Depression Following Spinal Cord Injury:

A Clinical Practice Guideline for Primary Care Physicians

consortium for SPINAL CORD MEDICINE
CLINICAL PRACTICE GUIDELINES

Administrative and financial support provided by Paralyzed Veterans of America

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This guide has been prepared based on scientific and professional information known about depression following spinal cord injury/dysfunction, its causes, and its treatments, in 1998. Users of this guide should periodically review this material to ensure that the advice herein is consistent with current reasonable clinical practice.

Contents

Foreword

Mood disturbances are common among individuals with chronic health conditions, including spinal cord injury (SCI) or impairment. More often than not, these experiences are described by the word "depression." Depression can be defined in a number of ways. These guidelines define depression using the diagnostic criteria established in the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition* (DSM-IV) and in the International Classifications of Impairments, Disabilities, and Handicaps published in 1989 by the World Health Organization (WHO).

DSM-IV states that depression is not a single entity, but rather a spectrum of depressive disorders. The specific constellation of symptoms determines whether an individual meets the criteria for a specific depressive disorder, and the origin of symptoms further dictates which depressive disorder is diagnostically appropriate for a specific individual.

WHO provides a model for understanding the impact of various disorders, including depressive disorders, by virtue of impairment, disability, and handicap. WHO defines **impairment** as an abnormality in body structure, appearance, or organ or system function resulting from any cause. **Disability** reflects the consequences of impairment in terms of functional performance and activity by the individual. **Handicaps** concern the disadvantages experienced by an individual as a result of impairments and disabilities. From the perspective of this model, depression is viewed as an impairment with obvious cascades into the disability and handicap sectors.

Depression can have devastating effects on an individual with a spinal cord injury. Depression can be a major factor in the higher utilization of health services and can be associated with suboptimal functional gains, increased complications such as pressure ulcers and urinary tract infections, compromised immune function, increased hospital stays, increased medical expenses, decreased social integration, compromised intimate relationships, and strained caregiver support. The comorbidity of depression and spinal cord injury can prove lethal. For this reason, it must be assessed quickly and addressed with skill, knowledge, and competence. Awareness of the specific risk factors for depression can hold significant clinical utility in the identification of individuals who may be at risk to incur a depressive disorder.

The assessment, diagnosis, and treatment of depression in people with spinal cord injury are multilayered processes, complicated by an interplay of biological, psychological, and social factors that are unique to individuals with a spinal cord injury. Awareness of the inherent complexities in the diagnostic process is essential to the successful identification and treatment of these disorders. To this end, panelists from a variety of disciplines and medical specialties, including primary care, physiatry, psychiatry, pharmacology, nursing, psychology, and social work met under the auspices of the Paralyzed Veterans of America to draft recommendations specific to people with spinal cord injuries. Their recommendations are presented in these clinical practice guidelines.

The guidelines cover the full range of the diagnostic and treatment processes, incorporating detailed information about differential diagnosis, multiple treatment options, and referrals to appropriate mental health providers. Recommendations are provided on the general risk factors for depression; on the signs and symptoms of depression in people with spinal cord injury; and on the identification of the biological, psychological, and social factors that cause or contribute to depression. Other recommendations focus on the use of psychopharmacological agents, on the selection of psychological interventions for specific problems, and on the utilization of treatment strategies aimed at strengthening an individual's social support system.

The guidelines are designed to be used primarily by primary care physicians, who typically represent the initial interface between an individual with spinal cord injury and the medical and mental health professions. In this position, primary care physicians confront the complexities of making clinical assessments, treatment decisions, and referrals. But without an accurate appreciation of these complexities, misdiagno-

sis, ineffective treatments, and inappropriate referrals can result. Therefore, primary care physicians need knowledge to make sound decisions when treating individuals with spinal cord injury who present with depressive symptoms.

The panelists are aware that these guidelines are but a beginning step in the ongoing process of developing useful tools that will assist primary care physicians in the diagnosis and treatment of depression in individuals with a spinal cord injury. It is the panel's hope that these guidelines will stimulate interest in this vital topic, provide guidance in dealing with the complexities of depression and spinal cord injury, and create ongoing interest in refining the knowledge and skill needed to accurately and appropriately assess and treat depression in this population.

Jason Mask, LCSW
Chair, Guidelines Development Panel

Preface

Depression is a treatable, even reversible form of impairment. It contributes to the disability and handicap of the person with SCI. To minimize the impact of depression, it must be correctly identified and successfully treated. *Depression Following Spinal Cord Injury: Clinical Practice Guidelines for Primary Care Physicians* defines the basis of diagnosis and treatment of depression in the context of the patient with SCI. While we have defined the intended audience as primary care physicians, all health-care professionals who interact with people with SCI will benefit from the content of this document.

By focusing on primary care physicians, the Consortium for Spinal Cord Medicine recognizes the reality of today's managed care environment. Inpatient rehabilitation services seem to become more restricted with each passing year and recently injured individuals are usually back in the home and community before confronting the full impact of their losses. By that time the patient may be cut off from the psychosocial support of the SCI rehabilitation environment. Later in life, depression may occur in the confounding environment of managing pain and spasticity. Loss of a caregiver or the onset of an acute illness may cause additional stress that precipitates an episode of depression. The primary care physician may be the first health-care professional with an opportunity to diagnose and treat this condition that may prove otherwise fatal.

This document defines depression and related disorders of mood. It also defines the contribution of commonly prescribed medications to the problem of depression. The implications of substance abuse and withdrawal phenomena are also explored. Suicide risk assessment is simplified and recommendations are made about when referral to mental health care services is appropriate. Also, depression is seen in a new light: as a contributing factor to pressure sores, the most expensive of complications after SCI. The guidelines in this document offer significant assistance in the evaluation and comprehensive treatment of these complicated patients.

Usually the diagnosis of depression has the physician reaching for the prescription pad for the favorite antidepressant of the week. However, the side effects of various antidepressants may have serious consequences with regard to appetite and its impact on weight gain in the sedentary wheelchair user, or weight loss in the emaciated patient with a pressure sore. Other problems of SCI may be amplified by the wrong choice of medication, such as urinary retention and constipation as a result of the use of a medication with strong anticholinergic side effects. The tables in this publication will help with the choice of medication, while allowing the avoidance of common pitfalls.

Also very important is the guidance to look for the specific stressors often found with persons with SCI. Inadequate housing, poverty-level income, or family dissolution are common conditions of handicap that result from the impairment of SCI. No prescribed drug will find the necessary resources to resolve these issues and get people moving with their lives. Following the guidance in this document will lead to a holistic approach to the person with the co-existence of SCI and depression and their additive effect on disability and handicap.

The Consortium for Spinal Cord Medicine is delighted to publish our fourth clinical practice guideline. As chairman of the steering committee I want to extend my special thanks to Jason Mask, LCSW, the panel chair, and Helen Bosshart, LCSW, the liaison with the steering committee. I also want to acknowledge all members of the panel for their expertise and collaboration in the process. All are commended for their excellent product. Also commended are the reviewers who brought forth their own contributions, professional and personal, to enrich our group effort. As in the past, the leadership and staff of the Paralyzed Veterans of America have once again provided invaluable support for the panel, the process, and the publication of this guideline. J. Paul Thomas and Dawn M. Sexton continue to provide the organizational skills to bring it all together. My thanks to you all.

By the publication of these guidelines, we hope to add to the quality of the care of people with spinal cord injury or disease. A broader application of this set of guidelines may also provide guidance for the diagnosis and treatment of depression among people with other serious physical impairments. If we have done our work well, then our patients will benefit.

Kenneth C. Parsons, MD
Chair, Steering Committee
Consortium for Spinal Cord Medicine

Acknowledgments

The chair and members of the Guidelines Development Panel wish to express special appreciation to both the individuals and the professional organizations that are involved in the Spinal Cord Medicine Consortium; to the expert clinicians and healthcare providers who reviewed the draft documents; and to the consumers and the staffs of the numerous medical facilities, spinal cord injury rehabilitation centers, and advocacy organizations who contributed their time and expertise to the development of these guidelines.

Timothy Elliott, Ph.D., served as the initial chair of the panel. His clinical knowledge and research expertise greatly contributed to guiding the panel at the beginning of its work. Robert Frank, Ph.D., is recognized for his contributions in focusing the panel on the relevancy of the depression literature to the person with spinal cord injury and on the existing gaps in our understanding of the interplay between depression and spinal cord injury. Lawrence Cohen, Pharm.D., served as a pharmacological consultant. His expert information regarding pharmacotherapy, pharmacokinetic profiles, dosing schedules, and adverse reactions was invaluable.

Mary McAweeney Ph.D., served as consultant methodologist. She masterfully conducted the initial and secondary-level literature searches, evaluated the quality and strength of evidence of the scientific investigations, constructed evidence tables, and graded the quality of research for all identified literature citations.

Members of the Consortium Steering Committee, representing 17 professional, payer, and consumer organizations, were joined by 51 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 guidelines development process.

The astute and perceptive legal review conducted by William H. Archambault, attorney at law for Piedmont Liability Trust, is hereby acknowledged. Mr. Archambault's analysis of the legal and other health policy issues associated with this multifaceted topic was essential in the development process.

The Guideline Development Panel is grateful for the many technical support services provided by 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 of the Consortium Coordinating Office; the astute analytic skills of John L. Carswell and the cogent consumer perspective offered by Fred Cowell of the Health Policy Department; the guidance in writing, formatting, and creating art provided by James A. Angelo, Patricia E. Scully, and Sarah E. Ornstein of the Communications Department; the excellent technical and editorial review of both the clinical practice guidelines and the consumer guide provided by medical writers Joellen Talbot and Barbara Shapiro; and the intensive effort of the PVA consultants and staff who 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 Spinal Cord Medicine Consortium and guidelines development process a successful venture.

Panel Members

Jason Mask, LCSW (Chair)
(Social Work)
Edward Hines Jr. VA Hospital
Hines, Illinois

Kimberly Arlinghaus, MD
(Psychiatry)
Houston VA Medical Center
Houston, Texas

Helen Bosshart, LCSW (Steering Committee Liaison)
(Social Work)
Augusta VA Medical Center
Augusta, Georgia

Lester Butt, PhD
(Medical Psychology)
Craig Hospital
Englewood, Colorado

Rebecca R. Clearman, MD
(Physical Medicine & Rehabilitation)
Houston VA Medical Center
Houston, Texas

Mary J. McAweeney, PhD (Consultant Methodologist)
Dexter, Michigan

Barbara Simmons, MSN, RN
(Spinal Cord Injury Nursing)
Tampa VA Medical Center
Tampa, Florida

Contributors

Consortium Member Organizations and Steering Committee Representatives

American Academy of Orthopedic Surgeons
Robert Waters, MD

American Academy of Physical Medicine and Rehabilitation
Margaret Turk, MD

American Association of Neurological Surgeons
Paul McCormick, MD

American Association of Spinal Cord Injury Nurses
Nahid Veit, RN, MSN

American Association of Spinal Cord Injury Psychologists and Social Workers
Helen Bosshart, LCSW

American Congress of Rehabilitation Medicine
Marilyn Pires, MS, RN

American Occupational Therapy Association
Susan Garber, MA, OTR, FAOTA

American Paraplegia Society
Todd Linsenmeyer, MD

American Physical Therapy Association
Montez Howard, PT, MEd

American Psychological Association
J. Scott Richards, PhD

American Spinal Injury Association
Kenneth C. Parsons, MD

Association of Academic Physiatrists
Kristjan Ragnarsson, MD

Association of Rehabilitation Nurses
Audrey Nelson, PhD

Congress of Neurological Surgeons
Paul McCormick, MD

Insurance Rehabilitation Study Group
Karen O'Malley, MA, CRC, LPC

Paralyzed Veterans of America
R. Henry Bodenbender, MD

U.S. Department of Veterans Affairs
Margaret C. Hammond, MD

Expert Reviewers

American Academy of Orthopedic Surgeons
Robert Waters, MD

American Academy of Physical Medicine and Rehabilitation
Daniel Clinchot, MD
Stanley Ducharme, Jr., Ph.D.
W. Jerry Mysiw, MD

American Association of Neurological Surgeons
Robert E. Florin, MD
Beverly C. Walters, MD

American Association of Spinal Cord Injury Nurses
Constance Captain, PhD, RN
Kathleen L. Dunn, MS, RN, CRRN
Audrey Nelson, PhD, RN

American Association of Spinal Cord Injury Psychologists and Social Workers
Gordon J. Casebolt, ACSW
Kristofer J. Hagglund, PhD

American Congress of Rehabilitation Medicine
Jessica Robins Miller, ACSW
Roberta Trieschman, PhD

American Occupational Therapy Association
Myrtice Atrice, PT
Franki Cassaday, OT
Sue Eberle, OT

American Paraplegia Society
Vidya Sridharan, MD
Robert W. Woolsey, MD

American Physical Therapy Association
Jill Koval, PhD
Maureen Lynch

American Psychological Association
Bruce Caplan, PhD
Kristofer J. Hagglund, PhD
Daniel E. Rohe, PhD, LP

American Spinal Injury Association
Daniel P. Lammertse, MD
Michael Y. Lee, MD
Marcia Sipski, MD

Association of Academic Physiatrists
Diana D. Cardenas, MD
Steven Kirschblum, MD
Michael Priebe, MD

Association of Rehabilitation Nurses
Joseph Adamski, MS, RN, CRRN
Jane Egan, MSN, RN
Rae Langford, EdD, MS, RN

Congress of Neurological Surgeons
Robert E. Florin, MD
Beverly C. Walters, MD

Insurance Rehabilitation Study Group
Suzanne Baillargeon
Colleen Flusk
Karen O'Malley, MA, CRC, LPC

Paralyzed Veterans of America
R. Henry Bodenbender, MD

U.S. Department of Veterans Affairs
Beth Budney, MS, RN, CNA, CRRN
Robert D. Hendricks, PhD
Suzanne Szollar, MD

Summary of Recommendations

Assessment

1. Perform routine screening for depression during the individual's initial visit and annually thereafter. Self-report measures of depression may be helpful in screening psychological status, but should never be used without a clinical interview to establish the existence or absence of a depressive disorder.

2. Assess the individual for the presence of the following general risk factors for depression:

   - Prior episodes of depression
   - Family history of depressive disorder or bipolar disorder
   - Family history of suicide attempts
   - Current suicidal ideation
   - Age of onset under 40
   - Chronic pain
   - Female gender
   - Lack of social support
   - Postpartum
   - Multiplicity of life stressors
   - Concurrent medical illness
   - Concurrent substance abuse

3. Assess individuals with spinal cord injury for the specific risk factors of depression, including:

   - Complete neurologic injury
   - Medical comorbidity, including but not limited to traumatic brain injury (TBI)

4. Assess the individual for signs and symptoms of depression and potential for suicide during a history and physical examination.

5. Identify the biological factors that may cause or contribute to depression, including the following physiological factors:

   - Biological effects of SCI, such as fatigue, anorexia, sleep disturbance, decreased energy
   - History of mood disorder
   - Family history of mood disorder
   - Presence of a general medical condition that may cause or contribute to depression
   - Presence of medications or drugs that may cause or contribute to depression

6. Conduct a comprehensive assessment of the social factors specific to spinal cord injury that contribute to depression to evaluate the adequacy of the individual's social support system in meeting basic needs and to determine the presence of depression in response to an inadequate support network. Specifically, the assessment should include but not be limited to:

   - The individual's social network, including family members, friends, and community organizations
   - The individual's financial resources
   - Vocational and avocational interests and issues
   - Current living arrangements, including wheelchair accessibility
   - Adaptive equipment needs and resources
   - Personal assistance needs and resources
   - Transportation needs and resources

7. Assess the psychological factors specific to spinal cord injury that contribute to depression, including the following:

   - Coping style
   - Self-blame for the injury
   - Unresolved conflicts from previous losses or traumas
   - Preinjury psychological or psychiatric impairment
   - Cognitive style
   - Grief and bereavement from SCI

Diagnosis

8. Use established diagnostic criteria to diagnose depression.

9. Identify the mental health factors that indicate referral to the appropriate mental health provider including:

   - Active suicidal ideation
   - Psychotic depression
   - Bipolar disorder

- Complex psychiatric diagnoses such as depression that are associated with post traumatic stress disorder, obsessive-compulsive disorder, eating disorder, schizophrenia, schizophreniform disorder, schizoaffective disorder, and personality disorders
- Persistent substance abuse complicating the diagnosis and/or management of depression (especially when detoxification or more intensive treatment beyond a 12-step program is needed)

Treatment

10. Formulate a treatment plan identifying:
    - Which treatments are to be provided by the primary care physician
    - What type of individual and family education needs to be provided and by whom
    - Who will address comorbid conditions and how those conditions will be treated
    - Specific criteria for referring the individual to a mental health provider
11. Provide or refer for psychotherapy by matching the type of psychological intervention to both the identified problem and the therapeutic capacity of the individual.

Psychopharmacological Agents

12. If indicated, select appropriate antidepressant medications. Psychopharmacological agents should be considered for individuals who present significant biological, somatic, and/or mood-related symptoms of sufficient severity to disrupt the person's life and activities of daily living. Selection of a specific agent should be predicated upon the unique characteristics of the individual and the presenting signs and symptoms of depression.

Environmental and Social Factors and Social Support System

13. Address environmental and social factors and refer to a social worker, rehabilitation counselor, or case manager, as appropriate. When problems in the individual's support system are identified, treatment interventions should be implemented to strengthen the social support system. These interventions should be directed at one or more of the following areas:
    - Education and information regarding available resources
    - Referrals to existing community resources
    - Development of alternatives to access services or assistance where no existing community resource is readily available
    - Advocacy to change public policy to ensure that individuals with SCI have the resources to meet their lifelong needs

Consumer and Family Education

14. Provide patient and family education on the following topics:
    - Signs and symptoms of depression
    - Treatment options
    - Medications, side effects, adverse reactions, and drug interactions
    - Effect of depression on individuals with SCI/D
    - Effect of depression on the family
    - Community resources

Evaluation and Modification of Treatment Plan

15. Evaluate treatment, focusing on the following elements:
    - Evaluation of treatment efficacy
    - Modification of treatment, as indicated
    - Follow-up with referral sources

The Spinal Cord Medicine Consortium

Seventeen organizations, including PVA, joined in a consortium in June 1995 to develop clinical practice guidelines in spinal cord medicine. A steering committee governs consortium operation, leading the guidelines development process, identifying topics, and selecting 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 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 completion of each set of guidelines.
- *Reality-based*, to make the best use of the time and energy of the busy clinicians who serve as panel members and field expert reviewers.

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 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 processes. The consortium expects to initiate work on four or more topics per year, with evaluation and revision of previously completed guidelines as new research demands.

Guideline Development Process

The guidelines 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, 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 their 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 bibliography, 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 databased 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 the following:

- Clinical practice options and care standards
- Medical and health professional education and training
- Building blocks for pathways and algorithms
- Evaluation studies of guidelines 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

Depression Guidelines Methodology

A comprehensive computer search of six database systems was completed for the years 1966 to 1998. The six databases were Medline, PsychLit, ERIC, NARIC, CINAIL, and Dissertation Abstracts. The search involved two general categories, spinal cord injury and depression. The keyword "depression" was cross-referenced with up to 14 related terms: psychological, psychosocial, adjustment, coping, counseling, family therapy, psycho-education, cognitive therapy, relaxation, assertiveness, social skills, group therapy, support groups, and behavior therapy. Because a few of the databases did not contain certain keywords, there was some variation in the specific keywords used (e.g., in ERIC, the term "spinal cord injury" was not an available keyword so it was replaced with the broader term "disability").

All abstracts of articles cited under these terms were screened. Articles were selected for this study based on four criteria: (1) the article had an experimental or quasi-experimental design with randomized assignment to a control and treatment group; (2) the article had an experimental or quasi-experimental design with no randomized assignment to group; (3) the article was a case series with no controls; or (4) the article was a review thought to have relevant information and citations. Quasi-experimental designs are studies that lack random assignment, that address correlational or normative questions, or that are descriptive in nature. Case study articles provide the reader with extremely useful information about clinical course and prognoses but only hint at efficacy (Sackett, 1989). One hundred and fifteen articles were identified through this screening process. An additional eight articles were identified through peer recommendations.

An evaluation tool was developed in part from evaluation tools that currently exist (Kohn and Suydam, 1970; Thomas and Lawrence, 1991), from a thorough review of credible statistics textbooks (Kirk, 1982; Keppel, 1982; Stevens, 1991), and from recently published articles identifying common deficiencies in research (Braddom, 1990; Dar, Serlin, and Omer, 1994; Ottenbacher, 1991; Thomas and Lawrence, 1991). The tool was divided into two broad sections, each containing separate criteria. Part one consisted of descriptive data, including variables that were investigated with depression, measures of depression, design of the study, retrieval form, and type of article. Part two consisted of 11 quantitative categories addressing a specific aspect of methodological standards: significance of problem or theoretical relevance, clarity of problem definition, scope of the literature review, adequacy of the research design, control of variables, sample selection and sample size, psychomotor properties of the instruments, analysis techniques, interpretations and generalizations from the results, limitations of the study, and adequacy of the research report.

The methodologist, panel chairperson, and PVA staff identified a core field of approximately 33 key papers that covered the major issues in SCI. These articles were sent to panel members for study and consideration. During the subsequent period, the methodologist evaluated the articles and consulted with the panel chair and panel members. In addition, another 30 articles were identified for evaluation with respect to pharmacological interventions. These articles, as well as the 33 spinal cord injury articles, are listed in Reference Section A. Other documents, primarily textbooks and individual book chapters, were used for additional information but were not applicable for review. These articles are found in Reference Section B.

Strength of Evidence

Three methods were used to evaluate the quality of the evidence on which the guidelines recommendations are based. First, a hierarchy of the levels of scientific evidence, proposed by Sackett (1989), was used. The hierarchy is presented in table 1.

TABLE 1
Hierarchy of the Levels of Scientific Evidence

Level	Description
I	Large randomized trials with definite results
II	Small randomized trials with uncertain results
III	Nonrandomized studies with concurrent controls
IV	Nonrandomized studies with historic controls
V	Case series with no controls

The resulting rankings were provided to the panel members during the deliberation process. Next, each of the recommendations was classified, depending upon the level of scientific evidence used in the development of the specific recommendations. The scheme used by the panel is shown in table 2, based on Sackett (1989), and used by prior PVA clinical practice guidelines (see *Acute Management of Autonomic Dysreflexia* and *Prevention of Thromboembolism*). 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.

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

Category	Description
A	The recommendation is supported by scientific evidence from properly designed and implemented controlled trials providing statistical results that consistently support the guidelines statement
B	The recommendation is supported by scientific evidence from properly designed and implemented clinical series that support the guidelines statement
C	The recommendation is supported by expert opinion

The strength of the recommendation is indicated by the language describing the rationale and is based on the discussion that occurred during the guideline panel's deliberations. Each panel member indicated his or her agreement to each recommendation by voting 1-5, with 1 corresponding to neutrality and 5 representing maximum agreement. The scores were then aggregated across the panel members, and an arithmetic mean was calculated. These scores were then translated into low, moderate or strong, as shown in table 3. All recommendations had strong expert panel agreement and support.

TABLE 3
Strength of Expert Panel Opinion

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

Treatment Recommendations

Assessment

1. **Perform routine screening for depression during the individual's initial visit and annually thereafter. Self-report measures of depression may be helpful in screening psychological status, but should never be used without a clinical interview to establish the existence or absence of a depressive disorder.** (Scientific evidence—III; Grade of recommendation—B; Strength of expert panel opinion—Strong)

In western, industrialized nations, the lifetime risk of a major depressive disorder is 7 to 12 percent in men and 20 to 25 percent in women (U.S. Dept. of HHS, 1993). Life stressors or psychosocial events can play a significant role in precipitating the first or second episode of major depression (U.S. Dept. of HHS, 1993). In that SCI assuredly represents a life stressor, limited research on the prevalence of depressive symptomatology in the SCI population varies. Estimates range up to 25 percent for men and 47 percent in women (Fuhrer et al., 1993), underscoring the necessity for accurate assessment and treatment planning.

Although self-report questionnaires, such as the Beck Depression Inventory or the Zung Self-Rating Depression Scale are low-cost, time-efficient tools, they are not diagnostic for depression (U.S. Dept. of HHS, 1993). Because these tests are generally nonspecific, they require a clinical interview to determine the presence of a depressive disorder (Coulehan, Schulberg, and Block, 1989).

2. **Assess the individual for the presence of the following general risk factors for depression:**

- Prior episodes of depression
- Family history of depressive disorder or bipolar disorder
- Family history of suicide attempts
- Current suicidal ideation
- Age of onset under 40
- Chronic pain
- Female gender
- Lack of social support
- Postpartum
- Multiplicity of life stressors
- Concurrent medical illness
- Concurrent substance abuse

(Scientific evidence—IV; Grade of recommendation—B; Strength of expert panel opinion—Strong)

A history of prior episodes of depression increases the risk for future major depressive episodes. One major depressive episode in the general population is associated with a 50 percent chance of a future episode; with 2 prior episodes there is a 70 percent chance of a future episode; and with 3 or more episodes there is a 90 percent chance of future depression over a lifetime (U.S. Dept. of HHS, 1993).

A history of depression in first degree relatives increases the probability of a person developing depression. For people with recurrent types of depression, genetic factors appear to play a significant role. However, for those with less recurrent types of depression, genetic factors are unclear. Strong scientific evidence points to genetic vulnerability to mood disorder in those with first degree relatives with mood disorder (Kaplan and Sadock, 1989).

A history of prior suicide attempts is a major risk factor of depression. Forty percent of people who commit suicide have made a previous attempt.

People without a social support network have a higher risk of depression (U.S. Dept. of HHS, 1993; Kaplan and Sadock, 1989). A strong and supportive social network allows people greater opportunity to build close and nurturing relationships. In addition, there is strong research evidence that depression is more prevalent in those of lower socioeconomic levels (Kaplan and Sadock, 1989).

Stressful life events put people at higher risk for depression. (Kaplan and Sadock, 1989). Many studies have demonstrated the relationship between stressful life events and major depression, although a causal relationship cannot be definitely concluded. The probability of developing depression in the face of stressful life events increases if there is no social support network or close confiding relationships. Individuals with spinal cord injury who incur significant life stress report more depressive behavior regardless of time since onset of injury (Frank and Elliott, 1987).

Alcohol or substance abuse should trigger an inquiry into symptoms of depression (Kaplan and Sadock, 1989; Elliott and Frank, 1996). Alcohol and substances may be used to self-medicate for symptoms of depression; prolonged alcohol and substance use can cause depression. The presence of alcohol abuse or substance use should alert the practitioner to look for signs and symptoms of depression.

3. Assess individuals with spinal cord injury for the specific risk factors of depression, including:

- Complete neurologic injury
- Medical comorbidity, including but not limited to traumatic brain injury (TBI)

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

A spinal cord injury changes an individual's life profoundly and generates a period of enforced helplessness postinjury, followed by a gradual resumption of limited independence. In a study of 30 individuals with spinal cord injury, depression was the most frequent postinjury diagnosis, usually appearing within the first month. The depressive symptoms remitted before discharge in all 9 people who developed depression. A greater frequency of depression was noted in those with complete injuries, possibly because those with incomplete injuries maintained more hope regarding rehabilitation and were, as a result, less depressed (Fullerton et al., 1981).

4. Assess the individual for signs and symptoms of depression and potential for suicide during a history and physical examination. (Scientific evidence—V; Grade of recommendation—C; Strength of expert panel opinion—Strong)

Depressive disorders are best assessed with a clinical interview exploring signs and symptoms of depression in the following spheres:

Mood: sadness, emptiness, irritability, loss of interest or pleasure in usual activities, worthlessness, hopelessness, helplessness, excessive or inappropriate guilt

Cognitive: diminished ability to think or concentrate, indecisiveness, recurrent thoughts of death, recurrent suicidal ideation, self-criticality

Somatic: change in appetite, unintentional weight loss or gain, sleeping more or less than usual, fatigue, low energy, decreased sexual interest, restlessness or agitation, being slowed down (psychomotor retardation) (Elliott and Frank, 1996; Frank et al., 1992)

Behavioral: suicide attempt and/or a specific plan for committing suicide, decreased attention to personal hygiene or self-care

Social: distress or impairment in social or occupational functioning, withdrawal or isolation, decreased performance at work or school

A means for readily holding key depressive symptoms in mind is served by the mnemonic **SIG: E CAPS:**

Sleep: insomnia or hypersomnia

Interest: loss of interest or pleasure in activities, poor hygiene

Guilt: excessive guilt, worthlessness, hopelessness, and helplessness

Energy: fatigue, loss of energy

Concentration: diminished ability to concentrate, indecisiveness

Appetite: decreased or increased; more than 5 percent weight loss or gain

Psychomotor: retardation or agitation

Suicidality: suicidal ideation or plan, access to lethal means, prior attempt, comorbid alcohol or drug abuse

Include a focused mental status examination screening for depression as part of the history and physical examination of an individual with spinal cord injury/dysfunction. The mental status examination is a systematic collection of data based on observation of the individual's behavior during an interview and on answers given to specific questions. These questions should focus on the individual's orientation to person, place, and time; on the person's immediate, recent, and remote memory; and on the person's affect/mood, judgment, thought processes, and attention span. The purpose of the examination is to obtain evidence of current signs and symptoms of mental disorders from which the individual might be suffering (APA, 1995). When screening for depression, it is important to evaluate appearance, mood, speech, rate of movement, thought content, perceptions, insight, judgment, and general cognitive function.

Assess the risk for suicide. Because of the substantial mortality associated with depressive disorders, it is critically important to assess suicidal risk within this population. Some 15 percent to 20 percent of individuals with unipolar depression die by suicide (Sederer and Rothschild, 1997). In a study examining primary care visits by 100 people who later committed suicide, two-thirds of the suicide victims had seen a family physician in the month before their suicide, and 40 percent had seen a physician within 1 week of their suicide (Barraclough et al., 1974). In another classic series by Murphy (1975), 82 percent of suicide victims had contacted a physician within 6 months of the act, and 53 percent had contact with a physician within 1 month. Although

two-thirds of the victims had previously attempted or threatened suicide, only 17 percent of nonpsychiatric physicians were aware of this history.

Early diagnosis and appropriate treatment of psychiatric disorders are the most important factors in suicide prevention (Vaillant and Blumenthal, 1990). But research shows that 50 percent of patients with depression who consult their primary care practitioner do not receive a diagnosis of a depressive disorder. The Barraclough (1974) study found that 80 percent of the 100 suicide victims were prescribed psychotropic medications, mostly hypnotics, yet only one-third of the depressed patients received antidepressants, usually in inadequate doses. Moreover, in a different series of patients who committed suicide, over half of those who died by overdose received their complete means for suicide in a single prescription provided by the primary care physician (Murphy, 1975). Because primary care physicians see more psychiatric patients than psychiatrists do (Kamerow, Pincus, and MacDonald, 1986), primary care physicians must learn how to accurately diagnose and treat depressive disorders to decrease morbidity and mortality and improve quality of life.

Depressive symptoms are most often associated with seriousness of suicide attempts (Kaplan and Sadock, 1989). A helpful mnemonic for suicide risk factors is **SAD PERSONS**:

Sex: most common among white males

Age: increasingly prevalent among adolescents; elderly at high risk

Depression: SIG:E CAPS

Previous attempts: increases risk 50 to 100 times

Ethanol abuse: 25 to 35 percent of suicides occur among people with alcohol dependency

Rational thinking: disorganized ideation, psychosis

Social supports: dearth of family or friends for comfort and support, living alone

Organized plan: method, time, setting, availability of means

No spouse: lack of an intimate system heightens risk

Sickness: 35 to 40 percent of suicides have significant chronic physical illness or disability

Several of the above risk factors apply to many individuals with SCI, because the majority are male, many divorce after their injuries occur and live alone, and many have a premorbid history of substance abuse and return to substance abuse postinjury. Previous suicides in the family are also a risk factor because such incidents weaken the taboo against suicide. The mortality suicide rate for individuals with SCI is five times the age-sex specific suicide rate in the United States (DeVivo et al., 1991). Suicide rates are highest 1 to 5 years postinjury. Some research has linked hopelessness to suicidal ideation among people with SCI in a manner observed among the general population (DeVivo et al., 1991).

5. Identify the biological factors that may cause or contribute to depression, including the following physiological factors:

- Biological effects of SCI, such as fatigue, anorexia, sleep disturbance, decreased energy
- History of mood disorder
- Family history of mood disorder
- Presence of a general medical condition that may cause or contribute to depression
- Presence of medications or drugs that may cause or contribute to depression

(Scientific evidence—IV; Grade of recommendation—C; Strength of expert panel opinion—Strong)

Because consensus has not been achieved on which depressive symptoms accurately predict depressive disorders in people with spinal cord injury, the origin of each symptom must be explored in order to maximize diagnostic accuracy and treatment efficacy. For example, the physiologic effects of acute SCI may include fatigue, anorexia, and weight loss (Frank et al., 1992), as well as disruptions in sleep and energy level (Elliott and Frank, 1996). But sleep disturbances may also result from multiple awakenings for turning or other medical interventions, and sleep deprivation alone can lead to changes in mood, motivation, and cognition (Elliott and Frank, 1996).

Other physiologic factors that may be direct sequelae of SCI include alteration of excretory rhythms of catecholamines and dampening of 17-hydroxycorticosteriod secretion (Frank et al., 1985). Such changes in the hypothalamic-pituitary-adrenal axis may cause or contribute to depression, as well as to adverse responses to stress.

Individuals with spinal cord injury may have concurrent medical conditions that can cause or contribute to depression. Examples of such medical conditions are listed in table 4. If a medical condition associated with depression is identified, treatment may include: (1) optimizing treatment of the medical disorder, and/or (2) providing specific treatment for the depression (U.S. Dept. of HHS, 1993). Unless the depression is mild and clearly related to the medical condition, it is generally recommended that treatment of the depression ensue without the delay imposed by optimizing treatment for the medical disorder.

TABLE 4
Medical Illnesses That May Cause or Contribute to Depression

Neurological system	Cerebrovascular diseases, dementias, epilepsy, Huntington's disease, hydrocephalus, infections (including HIV and neurosyphilis), migraines, multiple sclerosis, neoplasms, Parkinson's disease, progressive supranuclear palsy, subdural hematoma, traumatic brain injury
Endocrine system	Adrenal (Cushings, Addison's), diabetes mellitus, hypopituitarism, premenstrual dysphoric disorder, ovarian hypofunction, parathyroid disorders, postpartum depression, thyroid disorders
Infections and Inflammations	Acquired immune deficiency syndrome (AIDS), chronic fatigue syndrome, fibromyalgia, influenza (recovery phase), mononucleosis, systemic lupus erythematosus, ulcerative colitis
Metabolism	Hyponatremia, hypo- and hyperkalemia, hypercalcemia, hypomagnesemia, uremia
Neoplasms	Brain neoplasms, paraneoplastic effect (pancreas and small cell)
Nutrition	Thiamine deficiency, vitamin B-12 deficiency, folate deficiency, niacin deficiency
Cardiovascular system	Coronary artery disease

Clinically significant depressive symptoms are detectable in approximately 12 to 36 percent of people with medical diseases. Rates in people with specific medical disorders may be even higher (U.S. Dept. of HHS, 1993). On the other hand, most people with general medical conditions do not develop depressive disorders (U.S. Dept. of HHS, 1993).

An issue that almost always arises in the diagnosis of medically induced depression is the question of causality. How is a medical condition determined to cause a depression? A causal relation is postulated if the clinician can demonstrate the presence of a medical condition known to cause depression and if the symptoms improve as the medical condition is treated (Rundell and Wise, 1996). Clues that suggest an underlying medical cause for a depression are an atypical clinical picture, resistance to standard treatment modalities, unexplained personality changes, and subtle cognitive findings on mental status examination.

Because many medical diseases may produce somatic symptoms that overlap with neurovegetative symptoms of depression, clinicians may inquire about specific symptoms that are highly correlated with depression, such as a wish for death, suicidal thoughts, dysphoria, distractibility, and feelings of guilt, discouragement, hopelessness, helplessness, and worthlessness. To further distinguish neurovegetative symptoms from the physical symptoms of a medical condition, the following patterns drawn from Kaplan and Sadock (1989) may be helpful:

- Weight loss with a normal appetite suggests a medical condition
- Early-morning awakening is usually suggestive of primary depression
- Fatigue caused by primary depression is usually worse in the morning

However, distinguishing depressive symptoms from those caused by a medical illness is often difficult; therefore, empiric treatment of depression is often recommended.

Individuals with spinal cord injury may develop clinically significant depressive symptoms as a side effect of a variety of medications. However, clinicians should be aware that this effect is uncommon and usually occurs within days to weeks of starting a medication. More than 100 medications have been associated with depression, yet only a few have been clearly shown to cause depressive disorders (see table 5). The cause-and-effect connection between medications and depression is based largely on anecdotal reports, not on prospective data (U.S. Dept. of HHS, 1993; Rundell and Wise, 1996).

Even without data to suggest a causal relationship between a specific medication and depression, good clinical judgment dictates that the medication be stopped or changed to an alternate therapy if a person develops depression after beginning the medication (U.S. Dept. of HHS, 1993). If the medication cannot be changed, treatment for the depression should be instituted.

TABLE 5
Medications Implicated in Causing or Contributing to Depression

Drug/Drug Class	Support for Association	Strength of Evidence
Amphetamine withdrawal	E, C	Substantial
Anabolic Steroids	E, C	Substantial
Benzodiazepines	C	Inconclusive
Cimetidine, Rantidine	C	Inconclusive
Clonidine	A	Inconclusive
Cocaine withdrawal	E, C	Substantial
Cycloserine	C*	Inconclusive
Digitalis	E	Substantial
Glucocorticoids	E, C, A	Substantial
Levodopa	E, C, A	Inconclusive
Methyldopa	E, C, A	Inconclusive
Metoclopramide	C	Inconclusive
Oral contraceptives	C	Inconclusive
Propranolol (beta blockers)	E, C, A	Inconclusive
Reserpine	C, A**	Inconclusive dose-related
Verapamil (calcium channel blockers)	C	Inconclusive

Abbreviations:
E = epidemiological evidence (not necessarily conclusive)
C = multiple case reports in the literature
A = association widely accepted in assertions contained in reference texts and review articles
*Cycloserine symptoms may be dose-related.
**Reserpine-induced depression is dose-related and uncommon at dosages of 0.25 mg/day or less.

Adapted from *Major Depressive Disorder Clinical Guidelines: Major Depressive Disorder*, Department of Veterans Affairs, page 12.

Given the prevalence of substance abuse in individuals with spinal cord injury, clinicians must be aware of addictive, mood-altering medications—diazepam or baclofen for spasms and narcotic analgesics for pain—as well as other substances of abuse. Many addictive substances—including alcohol, sedatives, hypnotics, and narcotic analgesics—can cause or complicate depression (U.S. Dept. of HHS, 1993; Rundell and Wise, 1996; Kaplan and Sadock, 1994). In addition, withdrawal from stimulants such as amphetamines or cocaine is also associated with depression. Once the withdrawal symptoms dissipate, the depression may improve or resolve.

Thus, it is important to consider prescribed or illicit drugs as possible causative or exacerbating agents in every individual with SCI presenting with signs and symptoms of depression. This is especially important because of the high incidence of substance abuse in individuals with SCI, the possibility of self-medication with addictive substances, and the frequent necessity of prescribing addictive substances for conditions related to SCI. Prescription of addictive medications should be avoided if possible.

6. Conduct a comprehensive assessment of the social factors specific to spinal cord injury that contribute to depression to evaluate the adequacy of the individual's social support system in meeting basic needs and to determine the presence of depression in response to an inadequate support network. Specifically, the assessment should include but not be limited to:

- The individual's social network, including family members, friends, and community organizations
- The individual's financial resources
- Vocational and avocational interests and issues
- Current living arrangements, including wheelchair accessibility
- Adaptive equipment needs and resources
- Personal assistance needs and resources
- Transportation needs and resources

(Scientific evidence—II; Grade of recommendation—B; Strength of expert panel opinion—Strong.)

An individual with SCI can experience depression at any time. Although the assessment of contributing social factors should be completed during the individual's rehabilitation, it is equally important to continue the assessment after the individual has been discharged from the rehabilitation setting and returned

to community living. Because social factors can change, attention to key variables is essential to adequately assess their impact on individuals with SCI and depressive symptomatology. The issues associated with aging with a disability—decreased functional abilities, increased pain, increased need for personal care assistance and adaptive equipment, decreased energy, and aging caregivers—should be examined as possible contributing factors to depression.

Several studies have documented that a lack of social support is associated with depressive behavior. Schultz and Decker (1985) found that people who felt satisfied with their social contacts and persons who reported high levels of social support also reported high levels of psychological adjustment. Forty-one percent of the respondents named only one support person. Schultz and Decker (1985) concluded that many of the respondents would have numerous coping problems if they lost a spouse. Treischmann (1987) described similar scenarios for older people with disabilities. In a follow-up study drawing on the work of Schultz and Decker, it was determined that satisfaction with social contact is a significant predictor of depression (Crisp, 1992).

In an examination of the associations of different social relationships with depressive behavior among 182 people with acquired spinal cord injuries, individuals involved in relationships that reassured the worth of the individual were predictive of lower depression scores (Elliott et al., 1992). In a study of 34 individuals in SCI rehabilitation where the family was incorporated into the treatment program, better outcomes were revealed on six of seven dependent measures, when compared to the control group without the family involvement (Moore et al., 1994). Elliott and Shewchuk (1995) found that depressive behavior was associated with less leisure activities. Individuals with limited or poor social support, social isolation, and decreased opportunities for socialization may be at an increased risk for depression.

Individuals with spinal cord injury face exceptionally high costs for health care and meeting their lifetime medical care needs (DeVivo, 1990). Because of disability-related costs, e.g., transportation, personal assistance services, home modifications, adaptive equipment, the individual with spinal cord injury needs a higher income than his or her nondisabled counterpart to achieve the same standards of living (Treischmann, 1987). Studies by Means and Bolton (1994), McAweeney et al. (1996), and Tate et al. (1994) confirm that transportation services are an unmet need for many individuals with SCI. Access to assistive technology is essential for restoration of quality of life after an injury, and many individuals with spinal cord injury lack the health-insurance coverage to ensure that these resources are available to them (Tate et al., 1994). People who lack the resources to complete home modifications or to access the adaptive equipment necessary for increased independence may find their quality of life compromised and their risk for depression greater.

Fuhrer et al. (1993) noted that individuals who were not employed, in school, working as a volunteer, or involved in a self-improvement program reported increased depressive symptomatology. Using the World Health Organization's model of disability as a frame of reference, individuals with SCI may well face significant handicaps as a result of their disability and impairments. Architectural barriers in the community may compromise an individual's opportunities to work, socialize, go to school, or engage in recreational activities. Attitudinal barriers associated with society's lack of understanding or acceptance of individuals with disabilities can create additional challenges. Financial limitations, coupled with the high costs for meeting lifetime health-care needs, may jeopardize quality of life.

7. Assess the psychological factors specific to spinal cord injury that contribute to depression, including the following:

- Coping style
- Self-blame for the injury
- Unresolved conflicts from previous losses or traumas
- Preinjury psychological or psychiatric impairment
- Cognitive style
- Grief and bereavement from SCI

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

The risk for developing clinical depression involves genetic, psychological, and environmental factors. Specific psychological factors include coping style, character structure, and pattern of adaptation to stress. Other psychological factors contributing to depression after SCI include self-blame for the injury and unresolved conflicts from previous losses or traumas.

Individuals with preinjury histories of psychological or psychiatric impairment, particularly substance abuse, have greater difficulty adjusting to a spinal cord injury (Elliott and Frank, 1996; Judd et al., 1989; Tate, Maynard, and Forchheimer, 1993). Preinjury difficulties coping with the demands of life increase the risk of depression after injury. Elliott and Frank (1996) noted that although there is little direct empirical evidence in the literature, it is generally accepted that individuals who had effective coping skills prior to spinal cord injury are at less risk of developing psychopathology after injury.

There is also consistent evidence linking cognitive style to depression. Effective skills in problem solving and greater confidence in problem-solving abilities

have been found to be associated with lower rates of self-reported depression (Elliott and Harkins, 1991). Individuals who maintain higher levels of hope and of goal-directed behaviors evidence less distress, regardless of time since injury (Elliott and Harkins, 1991). Problem solving, hope, and locus of control (i.e., internal versus external) are thought to be responsive to clinical interventions (Elliott and Marmarosh, 1995).

As the primary care provider assesses an individual's psychological reaction to spinal cord injury, it is important to investigate how the individual is coping with the injury and specifically with the loss. Although the pattern of emotional reaction is unique to every person, coping with a spinal cord injury normally involves sadness, yearning, and intense feelings of loss. While bereavement may appear similar to depression, it does not ordinarily involve prolonged feelings of guilt, worthlessness, self-reproach, or thoughts of death, as seen in depressive disorders. Because grieving, or bereavement, is universal in the context of SCI, it is important to differentiate bereavement from a depressive disorder. Research indicates that depression is not a stage of recovery from SCI; in fact, most individuals with SCI will not become depressed (Cushman and Dijkers, 1991).

For years many people in the rehabilitation and lay community believed that a depressive episode was a valued reaction to SCI, one that enhanced eventual adjustment to the injury (Frank et al., 1987). However, examination of this assumption has shown that the occurrence of a depressive episode after SCI does not predict successful adjustment to the injury (Elliott and Frank, 1996). It is now clear that injury to the spinal cord does not predict the behavior of any individual. Behavior and emotion after injury are a complex interaction of personality, environment, and the nature of the injury.

Diagnosis

8. Use established diagnostic criteria to diagnose depression. (Scientific evidence—V; Grade of recommendation—C; Strength of expert panel opinion—Strong)

The DSM-IV criteria for a major depressive episode include the following:

- Five or more of the following symptoms that have been present during the same 2-week period and that represent a change from previous functioning. At least one of the symptoms must be either (1) depressed mood, or (2) loss of interest or pleasure. **Note**: Do not include symptoms that are clearly due to a general medical condition or to mood-incongruent delusions or hallucinations.

  - Depressed mood that lasts most of the day, nearly every day, as indicated either by subjective report (e.g., individual reports feeling sad or empty) or by observation (e.g., individual appears tearful). **Note:** In children and adolescents, the mood can be expressed as general irritability.

  - Markedly diminished interest or pleasure in all, or almost all, activities most of the day, nearly every day, as indicated either by subjective account or by observation.

  - Significant weight loss (when not dieting) or weight gain (i.e., a change of more than 5 percent of body weight in a month), or a decrease or an increase in appetite nearly every day. **Note:** In children, failure to make expected weight gains can be a symptom of depression.

  - Insomnia or hypersomnia nearly every day.

  - Psychomotor agitation or retardation nearly every day, which must be observed by others, not merely reported as subjective feelings of restlessness or of being slowed down by the individual.

  - Fatigue or loss of energy nearly every day.

  - Feelings of worthlessness or excessive or inappropriate guilt, which may be delusional, nearly every day; feelings that go beyond mere self-reproach or guilt about being sick.

  - Diminished ability to think or concentrate, or indecisiveness, nearly every day, as reported through subjective account or observation by others.

  - Recurrent thoughts of death, beyond a fear of dying; recurrent suicidal ideation without a specific plan; or a suicide attempt or a specific plan for committing suicide.

- Symptoms that do not meet the criteria for a mixed episode.

- Symptoms that cause clinically significant distress or impairment in social, occupational, or other areas of functioning.

- Symptoms that are not due to the direct physiological effects of a substance (e.g., a drug of abuse or a prescribed medication) or of a general medical condition (e.g., hypothyroidism).

- Symptoms that are not better accounted for by bereavement (i.e., after the loss of a loved one, symptoms that persist longer than 2 months or that are characterized by marked functional impairment, morbid preoccupation with worthlessness, suicidal ideation, psychotic symptoms, or psychomotor retardation).

The following sequence of steps may be particularly helpful in making informed diagnoses of a depressive disorder:

STEP 1.

Has there been at least one distinct period—either current or past—of persistent depressed mood or anhedonia (that is, markedly diminished interest or pleasure in almost all activities).

STEP 2.

Has there been a distinct period of elevated, expansive, or irritable mood present?

STEP 3.

Is the depressed mood or anhedonia due to either a general medical condition or a substance (as opposed to a primary depressive disorder)? In such cases, the following diagnoses may apply:

A. Mood disorder due to a general medical condition, with depressive features (or with major depressive-like episode):

1. Depressed mood or anhedonia.
2. Evidence from medical history, physical examination, or laboratory findings that symptoms are a direct physiological consequence of a general medical condition.
3. Not better accounted for by another mental disorder.
4. Not occurring exclusively during delirium.
5. Causes clinically significant distress or impairment in functioning.
6. In an individual case, the name of the particular medical condition is used (e.g., mood disorder due to hypothyroidism, with depressive features). Note that only the depressive mood or anhedonia needs to be present for a diagnosis of mood disorder due to a general medical condition, with depressive features; accompanying symptoms are not required (in contrast to the primary depressive disorders).
7. "With major depressive-like episode" means that the accompanying symptoms are present and meet the full criteria for a major depressive episode (see step 6 below), except for the exclusion criterion regarding general medical conditions.

B. Substance-induced mood disorder, with depressive features:

1. Depressed mood or anhedonia.
2. Evidence from history, physical examination, or laboratory findings that symptoms developed during or within a month of substance intoxication or withdrawal or that medication use is etiologically related to the symptoms.
3. Not better accounted for by a nonsubstance-induced mood disorder.
4. Not occurring exclusively during delirium.
5. Causes clinically significant distress or impairment in functioning.
6. Specify if onset occurred during intoxication (if substance is not being used therapeutically) or during withdrawal.
7. In an individual case, the name of the particular substance is used (e.g., prednisone-induced mood disorder, with depressive features). Only the depressed mood or anhedonia is necessary for a diagnosis of substance-induced mood disorder; accompanying symptoms are not required as they are for a "primary" depressive disorder.
8. There is no subtype of "with major depressive-like episode" in this category.

STEP 4.

Has the persistent depressed mood or anhedonia (with or without accompanying symptoms) occurred within 2 months after the loss of a loved one? In such cases this may be best described as bereavement.

A. Normal bereavement gradually dissipates with time. In individuals with SCI, grief or adjustment reactions to the injury may be treated with psychotherapeutic interventions to prevent the development of major depression, especially in those at particular risk for depression.

1. Psychopharmacologic interventions may be helpful if the symptoms related to grief or adjustment interfere with the ability to participate in rehabilitation.
2. If the individual with SCI remains incapacitated by grief for longer than 2 months, the diagnosis of a depressive disorder should be considered and treatment initiated if the diagnostic criteria are met (U.S. Dept. of HHS, 1993).

B. A diagnosis of major depressive disorder instead of bereavement is made if the grief symptoms meet the criteria for a major depressive episode (see step 6 below) and either:

1. Last more than 2 months, or
2. Include marked functional impairment, marked preoccupation with worthlessness,

suicidal ideation, psychotic symptoms, or psychomotor retardation.

3. Bereavement is not a disorder; it is a V code nonpathological condition that may be a focus on clinical attention.

STEP 5.

Have there been at least 2 years of depressed mood most of the day, for more days than not, with

A. Presence while depressed of at least 2 of the following: poor appetite or overeating; insomnia or hypersomnia; low energy or fatigue; low self-esteem; poor concentration or difficulty making decisions; feelings of hopelessness.

B. Symptom-free periods never lasting more than 2 months at a time; no major depressive episodes [see step 6 below] during the first 2 years.

C. No manic episode, mixed episode, hypomanic episode, or cyclothymic disorder at any time

D. Not occurring only during course of chronic psychotic disorder.

E. Not due to direct physiological effects of substance or general medical condition.

F. Clinically significant distress or impairment in functioning.

G. In such cases, this constellation of symptoms may be best diagnosed as a dysthymia disorder.

H. A current diagnosis of dysthymic disorder is still permissible if a major depressive episode occurred and fully remitted at least 2 months before the onset of the dysthymic disorder.

I. After the initial 2 years of dysthymic disorder, it is permissible to diagnose both dysthymic disorder and major depressive disorder (if major depressive episodes are superimposed after the initial 2 years).

J. If a major depressive episode does occur during the first 2 years of dysthymic-type symptoms, then the diagnosis of dysthymia is not made; rather, the diagnosis given is major depression, in partial remission (so-called “double depression”).

STEP 6

Is the number, intensity, and total constellation of symptoms characteristic of a major depressive disorder according to the following criteria? A major depressive episode includes:

A. At least 5 of the following nearly every day during the same 2-week period, with at least 1 being either depressed mood or loss of interest or pleasure:

1. Depressed mood most of day.

2. Markedly diminished interest or pleasure in almost all activities most of the day.

3. Appetite disturbance or significant weight change.

4. Insomnia or hypersomnia.

5. Observable psychomotor agitation or retardation.

6. Fatigue or loss of energy.

7. Feelings of worthlessness or excessive inappropriate guilt.

8. Trouble thinking or concentrating, or indecisiveness.

9. Recurrent thoughts of death or suicide without a plan; or a suicide attempt; or a specific plan for committing suicide.

B. Criteria not met for a mixed episode.

C. Clinically significant distress or impairment in functioning.

D. Not due to the direct physiological effects of a substance or a general medical condition.

E. Not better accounted for by bereavement.

STEP 7.

If the constellation of symptoms fails to meet the criteria for a major depressive disorder, do the symptoms meet the criteria for either of the following?

A. Adjustment disorder with depressed mood:

1. Depressed mood that occurs in response to and within 3 months of the onset of an identifiable external stressor

2. Significant impairment in functioning or marked distress in excess of what would be expected from the stressor

3. Symptoms that do not meet the criteria for another specific Axis I disorder and are not just an exacerbation of preexisting Axis I or II disorder

4. Symptoms that do not represent bereavement

5. Symptoms that do not persist for more than 6 months beyond the termination of the stressor (or its consequences).

6. Specify if acute (i.e., when the duration of symptoms is less than 6 months) or chronic (i.e., when the duration is 6 months or more). Chronic is used when the stressor or its consequences in the patient’s environment are enduring.

B. Depressive disorder not otherwise specified (NOS). This category is used to describe cases in which the constellation of symptoms fails to meet the criteria for dysthymia disorder, major depressive disorder, adjustment disorder with depressed mood, substance-induced depression, or depression secondary to a general medicine condition, either in terms of the number or the duration of symptoms. (DSM-IV)

9. Identify the mental health factors that indicate referral to the appropriate mental health-care provider including:

- Active suicidal ideation
- Psychotic depression
- Bipolar disorder
- Complex psychiatric diagnoses such as depression that are associated with post traumatic stress disorder, obsessive-compulsive disorder, eating disorder, schizophrenia, schizophreniform disorder, schizoaffective disorder, and personality disorders
- Persistent substance abuse complicating the diagnosis and/or management of depression (especially when detoxification or more intensive treatment beyond a 12-step program is needed)

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

When assessing individuals with SCI for depression, specific mental illnesses and other situations that call for a mental health referral must be recognized. Such scenarios might involve mental illnesses comorbid with depression, non-mood disorders that present with depressive symptoms, or particular presentations of mood disorders that mandate referral to a mental health provider. For example, a mental health provider must evaluate individuals with SCI who present with active suicidal ideation to determine if emergency psychiatric hospitalization is needed. Likewise, referral to a qualified mental health provider is generally indicated when individuals present with psychosis or bipolar disorder—at least until stabilized with the appropriate psychotropic medications. Mental health providers also should be consulted in situations in which complex psychiatric diagnoses are in play, complicating the diagnosis and management of depressive symptoms.

Treatment

10. Formulate a treatment plan identifying:

- Which treatments are to be provided by the primary care physician
- What type of individual and family education needs to be provided and by whom
- Who will address comorbid conditions and how those conditions will be treated
- Specific criteria for referring the individual to a mental health provider

(Scientific evidence—IV; Grade of recommendation—C; Strength of expert panel opinion—Strong)

If the primary care physician decides to treat the presenting individual, the physician needs to select treatment predicated upon the specific depressive symptom clusters. The following treatment modalities are highly generic and do not, therefore, cover the entirety of clinical situations:

- Cognitive symptoms
- Affective symptoms
- Somatic symptoms

Psychopharmacological agents may be particularly useful for individuals with spinal cord injury who present mood disturbance and vegetative symptoms of sufficient severity to disrupt their ability to perform in social, personal, and vocational roles. Such agents may be useful in cases in which few cognitive symptoms of behavior are manifested.

Nevertheless, available research suggests that such agents are not particularly useful over time in the treatment of depressive behaviors in which cognitive symptoms are salient (Antonuccio, Danton, and DeNelsky, 1995), although there are variable results in this area. Psychopharmacological agents alone are not designed to address cognitive-behavioral deficiencies that render an individual vulnerable to depressive symptomatology, nor can they resolve environmental and interpersonal issues that may contribute to the onset and maintenance of depressive behaviors. Thus, individuals who present cognitively based depressive symptoms may require cognitive-behavioral interventions designed to improve the individual's coping repertoire, thinking patterns, and problem-solving strategies in an inpatient, outpatient, or community setting. Community-based and social interventions may be required to address environmental conditions that may be contributing to the onset and maintenance of depressive behavior.

Finally, physical conditions—notably the experience of pain and physical discomfort—also will require appropriate pharmacological, cognitive-behavioral, and psychological interventions in order to alleviate painful sensations and augment coping skills, which could in turn alleviate depressive symptomatology (Elliott and Frank, 1996).

A specific treatment plan needs to be tailored to address the individual's presentation of depressive

signs and symptoms and the various phases of treatment. The treatment of depression usually consists of an acute phase lasting 4 to 12 weeks, during which treatment is directed at reducing or eliminating depressive symptomatology; a continuation phase lasting 4 to 9 months during which treatment is directed at preserving symptom reduction; and a maintenance phase during which treatment is directed at preventing the recurrence of subsequent depressive episodes (APA, 1993). In general, treatment efficacy has been demonstrated for the acute phase of treatment, depending on the severity of the depression, with the use of psychotherapy, psychopharmacology, and electroconvulsive therapy, but has not been definitively established for the continuation and maintenance phases of treatment (APA, 1993).

Early detection and treatment of depressive symptomatology can be instrumental in improving the patient's response to rehabilitation and to continued independent community living, as well as in reducing the patient's psychosocial sequelae of his or her injury. In the majority of cases, it is the primary care physician who initiates the initial assessment of depression. If clinically appropriate, the primary care provider should initiate psychopharmacological treatment and, if warranted, refer the individual to a mental health provider for supportive counseling. There must be a very low threshold for referral to a mental health provider. If any of the following features manifest, referral to a mental health provider is appropriate:

- Single antidepressant treatment and brief supportive counseling that prove ineffective
- Depressive symptoms that are multifaceted and complex
- Presence of comorbid psychiatric disorders

11. Provide or refer for psychotherapy by matching the type of psychological intervention to both the identified problem and the therapeutic capacity of the individual. (Scientific evidence—IV; Grade of recommendation—C; Strength of expert panel opinion—Strong)

Multiple psychotherapeutic modalities—such as insight-oriented, psychodynamic supportive or cognitive psychotherapy, or marital, family, or group therapy—can help individuals with SCI contend with depression. Although these guidelines discuss the various forms of therapy as distinct entities, in practice clinicians combine the techniques and strategies of the different schools. The choice of therapeutic modalities should be individually tailored based upon each individual's condition, coupled with his or her coping capability (APA, 1993).

Supportive psychotherapy focuses on helping the individual utilize existing personal strategies to cope with depression. All forms of psychotherapy include some support, some insight, and some assistance through a relationship with the clinician, but supportive psychotherapy puts greater emphasis on support than on insight. The person with SCI and the clinician identify the ways in which the person was best able to deal with stressors in the past and then practice using those methods to deal with current stressors. Healthy defenses are strengthened, and impaired integrative capacities are restored (Moore et al., 1994; Elliott et al., 1992; Crisp, 1992).

Individuals with spinal cord injury who benefit from insight-oriented psychotherapy share several characteristics. First, they have sufficient psychological insight to see the possibility of a psychological basis to their problems; second, they have an ability to form an alliance with the clinician; third, they have the capacity to form stable relationships; and fourth, they have an ability to tolerate painful or threatening ideas and feelings in therapy without losing their ability to function in their usual social and occupational roles between outpatient sessions (Flaherty, Davis and Janicak, 1993).

In insight-oriented psychotherapy, the clinician begins by fostering a therapeutic alliance with the client while trying to understand both the conflicts underlying the depression and the defenses used to maintain the conflicts. Defense mechanisms are specific, unconscious, intrapsychic adjustive efforts that are used to resolve emotional conflict and free the person from anxiety. In the course of the therapy, the clinician uses interpretations to clarify the client's thoughts or feelings about a particular subject or to compare the handling of past incidents to similar incidents in the present. Within the context of therapy, the person reexperiences with the clinician the same unconscious thoughts and feelings experienced earlier in life toward significant others (transference). In time, transference feelings are analyzed so as to reconstruct and resolve conflicts, thereby helping the person to live a life free of self-constructed barriers. Some evidence suggests that the interpersonal components of this type of psychotherapy may delay but not prevent a reoccurrence of depression symptomatology (Frank, et al., 1990). However, the efficacy of insight-oriented therapy in the treatment of depression, either in conjunction with pharmacotherapy or alone, has not been subjected to a significant number of controlled studies.

Brief psychodynamic psychotherapy uses transference, dreams, fantasies, verbal associations, and nonverbal behavior to explore earlier derivatives of current problems as they become evident in the person's relationship with the clinician. In a limited number of sessions, the clinician tries to establish a connection between the problem for which help is being sought and previous life experiences, so that the person can acquire insight into how problems originated and how they might be handled more effectively. The efficacy of brief psychodynamic psy-

chotherapy as a single modality in the treatment of depression has not been conclusively demonstrated by controlled studies (Thompson, Gallagher, and Breckenridge, 1987). Research on combined pharmacotherapy and brief psychodynamic psychotherapy is equally sparse and inconclusive (Daneman, 1961; Covi et al., 1974).

Cognitive therapy is an active, directive, time-limited treatment based on the rationale that a person's depressed mood and behavior are determined by cognitive or automatic thoughts, derived from previous experiences in life (Beck et al., 1979). The faulty perceptions that predispose a person to depression include a tendency to interpret ongoing experiences in a negative way, a negative evaluation of the self, and a negative view of the future. In cognitive therapy, the depressed person with SCI is helped to recognize the idiosyncratic way in which he or she interprets events because of a preoccupation with negative thoughts. The clinician helps the person to recognize and modify the central assumptions governing the person's life. In time, new assumptions are explored and experiments are devised as homework assignments to challenge old beliefs and change old behaviors. Some evidence suggests that cognitive therapy reduces depressive symptoms during the acute phase of less severe nonmelancholic forms of major depression (Rush et al., 1977), but not significantly differently from pill placebo therapy coupled with clinical management (Elkin et al., 1989).

The supportive-educational approach to therapy focuses on the present situational problem, aiming to control symptoms and enhance personal adaptation to stressors. Methods include traditional interviews, behavior therapy, relaxation techniques, biofeedback, somatic treatments, sex therapy, and group therapy. Any one or combination of methods may be used.

Marital and family problems are oftentimes present with a mood disorder. Comprehensive treatment requires that these problems be assessed and addressed. Marital and family problems can be a consequence of depression, but they can also increase vulnerability to depression and in some instances retard recovery. Research suggests that marital and family therapy may reduce depressive symptoms as well as the risk of relapse in people who have marital and family problems (O'Leary and Beach, 1990; Jacobson et al., 1991). The utility of these treatments for depressed people without specific family or marital discord is less clear.

Group therapy is particularly useful in the treatment of depression in the context of bereavement or other common stressors such as chronic illness (APA, 1993). In group therapy, members benefit from the example of others who have successfully dealt with the same or similar challenges. Survivors enhance their self-esteem by becoming role models for others. Consumer-oriented support groups for individuals with depression serve a similar function by enhancing the self-esteem of and support network for participants and their families.

Behavior therapy is based on a functional analysis of observed behavior and social learning theory (Ferster, 1973; Bandura, 1977). The techniques involve activity scheduling (Lewinsohn et al., 1984), self-control therapy (Rehm, 1979), social skills training (Bellack, Hersen, and Himmelhoch, 1983), and problem solving (Nezu, 1986). Several studies suggest that the combination of behavior therapy with pharmacotherapy over time may sustain a therapeutic response to treatment of mild to moderately severe depressions (Brown and Lewinsohn, 1990; Usaf and Kavanagh, 1990; Nezu and Perri, 1989).

Psychopharmacological Agents

12. If indicated, select appropriate antidepressant medications. Psychopharmacological agents should be considered for individuals who present significant biological, somatic and/or mood-related symptoms of sufficient severity to disrupt the person's life and activities of daily living. Selection of a specific agent should be predicated upon the unique characteristics of the individual and the presenting signs and symptoms of depression. (Scientific evidence—I; Grade of recommendation—A; Strength of expert panel opinion—Strong)

The biologic treatment of depression is based on the theory that depression is associated with a decreased concentration of serotonin and/or norepinephrine in the synapse (U.S. Dept. of HHS, 1993). Other possible neurochemicals relevant to the biology of depression include dopamine and neuropeptides such as corticotrophin-releasing factor (CRF) (U.S. Dept. of HHS, 1993). Later research suggests that antidepressants normalize the firing rate of serotonin and noradrenergic neurons. Regardless of the mechanism, all antidepressants exert their effects by facilitating neurotransmission of serotonin and/or norepinephrine (Eric, 1991; Goodman, Price, and Rasmussen, 1989; Hellerstein, Yanowitch, and Rosenthal, 1993; Keller and Hanks, 1994; Pigott, Pato, and Bernstein, 1990; Prien, Kupfer, and Mansky, 1984; Rickels et al., 1993; Walsh, Stewart, and Roose, 1984). Available agents and information concerning dosing, adverse effects, mode of action, pharmacokinetics, and drug interactions are listed in tables 6, 7, 8, and 9.

The newer antidepressants are safer and better tolerated than the older tricyclic antidepressants (TCAs) and monoamine oxidase inhibitors (MAOIs). Use of tricyclic antidepressants with this population is complicated by autonomic dysfunction related to the SCI, making individuals with SCI more vulnerable to anticholinergic side effects and orthostatic hypotension common with TCAs. Use of MAOIs in people

with SCI carries even greater risk than TCAs due to numerous drug interactions—some of which are lethal—and the need for dietary restrictions. However, MAOIs are powerful medications in the treatment of refractory depression. Given the multiple complexities inherent in the use of MAOIs, it is essential to obtain psychiatric consultation if these medications are indicated.

Although antidepressants are considered equally effective in the treatment of depressive disorders, selection of a particular agent is often guided by profiling the potential side effects. The goal is to utilize side effects—such as sedation or activation—as therapeutic effects.

The following somatic interventions for individuals with SCI are offered as general guidelines, given the high degree of variability among individuals. The medical literature is sparse regarding the use of antidepressants in the treatment of depression in people with SCI. Therefore, medication selection must be based on patient-specific variables with careful consideration given to the risks and benefits of these agents.

When prescribing an antidepressant, it is important to:

- Educate the individual about the medication
- Consider all of the risks and benefits of these agents
- Adjust dosages at appropriate intervals and in appropriate amounts, being careful not to undertreat
- Allow for an adequate trial (usually 4 to 6 weeks at therapeutic dose range)
- Continue treatment for 9 to 12 months when medicating a major depression
- Consider lifelong antidepressant therapy for people with recurrent depression (50 percent risk of recurrence after 1 major depressive episode (MDE), 80 to 90 percent after 2 MDEs) (Angst, 1990; Kupfer, 1991)
- Use medications that require once or twice per day dosing whenever possible to optimize compliance
- Consult a psychiatrist if the individual doesn't respond to one or two trials of antidepressants
- Consult a psychiatrist if augmentation strategies are needed
- Consult a psychiatrist if the individual becomes suicidal or psychotic. Typically, psychotic depression does not respond to antidepressants alone and, therefore, more complex psychotropic medication management by a psychiatrist is necessary. Individuals with psychotic depression are at a distinctly higher risk for suicide or homicide
- Taper medication rather than stopping abruptly, if possible
- When an individual is taking multiple medications, select an antidepressant with minimal interactions with other prescribed medications (for example, venlafaxine, mirtazapine, low-dose sertraline, and bupropion.)

TABLE 6
General Treatment Recommendations

Symptom	Suggested Antidepressant
Decreased appetite/weight loss	Mirtazapine, tertiary TCAs, some SSRIs (especially paroxetine)
Increased appetite/weight gain	Fluoxetine, perhaps other SSRIs and newer agents (except Mirtazapine)
Insomnia	Sedating antidepressants: Trazodone, nefazodon, mirtazapine, paroxetine, tertiary TCAs
Hypersomnia	Activating antidepressants: Fluoxetine, venlafaxine, bupropion, sertraline, secondary TCAs
Psychomotor agitation	Sedating antidepressants: Trazodone, nefazodone, irtazapine, paroxetine, tertiary TCAs
Psychomotor retardation	Activating antidepressants: Fluoxetine, venlafaxine, bupropion, sertraline, secondary TCAs
Fatigue/low energy	Activating antidepressants: Fluoxetine, venlafaxine, bupropion, sertraline, secondary TCAs
Decreased concentration	Activating antidepressants: Fluoxetine, venlafaxine, bupropion, sertraline, secondary TCAs
Suicidal ideation	Avoid TCAs due to risk of overdose
Neuropathic pain and depression	Amitriptyline, nefazodone, any serotonergic antidepressant

NOTE: Avoid bupropion, TCAs, and maprotiline in individuals at risk for seizure.

Psychopharmacological Guidelines

Medication selection should be based on patient-specific factors. Pharmacokinetics and pharmacodynamic variables should be considered, including, but not limited to half-life, presence of active metabolites, water/fat solubility, protein binding, therapeutic index, side-effect profile, drug interactions (i.e., drug-drug, drug-food, and drug-disease), and additional considerations in special populations (e.g., people with renal and/or hepatic impairment, the elderly, and medically frail individuals). In addition, antidepressants known to reduce seizure threshold (e.g., bupropion and maprotiline) should be avoided, especially in individuals with a history of stroke or head injury.

Mood disorder treatment is increasingly recognized as a long-term process because mood disorders appear to be chronic in nature and patients with mood disorders experience high rates of relapse on discontinuation of drug therapy (Angst et al., 1973; Frank et al., 1990; Keller et al., 1982; Kupfer et al., 1992; Montgomery and Montgomery, 1992; Prien and Kupfer, 1986; Prien et al., 1984). Currently available medications may restore function, but the disease process is not halted. In this context, Kupfer (1991) has proposed three phases of treatment: an acute treatment phase, a continuation phase, and a maintenance phase. The phases are defined in relation to the status of symptoms and involve the concepts of treatment response, relapse, remission, recurrence, and recovery (Frank et al., 1991; Kupfer, 1991).

Response refers to some effect following an action, in this case a decrease in clinically significant symptoms following initiation of treatment. Relapse involves the return of some symptoms during or upon cessation of treatment. Remission refers to a clinically meaningful decrease in symptoms. Recurrence describes the return of symptoms after a remission. Recovery describes a more sustained remission from the most recent episode.

Acute Phase. Acute treatment includes making a diagnosis; deciding whether to treat with medications; choosing an agent; initiating treatment; and monitoring the degree of symptom reduction, compliance, and side effects. This stage generally lasts 4 to 12 weeks.

Continuation Phase. If a response is obtained, then the continuation phase, which consists of monitoring for completeness of response and side effects, ensues. Discontinuation of medication during or before this phase is associated with a relatively higher rate of relapse, and the rate at which the medication is discontinued may affect the likelihood of relapse (Kupfer, 1991; Prien and Kupfer, 1986). Continuation lasts 4 to 9 months and can be thought of as a consolidation phase. A recent World Health Organization consensus meeting suggested that the minimum period of time for continuation treatment is 6 months if the target symptoms are in remission (World Health Organization, 1989).

Maintenance Phase. Maintenance phase is thought of as prophylactic, but it is becoming increasingly clear that for many people this phase is essential not only for preventing new episodes but also for maintaining the response, because the illness persists. Newer data suggest that medication dosing during maintenance should continue at the same level used during the acute phase (Frank et al., 1990; Kupfer et al., 1992).

Most experts now agree that the majority of individuals with a mood disorder will have more than one depressive episode. Recurrence rates for depression are estimated to be at least 50 percent for patients with one prior MDE and 80 percent to 90 percent for patients with two prior MDEs (Angst, 1990; Kupfer, 1991). These high rates of recurrence and relapse highlight the need for consideration of the efficacy of antidepressant treatment in the continuation and maintenance phases of treatment.

Antidepressant Pharmacotherapy

Acute Phase Treatment. It is generally accepted that individuals who show significant improvement during the acute treatment phase should be continued on antidepressant drugs for at least 6 months (Altamura and Percudani, 1993; Kupfer et al., 1992; World Health Organization, 1989). Drugs that are effective in the acute treatment of an MDE have generally been found to be efficacious in continuation treatment. Fortunately, the same appears to be true for these drugs' efficacy in maintenance treatment. The consistency of these findings, in the face of the relative safety of the majority of antidepressant drugs, underscores the importance of continuation and maintenance treatment. It should be noted, however, that a growing number of anecdotal cases concerning antidepressant "poop-out" are appearing in the medical literature. In these cases, the medications appear to lose efficacy after 9 or more months of continuous use. In some cases, temporary discontinuation followed by reinitiation of the same medication, introduction of various adjunctive therapies, and use of an alternative medication has been a successful strategy with some people.

Continuation Phase Treatment. Most classes of antidepressants have been studied in continuation treatment, although most of these studies were extensions of acute treatment studies and the number of participants in many of the studies was small. Imipramine, amitriptyline, desipramine, nortriptyline, maprotiline, lithium, bupropion, phenelzine, fluoxetine, sertraline, paroxetine, fluvoxamine, trazodone, nefazodone, and moclobemide have shown efficacy in continuation treatment.

Maintenance Phase Treatment. Maintenance treatment studies have taken on considerable importance in light of recent findings of high rates of relapse and recurrence in individuals with relatively uncomplicated MDE following continuation treatment

(Frank et al., 1990; Kupfer et al., 1992; Prien et al., 1984). Seventy to 90 percent of patients with a successfully treated MDE will experience a recurrence of illness when placebo is substituted for active medication during a 3-year maintenance phase, as opposed to only 15 percent to 20 percent taking full-dose imipramine (Frank et al., 1990). Newer antidepressants are generally being tested for efficacy in the maintenance treatment of MDE. All of the available SSRIs have been studied. Fluoxetine (Montgomery et al., 1988), sertraline (Doogan and Caillard, 1992), and paroxetine (Eric, 1991) all show a significantly lower rate of relapse than placebo. A large, multicenter maintenance study of sertraline in comparison with imipramine in people with double depression (i.e., dysthymia and MDE) or chronic MDE has demonstrated that both of these drugs are effective in the maintenance treatment of these chronic forms of depression (Keller and Hanks, 1994).

In summary, one can infer from the results of the studies that have been conducted to assess the efficacy of antidepressant drugs in the maintenance phase of treatment of MDEs that most medications are effective. These studies suggest that when maintenance treatment is initiated, full antidepressant doses should be continued. Rates of depressive relapse appear to be higher when antidepressant drugs are discontinued rapidly compared with a slow (3 to 4 weeks) taper (Kupfer, 1991; Robinson et al., 1991). Antidepressants can be safely used if practitioners consider the pharmacokinetics and pharmacodynamic variables, such as presence of active metabolites, water/fat solubility, protein binding, therapeutic index, side-effect profile (including seizure potential), drug interactions (e.g., drug-drug, drug-food, drug-disease), and additional considerations in special populations.

TABLE 7
Dosing Table for Antidepressants

Agent	Dose	Comments
Tricyclic Anti-depressants (TCAs)		
Amitriptyline	Initial: 50–100 mg hs Range: 50–100 mg/day Max: 300 mg/day **	Not recommended for use in the elderly
Desipramine	Initial: 100-200 mg/day Range: titrate as tolerated Max: 300 mg/day	—Serum levels are associated with efficacy —Therapeutic level 125-300 ng/mL —Initial adult dose may start lower
Doxepin	Initial: 75 mg/day Range: 75-150 mg/day Max: 300 mg/day	—Higher doses, (up to 300 mg/day) generally for more severe anxiety or depression —Although antidepressant effect is generally seen in 2-3 weeks, anxiolytic activity is rapid
Imipramine	Initial: 75 mg/day Range: titrate as tolerated Max: 200 mg/day	
Nortriptyline	Initial: 25 mg tid or qid Range: titrate as tolerated Max: 100 mg/day	—Serum levels are associated with efficacy —Therapeutic level 50-150 ng/mL
Protriptyline	Initial: 15-40 mg/day in 3-4 divided doses Range: titrate as tolerated Max: 60 mg/day	—FDA approved for obstructive sleep apnea —Monitor cardiovascular closely at 20 mg/day
Trimipramine	Initial: 75 mg/day in divided doses Range: 50-150 mg/day Max: 200 mg/day	
Selective Serotonin Reuptake Inhibitors (SSRIs)		
Fluoxetine	Initial: 20 mg/day Range: 20-40 mg/day Max: 80 mg/day	—Administer doses > 20 mg on a once (am) or twice (am, noon) daily schedule
Paroxetine	Initial: 20 mg/day Range: 20-60 mg/day Max: 50 mg/day	—Use elderly dosing for debilitated persons —Dose usually in the am —Titrate (in increments of 10 mg/day in 1-week intervals

Sertraline	Initial: 50 mg/day Range: 50-200 mg/day Max: 200 mg/day	—Titrate in elderly in increments of 25 mg/day q 2-3 days —Administer doses in the am or pm
Other Antidepressants		
Nefazodone	Initial: 200 mg/day in 2 divided doses Range: 300-600 mg/day	—Titrate 100-200 mg/day in 2 divided doses at (1 week after initiation
Trazodone	Initial: 150 mg/day Range: increase as tolerated Max: 400 mg/day in divided doses	—Titrate in 50 mg/day (q 3-4 days —Dose major portion of daily dose q hs due to sedation —Adequate therapeutic response may not be evident until the 1st and 2nd weeks
Venlafaxine	Initial: 75 mg/day in 2-3 divided doses Range: 150 mg/day Max: 225 mg/day	—Administer with food —Titrate in increments of 75 mg/day at $\geq$ 4 day intervals —For severely depressed, may increase dose to 375 mg/day in 3 divided doses —No dose adjustment is necessary for the elderly, but use care individualizing doses —In patients with hepatic impairment: decrease dose 50% —In patients with renal impairment (GFR 1-70 mL/min) decrease dose 25% —In patients on dialysis: decrease dose 50%
Amoxapine	Initial: 50 mg bid or tid Range: 200-300 mg/day Max: 400-600 mg/day if no history of seizures	—For the adult dosage, may increase dose to 100 mg bid or tid in first week —For the elderly, may increase to 50 mg bid or tid in first week —For the elderly, the maintenance dose usually yields adequate control but some may need higher doses Classified as category C, has been know to be ter atogenic
Bupropion	Initial: 100 mg bid Range: increase per response to 300 mg/day Max: 450 mg/day	—Do not exceed dose increase of 100 mg/day in a 3-day period —Doses of 300 mg/day should be given in 3 divided doses —Doses of 450 mg/day should be given in 3-4 divided doses —Use cautiously in patients with renal or hepatic impairment —Adequate therapeutic response may not be evident for 4 weeks
Maprotiline	Initial: 75 mg/day Range: 150 mg/day Max: 225 mg/day	—Maintain initial dose for 2 weeks due to long half-life. —Titrate in 25 mg increments —Adequate therapeutic response may not be evident for 1 week, usually 2-3 weeks

Source: Adapted from "Antidepressants." In: Olin Br. (Ed). Drug Facts and Comparisons, St. Louis, Missouri: Facts and Comparisons Inc.; 1996; and Semla, T.P., J.L. Beizer, M.D. Higbee, Geriatric Dosage Handbook. 2nd edition 1995-1996. APhA: Lexi-comp Inc., 1995.

* Range refers to usual therapeutic range
** Max dose of 300 mg/day should be reserved for severely ill patients

NOTE: SPECIAL CONSIDERATION SHOULD BE GIVEN WHEN USING ANY OF THESE MEDICATIONS IN THE ELDERLY AND IN INDIVIDUALS WITH HEPATIC OR RENAL INSUFFICIENCY AND CNS COMPROMISE (TBI, DEMENTIA, ETC.).

TABLE 8
Pharmacokinetic Profiles of Antidepressants

Drug	Half-Life of Parent Drug (h)	Plasma Protein Binding (%)	Active Metabolites	Half-Life of Metabolite (h)	Therapeutic Index
TCAs					Narrow
Amitriptyline	9-46	96	Nortriptyline	18-48	
Imipramine	6-28	80-96	Desipramine	12-28	
Nortriptyline	18-48	87-93	10-hydroxynortriptyline	18-48	
SSRIs					Wide
Fluoxetine	4-6d	95	Norfluoxetine	4-16d	
Fluvoxamine	15	77	None or inactive		
Paroxetine	21	95	None or inactive		
Sertraline	26	-95	Desmethylsertraline		
Venlafaxine	4	27	O-desmethylvenlafaxine	10	Unknown
Bupropion	10-21	80	Hydroxybupropion,	22	Narrow
			erythrobupropion,	27	
			threohydrobupropion	20	
Triazolopyridines			A		Wide
Trazodone	6-13	89-95	MCPP	4+	
Nefazodone	2-4	>95		4+	
			MCPP	4+	
			a	4+	
			hydroxynefazodone		
			triazoledione		

Adapted from Cohen, L.J.. C.L. DeVane. "Clinical Implications of Antidepressant Pharmacokinetics and Pharmacogenetics." The Annals of Pharmacotherapy, 1996 December, Volume 30, p. 1472.

NOTE: SPECIAL CONSIDERATION SHOULD BE GIVEN WHEN USING THESE MEDICATIONS IN THE ELDERLY AND IN PEOPLE WITH HEPATIC OR RENAL INSUFFICIENCY AND CNS COMPROMISE (TBI, DEMENTIA, ETC.).

TABLE 9
Antidepressant Drugs for Patients with Depressive Disorder

Antidepressant	Usual Starting Dosage (mg/day)	Usual Maintenance Dosage (mg/day)	Sedation	Anticholinergic Effects	Orthostatic Hypotension	Activation
SSRIs						
Fluoxetine	20	20-40	+	0	0	+
Sertraline	20	100-200	+	0	0	+++
Paroxetine	20	20-60	+	0/+	0	+
Tertiary amines						
Amitriptyline	75	75-300	+++	+++	+++	+
Imipramine	75	75-300	++	++	++	+
Doxepin	75	75=300	+++	++	+++	+
Secondary amines						
Nortriptyline	10-50	50-200	++	+	+	+/0
Desipramine	75	75-300	+	+	+	0
Monoamine Oxidase inhibitors (MAOIs)						
Phenelzine	0/5 mg/kg	1 mg/kg	+	0	+++	0
Tranylcypromine	20	20-30	0	0	+++	0
Second-generation agents						
Trazodone	50	50-400	+++	0	++	0
Bupropion	200	300-450	0	0	0	++
Venlafaxine	75	150-375	+	0	0	+
Nefazodone	200	300-600	++	0	+	0
Mirtazapine	15	15-45	+++	0/+	0	0

Adapted from Cohen, L.J. "Rational Drug Use in the Treatment of Depression," Pharmacotherapy ,1997 Vol. 17, No. 1, p. 48.

NOTE: SPECIAL CONSIDERATION SHOULD BE GIVEN WHEN USING THESE MEDICATIONS IN THE ELDERLY AND IN PEOPLE WITH HEPATIC OR RENAL INSUFFICIENCY AND CNS COMPROMISE (TBI, DEMENTIA, ETC.).

Environmental and Social Factors and Social Support System

13. Address environmental and social factors and refer to a social worker, rehabilitation counselor, or case manager, as appropriate. When problems in the individual's support system are identified, treatment interventions should be implemented to strengthen the social support system. These interventions should be directed at one or more of the following areas:

- Education and information regarding available resources
- Referrals to existing community resources
- Development of alternatives to access services or assistance where no existing community resource is readily available
- Advocacy to change public policy to ensure that persons with SCI have the resources to meet their lifelong needs

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

A lack of knowledge about community resources can contribute to depression in individuals with inadequate social support systems. Initiating referrals or providing information regarding the procurement of disability benefits, placement options, vocational and educational assistance, assistive technology resources, transportation resources, and personal care assistance resources can help eliminate some environmental stressors. DeJong et al. (1981) found that 6 variables—transportation, economic disincentives, education, level of physical function, number of vocational rehabilitation services received, and age—accounted for 60.7% of the variance in productivity outcomes and that marital status alone accounted for 24.2% of

the variance in independent living arrangements. The handicapping role of the environment needs to be addressed.

Involvement in peer support and other counseling groups, psychoeducational groups, and independent living center activities can provide opportunities for the individual to expand his or her social support network, explore alternative ways of assuming responsibility for directing his or her own life, and learn new coping strategies from other individuals with impairment (Treischmann, 1987). Gerhart et al. (1992) and Means and Bolton (1994) emphasize the importance of independent living services for individuals with spinal cord injury.

Treischmann (1987) states, "Reduced access to satisfying activity can certainly lower mood, which tends to lower a person's interest in activity, which further lowers mood. Thus, a vicious cycle evolves. Consequently, rather than focusing primarily on treating mood through pharmaceuticals or counseling, the key may be to reduce the environmental barriers to normal activity."

In a study involving 140 individuals with spinal cord injury residing in the community, Fuhrer et al. (1993) had results suggesting that the level of depressive symptomatology was higher for people with spinal cord injury who live in the community than for people in the general population. In terms of the World Health Organization's model of disablement, depression is viewed as an impairment. Fuhrer et al. (1993) found that depression independently contributed to three dimensions of handicap—social integration, occupation, and mobility—though it was not associated with greater disability. The potential additive influence of impairments associated with spinal cord injury and depression may warrant additional scientific investigation.

Although local, state, and federally funded resources may not be readily available to address the unmet social support needs of people with spinal cord injury, there may be alternative support opportunities in local communities. Churches, veterans service organizations, civic clubs, and other nonprofit organizations may be willing to assist in the areas of transportation, construction of ramps and other home modifications to eliminate architectural barriers, identification of employment opportunities, finances, social support visitation, and community activities (see table 10). Every effort should be made to decrease the environmental barriers that can contribute to depression and increase the handicap of the person with spinal cord injury.

Tate et al. (1994) concluded, "The source of rehabilitation insurance coverage appears to influence the amount of independent living benefits received after spinal cord injury, as well as one's ability to adequately perform societal roles without experiencing physical and social handicaps." Individuals who receive Medicaid experience the highest level of handicap. They advocate for a change in health-care policy to ensure that people with spinal cord injury receive the basic insurance coverage necessary to address their lifelong needs. In recognition of the known high lifetime costs associated with spinal cord injury, as well as the impediments to access to services, clinicians working with individuals with spinal cord injury need to advocate for health policies that eliminate barriers to equitable access to quality services.

TABLE 10
Resurces for Social Interventions

Social Factors	Interventions
Housing	Assisted living; personal care homes; return to own home; independent living center; state veterans homes; nursing home placement; housing authority for subsidized housing and rental assistance programs; local realtor
Transportation	Local public transit authority; area agency on aging; state division of rehabilitation services; Medicaid taxi services; referral to VA for veterans; independent living centers; churches; rental van services
Personal care assistance	Home health aencies; independent living centers; family members; train individuals who can hire/manage their own exmployee(s); Medicaid waiver programs; state funding options; referral to VA for veterans
Home accessibility	Independent living centers; civic groups; churches; state division of rehabilitation services; referral to VA for veterans; workers' compensation
Vocational services/employment	State employment agency; independent living centers; state division of rehabilitation services; referral to VA for veterans
Leisure/recreation	Independent living centers; Paralyzed Veterans of America; National Spinal Cord Injury Association; churches; YMCA/YWCA; local fitness centers; county parks and recreation service; Chamber of Commerce; state sports associations; senior citizens centers
Peer support	Independent living centers; local rehabilitation hospitals; Paralyzed Veterans of America; National Spinal Cord Injury Association; local SCI or PVA chapters; disability-specific support groups
Family support	Independent living centers; local rehabilitation hospitals; referral to mental health center/professional; local SCI or PVA chapters; disability-specific support groups; state protection and advocacy agencies
Finances	Supplemental Security Income; Social Security Disability Insurance; VA for veterans who served during wartime or who are service-connected; workers' compensation; food stamps; Aid to Families with Dependent Children
Adaptive equipment needs	Referral to VA for veterans; independent living centers; Paralyzed Veterans of America; National Spinal Cord Injury Association; Medicare; private insurance
Caregiver burnout	Referral to VA for respite for veterans; respite care through local hospitals/nursing homes; homemaker services through VA or state funding; local support groups; referral to mental health center/professional

NOTE: LISTINGS FOR FEDERAL, STATE, COUNTY, AND LOCAL GOVERNMENT AGENCIES CAN BE FOUND IN THE PHONE BOOK. THE NATIONAL SCI HOTLINE (AT (800) 526-3456 OR (800) 638-1733 IN MARYLAND) CAN BE A RESOURCE FOR REFERRAL TO LOCAL RESOURCES FOR ALL THE SOCIAL FACTORS LISTED. ADDITIONAL RESOURCES SUCH AS THE SPINAL CORD INJURY INFORMATION NETWORK (HTTP://WWW.SPINALCORD.UAB.EDU) CAN BE FOUND ON THE INTERNET.

Consumer and Family Education

14. Provide patient and family education on the following topics:

- Signs and symptoms of depression
- Treatment options
- Medications, side effects, adverse reactions, and drug interactions
- Effect of depression on individuals with SCI/D
- Effect of depression on the family
- Community resources

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

Individuals with spinal cord injury are often educated about the physical consequences of spinal cord injury as part of a routine inpatient rehabilitation program. However, instruction in the relationships among emotional adjustment, physical health, social and environmental factors, and well-being rarely occurs in a routine way; rather, these relationships are usually regarded as the domain of a mental health provider. Available data indicate that the emotional adjustment and personal well-being of a person with SCI are directly associated with health status during the acute inpatient phase and over time as the person resumes personal, social, and vocational roles in the community (Hancock et al., 1993). Craig et al. (1994) report that the experience of pain and of not being in control of one's life before discharge were predictive of higher levels of depression 2 years after spinal cord injury. According to Fuhrer et al. (1993), individuals who had fewer social relationships and who were less involved in work, school, or other occupational activities scored higher on the Center for Epidemiological Studies Depression Scale.

Coping strategies useful in adjusting to SCI should be reviewed with both the individual and the family. Signs and symptoms of depression also should be discussed to maximize the likelihood of early detection and treatment of affective, cognitive, and vegetative symptoms. This information constitutes primary prevention similar to educational strategies aimed at preventing the physical complications associated with SCI (Moore et al., 1994). Appropriate options for treatment in the community also should be provided, including services provided by a variety of agencies, including home health, social work, and rehabilitation.

Evaluation and Modification of Treatment Plan

15. Evaluate treatment, focusing on the following elements:

- Evaluation of treatment efficacy
- Modification of treatment, as indicated
- Follow-up with referral sources

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

Treatment of depressive disorders may include three phases: an acute phase lasting 4 to 12 weeks, during which the remission of depressive symptoms is induced; a continuation phase lasting 4 to 9 months, during which remission is preserved; and a maintenance phase, during which remission is sustained (Kupfer, 1991).

During the acute phase, a range of treatment interventions can be used, including somatic interventions, consisting of pharmacologic treatment or electroconvulsive therapy, and/or psychotherapeutic interventions, including cognitive therapy, behavior therapy, interpersonal therapy, or psychodynamic psychotherapy.

Continuation treatment is based on the premise that there is a period of time following symptomatic recovery during which discontinuation of treatment would likely result in relapse. Available data indicate that individuals who are treated for a first episode of uncomplicated depression and who demonstrate a satisfactory response to an antidepressant agent should continue to receive a full therapeutic dose of that agent for at least 16 to 20 weeks after achieving full remission (APA, 1993). Monitoring and evaluation of the person's response is critical. The first 8 weeks after symptom resolution is a period of particularly high vulnerability to relapse. When medication is ultimately tapered and discontinued, the individual should be carefully monitored during the time immediately after discontinuation to ensure that remission is stable (APA, 1993).

Individuals who have had multiple prior episodes of depression should be considered for maintenance treatment. Factors include the frequency and severity of past episodes, the efficacy and side effects of continuous treatment, and the potential effects of a recurrent episode in the person's current life context. Factors increasing the risk of recurrence are the persistence of dysthymic symptoms after recovery from a depressive episode, the presence of an additional nonaffective psychiatric diagnosis, the presence of a chronic general medical disorder, and/or a prior history of multiple episodes of depression (U.S. Dept. of HHS, 1993). Maintenance treatment aims at preventing a recurrence. For well-established, recurrent depressions, the recurrence rate may approach 75

depressions, the recurrence rate may approach 75 percent of cases (Frank et al., 1990).

Specific forms of psychotherapy or psychotherapeutic management may be used during the acute phase to increase the probability and breadth of response to treatment as well as to enhance the acceptability of treatment by helping individuals to accept medications and their side effects. Psychotherapeutic management may also be used during the continuation and maintenance phases to attenuate stresses and conflicts that might reexacerbate the depressive disorder or undermine medication compliance (APA, 1993).

The efficacies of psychotherapy, pharmacotherapy, and electroconvulsive therapy have been demonstrated for the acute phase of treatment for the general population, depending on the severity of the depression. Psychotherapy and pharmacotherapy appear to be effective for the treatment of mild to moderate depression. Pharmacotherapy alone or combined with psychotherapy appears to be effective with severe depression. There is some evidence that cognitive therapy may be the psychotherapy of choice in terms of effectiveness in the treatment of depression, but this evidence is not conclusive (U.S. Dept. of HHS, 1993).

Directions of Future Research

The key questions that need to be addressed by future research are not necessarily new questions. The age-old questions regarding which treatments work, on whom, and at what specific stage of depression, still apply and remain very pertinent, especially as they apply to the individual with spinal cord injury/impairment. To be more specific, future research needs to delineate the core elements of psychotherapeutic treatment to understand and identify the indications for the various types of psychotherapy. In addition, the therapeutic value of psychotherapy needs to be examined at the different stages of depression and with the various types of depressive disorders. Questions such as the following need to be raised and studied:

- Which treatments are most effective and at which specific stage of depression?
- Are specific treatments more effective with individuals who have spinal cord injury/impairment?
- What is the optimal frequency of psychotherapeutic contacts for the various types of psychotherapy, in acute, continuation, and maintenance phases?
- Should multiple forms or combinations of therapy be used, and if so, should they be given separately, in sequence, or combined together?

A great deal of recent research has identified specific clinical indications for the array of antidepressant agents for the general population, but little is known about the clinical indications of these agents for the spinal cord injury population. It is important to know which agents are effective for which type of depressive disorder and at which phase of depression.

- Is effectiveness applicable to the individual with spinal cord injury/impairment if a specific agent is identified for the general population, for a specific depressive disorder, or for a specific phase of treatment?
- What is the duration of treatment before an individual responds therapeutically or is considered medication resistant?
- What are the cardiotoxic effects of the newer agents in comparison to the older-generation antidepressants?
- Do specific SCI factors like level of injury or completeness of injury make a difference either in terms of antidepressant selection, dosage, or duration of treatment?
- Do individuals with SCI respond similarly or differently than the general population during the three phases of treatment?

SCI literature has also underscored the need to address environmental and social factors, since such factors can play a major role in independent community living, in adjustment to one's injury or illness, and in the onset of depression for the individual with SCI. Questions such as the following need to be answered:

- In what specific ways do these factors contribute to the onset of depression?
- Are there specific environmental or social factors, for example inaccessible housing or an inadequate caregiver or social support system, which have a greater impact in generating depressive reactions?
- Are there separate and distinct interventions which can strengthen the individual's social support system or lessen the negative impact of environmental barriers or are psychopharmacological and psychotherapy treatments sufficient?
- If such interventions exist, are certain interventions more effective than other interventions?

Lastly, future research needs to address the needs of primary care physicians in successfully diagnosing and treating depression in individuals with SCI.

- What specific skills do primary care physicians need to possess to accurately identify a depressive disorder?
- Are these skills primarily in the area of psychodiagnostic ability or do they include interviewing and active listening skills, or are other unidentified skills necessary?
- Are these skills just pertinent to the general population or are they also applicable to the SCI population?
- Are there specific psychodiagnostic skills needed to treat the SCI population?
- Are there shortcomings in current primary care practice with respect to recognition of depression and quality treatment?
- If so, how can these shortcomings be remedied?

- What factors, symptoms or conditions dictate the need to refer an individual to a mental health provider for treatment?
- When a referral is made, what type of collaborative relationship needs to exist between the primary care physician and the mental health provider?
- What type of collaborative monitoring needs to occur regarding follow-up care and treatment adherence?
- Who assesses treatment outcomes and how are those outcomes determined?

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