
Although depression has been widely studied among persons with spinal cord injury, the ubiquitous and unsophisticated use of the term and presumptions about its manifestations in the rehabilitation setting have needlessly encumbered the understanding and treatment of depression. Major themes and issues in the study, measurement, and treatment of depression among persons with spinal cord injury are reviewed. Greater precision is recommended in distinguishing diagnosable depression from displays of negative affect, anxiety, distress, and dysphoria. Correlates of depressive behavior among persons with SCI are surveyed, and guidelines for research and practice in the SCI setting are explicated.

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DEPRESSION is probably the most frequently studied psychological variable among persons with spinal cord injury (SCI).1 In the 8 years since Frank and colleagues2 reviewed the existing literature, 36 additional studies addressing depression after SCI have appeared in MEDLINE and 28 studies are cited in PSYLIT. Despite the addition of 64 studies, many of the problems identified in the articles cited in 1987 still characterize the literature on SCI and depression.3,4 Methodologic issues that continue to constrain the study of depression following SCI include: (1) unclear or inconsistent use of diagnostic criteria; (2) use of imprecise and ambiguous definitions of depression; (3) inaccurate measurement of depression; (4) lack of cogent, testable theoretical approaches regarding depression after SCI; (5) failure to integrate models of depression after SCI with the extant literature on depression; and (6) a lack of rigorous longitudinal research on the development, course, resolution, and treatment of depression among persons with SCI. Researchers in this area have too often conducted work without regard to history, ignoring substantial findings and commentary in previous research.5,6 These recurrent problems have contributed to a general lack of integration and progress in this area.

In this article, we review the clinical aspects of depression and briefly describe the historical view of depression in SCI rehabilitation prior to 1987, then we address issues in the definition, measurement, and description of depression, depressive behavior, and distress following SCI. We survey empirical research to date concerning clinical correlates of depression and distress among persons with SCI, with particular attention to personal, environmental, and psychophysiological factors. Finally, we summarize the major unresolved issues concerning measurement and treatment of depression after SCI and provide specific recommendations for future research.

CLINICAL ASPECTS OF DEPRESSION: AN OVERVIEW

Depressed mood or affect refers to a state of dysphoria that occurs routinely and is a normal process. Depressed mood accompanied by persistent and pervasive loss of emotional involvement with other people, objects, or activities distinguishes a normal mood state of sadness, demoralization, or other negative affects such as anxiety from the syndrome of depression. A diagnosable depressive syndrome refers to a constellation of observable symptoms that may encompass tearfulness, apathy, irritability, loss of appetite, disturbed sleep, lack of energy, death thoughts, etc, in addition to depressed mood. For an individual, a depressive syndrome is recognized when the behavioral characteristics deviate from the norm in several realms of functioning. Depressive disorders are categorized according to specific criteria constituting psychiatric diagnoses in Diagnostic and Statistical Manual of Mental Disorders (DSM-IV).5,7

In general, the incidence of depressive disorders is increasing (see Fombonne7 for a comprehensive review). Several studies of families have shown increased incidence of new-onset depressive disorders in the youngest cohort (younger than 40 years) who had rates 3 times as high as the oldest cohort.8 At any age the youngest birth cohorts completing a period at risk have higher rates of disorders than the older cohorts and have progressively earlier age of onset.9 Surprisingly, the lifetime rate of any depressive disorder declines with age, with lower rates found among elderly people.8 Although most evidence continues to suggest that women have twice the risk of a depressive disorder, among the younger cohorts the gap between the two genders may be diminishing.8

Depression is a disabling syndrome.10 Hays and associates10 recently found that depressed individuals were less able to perform activities of daily living than patients with chronic medical conditions such as diabetes and arthritis. After 2 years, patients with dysthymia were significantly more limited in physical and role functioning than patients with hypertension despite significant deterioration of functioning for the hypertensive patients relative to their baseline.6 Outcome data indicate that individuals who recover from depressive episodes tend to be employed, have a lower intake of alcohol, use active coping strategies, and have higher levels of physical activity and social support in comparison with persons who do not recover over time.5

Depression is a major public health problem associated with excessive mortality and morbidity.11 Impairment and disability associated with depression is equal to that attributed to cardiovascular disease, and greater than that caused by other chronic physical disorders such as hypertension, diabetes mellitus, and arthritis.11 Although 6% to 10% of medical patients have Major Depressive Disorder, depression is often undetected and untreated among these persons.11

The advances in the pharmacotherapy of affective disorders over the last four decades have helped illuminate the psychobiology of depressive disorders. Beginning in the 1950s, tricyclic
antidepressants (TCAs) replaced monoamine oxidase inhibitors (MAOs) as the treatment of choice for unipolar depression. TCAs have a relatively high affinity for muscarinic, histaminergic, and adrenergic receptors causing the side effects and toxicity associated with this class of drugs. In 1987, the new selective serotonin reuptake inhibitors (SSRIs) were introduced. More recently, dual action serotonin-norepinephrine reuptake inhibitors have been introduced. These two classes of drugs have supplanted TCAs. Adoption of SSRIs reflects their improved ease of administration and the reduced side effects experienced by patients using the drugs. Despite these advantages, as many as 30% to 40% of patients fail to respond satisfactorily to an adequate trial of SSRIs and a significant number of patients fail to achieve long-term remission.

The development of antidepressant medications has underscored the role of neurotransmitters in the pathophysiology of depression. It is now well accepted that serotonin (5HT) neuronal systems are involved in many episodes of depression. It is unlikely, however, that only a single neurotransmitter system underlies the neurochemical basis of all depressions. Other neurotransmitters systems likely to be involved in the pathogenesis of depression include norepinephrine (NE), dopamine, and a variety of neuropeptides, most prominently corticotropin-releasing factor (CRF). More than 75 neurotransmitters have been identified to date. Two of the identified transmitters, NE and 5HT, are especially important in the pathogenesis of depression. NE is a catecholamine neurotransmitter synthesized from l-tyrosine, while 5HT is an indoleamine synthesized from 5-tryptophan. Both of these amino acids are derived from dietary sources, although tyrosine can be synthesized from phenylalanine in vivo. Norepinephrine is inactivated by the degradative enzymes monoamine oxidase (MAO) and catechol-O-methyltransferase (COMT), whereas 5HT is inactivated only by MAO. After neurotransmitter release from the presynaptic terminal, 5HT and NE are taken into the presynaptic terminal and recycled into storage vesicles or are degraded by MAO in the nerve terminal. Reuptake is the principal mechanism for terminating noradrenergic and serotonergic transmission. Enzymatic degradation provides a secondary route for inactivation.

DEPRESSION AND SCI: A HISTORICAL BACKDROP

Before 1987, much of the work concerning depression after SCI was embedded in nonempirical models preoccupied with a direct relationship between the SCI and psychological adjustment. Individual and environmental characteristics of the patient were largely ignored: presumed mechanisms that drive human behavior were generally disregarded or deemed superfluous. The presence of SCI was essentially considered the most salient, distinguishing feature—and the most powerful predictor—of the individual’s subsequent behavior and experience. Classically defined stage models—derived loosely from psychoanalytic conceptions of loss—posed that a person with recent-onset SCI should be expected to pass through several stages of psychological adjustment. A depressive phase was to be expected—if not encouraged—and the experience of depression was basically construed as a therapeutic prerequisite for optimal adjustment. In contrast, the absence of depression was arguably maladaptive and indicative of an unhealthy denial of the concomitants and finality of the injury (for a synopsis of these, see our earlier reviews). To a great extent, these assumptions may have been influenced by the stereotypical expectation that people with SCI are consumed with the trauma of physical disability. Such assumptions could also reflect gross extrapolations from personal experience with distressed patients in rehabilitation units, or reckless generalizations from studies with small sample sizes and inadequate measures. Consequently, the association between depression and SCI was rarely questioned in the rehabilitation clinic prior to 1987.

Nevertheless, our earlier critiques melded a disjointed yet developing empirical literature that converged to contradict the basic tenets of stage of model mentality. In sum, these data-based studies systematically demonstrated a lack of objective, empirical support for stage models in our understanding of psychological adjustment following SCI. We found the empirical evidence warranted a greater clinical and theoretical appreciation of the individual differences, environmental characteristics, and physiological parameters associated with depression in this population.

DEFINING AND ASSESSING DEPRESSION IN SCI REHABILITATION

A major depressive episode is categorized as one of four different types of mood disorder. A diagnosis of Major Depressive Disorder must involve either the presence of a depressed mood or loss of pleasure or interest over a 2-week span and at least five of the following symptoms ‘...most of the day, nearly every day’ within the same time frame: significant weight loss or gain, decreased or increased appetite, insomnia or hypersomnia, psychomotor agitation or retardation, fatigue or energy loss, feelings of worthlessness, excessive or inappropriate guilt, diminished ability to think or concentrate, indecisiveness, and recurrent thoughts of death or suicide. These symptoms must also cause significant impairment in social, occupational, or other roles; they must not be due to a ‘general medical condition’ (eg, hyperthyroidism), bereavement, or the physiological effects of a mood-altering substance.

Despite the refinements in diagnosis and understanding of depression, clinical practice in the rehabilitation of individuals with SCI and depression has been historically guided by anecdotal and impressionistic notions of “depression.” Rehabilitation professionals are particularly reactive to displays of depressed mood and dysphoria. Nevertheless, depressed mood alone does not warrant a diagnosis of Major Depressive Disorder; it may only represent a transient experience of negative affect in reaction to a wide array of personal or environmental circumstances, of which SCI might be but one. As such, this behavior might reflect symptomology subsumed under other diagnoses. For example, when fewer than five symptoms are observed, the appropriate diagnosis might be Depressive Disorder Not Otherwise Specified (eg, Minor Depressive Disorder). The presence of a downcast mood and corresponding behaviors within 3 months of injury onset might be best described as an Adjustment Disorder with Depressed Mood, if other criteria for Major Depressive Disorder or Dysthymic Disorder are not satisfied.

Studies relying on DSM-III criteria using relatively small samples of recently injured individuals and stringent interview systems have found that the rate of major depressive episodes among persons with SCI ranges from 22.7% to over 50%.

Lower rates have been observed in studies utilizing less stringent interview methods (13.7%; total sample N = 227), and with self-report measures based on DSM-IIIIR criteria with a sample varying considerably in time since injury onset (11%). Recent data suggest that many among newly injured persons who have diagnosed problems with Major and Minor Depressive Disorders may remit within 3 months of injury onset, but these studies have yet to be replicated. These findings have several implications. First, depressive
What To Do With Vegetative Symptoms?

Acute SCI is typically marked by weight loss, disruptions in appetite, sleep cycles, physical sensations, energy levels, and mobility problems. These symptoms are also symptomatic of depressive conditions. This overlap has prompted some to advocate for revision of diagnostic criteria and self-report items that are confounded by the physical sequelae of acute SCI.1

Sleep disturbance, disruptions in weight and appetite, and dramatic fluctuations in energy level and activity have been found to reflect the severity of the mood disturbance in everyday life. Research to date has not clarified symptom differences for persons with recent-onset SCI and others who have been injured for many years. If SCI affects vegetative/somatic symptoms, then evaluating these symptoms should be elevated among newly injured individuals. Available research indicates that vegetative/somatic symptoms are not inflated on self-report depression measures among persons with general medical conditions.30 Despite longstanding concern that vegetative and somatic items taint the parallel depression, a series of studies has found that vegetative/somatic items may be less of a confound in the diagnosis of depression among persons with SCI than often suspected. It now appears that symptoms—represented by symptoms of negative self-evaluations, depressed mood, and suicidal ideation—constitutes a core element of depression common to persons with SCI, individuals with rheumatoid arthritis, college students, and community-residing adults.21

Deleting items that assess sleep disturbance may deprive a clinician of potentially valuable information. Sleep disturbance may reflect a sleep cycle disrupted by initial trauma, medical intervention, or transitions from intensive care units to the rehabilitation center. Such disruptions can adversely affect patient mood, motivation, and cognition.1尽管 systematic research demonstrating the efficacy of behavioral interventions for insomnia among medical patients generally,23 this work has not yet been extended to the rehabilitation setting.

The clinical interpretation and recognition of vegetative/somatic symptoms varies between patients. Many patients recognize these symptoms as normal reactions to the trauma and sudden changes in physical functioning and may in turn experience less overall distress. Interpretation of vegetative symptoms has been correlated with general cognitive style. For example, individuals with adaptive reality negotiation strategies may experience less distress by refusing to acknowledge certain aspects of their injury until they can find appropriate goals to pursue. In contrast, those persons who lack goal-directed energy are more likely to acknowledge somatic symptoms and demonstrate elevated levels of distress regardless of the time since the onset of injury. Clearly, it is important to understand how the patient recognizes, understands, interprets, and experiences vegetative/somatic symptoms.

Although somatic criteria for depression may bias the prevalence of estimates of depression among patients with chronic physical conditions, correlations between depression scores and other psychological variables are not likely to be affected.22 Reidy and Caplan,23 for example, found self-blame for the incident culminating in SCI significantly correlated with the cognitive/affective and the total score of the Beck Depression Inventory (which contained the vegetative/somatic items).25 When evaluating mood after catastrophic injury, it is important for clinicians to examine reported problems from the patient’s perspective. Systematic evaluations of the separate domains of cognitive (low self-worth, hopelessness), affective (sad mood, loss of pleasure, irritability), and somatic (sleep disturbance, appetite disruption) symptoms are vital for planning effective interventions in SCI rehabilitation. Cognitive aspects of depression are typically less amenable to pharmacological strategies and warrant psychological interventions. Sleep disturbance in the period following injury may be treated with psychopharmacological agents, then with behavioral strategies. The use of self-report depression scales has been common in rehabilitation settings. As has been described, these scales reflect a confluence of variables which may affect their reliability. Clinicians must scrutinize symptom patterns on any depression measure and rely less on any total score. Studies that examine separate symptom patterns and their relation to other variables will undoubtedly expand our understanding of this general issue.

Diagnosable Depression, Depressive Behavior, and Distress

The basic assumptions guiding the use of self-report depression measures in SCI rehabilitation need to be examined. Too often data from these scales are considered synonymous with diagnosable depression, i.e., depression defined and diagnosed according to existing nosological criteria (e.g., DSM-IV). Thoughtful researchers and clinicians have recently pointed out the many flaws that negate this assumption. Self-report depression measures are susceptible to a host of
negative affects including transitory distress in reaction to stress and life events, mood changes attributable to alcohol or drug use, or displays of anxiety, tension, or fear. A total score on a self-report depression measure may reflect a “…summary of feelings, symptoms, and attitudes” tangentially related to diagnosable depression. Furthermore, practically all self-report measures lack adequate time parameters and referents to determine duration necessary to diagnose depression, and they inadequately sample salient features crucial to a diagnosis. 

Self-report depression instruments are best construed as measures of distress, unhappiness, dysphoria, or depressive behavior. Of these terms, distress and depressive behavior describe with greater accuracy what is being measured by self-report measures. Scores on the Beck Depression Inventory, for example, can reflect a cluster of behaviors that might be associated with diagnosable depression or another condition, such as an anxiety disorder. The term dysphoric is somewhat misleading because it implies a sense of sadness, hopelessness, or helplessness; a high score can be attained on any self-report depression measure without endorsement of any of these symptoms. Unfortunately, many who are aware of the distinctions between diagnosable depression and scores from a self-report instrument still lapse into “depression” terminology. This regrettable imprecision limits clinical and theoretical understanding of depression and distress in rehabilitation and in the larger scientific community. In this article, we distinguish between studies that address depression as a diagnosable condition, and those using a self-report measure as a proxy of depression. In this way, the measurement of depressive behavior or distress will be differentiated from a diagnosable depression syndrome.

**Clinical Correlates of Depressive Behavior and Distress After SCI**

Empirical studies have supplied information that contradicts stage-model anecdotes about depression that extoll the beneficial properties of “depression” soon after injury onset. In contrast to these models, depressive behavior has been associated with increased hospital stays and fewer functional improvements in SCI rehabilitation. Patients who exhibit more depressive behavior tend to expect a longer hospitalization and in turn evidence lower levels of functional independence and mobility at discharge than those without these problems. In comparison, those with the highest expectations for recovery of functional abilities report less depressive behavior and subsequently demonstrate a greater change in activities of daily living after rehabilitation.

Cross-sectional research has also painted an equally dismal view of depressive behavior among those injured for longer periods of time. Depressive behavior has been associated with the occurrence of secondary complications (eg, pressure sores, urinary tract infections), and poor self-assessed health status. Depressive behavior is a powerful predictor of decreased spare time activities. Community-residing adults with SCI who report problems with depressive symptoms also demonstrate decreased mobility, poorer social integration, and a greater haphazard use of many situation-specific coping efforts. In the most extensive documentation to date, Tate and coworkers found that persons with higher self-report depression scores spent more days in bed, fewer days outside the home, and had greater use of paid personal care attendants and greater general medical expenses than those with lower levels of depression.

These studies suggest that depressive behavior does not appear to signal “acceptance” of disability. Depressive behavior has been consistently associated with an array of psychosocial and medical complications. These complications occur at high cost to the person, the rehabilitation program, and the community.

**Theoretical Explanations of Depression and Distress After SCI**

The findings concerning the correlates of depressive behaviors do not clarify why some individuals develop problems with depression and distress. In the study of behavioral disorders, of which depression is but one, there is a tendency to develop explanatory models that are unique to a given population but divorced from contemporary perspectives of behavior and behavioral disorders. For example, impressionistic stage models have historically guided clinical judgements in absence of cogent, testable, theoretical frameworks. The weaknesses of these impressionistic models have been addressed in earlier reviews. Elliott argued that theoretical explanations of behavioral phenomena in general apply to individuals with SCI, theoretical models are ultimately superior to others in guiding interventions, and theoretical explanations provide potentially testable hypotheses necessary to advance science. Ideally, contemporary theories of adjustment to physical disability take into account the interactive effects of disability parameters, psychosocial stressors, and person/environment factors that culminate in the prediction of depression and other aspects of adaptability.

A biopsychosocial perspective appears to be the most relevant model of adjustment after SCI. From this perspective, unique features of the individual, the environment, and the spinal cord injury—its physical concomitants and other medical conditions—are taken into consideration to understand a given behavior. Accumulating evidence has supported such a view at each separate domain.

**Personal Factors**

Some individuals are more likely to exhibit depressive behavior following SCI than others for a variety of reasons. Individuals with preinjury histories of social maladjustment, psychological disorders, and alcohol/substance abuse have particular difficulties adjusting to SCI, and depressive behavior is often manifested in these cases. These data imply that individuals who have exhibited difficulties coping with the general demands of life will have greater difficulty coping after SCI. It logically follows that those with greater personal resources and those who have demonstrated an optimal level of adjustment before injury would be less likely to display depressive behaviors after SCI. Although this latter hypothesis has not been tested directly, several cross-sectional studies lend indirect support. Self-appraised skills in solving everyday problems are inversely related to depressive behavior among people in general. Elliott and colleagues found effective skills in solving problems and a greater confidence in problem solving were significantly associated with lower scores on a self-report depression measure among persons with SCI, and this relationship was not mediated by time since injury onset. This study suggests that those with useful, goal-directed strategies may be less likely to have problems with depression and distress. It has also been found that depressive behavior after SCI is associated with a heightened and haphazard use of many situation-specific coping efforts. Those who maintained internal expectancies for control over reinforcers and outcomes for their behavior were less distressed, however. Other studies indicate that higher levels of hope and goal-directed energy are associated with less distress, regardless of time since injury onset. Social-cognitive characteristics such as problem solving, hope, and locus of control are particularly valuable to clinicians; these variables are embedded in a...
learning tradition and are thus theoretically amenable to psychological interventions in the medical setting.57

Environmental Factors

Several studies have documented that a lack of social support is associated with depressive behavior.61,62 Unfortunately, the mechanisms by which social support is enhanced or eroded after SCI are not well understood. It is possible, for example, that chronic strain following SCI has a deteriorating effect on the quality of intimate relationships (i.e., the deterioration hypothesis).63 Preliminary research indicates that many family caregivers of persons with SCI encounter difficulties in helping with activities of daily living, personal hygiene, and self-care regimens.64 Other data indicate that unique characteristics of the person with SCI might moderate the effectiveness of social support. Those with more assertive mannerisms might be able to marshal available social support in certain situations; others may actually alienate otherwise supportive relationships.65

A series of laboratory studies suggest that displays of depressive behavior can have aversive ramifications for a person with SCI. Persons appearing to have SCI and who exhibit depressed mood and dysfunctional cognition elicit greater rejection, interpersonal avoidance, and stereotypic attitudes toward persons with physical disability.66,67 Rehabilitation staff members feel greater hostility and decline interaction with a person with SCI who acts in a depressed fashion68; persons with SCI are also inclined to reject and devalue a depressed peer.69 Conversely, persons with SCI who are not depressed and who display socially appropriate behaviors engender more positive attitudes and cognitions.70,71 Interpersonal displays of depressive behavior might contribute to subsequent social isolation and strains on intimate relationships, and culminate in chronic problems with depression.72

Person and environment variables can also interact with deleterious effects. Frank and Elliott73 found persons with SCI differed considerably in their recent experience of major life events. Participants who reported a higher level of life stress from such events as death of a loved one, death of a close friend, loss of finances, etc., reported more depressive behavior than other patients regardless of time since injury onset. Other data have confirmed that the subjective perception of stress and setbacks are related to patient distress.74 The need to consider phenomenological stressors other than the SCI is crucial in appreciating the nature of depressive behavior in SCI rehabilitation.

Psychoimmunological and Physiological Factors

Many attempts have been made to explain possible psycho-physiological consequences of SCI that may moderate depression after SCI. For example, it has long been assumed that persons with SCI have a decreased ability to experience intense emotions, presumably due to "physiological suppression" that diminishes the physical experience of emotion.75,76 Contemporary research has not supported this position. Chwalisz and colleagues77 found that persons with SCI reported many intense experiences of emotion regardless of their level of autonomic feedback. Their ratings of positive and negative emotions were basically equivocal to those culled from a group of participants with other physical disabilities and a group with no physical conditions. These researchers concluded that autonomic and visceral feedback was not crucial for the experience of emotion after SCI, and noted that cognitive arousal theories may hold more promise for understanding emotion after SCI.

Palmer78 reasoned that presumed disruptions in adrenocortical functions following SCI could affect the experience of depression among these patients. Biochemical indicators like the dexamethasone suppression test (DST) were recommended for clinical trials. Yet Frank and coworkers14 found the DST was not a sensitive indicator of depression among 32 patients with SCI. DST results were unrelated to level of lesion or time since injury. These authors speculated that continued study of the hypothalamic-pituitary-adrenal axis might eventually elucidate our understanding of depression following SCI. Few have attended to these issues; efforts to continue this line of inquiry have failed to establish a relationship between patient depressive behavior and circulating plasma levels of beta-endorphin and cortisol.79

Recent developments in the psychopharmacology of depressive disorders have received scant attention in the SCI literature. The SSRIs were the first class of drugs developed to target the 5HT reuptake carrier. These drugs have few if any effects on histaminergic, muscarinic, and a, adrenergic receptors and sodium fast channels, thereby avoiding the side effects that plague use of TCAs (e.g., sedation anticholinergic affects, orthostatic hypotension and cardiotoxicity).80 These side effects are especially problematic for persons with SCI. Because of these favorable side effects profiles, SSRIs have replaced TSAs as the drug of choice in the treatment of major depression. Indeed, clinical anecdotes suggest that these drugs have gained popularity in the treatment of depression associated with SCI, although there does not appear to be any clinical outcome research to verify this assumption. Despite advances associated with the introduction of SSRIs, drugs that act on 5HT and other transporters may be even more advantageous in the treatment of depression. These dual action serotonin-norepinephrine reuptake inhibitors include venlafaxine, duloxetine, and milnacipran. Venlafaxine is a unique inhibitor of both 5HT and NE reuptake. This class of drugs retains the safety profile of the SSRIs while possibly offering more antidepressant advocacy including increased onset of action and dosage flexibility. Unfortunately, the lack of applicable research in SCI rehabilitation limits our understanding of the relation of these neurotransmitters to the occurrence of diagnosable depression among persons with SCI.

Other lines of research have expanded into areas that were previously ignored before our 1987 articles. Initial evidence indicates that neuropsychological concomitants sustained in trauma that resulted in SCI are unrelated to self-report depression scores.81 However, patients who report a persistent experience of pain exhibit more depressive behavior than those who report no pain.82,83 Complaints of pain among newly injured patients can be a significant predictor of distress 2 years after injury onset.84 Chronic pain following SCI is not well understood, but it is clearly emerging as a major correlate of well-being and adjustment that will likely require an array of intervention strategies.85

SUMMARY

Despite frequent discussion in the literature, little effort has been directed toward systematically assessing treatment of depression and distress associated with spinal cord injury. In a recent review of the literature, no documented study of the treatment of depression or distress following SCI with either behavioral or psychopharmacological strategies was found. The outcome research that has been conducted has generally focused on skill-building (e.g., assertion training)86 or well-being (eg, acceptance of disability).87 In light of our knowledge about the detrimental concomitants of depressive behavior following SCI, it is imperative that the next wave of research focus on the prevention and treatment of depression and distress among these patients.
In order to conduct this work, it is important to test assumptions gleaned from clinical practice and available theory. For example, do antidepressants that regulate the sleep cycle prove more efficacious in the treatment of diagnosable depression among acute injuries in SCI rehabilitation? Do these agents have a therapeutic effect on subclinical displays of dysphoria and distress? Are these agents ably supplemented or replaced by behavioral strategies designed to develop adaptive sleep habits? Are other agents more efficacious among outpatients who have been injured for longer periods of time and who might have more recurrent, chronic problems with dysfunctional moods and cognition? Are these patients better served by interventions that attempt to improve interactions with the environment in terms of relationships, meaningful leisure activities, or other pursuits that might heighten a sense of competence?

From a behavioral standpoint, it is important to know if cognitive-behavioral strategies (eg, problem solving training) are helpful in preventing and alleviating depressive behavior among persons with recent-onset SCI. Are psychoeducational and support groups equally effective in working with family members and identified caregivers during acute SCI rehabilitation? Do group approaches offer the most efficacious format for treating depression and distress after SCI? Available evidence concerning the social aspects of depressive behavior imply that strategies that reconstruct and promote a therapeutic milieu might be a potent agent of change for these persons. It seems that coping strategies and cognitive beliefs vary in effectiveness at different times following SCI; it may be necessary to train patients with a more flexible repertoire of available strategies.

We need to clarify relations between depression and other variables crucial to quality of life and potentially amenable to therapeutic interventions. For example, we have only a cursory understanding of hopelessness and suicidal ideation among persons with SCI. Similarly, little is known about anxiety disorders and social phobias in this area. The mechanisms by which dysphoria and distress exert a detrimental influence on health status, secondary complications, immune functioning, and intimate relationships over time have not been explicated.

The development and treatment of depression and distress among children and adolescents who acquire SCI is also poorly understood. It is probable that family dynamics are salient in this particular arena; unfortunately, researchers have neglected this challenging area of inquiry. SCI might increase an overall vulnerability to life's routine stresses and strains. Families that have more adaptive and expansive strategies in coping with stress might be more adaptive following pediatric SCI, resulting in a more optimal outcome for the child. In this scenario, it seems more profitable to study family, rather than individual, reactions to SCI, taking into account family dynamics, division of tasks, and the experience of strain and stress on the system. Despite several interesting studies, little is known about alterations in the hypothalamic-pituitary-adrenal axis after SCI. Changes in neuroendocrine function after SCI warrant further investigation. The new classes of SSRIs offer a method of elucidating the action of receptors after SCI. Along the same line sympathetic assessment of efficacy of SSRIs in the treatment of major depression after SCI is justified.

Measures that cull from the patient's perspective and that of a significant other are definitely preferred over staff ratings, which are undoubtedly contaminated by staff issues about adjustment following SCI. Studies that insist on examining presumed symptom patterns piecemeal may be more convenient academically and clinically. Intervention studies and investigations of the mechanisms of depression and distress on daily function and the treatment of these problems are ultimately more demanding, worthwhile, and deserving of high priority from funding agencies. Research of this nature is of greater potential value at this juncture.

Specific Recommendations for Research and Practice

It is important that rehabilitation professionals dispense with pseudodiagnostic language and use precise descriptions of behavior. Research participants and patients who fail to meet diagnostic standards for depression may be best described as distressed; dysphoric may be the best available adjective to describe those who emit downturned or sad mood, hopelessness, or helplessness in absence of other symptomology necessary for diagnosis. Practitioners should carefully consider the presence of behaviors characteristic of anxiety (eg, worry, dread, tension) that might differentiate the clinical picture from a diagnosable depression.

It is time for us to establish standards for the measurement of depression in SCI research congruent with contemporary diagnostic criteria and nomenclature. If diagnosable depression is to be the focus of inquiry, then studies at a minimum should (1) articulate clear diagnostic parameters (2) by established and accepted criteria (3) using standard interview systems. Furthermore, (4) evidence of interrater reliability should be provided and (5) efforts should be conducted to differentiate depression from other conditions (eg, generalized anxiety disorder) and rule out mood-related changes attributable to drug or alcohol use, or to the general medical condition.

Self-report depression measures should be used with certain caveats. In clinical practice, clinicians should take care to describe endorsed items that approximate depressive symptomatology and those that could be associated with other syndromes (eg, sleep disturbance, worry, restlessness, irritability, anxiety). Patients should not be categorized as depressed on the basis of self-report measures alone. If a self-report measure of depression is to be used to classify participants for study, then researchers should comply with recommended procedures and corresponding terminology to prevent erroneous and misleading interpretations. For example, if the Beck Depression Inventory is to be used to classify participants the recommendations from Kendall and colleagues should be employed. Interested researchers should also consider the Inventory to Diagnose Depression. This scale provides information essential for the diagnosis of major depression and dysthymia, and it has been used successfully in recent studies of depression following SCI. This instrument can also be administered as a semi-structured interview.

Research projects that examine the predicted relation of a theoretically derived variable to self report depression measures can be enlightening. However, the report should not lapse into language reserved for a diagnosable depression condition, and the explicit focus and language of the study should be confined to the distress and/or depressive behavior. It is important that we identify precursors of distress, anxiety, and depression to guide prevention and intervention strategies. Whenever possible, it would be advantageous to include measures of trait negative affectivity, trait anxiety, or neuroticism to examine and partial out the confounding effects of these variables on depressive behavior.

Critics of the depression literature have lamented the overreliance on samples of convenience (eg, college students) and from the mental health clinic. Results from these studies do not readily generalize to the community in general, nor do they
accurately capture the nature of depression observed in the medical setting. In our opinion, the SCI rehabilitation setting provides a fertile laboratory for studying the course, correlates, and treatment of depression in a rigorous yet stimulating environment. Increasing our precision and sophistication in the study and treatment of depression following SCI could potentially enrich our understanding of this disorder in other contexts.

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