

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.

Ms. Chriki is a Research Assistant in the Department of Psychiatry at MGH. Dr. Bullain is a Postdoctoral Research Fellow at the MGH-Harvard Medical School Center for Nervous System Repair in the Departments of Neurology and Neurosurgery at Harvard Medical School. Dr. Stern is Chief of the Psychiatric Consultation Service at MGH and a Professor of Psychiatry at Harvard Medical School.

Corresponding author: Theodore A. Stern, M.D., Massachusetts General Hospital, Fruit St., WRN 605, Boston, MA 02114 (e-mail: tstern@partners.org).

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

Lyvia S. Chriki, B.A.; Szofia S. Bullain, M.D.;
and Theodore A. Stern, M.D.

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 Koloff¹⁹ 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 colleagues⁶ 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 colleagues¹⁰ 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 associates⁵ 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 colleagues⁶ 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 post-stroke 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 colleagues³⁵ 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 colleagues³⁶ 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 population⁴² 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.

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 –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.