in the individual's medical record. This record should include the presenting signs and symptoms and their course, treatment instituted, recordings of blood pressure and pulse, and response to treatment.**

    The effectiveness of the treatment may be evaluated according to the level of outcome criteria reached:

    - ***The cause of the autonomic dysreflexia episode has been identified.***
    - ***The blood pressure has been restored to normal limits for the individual (usually 90 to 110 systolic mm Hg for a tetraplegic individual in the sitting position).***
    - ***The pulse rate has been restored to normal limits.***
    - ***The individual is comfortable, with no signs or symptoms of autonomic dysreflexia, of increased intracranial pressure, or of heart failure.***

    (Expert consensus.)

24. **Once the individual with spinal cord injury has been stabilized, review the precipitating cause with the individual, members of the individual's family, significant others, and care givers. This process entails adjusting the treatment plan to ensure that future episodes are recognized and treated to prevent a medical crisis or, ideally, are avoided altogether. The process also entails discussion of autonomic dysreflexia in the spinal cord injury individual's education program, so that he or she will be able to recognize early onset and obtain help as quickly as possible. An individual with a spinal cord injury should be given a written description of treatment for autonomic dysreflexia at the time of discharge that can be referred to in an emergency.** (Expert consensus.)

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Glossary of Terms

Autonomic dysreflexia: also known as hyperreflexia, an uninhibited sympathetic nervous system response to a variety of noxious stimuli occurring in persons with spinal cord injury at the thoracic 6 (T-6) level and above.

Autonomic spasticity: a state of increased muscular tone with exaggeration of the tendon reflexes having independence or freedom from control by external forces.

Coude catheter: a urethral catheter that has a slight upward bend and narrowing at the tip to allow easier passage through the urethra past the sphincter and prostate into the bladder.

Dysreflexia: another term used to describe autonomic dysreflexia

Evidence tables: charts developed by methodologists supporting guideline development that describe scientific literature citations and the type and quality of the reported research for use in developing clinical practice guidelines.

Heterotopic bone: bone formation in abnormal soft tissue; locations documented by radiograph or bone scan; common locations include the hip and/or knee, which can restrict flexion to less than 90%.

Hyperreflexia: a condition in which the deep tendon reflexes are exaggerated.

Hypotension: subnormal arterial blood pressure.

Meta-analysis: the process of using statistical methods to combine the results of different studies; systematic, organized, and structured evaluation of a problem using information, commonly in the form of statistical tables, etc., from a number of different studies of the problem.

Neurovegetative syndrome: another term used to describe autonomic dysreflexia.

Paroxysmal neurogenic hypertension: another term used to describe autonomic dysreflexia.

Poiseuille's formula: in the centimeter-gram-second (CGS) system, the unit of viscosity equal to 1 dyne-second per square centimeter and to 0.1 pascal second.

Spinal shock: a temporary flaccid paralysis and loss of all reflex activity (below the level of spinal cord injury). This occurs at the time of injury and appears to be the result of sudden loss of supraspinal excitatory activity. Sacral parasympathetic activity is diminished accounting for bowel and bladder atony. After a period of spinal shock, reflex activity returns—usually within 6 months.

Splanchnic sympathetic outflow: sympathetic nerve outflow from the thoracic sympathetic ganglia (T6 through the second lumbar (L2)) to the viscera and blood vessels within the gastrointestinal (GI) tract.

Strength of evidence: a method for grading the type and quality of research reported in the scientific literature for a given topic. These levels of evidence are used by methodologists to construct evidence tables for development of clinical practice guideline recommendations.

Sympathetic hyperactivity: denoting the sympathetic part of the autonomic nervous system having abnormally great activity.

Sympathetic hyperreflexia: another term used to describe autonomic dysreflexia.

Tetraplegia: impairment or loss of motor and/or sensory function below the cervical segments of the spinal cord due to damage of the neural elements within the spinal cord.

Vegetative dysregulation: another term used to describe autonomic dysreflexia.

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NOTES

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