LESSONS LEARNED AT THE INTERFACE OF MEDICINE AND PSYCHIATRY

The Psychiatric Consultation Service at Massachusetts General Hospital (MGH) sees medical and surgical inpatients with comorbid psychiatric symptoms and conditions. Such consultations require the integration of medical and psychiatric knowledge. During their thrice-weekly rounds, Dr. Stern and his staff discuss the diagnosis and management of conditions confronted. These discussions have given rise to rounds reports that will prove useful for clinicians practicing at the interface of medicine and psychiatry.

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The Recognition and Management of Psychological Reactions to Stroke: A Case Discussion

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Have you ever wondered how people cope with the devastating sequelae of strokes? Have you wondered whether you can predict who is likely to cope poorly with physical impairments? Have you wondered how you can distinguish psychological reactions from neuropsychiatric and neurologic reactions? If you have, then the following case vignette (of a man who developed poststroke depression) should provide the forum for answers to these and other questions related to the psychological sequelae of stroke and comorbid neuropsychiatric syndromes.

Cerebrovascular accidents (CVAs) disable thousands of people each year and are a major cause of death in this country.[1] CVAs lead to physical limitations in daily living and to psychological disorders, expressed in alterations to an individual's behavior and emotion.[2] However, in the recovery process after stroke, many patients and their caregivers focus on the patient's physical disabilities and fail to appreciate that the psychological complications of stroke can hinder a patient's recovery. Psychological reactions to stroke are manifested in myriad ways in the days, months, and years after a stroke.[2–7] Although not every patient develops intense emotional responses to stroke, those who do often have risk factors that make them more vulnerable to psychological consequences.[3,4,8–11] Attention paid to those patients who may benefit from psychotherapeutic treatments as well as from psychotropic medications can facilitate effective treatment.

The patient we present sustained a stroke and had severe psychological reactions to it. We will highlight symptom recognition, review the risk factors for distress and dysfunction, and discuss the treatment of psychological reactions to stroke.

Case Vignette

Mr. B, a previously healthy, right-handed, 60-year-old man, suffered a CVA in his sleep. A sudden noise awakened him; his head ached, and he realized that he could barely move his right arm. Despite this, he managed to call for an ambulance. On arrival at the emergency room, his vital signs revealed a heart rate of 120 beats per minute, a respiratory rate of 26 breaths per minute, and a blood pressure reading of 172/112 mm Hg. The physical examination revealed decreased muscle tone, 1/5 muscle strength, and hyperreflexia in the right arm. A noncontrast computed tomography scan of Mr. B's head confirmed the diagnosis of stroke. Results of the rest of his examination were within normal limits. After 4 days in the hospital, Mr. B was discharged to his home and began a course of physical therapy as arranged by his family practitioner. Mr. B struggled to carry out the basic activities of daily living (ADLs); he had difficulty dressing himself and preparing his own food, and he was unable to write.

Unfortunately, Mr. B denied his physical limitations and his need for therapy. Despite his paresis, he attempted to drive his stick-shift car. An inability to perform effectively resulted in emotional outbursts (i.e., he suddenly became angry, irritable, and tearful).

Divorced, and with both of his children away at college, Mr. B's social support was less than ideal. He had few friends, and he spent much of his time alone.

At a follow-up visit several months after the stroke, his physician identified that Mr. B was anxious and depressed (believing that "he will never get better"), and his primary care physician prescribed a selective serotonin reuptake inhibitor (SSRI), fluoxetine, 40 mg/day.

What Are the Psychological Reactions to Stroke and When Do They Occur?

Strokes are life-threatening, traumatic, and debilitating. As in Mr. B's case, the resulting impairment after stroke is composed of physical, emotional, and behavioral components. Mr. B's hopelessness, anxiety, refusal of therapy, and emotional outbursts were consistent with common psychological reactions to stroke. The most common psychological symptoms include affective (including the experience of deep sadness, anger, or anxiety), behavioral (such as sudden outbursts of anger or crying, denial of disability, or withdrawal), and cognitive symptoms (e.g., diminished attention, decreased memory, or aphasia).[2,6,11–13]

Poststroke depression. Poststroke depression (PSD) is a common and well-studied reaction to stroke.[7,14,15] Depending on the sample size and the assessment tools used, researchers have found that PSD occurs in 20% to 40% of stroke patients.[4,12,14] PSD has the same signs and symptoms (including disturbed sleep, a lack of interests, guilt or a preoccupation of thought, reduced energy, diminished concentration ability, disturbed appetite, psychomotor agitation or retardation, and thoughts of death or suicide) as does major depressive disorder (MDD). Four or more of these symptoms, in the presence of depressed mood or anhedonia (the loss of pleasure), for a duration of 2 weeks or longer will satisfy criteria for MDD.[16] PSD adversely affects a patient's chance (and rate) of recovery and has been associated with more than a 3-fold increase in mortality rates for as long as 10 years after stroke.[7,14]

Generalized anxiety disorder. Another common sequela of stroke is generalized anxiety disorder (GAD), which occurs in 20% to 30% of patients, depending on the time that has elapsed since the stroke.[3,5] GAD is characterized by frequent and uncontrollable worry that occurs most of the time for at least 6 months and includes feelings of restlessness, impaired concentration ability, irritability, muscle tension, and/or a sleep disturbance.[16] Anxiety after stroke can adversely impact recovery from stroke and is associated with decreased ADLs and social dysfunction.[3]

Both PSD and poststroke anxiety can occur early after stroke, or develop months or years following stroke, and may become chronic.[3–5,7] Early onset of either depression or anxiety in the first year after stroke has been associated with persistent anxiety and depression.[3,4] Therefore, early treatment of PSD and GAD after a stroke is advised.

Catastrophic reactions and emotional incontinence (pseudobulbar affect). Catastrophic reactions and pseudobulbar affect are hard to distinguish from one another, as they have similar symptoms[2,10]; patients present with outbursts of emotion (e.g., sudden crying, anger or aggression, uncontrollable laughing, or sudden surges of anxiety). The differences are related more to their triggers than to the behaviors associated with them.[10]

Catastrophic reactions, a term first coined by Goldstein,[17] are defined as the intense emotional reactions that patients have when presented with a task that they are unable to perform due to neurologic deficits.[10] Research has found that catastrophic reactions are prevalent and related to lesion location; they occur in about 4% to 20% of stroke survivors.[11,18] Denial of physical impairment can also lead to catastrophic reactions: when denial fails, anger arises.[17,19]

Inappropriate or uncontrollable laughing or crying (also referred to as pseudobulbar affect) can be triggered by a stimulus that is related, yet disproportionate, to the behavior it elicits.[10] The condition occurs in 20% to 30% of stroke survivors and can be associated with intrusive thoughts about the stroke and with feelings of helplessness and hopelessness[20,21] or in response to a stimulus that has little or no emotional value and that does not induce a subjective experience of emotion (e.g., a patient may cry but not feel sad).[10] Afflicted individuals are often disinhibited or unable to control their emotional expressivity, and there is a disconnection between external stimuli and the emotional responses.[10,22,23]

These reactions (which cause distress, embarrassment, and avoidance of social situations and have been linked to depression)[6,10,18,20] often occur in the first weeks after the stroke and are acute, short-lived, and intense.[2,11,20,24] These features differentiate them from other psychological reactions (e.g., anxiety and depression) that can become chronic.[2]

Denial. *Denial* and *anosognosia* are terms that have often been used interchangeably since they were first applied to the medically ill.[6,19,25,26] Anton and Pick first described the phenomenon of denial in 1898, and in 1914, Babinski coined the term *anosognosia* (to describe brain-injured patients with hemiplegia who did not acknowledge their illness).[27] Since then, anosognosia has been used to describe patients with various neurologic disorders who lack awareness and insight into their disability.[26]

Freud viewed the lack of awareness as a defense mechanism that serves to protect the individual from anxiety.[19] In 1955, Weinstein and Kahn observed that there might be different types of anosognosia, though they failed to find a specific neurologic function or territory that was associated with anosognosia.[19]

Prigatano and Koloff[19] observed that patients with denial demonstrated some knowledge about their illness, showed resistance and anger when receiving feedback about their condition, and struggled when trying to work with new information about themselves. Patients with anosognosia seemed oblivious to their illness and showed surprise when unable to perform simple tasks.[19]

Ghika-Schmid and colleagues[6] found that denial was correlated with decreased fear, which was associated with a delay in seeking medical care and with poor outcome. Therefore, it is important to help patients overcome their fear of illness so that they may seek the help they need.

Comorbidity. Patients who experience problematic psychological reactions to stroke are likely to have comorbid mental disorders.[3,5,10,18,20,28] All of the short-lived reactions to stroke (such as catastrophic reactions and pseudobulbar affect) often correlate with PSD. Studies have found that 41% to 75% of patients with catastrophic reactions also manifest PSD.[10,18] Additionally, Carota and colleagues[10] found that within their pool of 326 stroke patients, 63% had heightened emotions, and 38% had pathologic laughing and crying soon after their stroke and developed PSD within a year. Comorbidity in stroke survivors may worsen chances of recovery, both physically and mentally.[3,28] Moreover, patients with heightened emotions poststroke are prone to develop GAD.[20]

Which Patients Are More Likely to Develop Adverse Psychological Reactions to Stroke?

In the process of identifying those patients who are likely to develop psychological reactions to stroke, physicians must consider the amount of time that has elapsed since stroke, the current risk factors and symptoms, the patient's history, and the patient's personality.

The amount of time that has elapsed since the stroke affects one's susceptibility to PSD or GAD. Astrom and colleagues[3,4] followed 80 stroke patients throughout the first 3 years after stroke to observe the relationship of neurobiological, functional, and psychosocial factors to PSD and GAD. Both PSD and GAD that arose in the first weeks after stroke were associated with living alone and with dysphasia. At 3 months, dysphasia was related to both PSD and GAD, whereas in the remaining 3 years few social contacts and the level of dependence in ADLs were significantly correlated with these disorders. Other studies[9,12] that investigated the factors associated with PSD 3 and 4 months poststroke found that impairment in function, as measured by ADLs and/or social function, was closely related to PSD. Thus, close attention to a patient's social and living situation as well as his or her facility with ADLs in the early stages following stroke is warranted.

Other psychological reactions that occur in the acute stages of stroke may also predict later development of depression or anxiety. Castillo and associates[5] found that, in some cases, early onset of PSD preceded the development of GAD. Additionally, in a 3-month follow-up study, Ghika-Schmid and colleagues[6] found that denial in the acute stages of stroke was associated with the development of depression and anxiety at 3 months following stroke.

Factors have also been identified that may help recognize those patients susceptible to experience catastrophic reactions and pseudobulbar affect. As expected, patients who experienced catastrophic reactions had less ability to perform ADLs.[18] Additionally, catastrophic reactions often occur in patients with poststroke aphasia.[11] Although pseudobulbar affect has not been linked to the level of ADLs, it has been found to be more prevalent in individuals with severe motor dysfunction (weakness of the limbs) and in those who have suffered an ischemic stroke (as opposed to a hemorrhagic stroke).[22]

Premorbid factors. Certain premorbid conditions may increase the risk of psychological reactions to stroke. Prior stressors (e.g., divorce, ongoing excessive alcohol intake, or previous incidents of CVAs) can influence a person's chances of experiencing PSD or poststroke anxiety.[9,12] Patients with a personal or family history of psychiatric illness are also at a higher risk for both early-onset and late-onset (3 months poststroke) PSD or anxiety,[5,29] as well as for catastrophic reactions.[18] Additionally, certain demographic factors (such as being female or younger) may increase vulnerability to an array of psychological reactions after stroke.[10,13,20,22]

Psychological reactions to stroke are in large measure determined by an individual's premorbid thought processes, personality, and coping mechanisms (i.e., the thoughts and behaviors employed by a person in an effort to manage a stressful situation).[8,19,30] Individuals who are predisposed to feeling distressed and who tend to be highly emotional in reaction to stress are more likely to interpret or to appraise their condition as overly stressful and to feel that they lack control over it.[30] The ability to cope with the illness, therefore, depends both on the appraisal of the event as stressful and on the capacity to utilize effective strategies in changing one's relationship to the situation and regarding it as manageable.[30]

Depression is often linked to maladaptive thinking processes (termed *cognitive distortions*) that adversely influence a person's appraisal processes and lead to an appraisal of problems as unsolvable.[31] Cognitive distortions include "black-and-white thinking," "catastrophizing,"

and "future-telling." Therefore, a patient who expresses thoughts such as "I will never get better" or "This is out of my control, and there's nothing I can do about this" is more likely to feel overwhelmed and to become depressed or anxious.[31]

The patient we described (who presented with poststroke catastrophic reactions) lived alone, did not appear to have a stable social network, denied his disabilities, and felt overwhelmed by his illness. As a result, Mr. B was at high risk for depression and anxiety.

How Do We Distinguish Psychological Reactions From Neuropsychiatric Reactions to Stroke?

It is not clear that psychological and neuropsychiatric reactions to stroke are independent from each other.[32] Some believe that neuropsychiatric reactions to stroke are direct and primary manifestations of the biological mechanisms affected by the stroke.[15,17] Others believe that psychological reactions to stroke are indications of poor coping mechanisms.[17,19]

In both PSD (which the DSM would categorize as depression secondary to a general medical illness [stroke]) and poststroke anxiety, a relationship to the anatomical location of the lesion has been proposed.[33] Some studies have linked PSD with left-sided anterior lesions and lesions to the left basal ganglia,[14] whereas others have found that some emotional reactions to stroke are correlated with right-sided lesions.[31] Others have found that a relationship between PSD and lesions of the anterior frontal cortex is independent of laterality or size of the lesion.[22]

It is becoming clearer that psychological reactions are complex and may be directly related to physiologic mechanisms; this blurs the boundary between neuropsychiatric and psychological reactions. For example, cortisol affects regions of the brain involved in attention, perception, memory, and appraisal of events.[34] Furthermore, abnormal cortisol levels are often found in depressed individuals.[34] Dysregulations in cortisol levels may play a role in the perceptual bias depressed individuals show toward negative or neutral events (i.e., cognitive distortions).[34] Astrom and colleagues[35] found that increased levels of cortisol during the first 3 months after stroke predict later development of depression.

Psychological and neuropsychiatric mechanisms are intertwined.[15,33,36,37] In a study comparing cognitive behavioral therapy (CBT)[38] and paroxetine as treatments for depression, Goldapple and colleagues[36] found that CBT and psychotropic medications may initially affect similar brain pathways.

How Do We Treat Psychological Reactions to Stroke?

Although primary care physicians do not have specialized psychiatric training, they can provide effective and compassionate care for patients who experience psychological reactions to stroke. First, primary care physicians can pay close attention to the behaviors that occur early after stroke (e.g., denial or overt sadness, the catastrophic reactions to stroke) that may herald the onset of depression or anxiety. Physicians can also look for risk factors for inadequate coping (such as social isolation, negative thoughts, severity of disability, or previous psychiatric history), which will increase a patient's susceptibility to mental illness.

When a primary care physician encounters a patient like Mr. B, treatment approaches may include those that directly alter the individual's physiology, such as medication, and approaches that emphasize the patient's thoughts, behaviors, and coping methods. Psychotropic medications (e.g., SSRIs, tricyclic antidepressants, stimulants) or other therapeutic approaches (such as electroconvulsive therapy or CBT) can be effective for PSD, anxiety, and pseudobulbar affects.[14,39,40,41] Still, in cases in which medications do not seem to help, or in which a patient experiences extreme side effects, other forms of psychological treatment may be useful.

Although CBT has not been widely studied in stroke patients, it has been useful in the treatment of depression in the general population[42] and in patients suffering from other physical illnesses.[43] CBT is a type of psychotherapy based on the idea that reciprocal relationships exist among humans' thoughts, emotions, behaviors, and physiology. CBT stems from cognitive therapy, which focuses mostly on the individual's thought processes. It emphasizes both thoughts and behaviors in treatment of psychological disorders.[38,42] According to these theories, changing ineffective thoughts or behaviors will affect mood and alleviate depression.[38]

In CBT, individuals learn to alter maladaptive thoughts that amplify feelings of helplessness and to restore their sense of control. They learn to perceive situations as more manageable and respond to these situations effectively, thereby improving their mood and altering their physiology.[38] Patients with catastrophic reactions or heightened emotionality may also benefit from CBT, as these reactions may represent a lack of effective coping strategies to stress.[19,30,38,44]

Although CBT training may not always be accessible to physicians, primary care physicians can help patients enhance coping mechanisms and engage in effective thought processes and behaviors by keeping in mind the fundamental concepts of CBT. Physicians facilitate coping processes whenever they help their patients understand what part of the illness is under their control (e.g., by supplying them with resources that they can draw on in the process of rehabilitation and by teaching them constructive problem-solving strategies). Physicians can also help their patients by reframing the illness in ways that

allow patients to perceive it as manageable. Finally, helping a patient find a supportive social network may alleviate depression and facilitate coping with illness.

When Mr. B did not respond to the antidepressants and continued to express negative thoughts, his physician challenged those thoughts and repeatedly asked him to point out something positive in his life. Often resistant, Mr. B usually gave in and answered that his children were a positive part of his life. In time, Mr. B grew to expect these exercises at follow-up visits and was able to think of additional positive aspects of his life. His doctor also asked Mr. B to spend a certain amount of time each week with his children and encouraged him to contact old friends and to actively participate in the rehabilitation process, even when he experienced frustration. Whenever the patient denied his disabilities or illness, his physician would state that it was understandable that Mr. B did not believe that he needed help but asked that he continue the social interactions and activities associated with rehabilitation anyway. Reading material relating to stroke and the resources available for stroke survivors in the area was also provided. Slowly, Mr. B began to feel that he could manage his illness, and his denial decreased.

Conclusion

Psychological reactions to stroke are varied; however, psychological reactions may serve as a prominent obstacle to a patient's recovery and health. Although these reactions may be a direct manifestation of physiologic damage to the brain, they may also result from ineffective thought processes, skewed perceptions, and poor coping skills. Since stroke affects complex neural mechanisms involved in human information processing, behavior, and emotion, our ability to determine whether a patient will benefit from psychological treatments requires a consideration of many factors, including the patient's background and circumstances. Although CBT has not been widely studied in stroke patients, it has been found to be highly effective in depressed individuals, and it may be useful in this population. Helping a patient gain access to appropriate coping strategies and to constructive ways of thinking is crucial to recovery and may be a part of treatment even when psychiatric medications are employed.

REFERENCES

1. Jemal A, Ward E, Hao Y, et al. Trends in the leading causes of death in the United States, 1970–2002. JAMA 2005;294:1255–1259
2. Bogousslavsky J. Emotions, mood, and behavior after stroke. Stroke 2003;34:1046–1050
3. Astrom M. Generalized anxiety disorder in stroke patients: a 3-year longitudinal study. Stroke 1996;27:270–275
4. Astrom M, Adolfsson R, Asplund K. Major depression in stroke patients. Stroke 1993;24:976–982
5. Castillo CS, Schultz SK, Robinson RG. Clinical correlates of early-onset and late-onset poststroke generalized anxiety. Am J Psychiatry 1995;152:1174–1179
6. Ghika-Schmid F, van Melle G, Guex P, et al. Subjective experience and behavior in acute stroke: the Laussanne Emotion in Acute Stroke Study. Neurology 1999;52:22–28
7. Morris PLP, Robinson RG, Andrzejewski P, et al. Association of depression with 10-year poststroke mortality. Am J Psychiatry 1993;150:124–129
8. Aben I, Denollet J, Lousberg R, et al. Personality and vulnerability to depression in stroke patients: a 1-year prospective follow-up study. Stroke 2002;33:2391–2395
9. Burvill P, Johnson G, Jamrozik K. Risk factors for post-stroke depression. Int J Geriatr Psychiatry 1997;12:219–226
10. Carota A, Berney A, Aybek S. A prospective study of predictors of post-stroke depression. Neurology 2005;64:428–433
11. Carota A, Rossetti AO, Karapanayiotides T, et al. Catastrophic reaction in acute stroke: a reflex behavior in aphasic patients. Neurology 2001;57:1902–1905
12. Hosking SG, Marsh NV, Friedman PJ. Depression at 3 months poststroke in the elderly: predictors and indicators of prevalence. Aging, Neuropsychology, and Cognition 2000;7:205–216
13. Kadojic D, Vladetic M, Candrlic M. Frequency and characteristics of emotional disorders in patients after ischemic stroke. Eur J Psychiatry 2005;19:88–95
14. Robinson RG. Poststroke depression: prevalence, diagnosis, treatment, and disease progression. Biol Psychiatry 2003;54:376–387
15. Whyte EM, Mulsant BH. Post stroke depression: epidemiology, pathophysiology, and biological treatment. Biol Psychiatry 2002;52:253–264
16. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition. Washington, DC: American Psychiatric Association; 1994
17. Goldstein K. Origin of symptoms. In: Aftereffects of Brain Injuries in War: Their Evaluation and Treatment: The Application of Psychologic Methods in the Clinic. New York, NY: Grune & Stratton; 1942:69–79
18. Starkstein SE, Fedoroff JP, Price TR, et al. Catastrophic reaction after cerebrovascular lesions: frequency, correlates, and validation of scale. J Neuropsychiatry Clin Neurosci 1993;5:189–194
19. Prigatano GP, Klonoff PS. A clinician's rating scale for evaluating impaired self-awareness and denial of disability after brain injury. Clin Neuropsychol 1998;12:56–67
20. Calvert T, Knapp P, House A. Psychological associations with emotionalism after stroke. J Neurol Neurosurg Psychiatry 1998;65:928–929
21. Eccles S, House A, Knapp P. Psychological adjustment and self reported coping in stroke survivors with and without emotionalism. J Neurol Neurosurg Psychiatry 1999;67:125–126
22. Kim JS, Choi-Kwon S. Poststroke depression and emotional incontinence: correlation with lesion location. Neurology 2000;54:1805–1810
23. Kim JS, Choi S, Kwon SU, et al. Inability to control anger or aggression after stroke. Neurology 2002;58:1106–1108
24. Paradiso S, Robinson RG, Arndt S. Self-reported aggressive behavior in patients with stroke. J Nerv Ment Dis 1996;184:746–753
25. Katz N, Fleming J, Keren N, et al. Unawareness and/or denial of disability: implications for occupational therapy intervention. Can J Occup Ther 2002;69:281–292
26. Kortte KB, Wegener ST. Denial of illness in medical rehabilitation populations: theory, research and definition. Rehabil Psychol 2004;49:187–199
27. Ellis SJ, Small M. Denial of illness in stroke. Stroke 1993;24:757–759
28. Shidoma K, Robinson RG. Effects of anxiety disorder on impairment and recovery from stroke. J Neuropsychiatry Clin Neurosci 1998;10:34–40
29. Pohjasvaara T, Leppavuori A, Siira I, et al. Frequency and clinical determinants of poststroke depression. Stroke 1998;29:2311–2317
30. Folkman S, Lazarus RS. The relationship between coping and emotion: implications for theory and research. Soc Sci Med 1988;26:309–317
31. Kovacs M, Beck AT. Maladaptive cognitive structures in depression. Am J Psychiatry 1978;135:525–533
32. Gainotti G, Azzoni A, Marra C. Frequency, phenomenology and anatomical-clinical correlates of major post-stroke depression. Br J Psychiatry 1999;175:163–167
33. Carson AJ, MacHale S, Allen K, et al. Depression after stroke and lesion location: a systematic review. Lancet 2000;356:122–126
34. Erickson K, Drevets W, Schulkin J. Glucocorticoid regulation of diverse cognitive functions in normal and pathological emotional states. Neurosci Biobehav Rev 2003;27:233–246

35. Astrom M, Olsson T, Asplund K. Different linkage of depression to hypercortisolism early versus late after stroke: a 3-year longitudinal study. Stroke 1993;24:52–57
36. Goldapple K, Segal Z, Garson C, et al. Modulation of cortical-limbic pathways in major depression: treatment-specific effects of cognitive behavior therapy. Arch Gen Psychiatry 2004;61:34–41
37. Roffman JL, Marci CD, Glick DM, et al. Neuroimaging and the functional neuroanatomy of psychotherapy. Psychol Med 2005;35:1385–1398
38. Rupke SJ, Blecke D, Renfrow M. Cognitive therapy for depression. Am Fam Physician 2006;73:83–86
39. Teasell RW, Merskey H, Deshpande S. Antidepressants in rehabilitation. Phys Med Rehabil Clin N Am 1999;10:237–253
40. Kim SW, Shin IS, Kim JM, et al. Mirtazapine for pathological laughing and crying after stroke. Clin Neuropharmacol 2005;28:249–251
41. Currier MB, Murray GB, Welch CC. Electroconvulsive therapy for post-stroke depressed geriatric patients. J Neuropsychiatry Clin Neurosci 1992; 4:140–144
42. Beck AT. The current state of cognitive therapy: a 40-year retrospective. Arch Gen Psychiatry 2005;62:953–959
43. Mohr DC, Boudewyn AC, Goodkin DE, et al. Comparative outcomes for individual cognitive-behavior therapy, supportive-expressive group psychotherapy, and sertraline for the treatment of depression in multiple sclerosis. J Consult Clin Psychol 2001;69:942–949
44. Nicholl CR, Lincoln NB, Muncaster K, et al. Cognitions and post-stroke depression. Br J Clin Psychol 2002;41:221–231

ANNOTATED BIBLIOGRAPHY

Folkman S, Lazarus RS. The relationship between coping and emotion: implications for theory and research. Soc Sci Med 1988;26:309–317
–A detailed review of Folkman and Lazarus's theory of stress and coping. The authors present the traditional views of coping—the animal and ego models. These traditional theories view coping as a response to emotion. The authors propose a theory, which regards coping and emotion as reciprocal entities that influence each other and give a detailed explanation of how coping can affect emotion. The terms *emotion*, *coping*, and *cognitive appraisal* are defined and discussed. Additionally, emotion-focused and problem-focused coping styles are presented, as well as the idea that a person's interoperations and appraisals of situations will affect the coping style that is employed by that individual.

Klonoff PS, Lage GA. Varieties of the catastrophic reaction to brain injury: a self psychology perspective. Bull Menninger Clin 1993;57:227–242
–A presentation of several case studies demonstrating an inability to confront disabilities after traumatic brain injury. Although these cases involved traumatic brain injury, the reactions described are similar to those seen in stroke patients. The authors explain that the presentation of catastrophic reactions may vary (depending on the individual) and discuss important elements in the treatment of individuals with these reactions. Empathic care, education of patients about their condition, modification of the environment so that it is not overwhelming to patients, creation of an alliance with the family, and promotion of staff communication and consultation are all pertinent to adequate care of these patients.

Astrom M. Generalized anxiety disorder in stroke patients: a 3-year longitudinal study. Stroke 1996;27:270–275
–Two follow-up studies (using the same sample) investigated poststroke depression and anxiety during the first 3 years of recovery from stroke. The sample comprised 80 patients, with a mean age of 73 years, who suffered an acute CVA. Patients were followed intermittently during the first 3 years after stroke. Both studies found that the elements associated with PSD and GAD after stroke vary as a function of the time that has passed since the incident.

Ghika-Schmid F, Bogousslavsky J. Affective disorders following stroke. Eur Neurol 1997;38:75–80
–A concise review of poststroke affective disorders and their etiology. The authors describe pseudo-depressive manifestations that are prevalent after stroke. The article outlines diagnosis methods, risk factors, prevalence, prognosis, and lesion location associated with PSD and pseudo-depressive disorders.

Prigatano GP, Klonoff PS. A clinician's rating scale for evaluating impaired self-awareness and denial of disability after brain injury. Clin Neuropsychol 1998;12:56–67
–An article presenting the development of a tool used to differentiate patients suffering from anosognosia from those in denial. The author introduces 3 guidelines that may be followed to ascertain which patients deny their illness. On the basis of these criteria, 56 subjects enrolled in a rehabilitation center were included in the sample. Using the 3 broad criteria, two 10-item scales were developed. Statistical analysis demonstrated that the scales were reliable and valid in the discrimination of patients with denial from those with anosognosia.

Kneebone II, Dunmore E. Psychological management of post-stroke depression. Br J Clin Psychology 2000;39:53–65
–A clearly written review of the research conducted on psychological treatments for PSD. The article summarizes the difficulties in assessing depression in stroke patients and names a number of assessment tools that have been developed for this purpose. The authors conclude that further empirically based research is needed; they emphasize that CBT holds great promise as an empirically supported treatment for PSD.

Alexopoulos GS, Raue P, Arean, P. Problem-solving therapy versus supportive therapy in geriatric major depression with executive dysfunction. Am J Geriatr Psychiatry 2003;11:46–53
–A comprehensive study that tested the hypothesis that problem-solving therapy is more effective than supportive therapy in reducing disability and reducing depressive symptoms mediated by improved decision making and generation of alternatives in executive dysfunction. Twenty-five depressed elderly individuals with executive dysfunction (and therefore who were less likely to respond to pharmacotherapy) were included in the study. Subjects were given a multitude of assessment measures. The results confirmed the initial hypothesis that problem-solving therapy was more effective than supportive therapy. These results may be relevant to stroke patients with cognitive impairments following stroke.

Bogousslavsky J. Emotions, mood, and behavior after stroke. Stroke 2003; 34:1046–1050
–An overview of the various emotional reactions and mood disorders following stroke, with an emphasis on the need to distinguish psychological reactions in the acute stages after stroke from those that are chronic, and the need to reframe them rather than categorize them under the single category of depression. An easy-to-follow measurement for the assessment of psychological reactions in the acute stages of stroke is presented. The author also discusses PSD (and its diagnosis, prognosis, and etiology).

Erickson K, Drevets W, Schulkin J. Glucocorticoid regulation of diverse cognitive functions in normal and pathological emotional states. Neurosci Biobehav Rev 2003;27:233–246
–A well-written and intricate review of the literature on the role of cortisol in the brain, its interactions with various chemicals and brain structures, and the influence these interactions may have on pathologic information processing and cognition and mood disorders. Cortisol regulates a number of chemicals (including serotonin, norepinephrine, and dopamine) and is involved in human motivation, attention, and cognitive functions. The review shows that abnormal cortisol levels in the brain may contribute to negative interpretation of events, distorted perceptions, and an increase in the salience of negative events.

Turner-Stokes L. Post stroke depression: getting the full picture. Lancet 2003;361:1757–1758
–A commentary on the literature investigating the biological etiology of PSD. The author presents the idea that biochemical change, and not exclusively localized structural damage, may lead to depression after stroke. Affected individuals are unable to communicate adequately and to complete common questionnaires used to assess depression.

Goldapple K, Segal Z, Garson C, et al. Modulation of cortical-limbic pathways in major depression: treatment-specific effects of cognitive behavior therapy. Arch Gen Psychiatry 2004;61:34–41
–A fascinating study examining the effects of CBT and pharmacotherapy on regional changes in the brains of those treated for depression. Positron emission tomography (PET) scans of 14 depressed patients treated with CBT were compared with scans of 13 depressed patients treated with paroxetine in a previous study. Statistical analyses confirmed that the 2 groups were comparable in their severity of illness and in the results of the PET scans at baseline. Results showed that CBT had an effect on regional brain structures. Although the 2 therapies affected different primary brain structures, the treatments caused effects in opposite directions; both resulted in a net change in critical prefrontal hippocampal pathways.

Kortte KB, Wegener ST. Denial of illness in medical rehabilitation populations: theory, research and definition. Rehabil Psychol 2004;3:187–199
–A useful review in understanding the differences between denial and anosognosia. The article discusses the history behind development and evolution of the 2 terms. A review of the literature on denial of illness and its impact on treatment outcome concerning heart disease, cancer, acquired brain injury, and spinal cord injury is provided. The authors conclude that denial is multidimensional and propose a taxonomy of the constructs of denial of illness and anosognosia.

Hackett ML, Anderson CS. Predictors of depression after stroke: a systematic review of observational studies. Stroke 2005;36:2296–2301
–A systematic review assessing the validity and reliability of studies investigating the predictors for PSD. Data were drawn from population-based, hospital-based, and rehabilitation-based studies. The authors present the shortcomings of the PSD studies and conclude that there are not enough well-designed studies to identify conclusively predictors of PSD. Nevertheless, variables such as severity of stroke, physical disability, and cognitive impairment were most consistently reported as correlates of the later development of PSD, with severity of stroke holding the strongest relationship.

Rupke SJ, Blecke D, Renfrow M. Cognitive therapy for depression. Am Fam Physician 2006;73:83–86
–A clear review intended for family physicians that summarizes the use of CBT for the treatment of depression. The authors give a brief description of the development of CBT and present the nature of CBT and its effectiveness for various types of depression. Guidelines for family practitioners are provided to assess the usefulness of CBT for their patients, as well as useful ways to present this form of therapy to patients and their families. The article emphasizes that CBT is a useful alternative to psychotropic medication and presents studies that compare CBT and antidepressants.