SPINAL CORD MEDICINE

AUTONOMIC DYSREFLEXIA

CLINICAL PRACTICE GUIDELINES

Acute Management of Autonomic Dysreflexia:

Individuals with Spinal Cord Injury Presenting to Health-Care Facilities

2nd Edition

consortium for SPINAL CORD MEDICINE
CLINICAL PRACTICE GUIDELINES

Administrative and financial support provided by Paralyzed Veterans of America
Printing provided by Eastern Paralyzed Veterans Association

Consortium for Spinal Cord Medicine Member Organizations

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This guide has been prepared based on scientific and professional information known about the treatment of autonomic dysreflexia following spinal cord injury in 2000. Users of this guide should periodically review this material to ensure that the advice herein is consistent with current reasonable clinical practice.

July 2001

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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 thoracic level six (T6). The resolution of AD requires quick and decisive treatment. Spinal cord medicine health-care providers are very familiar with the diagnosis and treatment of AD. However, because of the rapid onset of AD and the potentially severe symptoms, individuals with this condition are often rushed to the nearest health-care facility that may be staffed by health-care providers who have little or no experience in the treatment of AD. The purpose of these guidelines is to make available information that can be used by health-care providers when an individual with signs and symptoms of autonomic dysreflexia presents to their facility.

The 1st edition was published in 1997. In order to make sure these guidelines were kept up-to-date, the Consortium steering committee decided that it was time to update the first edition. The 2nd edition underwent the same detailed development process as did the 1st edition. An extensive literature search and methodological review were undertaken and the members of the 1st edition development panel reconvened. The 2nd edition takes into consideration children, adolescents, and pregnant women with SCI. Therefore, an expert in pediatric rehabilitation, Lawrence Vogel, MD, and an expert in obstetrics and gynecology with a special interest in women with SCI, Emily Baker, MD, joined the panel. The draft was circulated to expert field reviewers and further revisions were made based on their comments and the literature.

Overall, no significant changes in evaluation or management were identified in the literature. Therefore, the management of adults presenting to health-care facilities with acute AD is essentially unchanged. There were, however, a number of excellent suggestions from field reviewers that helped "fine tune" these guidelines. Detailed literature reviews were undertaken in the areas of pediatrics and obstetrics as they related to AD. The literature regarding evaluation and management of AD was extremely limited in these areas. This was of particular concern with regards to making recommendations on the diagnosis and management of AD in pregnant women. During pregnancy there are a number of other causes and treatments, depending on the type of hypertension. Therefore, the consortium steering committee recommended that these guidelines be limited to "when to refer" a pregnant woman exhibiting signs and symptoms of AD.

As with the 1st edition, this publication would not have been possible without the strong support and leadership of Paralyzed Veterans of America (PVA). Eastern Paralyzed Veterans Association (EPVA) generously volunteered to print this edition. Members of the AD panel wish to extend a special thanks to Dawn M. Sexton and J. Paul Thomas for the countless hours they spent on this project.

We hope that these guidelines play an important role in the evaluation and management of individuals presenting with signs and symptoms of AD. Moreover, it is the panel's hope that these guidelines stimulate further clinical studies in this important area.

Todd A. Linsenmeyer, MD
Chairman, Autonomic Dysreflexia Guideline Development Panel

Preface

The 2nd edition of *Acute Management of Autonomic Dysreflexia: Individuals with Spinal Cord Injury Presenting to Health-Care Facilities* marks a significant milestone for the Consortium for Spinal Cord Medicine. When we first began developing clinical practice guidelines (CPGs) in 1994, we recognized that the state-of-the-art of knowledge and practice should evolve as new research is published. From the earliest days, we recognized that periodic review and updating of each CPG would be necessary. To that end, the autonomic dysreflexia (AD) panel reconvened, under continuing leadership of the very capable Todd Linsenmeyer, MD. We are proud of the work of that panel and of the methodologists at Duke University for this second edition. We also appreciate the efforts of our expert reviewers, who contribute so much to the quality of the final product.

Information about AD in women with spinal cord injury (SCI) who are pregnant has been added to this document, but the dearth of good clinical research and publications prevents comprehensive guidelines development. By setting a research agenda in this edition, we hope to stimulate experts in obstetrics to publish future studies. Likewise, information about identification and treatment of AD in children with SCI has been added to this edition, but future research is also necessary in this population.

So now the challenge to you, the reader, is to incorporate the information in these clinical practice guidelines into your practice and your educational programs for patients, their families and health-care providers in training. The steering committee requests your comments about this edition. Do you find it to be useful? What related publications would increase the effectiveness of these guidelines in your environment? Would a downloadable presentation be of help to you, as an educator of health-care professionals? Would a poster or other graphic depiction be helpful?

We plan to publish an updated consumer guide for AD in the future to help consumers educate themselves and their health-care providers about this crucial topic. What additional materials would you find helpful for education of consumers?

I am profoundly thankful to the leadership of the Paralyzed Veterans of America—including the entire PVA board of directors and of PVA's senior officers, specifically National President Joseph L. Fox, Sr., Immediate Past President Homer S. Townsend, Jr., Executive Director Keith W. Wingfield, Deputy Executive Director John C. Bollinger, and Associate Executive Director for Health Policy John L. Carswell—for providing vision and support to the Consortium for Spinal Cord Medicine. Without PVA, the consortium and this publication would not exist. Likewise, I am grateful to my colleagues on the steering committee who donate their time and expertise to the development, review, and revision of each set of guidelines. Finally, I am deeply indebted to J. Paul Thomas and Dawn M. Sexton for their tireless efforts on behalf of the consortium, the writing panel, and the reviewers. You all have accomplished so much on behalf of those whom we serve.

Kenneth C. Parsons, MD
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Acknowledgements

The Consortium for Spinal Cord Medicine Steering Committee representatives and Autonomic Dysreflexia panel chair and members of the AD guideline development panel wish to express special appreciation to the individuals and professional organizations who are members of the Consortium for Spinal Cord Medicine and to the expert health-care providers, researchers, and other professionals who critiqued the draft documents. Special thanks go to the consumers, advocacy organizations, and staffs of the numerous medical facilities and spinal cord injury rehabilitation centers who contributed their time and expertise to the development of both the 1st and 2nd editions of these guidelines.

The consortium and the guidelines development panel would like to express their gratitude and appreciation to Eastern Paralyzed Veterans Association (EPVA) (a consortium member organization) for magnanimously funding the printing of this second edition. Without EPVA's generosity these guidelines would not be in print and available.

Kit N. Simpson, PhD, Andrea K. Biddle, PhD, and their fine staff in the Health Policy and Administration Department at the University of North Carolina (UNC) 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 for the 1st edition. UNC's fine work was supplemented by David Matchar, MD, and his staff at Duke University in Durham, North Carolina. Special appreciation must be expressed for Duke's flexibility in meeting the goals and demands of the panel in performing the additional literature searches and evaluations of quality and strength of the evidence found in the areas of pregnancy and pediatrics.

Members of the Consortium Steering Committee, representing 19 professional organizations, were joined by thirty four expert reviewers who provided outstanding scientific and clinical analysis for the 1st edition. A second team of 38 expert reviewers assisted the guidelines development panel with their insight and analysis on the 2nd edition. Their valuable comments have 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 guidelines development process. Attorney William H. Archambault, of Goodman, West & Filetti, PLLC, in Charlottesville, Virginia, conducted a comprehensive analysis of the legal and health policy issues associated with this complex, multifaceted topic.

The panel would again like to express its gratitude to the Clinical Practice Committee of the American Paraplegia Society (APS) for turning its AD guidelines over to the consortium in 1996, resulting in the 1st edition of these guidelines, published by the consortium in February 1997.

The AD guidelines development panel is grateful for the many administrative, organizational, and technical support services provided by various departments of the Paralyzed Veterans of America (PVA). The panel recognizes J. Paul Thomas and Dawn M. Sexton in the Consortium Coordinating Office and the Health Policy Department for their untiring efforts in guiding the panel through the guidelines development process; Fred Cowell in the Health Policy Department for his cogent comments reflecting the perspective of consumers; James A. Angelo and Patricia E. Scully in the Communications Department for their guidance in writing, editing, and indexing; medical editor Joellen Talbot for her excellent technical review and editing of both editions of the guidelines; and graphic designer

Chris Campbell for designing the document. Appreciation is expressed for the steadfast commitment and enthusiastic advocacy of the entire PVA board of directors and of PVA's senior officers, including National President Joseph L. Fox, Sr., Immediate Past President Homer S. Townsend, Jr., Executive Director Keith W. Wingfield, Deputy Executive Director John C. Bollinger, and John L. Carswell, Associate Executive Director for Health Policy. Their generous financial support has made the consortium and its guidelines development process a successful venture.

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Summary of Recommendations

1. Recognize the signs and symptoms of autonomic dysreflexia.

   [**Note:** See *Signs and Symptoms* on page 9 and *Recommendations and Supporting Evidence* on page 12.]

2. Check the individual's blood pressure.

   - A sudden, significant increase in both the systolic and diastolic blood pressure above their usual levels, frequently associated with bradycardia. An 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.
   - Systolic blood pressure elevations more than 15-20 mm Hg above baseline in adolescents with SCI or more than 15 mm Hg above baseline in children with SCI may be a sign of AD.

3. If a pregnant woman with a spinal cord injury at T6 or above presents with signs and symptoms of autonomic dysreflexia, consider referral to an obstetric health-care provider under the following circumstances:

   - Determination of choice of antihypertensive medication.
   - Persistent hypertension after resolution of the acute autonomic dysreflexia episode.
   - Persistent symptoms of autonomic dysreflexia despite acute care measures.
   - Life-threatening autonomic dysreflexia.
   - Autonomic dysreflexia episode occurring in the third trimester of pregnancy.
   - Hypotension requiring pharmacological treatment.
   - First episode of autonomic dysreflexia during the pregnancy.
   - Presence of vaginal bleeding or suspicion of labor.
   - Decisions to be made about long-term medication use.
   - Unclear about the causes, signs, and symptoms, despite a normal blood pressure.

4. If signs or symptoms of AD are present, but the blood pressure is not elevated and the cause has not been identified, refer the individual to an appropriate consultant depending on symptoms.

5. If the blood pressure is elevated, immediately sit the person up if the individual is supine.

6. Loosen any clothing or constrictive devices.

7. Monitor the blood pressure and pulse frequently.

8. Quickly survey the individual for the instigating causes, beginning with the urinary system.

9. If an indwelling urinary catheter is not in place, catheterize the individual.

10. Prior to inserting the catheter, instill 2 percent lidocaine jelly (if immediately available) into the urethra and wait 2 minutes, if possible.

11. 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. If a problem is found, correct it immediately.

12. If the catheter appears to be blocked, gently irrigate the bladder with a small amount (10-15 cc) of fluid, such as normal saline at body temperature. Irrigation should be limited to 5-10 ml for children under 2 years of age and to 10-15 ml in older children and adolescents. Avoid manually compressing or tapping on the bladder.

13. If the catheter is draining and the blood pressure remains elevated, proceed with recommendation 18.

14. If the catheter is not draining and the blood pressure remains elevated, remove and replace the catheter.

15. Prior to replacing the catheter, instill 2 percent lidocaine jelly (if immediately available) into the urethra and wait 2 minutes, if possible.

16. If difficulties arise in replacing the catheter, consider attempting to pass a coude catheter or consult a urologist.

17. Monitor the individual's blood pressure during bladder drainage.

18. If acute symptoms of autonomic dysreflexia persist, including a sustained elevated blood pressure, suspect fecal impaction.

19. 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 recommendation 22.

20. Use an antihypertensive agent with rapid onset and short duration while the causes are being investigated.

21. Monitor the individual for symptomatic hypotension.

22. If fecal impaction is suspected and the elevated blood pressure is less than 150 mm Hg systolic, 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 2 minutes if possible 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.

23. If the precipitating cause of the AD episode has not yet been determined, check for less frequent causes. (See *Potential Causes* on page 10.) The individual may first need to be admitted to the hospital for monitoring to maintain pharmacological control of the blood pressure. Particularly if there is a poor response to the treatment specified above.

24. Following an episode of autonomic dysreflexia, instruct individuals who are outpatients to monitor symptoms and blood pressure for at least 2 hours after resolution of the episode to make sure that it does not reoccur.

    - Educate the individual to seek immediate medical attention if it reoccurs.
    - Monitor inpatients closely for at least 2 hours, as deemed necessary by the health-care provider.
    - Seek the pregnant woman's obstetrical-care provider for evaluation.

25. Consider admitting the individual to the hospital for monitoring to maintain pharmacologic control of the blood pressure, and to investigate other causes:

    - If there is poor response to the treatment specified above.
    - If the cause of the dysreflexia has not been identified.
    - If there is suspicion of an obstetrical complication.

26. Document the episode in the individual's medical record, including:

    - Presenting signs and symptoms and their course.
    - Treatment instituted.
    - Recordings of blood pressure and pulse.
    - Response to treatment.

    Evaluate effectiveness of the treatment 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.
    - An education plan has been completed and included preventive and emergency management guidance.

27. Once the individual with spinal cord injury has been stabilized, review the precipitating cause of the AD episode with the individual, family members, significant others, and care givers. This preventive 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.
    - Discussing autonomic dysreflexia during the individual's education program, so that he or she will be able to minimize risks known to precipitate AD, solve problems, recognize early onset, and obtain help as quickly as possible.
    - Providing the individual with education about the prevention and treatment of autonomic dysreflexia at the time of discharge that can be referred to in an emergency.
28. Schedule detailed medical evaluations for individuals with recurrent autonomic dysreflexia.

The Consortium for Spinal Cord Medicine

Seventeen organizations, including PVA, joined together to form a consortium in June 1995 to develop evidence-based clinical practice guidelines in spinal cord medicine. Today, nineteen organizations compose the consortium. A steering committee governs its operation, leads the guideline development process, identifies topics, and selects panels of experts for each topic. The steering committee is composed of one representative with clinical practice guideline experience from each consortium member organization. PVA provides financial resources, administrative support, and programmatic coordination of consortium activities.

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 the Agency for Health Care Policy and Research (AHCPR). The consortium's model is interdisciplinary, in that it reflects the multiple information needs of the spinal cord practice community; it is responsive, with a 12-month timeline for completion of each set of guidelines; and it is reality-based, in that the time and energy of the busy health-care professionals who serve as panel members and expert reviewers are used efficiently and effectively.

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

The Consortium for Spinal Cord Medicine is unique to the clinical practice guideline development field. 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 processes. The consortium expects to initiate work on two or more 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 Consortium for Spinal Cord Medicine consists of twelve steps, leading to panel consensus and organizational endorsement. After the steering committee chooses a topic, a panel of experts is selected. Panel members must have demonstrated leadership in the topic area through independent scientific investigation and publication. Following a detailed explication and specification of the topic by select steering committee and panel members, consultant methodologists review the international literature, prepare evidence tables that grade and rank the quality of research, and conduct statistical meta-analyses and other specialized studies, as needed. The panel chair then assigns specific sections of the topic to the panel members, based on area of expertise. Writing begins on each component using the references and other materials furnished by the methodology support group.

After the panel members complete their sections, a draft document is generated during the first full meeting of the panel. The 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 list of references, are added, and the full document is sent to legal counsel for review.

After legal analysis to consider antitrust, restraint-of-trade, and health policy matters, the draft document is reviewed by clinical experts from each of the consortium organizations plus other select clinical experts and consumers. The review comments are assembled, analyzed, and entered in to a database, and the document is revised to reflect the reviewers' comments. Following a second legal review, the draft 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 receives 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 medicine practice community are numerous. Among the more significant applications and results are:

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

Methodology

The overall strategy for finding evidence relevant to the management of autonomic dysreflexia in individuals with SCI paralleled that used in earlier guidelines (Consortium for Spinal Cord Medicine, 1997) and is modeled after the methods recommended by the Agency for Health Care Policy and Research (1993) and the Institute of Medicine (1990). Three separate search strategies were developed to find literature addressing each of the three foci of the guidelines revision. These literature searches provide a general update of the literature on autonomic dysreflexia since the original guidelines were published and comprehensive reviews of the literature on the management of autonomic dysreflexia during pregnancy and delivery and on the use of sildenafil by men with SCI.

First, a search of the MEDLINE database from January 1996 to June 1999 was conducted to identify articles published since the original guidelines (Consortium for Spinal Cord Medicine, 1997). To identify issues related to autonomic dysreflexia during pregnancy and delivery in women with SCI, a topic not covered in the original guidelines, searches were conducted for the period 1966 through June 1999. To address the use of sildenafil in men with SCI, searches were conducted from June 1996 (the time at which the first article appeared on its use for erectile dysfunction in general) through June 1999.

Because no Index Medicus subject headings (MeSH) existed until recently for autonomic dysreflexia, text word searches were conducted using the following key words: autonomic dysreflexia, autonomic hyperreflexia, paroxysmal hypertension, paroxysmal neurogenic hypertension, autonomic spasticity, sympathetic hyperreflex, mass reflex, neurovegetative syndrome, and vegetative dysregulation. To identify autonomic dysreflexia occurring during pregnancy and labor in women or associated with the use of sildenafil by men, the text word searches were combined with the MeSH subheadings pregnancy, eclampsia/preeclampsia, sildenafil, erectile dysfunction, and impotence.

As was the case in the original guidelines, inclusion and exclusion criteria were established for the literature searches. Articles involving nontraumatic paralysis were excluded, as were articles that focused on pediatric patients or that considered differential diagnoses without mention of autonomic dysreflexia. Case series and small cohort studies were included because the literature is relatively lacking in nonobservational studies. Animal studies were included because of the uncertainty of the disease pathophysiology. Unlike the original guidelines, only articles published in English were included.

More than 366 abstracts from the literature searches were reviewed, using the inclusion and exclusion criteria, to determine relevance to management of autonomic dysreflexia in general, during pregnancy and delivery for women, and following use of sildenafil by men. Those abstracts that met the criteria were retrieved. If an article did not have an abstract or if its relevance was unclear, the article was retrieved for further evaluation. Additionally, the reference lists of all relevant articles were reviewed to identify additional or "fugitive" articles.

The data extraction forms developed for the original guidelines were enhanced to further standardize the data used for extraction. These extraction forms were used to evaluate the 32 articles that met the stated inclusion/exclusion criteria. Extracted information was compiled into evidence tables according to topic area and disseminated to panel members for use in writing the revised guideline recommendations.

Supplementary Literature Review

Following preliminary discussions by the expert panel, it was decided to expand the guideline to include the pediatric population and pregnancy as well as update the overall search on AD to extend from January 1966 to May 2000. For the pediatric search, 41 articles were identified

and all were excluded (i.e., were either not relevant or had been previously identified in the nonpediatric searches). For the extended search, 64 articles were identified, of which 19 were retained and summarized in evidence tables.

Strength of Scientific Evidence for the Recommendations

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), presented in table 1. 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 individuals and 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 were 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 writing and deliberation process. If the literature supporting a guideline recommendation came from two or more levels, the level of each study is reported (e.g., in the case of a guideline recommendation that was supported by two studies, one a level III, the other a level V, the scientific evidence was indicated as "III/V").

Next, each of the guideline recommendations was classified, according to the level of scientific evidence used in the development of the recommendation. The schema used by the panel is shown in table 2. 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.

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 statement. 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 for which no controls were used (observational studies).

If a guideline recommendation was supported by literature that crossed two categories, both categories were reported (e.g., a guideline recommendation that included both level II and III studies would be classified as categories B/C and be indicated as "grade of recommendation–B/C"). In situations where no published literature existed, consensus of the panel members and outside expert reviewers was used to develop the guideline recommendation and the grade of recommendation is indicated as "expert consensus."

TABLE 1
Hierarchy of the Levels of Scientific Evidence

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

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*, 2nd Edition (Baltimore: Williams and Wilkins, 1996).

TABLE 2
Categories of the Strength of Evidence Associated with the Recommendation

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*, 2nd Edition (Baltimore: Williams and Wilkins, 1996).

Strength of Panel Opinion

After deliberation and discussion of each guideline recommendation and the supporting evidence, the level of expert panel agreement with the recommendation was assessed as either low, moderate, or strong. In this assessment, each panel member was asked to indicate his or her level of agreement on a 5-point scale, with 1 corresponding to neutrality and 5 representing maximum agreement. Panel members could abstain from this voting process for a variety of reasons, such as lack of expertise associated with a particular guideline recommendation. Subsequently, the scores were aggregated across the panel members, and an arithmetic mean was calculated. This mean score was then translated into low, moderate, or strong, as shown in table 3.

TABLE 3
Levels of Panel Agreement with the Guideline Recommendation

Level	Mean Agreement Score
Low	1.0 to less than 2.33
Moderate	2.33 to less than 3.67
Strong	3.67 to 5.0

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 (Erickson, 1980; Kurnick, 1956). 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 (AD) 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). Bladder and bowel distention are the most common causes of AD (Colachis, 1992).

Pathophysiology of Autonomic Dysreflexia

Autonomic dysreflexia does not occur until after the phase of spinal shock when 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 vertebral levels. Intact sensory nerves below the level of the injury transmit noxious afferent 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. It is important to note that this may be a "relative" slowing of the heart; however, the heart rate may not drop as low as 60 per minute (which is generally defined as being bradycardia). This drop in heart rate 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, inhibitory impulses with a negative feedback are unable to pass below the injury and cannot dilate the splanchnic bed to accommodate the excessive amount of circulating blood resulting from increased peripheral resistance. Above the level of injury there may be profuse sweating and vasodilation with skin flushing (Erickson, 1980; Kurnick, 1956).

Pregnant Women

Pregnancy is associated with changes in every organ system in the body, discussion of which is beyond the scope of this document. Information can be found in basic obstetric textbooks and in review articles regarding spinal cord injury in pregnancy (see Baker and Cardenas, 1996). Cardiovascular changes include the following: (a) expansion of plasma and blood volume, (b) significant increase in cardiac output and heart rate, (c) decreased systemic resistance, and (d) decreased blood pressure until the third trimester, when the blood pressure returns to baseline. Hypertension is common in pregnancy due to preeclampsia, gestational hypertension, and chronic hypertension.

Nonobstetrical care providers who work with SCI pregnant women need to be aware of the predictable physiologic and anatomic changes of pregnancy. Obstetrical care providers need to be aware of the diagnosis, prevention, and treatment of AD and the risk of unattended birth. Failure to recognize and treat AD has caused intracranial hemorrhage and death in pregnant women

(Abouleish et al., 1989, McGregor and Meeuwsen, 1985).

The pathophysiology, signs, and symptoms of AD in pregnant women are the same as in women who are not pregnant. In spinal cord injured women, hypertension would prompt a consideration of AD. The physical changes of pregnancy prompt many stimuli that are also potent triggers of AD. The process of labor and delivery is the most intense stimulus and the time of greatest risk for AD. Cases of AD have occurred antepartum, intrapartum, and postpartum.

There are some clinical features that make distinguishing preeclampsia from autonomic dysreflexia difficult. Both preeclampsia and AD are often diagnosed while the woman is in labor. The clinical presentation of preeclampsia may vary from the typical triad of hypertension, proteinuria, and edema. Some clinical facts may be helpful. Preeclampsia essentially never occurs prior to 24 weeks' gestation. The major morbidity of preeclampsia is caused by hypertension, so treatment of hypertension is needed regardless of the diagnosis. In addition, an obstetrical care provider should be involved to diagnose preeclampsia, assess fetal well-being, and consider the potential of delivery.

Given the current data, it is not possible to recommend the best mode of delivery or comment on incremental risks for AD with induction of labor and delivery. Epidural anesthesia is very effective in controlling the hypertension associated with uterine contractions (Colachis, 1992; Ravindran et al.; 1981).

Children and Adolescents

Approximately 20 percent of all spinal cord injuries that occur in the United States affect children and adolescents (Hadley et al., 1988; Haffner et al., 1993; Hamilton and Myles, 1992; Kewalramani et al., 1980; Nobunaga et al., 1999; Osenbach and Menezes, 1992; Ruge et al., 1988; Vogel and DeVivo, 1996, 1997). Manifestations and complications of SCI in children and adolescents are unique because of the distinctive anatomic and physiologic features related to growth and development inherent in the pediatric population (Betz and Mulcahey, 1996; Massagli, 2000; Vogel, 1997; Vogel et al., 1997).

Autonomic dysreflexia occurs in approximately 16% of children and adolescents with SCI (The Annual Statistical Report for the Shrine Spinal Cord Injury Units, May 2001, published by the National Spinal Cord Injury Statistical Center, Birmingham, AL). As with adults with SCI, bladder and bowel issues are the most common inciting factors of autonomic dysreflexia in children and adolescents with SCI.

The pathophysiology, signs and symptoms, and management of AD in children and adolescents with spinal cord injuries are similar to those of the adult SCI population. The major differences relate to developmental variations in blood pressure in children and adolescents, appropriate sizing of blood pressure cuffs, the relative inability of children to communicate their symptoms, and the varying dependence of children and adolescents upon their parents or guardians.

The care and management of children and adolescents with SCI must be responsive to developmental changes and therefore must vary as a child grows. Because of the central role of the family in a child's life, care must be family-centered, which means that parents must be integrally involved in decision making (Betz and Mulcahey, 1994; Bray, 1978; Shelton et al., 1989). In addition, children and adolescents must be included in a significant and meaningful way in health-care and decision-making processes.

All spinal cord-injured individuals, 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.

Signs and Symptoms

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

- A sudden, significant increase in both the systolic and diastolic blood pressure above their usual levels, frequently associated with bradycardia. An 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).
- Systolic blood pressure elevations more than 15–20 mm Hg above baseline in adolescents with SCI or more than 15 mm Hg above baseline in children with SCI may be a sign of AD.
- Pounding headache.

- Bradycardia (may be a relative slowing so that the heart rate is still within the normal range).
- Profuse sweating above the level of the lesion, especially in the face, neck, and shoulders, or possibly below the level of the lesion.
- Piloerection or goose bumps above or possibly below the level of the lesion.
- Cardiac arrhythmias, atrial fibrillation, premature ventricular contractions, and atrioventricular conduction abnormalities.
- 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 visual fields.
- Nasal congestion.
- Feelings of apprehension or anxiety over an impending physical problem.
- Minimal or no symptoms, despite a significantly elevated blood pressure (silent autonomic dysreflexia).

Potential 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 AD and to prevent recurrence. Any painful or irritating stimuli below the level of injury may cause AD. Bladder and bowel problems are the most common causes of AD. Following are some of the more common potential causes (Kuric and Hixon, 1996):

Urinary System

- Bladder distention.
- Bladder or kidney stones.
- Blocked catheter.
- Catheterization.
- Urologic instrumentation, such as cystoscopy or testing requiring catheterization.
- Detrusor sphincter dyssynergia.
- Shock wave lithotripsy.
- Urinary tract infection.

Gastrointestinal System

- Appendicitis.
- Bowel distention.
- Bowel impaction.
- Gallstones.
- Gastric ulcers or gastritis.
- GI instrumentation.
- Hemorrhoids.

Integumentary System

- Constrictive clothing, shoes, or appliances.
- Contact with hard or sharp objects.
- Blisters.
- Burns, sunburn or frostbite.
- Ingrown toenail.
- Insect bites.
- Pressure ulcers.

Reproductive System

- Sexual intercourse.
- Sexually transmitted diseases (STDs).

MALE

- Ejaculation.
- Epididymitis.
- Scrotal compression (sitting on scrotum).
- Electroejaculation and vibratory stimulation to induce an ejaculate.

FEMALE

- Menstruation.
- Pregnancy, especially labor and delivery.
- Vaginitis.

Other Systemic Causes

- Boosting (an episode of AD intentionally caused by an athlete with SCI in an attempt to enhance physical performance).
- Deep vein thrombosis.
- Excessive alcohol intake.
- Excessive caffeine or other diuretic intake.
- Fractures or other trauma.
- Functional electrical stimulation.
- Heterotopic bone.
- Over-the-counter or prescribed stimulants.
- Pulmonary emboli.
- Substance abuse.
- Surgical or invasive diagnostic procedures.

Recommendations and Supporting Evidence

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

1. **Recognize the signs and symptoms of autonomic dysreflexia, including:**

   - **Elevated blood pressure.**
   - **Pounding headache.**
   - **Bradycardia (may be a relative slowing so that the heart rate is still within the normal range).**
   - **Profuse sweating above the level of the lesion, especially in the face, neck, and shoulders, or possibly below the level of the lesion.**
   - **Piloerection or goose bumps above or possibly below the level of the lesion.**
   - **Cardiac arrhythmias, atrial fibrillation, premature ventricular contractions, and atrioventricular conduction abnormalities.**
   - **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 a significantly elevated blood pressure (silent autonomic dysreflexia).**

   (Scientific evidence–None; Grade of recommendation–Expert consensus; Strength of panel opinion–Strong)

An individual may have one or more of these signs and symptoms when experiencing an episode of autonomic dysreflexia. Symptoms may be minimal or even absent, despite an elevated blood pressure.

Health-care providers should be aware that the varying cognitive and verbal communication abilities of adults, children, and adolescents can cause the symptoms of AD to be absent, subtle, vague, or expressed imperfectly. Because of the varying cognitive and verbal communication abilities of individuals as they progress through infancy, childhood, and adolescence, symptoms of AD may not be expressed or may be communicated in a less articulate manner compared to a cognitively intact adult with SCI. For instance, preschool-aged children, even though they are verbal, may present with vague complaints; they are not able to accurately articulate that they are experiencing a pounding headache—a cardinal feature of autonomic dysreflexia.

2. **Check the individual's blood pressure.**

   - **A sudden, significant increase in both the systolic and diastolic blood pressure above their usual levels, frequently associated with bradycardia. An 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.**
   - **Systolic blood pressure elevations more than 15–20 mm Hg above baseline in adolescents with SCI or more than 15 mm Hg above baseline in children with SCI may be a sign of AD.**

   (Scientific evidence–III/V; Grade of recommendation–C; Strength of panel opinion–Strong)

Elevated blood pressures can be life-threatening and need immediate investigation and treatment (Cole et al., 1967; Guttman et al., 1965). For children and adolescents, age and body size are determinants of normal blood pressures, with increasing blood pressures advancing with age and approximating adult norms in older teenagers (National High Blood Pressure Education Program, 1996). Similar to adults with SCI, children and adolescents with cervical and upper thoracic SCI would be expected to have lower baseline blood pressures compared to the general population. Therefore, it is important to determine and document baseline blood pressures on an annual basis or as needed, as the child or adolescent with SCI ages. For the purposes of these guidelines, the panel agreed that systolic blood pressures at or above 150 mm Hg in adults, 120 mm Hg in chil-

dren under 5 years old, 130 mm Hg in children 6–12 years old, and 140 mm Hg in adolescents is when pharmacological agents should be considered. See recommendation 19 on page 15.

Be calm and maintain a reassuring environment in the presence of the child's parents/caregiver when obtaining blood pressures. Any anxiety associated with obtaining blood pressures in children and adolescents may make it difficult to obtain accurate measurements both for baseline determinations as well as during an episode of autonomic dysreflexia. Teaching parents how to obtain blood pressures or having school nurses obtain baseline blood pressures may be beneficial. It is important that all health-care professionals remain calm and maintain a relaxing atmosphere.

Use appropriately sized blood pressure cuffs when measuring blood pressure in children and adolescents. The width of the blood pressure cuff should be approximately 40 percent of the arm circumference, measured midway between the olecranon and the acromiom (Perloff et al., 1993). The cuff bladder will cover 80 to 100 percent of the circumference of the arm. A blood pressure cuff that is too small may result in an overestimation of the individual's blood pressure. In contrast, a blood pressure cuff that is too large may result in an underestimation of the blood pressure, which is less than the error of overestimation with a cuff that is too small. If an appropriately sized blood pressure cuff is not available, interpretation of the blood pressure is complicated. However, it is important for the health-care professional or caregiver to remember that small blood pressure cuffs tend to overestimate and large cuffs tend to underestimate the true blood pressure.

3. **If a pregnant woman with a spinal cord injury at T6 or above presents with signs and symptoms of autonomic dysreflexia, consider referral to an obstetric health-care provider under the following circumstances:**

   - **Determination of choice of antihypertensive medication.**
   - **Persistent hypertension after resolution of the acute autonomic dysreflexia episode.**
   - **Persistent symptoms of autonomic dysreflexia despite acute care measures.**
   - **Life-threatening autonomic dysreflexia.**
   - **Autonomic dysreflexia episode occurring in the third trimester of pregnancy.**
   - **Hypotension requiring pharmacological treatment.**
   - **First episode of autonomic dysreflexia during the pregnancy.**
   - **Presence of vaginal bleeding or suspicion of labor.**
   - **Decisions to be made about long-term medication use.**
   - **Unclear about the causes, signs, and symptoms, despite a normal blood pressure.**

   (Scientific evidence–None; Grade of recommendation–Expert consensus; Strength of panel opinion–Strong)

Care of pregnant women with AD should take into account that, due to compression of the vena cava, hypotension may occur if the woman is in supine position. A lateral tilt or upright position facilitates resolution of the hypotension and improves uterine blood flow.

4. **If signs or symptoms of AD are present, but the blood pressure is not elevated and the cause has not been identified, refer the individual to an appropriate consultant depending on symptoms.**

   (Scientific evidence–None; Grade of recommendation—Expert consensus; Strength of panel opinion–Strong)

Other medical problems may be causing the signs and symptoms of autonomic dysreflexia.

5. **If the blood pressure is elevated, immediately sit the person up if the individual is supine.**

   (Scientific evidence–III/V; Grade of recommendation–C; Strength of panel opinion–Strong)

Performing this maneuver may allow a pooling of blood in the lower extremities and may reduce the blood pressure (Cole et al., 1967; Guttman et al., 1965). If possible, in addition to sitting the person up, lower their legs as well.

6. **Loosen any clothing or constrictive devices.**

   (Scientific evidence–III/V; Grade of recommendation–C; Strength of panel opinion–Strong)

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

7. **Monitor the blood pressure and pulse frequently.**

   Scientific evidence–III/V; Grade of recommendation–C; Strength of panel opinion–Strong)

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 (Colachis, 1992; Cole et al., 1967; Erickson, 1980; Guttman et al., 1965; Kewalramani, 1980; Kuric and Hixon, 1996; Kurnick, 1956; Pollock et al., 1951).

8. **Quickly survey the individual for the instigating causes, beginning with the urinary system.**

   (Scientific evidence–III/V; Grade of recommendation–C; Strength of panel opinion–Strong)

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

9. **If an indwelling urinary catheter is not in place, catheterize the individual.**

   Scientific evidence–None; Grade of recommendation–Expert consensus; Strength of panel opinion–Strong)

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

10. **Prior to inserting the catheter, instill 2 percent lidocaine jelly (if immediately available) into the urethra and wait 2 minutes, if possible.**

    (Scientific evidence–None; Grade of recommendation–Expert consensus; Strength of panel opinion–Strong)

Catheterization can exacerbate autonomic dysreflexia. The use of lidocaine jelly may decrease the sensory input and relax the sphincter to facilitate catheterization. The peak effect of lidocaine jelly is between 2–5 minutes. Exercise clinical judgment regarding elevated blood pressure; immediate catheterization may be necessary.

11. **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. If a problem is found, correct it immediately.**

    (Scientific evidence–None; Grade of recommendation–Expert consensus; Strength of panel opinion–Strong)

12. **If the catheter appears to be blocked, gently irrigate the bladder with a small amount (10–15 cc) of fluid, such as normal saline at body temperature. Irrigation should be limited to 5–10 ml for children under 2 years of age and to 10–15 ml in older children and adolescents. Avoid manually compressing or tapping on the bladder.**

    (Scientific evidence–None; Grade of recommendation–Expert consensus; Strength of panel opinion–Strong)

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. Do not continue to irrigate the bladder if the fluid is not draining.

13. **If the catheter is draining and the blood pressure remains elevated, proceed to recommendation 18.**

    (Scientific evidence–None; Grade of recommendation–Expert consensus; Strength of panel opinion—Strong)

14. **If the catheter is not draining and the blood pressure remains elevated, remove and replace the catheter.**

    (Scientific evidence–None; Grade of recommendation–Expert consensus; Strength of panel opinion—Strong)

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. Refer to Recommendation 19, page 15, and its accompanying rationale for guidance on pharmacologic management.

15. **Prior to replacing the catheter, instill 2 percent lidocaine jelly (if immediately available) into the urethra and wait 2 minutes, if possible.**

    (Scientific evidence–None; Grade of recommendation–Expert consensus; Strength of panel opinion–Strong)

Catheterization can exacerbate autonomic dysreflexia. The use of lidocaine jelly may decrease sensory input and relax the sphincter to facilitate catheterization. The peak effect of lidocaine jelly is between 2–5 minutes. Exercise clinical judgment regarding elevated blood pressure and the use of lidocaine; immediate catheterization may be necessary.

16. If difficulties arise in replacing the catheter, consider attempting to pass a coude catheter or consult a urologist.

(Scientific evidence–None; Grade of recommendation–Expert consensus; Strength of panel opinion–Strong)

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

17. Monitor the individual's blood pressure during bladder drainage.

(Scientific evidence–None; Grade of recommendation–Expert consensus; Strength of panel opinion–Strong)

Sudden decompression of a large volume of urine would be expected to normalize blood pressure. However, this may cause hypotension if the individual has already been given pharmacological agents to decrease blood pressure. (See Recommendation 21, page 16.)

18. If acute symptoms of autonomic dysreflexia persist, including a sustained elevated blood pressure, suspect fecal impaction.

(Scientific evidence–II/V; Grade of recommendation–B/C; Strength of panel opinion–Strong)

Fecal impaction is the second most common cause of autonomic dysreflexia (Colachis, 1992; Lee et al., 1995). Refer to the clinical practice guidelines titled *Neurogenic Bowel Management in Adults with Spinal Cord Injury*, pages 26–27 (Consortium for Spinal Cord Medicine, 1998), for information on autonomic dysreflexia precipitated by neurogenic bowel conditions. All Consortium publications are available on the Paralyzed Veterans of America's web site at **www.pva.org**. Click on "publications" and then "consortium publications."

19. 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.

(Scientific evidence–None; Grade of recommendation–Expert consensus; Strength of panel opinion—Strong)

If the blood pressure remains elevated but is less than 150 mm Hg systolic, proceed to recommendation 22.

(Scientific evidence–V; Grade of recommendation–C; Strength of panel opinion–Strong)

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. This is true for nonpregnant adults, pregnant women, and children and adolescents, as well.

There are no studies showing the exact point at which blood pressure becomes dangerous. For this recommendation, 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). An adult with an injury at or above T6 would be expected to have a baseline systolic 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.

Pharmacological management of AD in children and adolescents should be considered prior to checking for fecal impaction if the blood pressure is excessively elevated for the child's or adolescent's age and height. Knowing the child's baseline blood pressure is very important when deciding whether to intervene with antihypertensive medications. Indications for pharmacological intervention may include a systolic blood pressure of 120 mm Hg in infants and younger children (under 5 years old), 130 mm Hg in older children (6–12 years old), and 140 mm Hg in adolescents.

20. Use an antihypertensive agent with rapid onset and short duration while the causes are being investigated.

(Scientific evidence–V; Grade of recommendation–C; Strength of panel opinion–Strong)

Nifedipine and nitrates are the most commonly used agents (Braddom and Rocco, 1991; Dykstra et al., 1987; 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 from 1966 through December 2000 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.

There is increasing use of sildenafil in those with spinal cord injury. The use of medications containing nitrates is contraindicated when a person has taken sildenafil. Medications containing nitrates are sometimes used for the treatment of acute autonomic dysreflexia. Prior to the use of nitrates, such as nitroglycerin, isosorbide dinitrate, or sodium nitroprusside, a person with SCI presenting with acute autonomic dysreflexia should be questioned regarding sildenafil. If this agent has been used within the last 24 hours it is recommended that an alternative short-acting, rapid-onset antihypertensive agent be used.

Examples of agents with such characteristics are prazosin and captopril. Both have an onset within thirty minutes, achieve peak serum levels within 1-3 hours, and have elimination rate half-lives of 2-4 hours.

21. Monitor the individual for symptomatic hypotension.

(Scientific evidence–None; Grade of recommendation–Expert consensus; Strength of panel opinion–Strong)

Treat severe (symptomatic) hypotension by laying the individual down 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).

22. If fecal impaction is suspected and the elevated blood pressure is less than 150 mm Hg, 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 2 minutes if possible 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–II/V; Grade of recommendation–B/C; Strength of panel opinion–Strong)

A rectal examination may exacerbate autonomic dysreflexia (Bors and French, 1952; Watkins, 1938). Instillation of a local anesthetic agent may decrease the occurrence of autonomic dysreflexia during the exam. For additional information about neurogenic bowel, refer to *Neurogenic Bowel Management in Adults with Spinal Cord Injury* (Consortium for Spinal Cord Medicine, 1998). All Consortium for Spinal Cord Medicine publications can be downloaded from the Paralyzed Veterans of America's web site at **www.pva.org**. Click on "publications" and then "consortium publications."

23. If the precipitating cause of the autonomic dysreflexia episode has not yet been determined, check for less frequent causes. The individual may need to be admitted to the hospital; see recommendation 25 for considerations.

(Scientific evidence–None; Grade of recommendation–Expert consensus; Strength of panel opinion—Strong)

As the most common causes of AD are related to bladder and bowel problems, further assessment may need to include more advanced diagnostic procedures. For example, cystoscopies or urodynamic studies may detect urinary system pathology or dysfunction.

Other causes of AD need to be investigated to determine appropriate treatment and to resolve

the episode. Further causes related to the urinary, gastrointestinal, integumentary, reproductive, and other body systems are detailed in *Potential Causes* on page 10.

24. Following an episode of autonomic dysreflexia, instruct the individual to monitor symptoms and blood pressure for at least 2 hours after resolution of the episode to make sure that it does not reoccur.

- **Educate the individual to seek immediate medical attention if it reoccurs.**
- **Monitor inpatients closely for at least 2 hours, as deemed necessary by the health-care provider.**
- **Seek the pregnant woman's obstetrical-care provider for evaluation.**

(Scientific evidence–None; Grade of recommendation–Expert consensus; Strength of panel opinion–Strong)

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.

25. Consider admitting the individual to the hospital for monitoring to maintain pharmacologic control of the blood pressure, and to investigate other causes:

- **If there is poor response to the treatment specified above.**
- **If the cause has not been identified.**
- **If there is suspicion of an obstetrical complication.**

(Scientific evidence–V; Grade of recommendation–C; Strength of panel opinion–Strong)

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.

26. Document the episode in the individual's medical record, including

- **Presenting signs and symptoms and their course.**
- **Treatment instituted.**
- **Recordings of blood pressure and pulse.**
- **Response to treatment.**

Evaluate the effectiveness of the treatment according to the level of outcome criteria reached:

- **Cause of the episode has been identified.**
- **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).**
- **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.**
- **An education plan has been completed and included preventive and emergency management guidance.**

(Scientific evidence–None; Grade of recommendation—Expert consensus; Strength of panel opinion–Strong)

27. Once the individual with spinal cord injury has been stabilized, review the precipitating cause of the AD episode with the individual, family members, significant others, and caregivers. This preventive 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.**
- **Discussing autonomic dysreflexia during the individual's education program, so that he or she will be able to minimize the risks known to precipitate AD, solve problems, recognize early onset, and obtain help as quickly as possible.**
- **Providing the individual with education about the prevention and treatment of autonomic dysreflexia at the time of discharge that can be referred to in an emergency.**

(Scientific evidence–None; Grade of recommendation–Expert consensus; Strength of panel opinion–Strong)

Health-care professionals should refer to the consumer guide *Autonomic Dysreflexia: What You Should Know* (Consortium for Spinal Cord Medicine, 1997a), to provide individuals a tool to help guide their own treatment of autonomic dysreflexia. This consumer guide is written in such a way that both adults and children will find it helpful. It can be downloaded from the Paralyzed Veterans of America's web site at **www.pva.org**. Click on "publications" and then "consortium publications."

A written guide or alert, such as the wallet card found in the above referenced consumer guide, may help them in communicating with their health-care providers. Such an alert system is especially needed when individuals with concomitant injuries that have resulted in reduced or limited cognition and verbal skills may be hindered in their ability to communicate that they are experiencing autonomic dysreflexia.

A written treatment plan for autonomic dysreflexia prepared for children and adolescents with SCI should include:

- The child's normal blood pressure, which is updated annually or more frequently as needed.
- Diagnostic criteria.
- An emergency management plan.

Limited cognition and verbal skills hinder the ability of younger children to communicate that they are experiencing autonomic dysreflexia with health-care providers, teachers, and other adults who are responsible for supervising their activities.

In addition to the signs and symptoms seen in adults, infants and children may present with nonspecific symptoms, such as crying, irritability, or somnolence. Parents of young children should consider using some form of medical alert identification as well as ensure that appropriate education is provided to those adults who have significant interactions with and responsibility for their child with SCI, such as teachers, school nurses, coaches, and community-based health-care providers.

When a woman with spinal cord injury at T6 and above becomes pregnant, her care should be coordinated by an interdisciplinary team. It is recommended that the team develop a plan regarding management of autonomic dysreflexia.

28. Schedule detailed evaluations for individuals with recurrent autonomic dysreflexia.

(Scientific evidence–None; Grade of recommendation–Expert consensus; Strength of panel opinion–Strong)

There may be subtle changes in an individual's medical condition, such as a worsening of detrusor sphincter dyssynergia or an expanding syrinx that is causing recurrent autonomic dysreflexia. Therefore, a detailed medical evaluation is warranted.

Recommendations for Future Research

The first comprehensive literature review was completed on AD in 1997. The literature review for the second edition, completed in 2000, found new citations on pediatric autonomic dysreflexia, obstetric considerations, and the effect of pharmacologic agents such as sildenafil on risk. The literature review and grading of evidence were performed by the Duke University methodology team. Knowledge gaps identified in the first edition still need further investigation:

- Studies demonstrating when blood pressure becomes dangerous. (A person with SCI at or above T6 would be expected to have a baseline systolic BP between 90 and 110 mm Hg.)
- Definitive research on the common causes of and risk factors for AD.
- Research on the implications of pregnancy and of obstetric complications and delivery for risk of AD.
- Studies that define the special aspects of AD in children and adolescents.

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Glossary

autonomic dysreflexia: also known as hyperreflexia, an uninhibited sympathetic nervous system response to a variety of noxious stimuli occurring in people with spinal cord injury at the thoracic 6 (T6) 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.

boosting: a term used by some SCI athletes to describe an episode of AD that is purposefully instigated by the individual in an attempt to enhance athletic performance.

coude catheter: a urethral catheter that has a slight upward bend and a 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 percent.

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 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 the 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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