

Poststroke Depression Incidence and Risk Factors: An Integrative Literature Review



Jessica L. Johnson, Pamela A. Minarik, Karin V. Nyström, Cynthia Bautista, Mark J. Gorman

Abstract: Depression is a frequent complication of stroke, but few nurse researchers have studied poststroke depression (PSD). We reviewed all published research (January 1980–March 2005) that examined the incidence of and risk factors for depression among stroke survivors during the first 3 months after stroke. Many of the 49 studies reviewed were complicated by methodological limitations, including differing definitions of stroke and depression, the use of screening instruments to diagnose depression, selection bias, assessment at different time intervals poststroke, exclusion of patients with physical or cognitive impairments, and failure to control for associated variables. The incidence of PSD ranged from 5% to 63%. A history of depression, increased stroke severity, and poststroke cognitive or physical impairment were found to be risk factors for PSD.

Stroke is the third leading cause of death and a major cause of serious, long-term disability in the United States (American Heart Association [AHA], 2005). An estimated 5.4 million stroke survivors are living in the United States today (AHA, 2005). More than half of all stroke survivors may be at risk for developing depression within 3 months of the onset of stroke (Angelelli et al., 2004).

Depression is among the most common psychiatric disorders and is the leading cause of disability in the United States, affecting more than 18 million Americans

Questions or comments about this article may be directed to Karin V. Nyström, MSN APRN, at karin.nystrom@yale.edu. She is the clinical coordinator of the Primary Stroke Center at Yale–New Haven Hospital and assistant clinical professor at Yale University School of Nursing, New Haven, CT.

Jessica L. Johnson, MSN APRN, is a nurse practitioner in the Department of Neurology at Yale University School of Medicine.

Pamela A. Minarik, MSN CS FAAN, was a professor at Yale University School of Nursing and director of its Office of International Affairs when this study was completed. She currently is a doctoral candidate at the University of California–San Francisco.

Cynthia Bautista, PhD RN CNRN, is a neuroscience clinical nurse specialist at Yale–New Haven Hospital.

Mark J. Gorman, MD, was director of the Stroke Program at Yale–New Haven Hospital when this study was completed. He currently is an associate professor in the Department of Neurology at the University of Vermont, Burlington, VT.

Copyright © 2006 American Association of Neuroscience Nurses 0047-2606/06/3804(Suppl.)/00316\$5.00

(National Institute of Mental Health, 2005). Considerable controversy exists about whether the depression that occurs after stroke is a diagnostic category, distinct from depression in other populations. Relatively little is known about the specific effects of comorbid illnesses on the course and outcome of depression, except that comorbidities have negative effects on outcomes (Boland & Keller, 2002; Gotlib & Hammen, 2002). Poststroke depression (PSD) is associated with increased mortality (Everson, Roberts, Goldberg, & Kaplan, 1998; House, Knapp, Bamford, & Vail, 2001), poor cognitive function (Jorge, Robinson, Arndt, & Starkstein, 2003; Murata, Kimura, & Robinson, 2000; Narushima, Chan, Kosier, & Robinson, 2003), physical impairment (Chemerinski, Robinson, Arndt, & Kosier, 2001; Narushima & Robinson, 2003), less participation in rehabilitation (Gonzales-Torrecillas, Mendlewicz, & Lobo, 1995; Herrmann, Black, Lawrence, Szekely, & Szalai, 1998; Kotila, Numminen, Waltimo, & Kaste, 1998, 1999), and decreased quality of life (King, 1996; Sturm et al., 2004). For these reasons, recognition, prevention, and treatment of PSD are critical in reaching optimal patient outcomes after stroke. This integrative literature review identifies the incidence of and risk factors for the development of PSD within the first 3 months following stroke.

Definitions

Stroke is a clinical syndrome of neurological impairment that results from an impairment of cerebral blood flow for a period of time, which causes cellular injury and death. Perfusion may be impaired by blockage of blood flow by a clot (ischemic stroke) or by blood-vessel hemorrhage (hemorrhagic stroke). Patients typically experience sudden onset of one or more focal neurological deficits that persist for more than 24 hours (Hickey & Hock, 2003).

Depression is a term that has both lay meanings and meaning as a psychiatric diagnosis. Periods of sadness or “feeling blue,” as well as feelings of loss and bereavement, are a normal part of the human condition. Depressive symptoms can be a response to stressful life events, such as illness or hospitalization. Patients may also be diagnosed with depression when they meet the criteria for major depressive disorder. Thus, depression can be an affective experience (such as sadness), a symptomatic complaint, or a clinical syndrome (Rouchell, Pounds & Tierney, 2002). The boundary between normal and

abnormal symptoms is often unclear and may be arbitrary. It is usually determined by criteria for symptom severity, duration, and clinically significant distress or impairment and must be viewed in the context of the patient's personal situation (American Psychiatric Association [APA], 2000; Gotlib & Hammen, 2002; Pasacreta, Minarik, & Nield-Anderson, 2000).

Widely recognized as having a neurobiological basis, depression relates to other medical illness in four ways. It may be a cause or early manifestation of a medical condition. When depression occurs after the medical condition, it may be a pathophysiologic consequence of the medical illness or a reaction to prescribed drugs or substances. Finally, it may be a psychological reaction to the medical illness. Depression and medical illness also can coexist but be etiologically unrelated (Rouchell et al., 2002).

Diagnostic terms for depression include *major depressive episode or disorder, adjustment disorder with depressed mood, dysthymia or dysthymic disorder, mood disorder due to a general medical condition, and substance-induced mood disorder*. Also relevant to the discussion of poststroke depression are *minor depression* and *subsyndromal depression*. Major depressive disorder (MDD) is distinguished by one or more major depressive episodes lasting at least 2 weeks and characterized by depressed mood or diminished interest or pleasure for most of the day almost every day. It is accompanied by at least four out of nine depressive symptoms: depressed mood; diminished interest or pleasure; changes in appetite or weight; insomnia or hypersomnia; psychomotor agitation or retardation; fatigue or loss of energy; feelings of worthlessness or excessive or inappropriate guilt; diminished ability to think or concentrate, or indecisiveness; and recurrent thoughts of death, recurrent suicidal ideation, suicide attempt, or specific plan for suicide. The episode includes clinically significant distress or impairment in social, occupational, or other important areas of functioning. The symptoms cannot be due to the direct effects of a substance or a general medical condition (APA, 2000; Rouchell et al., 2002).

Minor depression is a term used in clinical practice in general hospital settings and in research. *Minor depressive disorder* is included in the current edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM) as a research diagnosis, meaning that there was insufficient evidence or professional agreement to include it as an official diagnosis at the time of publication (APA, 2000). *Minor depressive disorder* refers to one or more periods of depressive symptoms lasting at least 2 weeks but involving fewer symptoms and less impairment than MDD.

Subsyndromal depression—the presence of symptoms that do not meet the criteria for a diagnosis—is of growing interest in research and clinical practice. Kessler (2002) notes that research using screening scales reveals a high prevalence of symptoms and a comparatively low prevalence of depressive disorder. This pattern suggests

that many people have depressive symptoms that do not meet the criteria for a diagnosis of MDD.

Poststroke depression, for the purpose of this article, refers to depression, as determined by the authors of the studies reviewed, occurring within 3 months of the onset of a clinically apparent stroke.

Table 1. Comparison of the Literature on Incidence of and Risk Factors for Acute-Onset Poststroke Depression

Table 1 is published online at www.aann.org/journal (Continuing Education).

Methods

To identify relevant studies for this review, we initially searched the reference lists of the Cochrane Reviews on treatment and prevention of poststroke depression (Anderson, Hackett, & House, 2004; Hackett, Anderson, & House, 2004). In December 2004 we searched the computerized databases MEDLINE, CINAHL, and PsychINFO, from their respective start dates (1966, 1982, 1967), using the following keywords for stroke: *stroke, cerebral vascular accident, cerebral infarction, brain ischemia, transient ischemic attack, intracranial hemorrhage, intracranial embolism and thrombosis, intracranial hypertension, intracranial vasospasm, hypoxia-ischemia brain, cerebrovascular trauma, brain attack, aphasia*. We also used the following keywords for depression: *depression, depressive disorder, self-injurious behavior, mental health, antidepressive agents, central nervous system agents*. The literature search was restricted to English-language papers that focused on adults and excluded case reports, editorials, letters, interviews, patient handouts, newspaper articles, and commentaries. The search yielded 303 hits in MEDLINE, 257 hits in CINAHL, and 40 hits in PsychINFO. It was updated monthly until March 2005 to identify newly published research. The review included studies conducted outside the United States, because they constituted the majority of the research literature on PSD.

A literature search of the same databases was conducted in January 2005 using these additional search terms: *epidemiology, demography, risk factors, comorbidity, probability, age of onset, delayed onset, surveys, health screening, mental disorders, patient history taking, life history, family history, substance abuse, substance dependence, alcohol abuse, alcoholism, cocaine, addictive behavior, age factors, race factors, sex factors, minority groups, marital status, geographic factors, socioeconomic factors, social class, ethnic groups, employment, educational status, disease progression, recovery*. This search yielded 226 articles in MEDLINE, 133 articles in CINAHL, and 6 articles in PsychINFO. This search was also updated monthly until March 2005 to identify newly published research.

English-language papers published after 1980 were obtained. From these papers, ancestry searches were conducted, and additional papers were identified through

author searches. Studies that examined PSD beyond 3 months poststroke and those that did not specify the length of time between stroke and depression evaluation were excluded. The following 49 studies made up the final set that matched criteria for inclusion in this review (i.e., studies that were published after 1980 in English and that examined PDS within 3 months of stroke; Table 1)

Incidence of PSD

Estimates of the incidence of PSD ranged from 5% (Aben et al., 2002) to 63% (Gottlieb, Salagnik, Kipnis, & Brill, 2002). Although in the majority of studies researchers described the instruments used to measure depression among participants, the psychiatric diagnosis of MDD was made only in 24 studies. For those 24 studies, the reported incidence ranged from 6% (Berg, Palomaki, Lehtihalmes, Lonnqvist, & Kaste, 2001) to 35% (Weg & Kulk, 1999). Seventeen studies described subjects who met the criteria for minor depression. The reported incidence of minor depression ranged from 11% (Gonzales-Torrecillas et al., 1995) to 44% (Kauhanen et al., 1999, 2000).

The wide range of incidence rates of PSD is related to methodological differences among studies, including differing definitions of *stroke* and *depression*, the use of screening instruments to diagnose depression (Spencer, Tompkins, & Schultz, 1997), selection bias inherent in convenience samples (Aben et al., 2001), depression assessment at different time intervals poststroke (Anderesen, 1997), exclusion of stroke patients with physical or cognitive impairments (Nelson et al., 1993), and failure to control for associated variables, such as depression risk factors and premorbid functioning (House, 1987a; Whyte & Mulsant, 2002).

Operational Definitions of Stroke

Most PSD studies did not define *stroke* based upon consensus standards. As a result, subjects may not have been representative of the general population of stroke patients, and individual studies may not be comparable. For example, some studies included only stroke patients who had a visible lesion on CT scan or magnetic resonance imaging (MRI) scan (Aben, Denollet, et al., 2002; Aben, Verhey, et al., 2003; Angelelli et al., 2004; Berg et al., 2001; Herrmann, Bartels, Schumacher, & Wallesch, 1995; Morris, Robinson, de Carvalho et al., 1996; Morris, Robinson, Raphael, & Hopwood, 1996; Nannetti, Paci, Pasquini, Lombardi, & Taiti, 2005; Niedermaier, Bohrer, Schulte, Schlattmann, & Heuser, 2004; Nys et al., 2005; Robinson, Kubos, Starr, Rao, & Price 1984; Shimoda & Robinson, 1999; Singh et al., 2000; Sinyor, Jacques, Kaloupek, Becker, Goldenberg, et al., 1986a, 1986b; Spalletta, Ripa, & Caltagirone, 2005; Starkstein, Robinson, Berthier, Parikh, & Price, 1988; Starkstein, Robinson, & Price, 1987; Vataja et al., 2004), whereas other studies included all patients admitted to or discharged from a hospital with a diagnosis of

stroke (Eriksson et al., 2004; Hayee, Akhtar, Hague, & Rabbani, 2001; Kotila et al., 1998, 1999; Wade, Legh-Smith, & Hewer, 1987). Some PSD studies included only those patients with a "first-ever stroke" (Aben et al., 2002, 2003; Angelleli et al., 2004; Berg et al., 2001; Bolla-Wilson, Robinson, Starkstein, Boston, & Price, 1989; Carota et al., 2005; Hayee, Akhtar, Hague, & Rabbani, 2001; Herrmann et al., 1995; House, Dennis, Warlow, Hawton, & Molyneux, 1990a, 1990b; House et al., 1991; Kauhanen et al., 1999, 2000; Kotila et al., 1998, 1999; Morris, Robinson, de Carvahlo, et al., 1996; Morris, Robinson, Raphael, et al., 1996; Nannetti et al., 2005; Nys et al., 2005; Singh et al., 2000; Sinyor, Amato, et al., 1986; Sinyor, Jacques, et al., 1986; Starkstein, Robinson, Berthier, et al., 1988; Starkstein, Robinson & Price, 1988a; Starkstein et al., 1989; Spalletta et al., 2005; Tang et al., 2002), while others included individuals with a history of previous strokes (Astrom, Adolfs-son, & Asplund, 1993; Gonzalez-Torrecillas et al., 1995; Hosking, Marsh, & Friedman, 2000; Morris, Robinson, & Raphael, 1990; Morris, Robinson, Raphael, Samuels, & Molloy, 1992; Morris & Robinson, 1995; Nelson et al., 1993; Ramasubbu, Robinson, Flint, Kosier, & Price, 1998; Robinson, Starr, Kubos, & Price, 1983; Robinson, Starr, Lipsey, Rao, & Price, 1984, 1985). Additionally, two PSD studies included patients with a transient ischemic attack (Astrom et al., 1993; Ramasubbu et al., 1998), rather than a stroke.

Operational Definitions of Depression

There is no expert consensus on the definition of *PSD*. Methods used to measure depression in PSD research varied greatly among studies, thereby preventing the pooling of results. Several researchers studying PSD used standardized scales, such as the Beck Depression Inventory or the Hamilton Depression Rating Scale, which were designed to measure depression severity, rather than to diagnose depression. These studies showed a higher prevalence of depression (Nys et al., 2005; Ramasubbu, Robinson, Flint, Kosler, & Price, 1998). Other researchers designed their own instruments to measure depression (Wade et al., 1987), relied on depression self-rating scales (Eriksson et al., 2004), or based depression assessments on information provided by individuals other than the patient (Nelson et al., 1993). In other studies, stroke patients were interviewed by a qualified mental health professional and diagnosed with depression based upon the most recent *DSM* or *International Classification of Diseases (ICD; World Health Organization, 1990)* diagnostic criteria. The *DSM* criteria for depression may not be valid for PSD, as they include somatic symptoms, such as psychomotor retardation, and disturbances in appetite, sleep, and sexual interest that are also seen in nondepressed stroke patients. Fedoroff, Starkstein, Parikh, Price, and Robinson (1991) demonstrated that when the diagnosis of PSD was based solely upon the

nonsomatic *DSM* criteria, there was no significant change in the incidence of PSD, suggesting that somatic symptoms do not lead to overdiagnosis of PSD.

Selection Bias

PSD may be more common among patients living in a hospital or rehabilitation setting than among those living in the general community, because patients who required admission to neurological units or rehabilitation services may have suffered more severe strokes than those who were able to receive care on an outpatient basis in the community. For PSD studies, stroke patients were typically recruited from an acute care hospital, a rehabilitation facility, or the general community. Study samples may have excluded those with the most severe strokes, who may have gone to hospice or extended care facilities. When we compared research on PSD on the basis of the location of sample recruitment, it appeared there may be a trend for community-based studies to show lower incidences of PSD, but it is difficult to attribute differences solely to this factor. Of the studies included in this review, those that used community samples had an incidence of PSD of approximately 15%–20% (Ghika-Schmid, van Melle, Guex, & Bogousslavsky, 1999; House et al., 1990a, 1990b, 1991). Those based on hospitalized patients yielded a higher but relatively consistent 1-month poststroke incidence rate of approximately 25% (Aben et al., 2002, 2003; Andersen, Vestergaard, Riis, & Lauritzen, 1994; Astrom, Adolfsson, & Asplund, 1993; Berg et al., 2001; Fedoroff et al., 1991; Herrmann et al., 1995; Morris, Robinson, de Carvalho, et al., 1996; Paradiso & Robinson, 1998). Studies of stroke patients in rehabilitation settings showed a wider range, with incidence rates at 25%–50% (Nannetti et al., 2005; Robinson-Smith, Johnston, & Allen, 2000; Sinyor, Amato, et al., 1986; Sinyor, Jaques, et al., 1986; Weg & Kulk, 1999). Although it is commonly thought that those in rehabilitation are more likely to suffer from PSD because of the increased severity of stroke, a group of researchers in Finland (Kotila et al., 1998) showed that after discharge from the hospital, stroke patients in districts with active outpatient rehabilitation had lower rates of PSD than those without such resources.

PSD Assessment at Different Time Intervals Poststroke

The timing of PSD assessment is likely to affect research findings. Because the diagnosis of MDD requires the presence of symptoms for a minimum of 2 weeks, patients should be assessed at least 14 days poststroke. For studies that examined PSD within 1 month poststroke, the incidence of depression ranged from 12% to 52% (Aben et al., 2002, 2003; Andersen et al., 1994; Astrom et al., 1993; Berg et al., 2001; Bush, 1999; Fedoroff et al., 1991; Gillen, Tennen, McKee, Gernert-Dott, & Affleck, 2001; Gonzalez-Torrecillas et al., 1995; House et al., 1990a, 1990b; House, Dennis, et al., 1991; Morris,

Robinson, de Carvahlo, et al., 1996; Nannetti et al., 2005; Niedermaier et al., 2004; Nys et al., 2005; Palomaki et al., 1999; Paradiso & Robinson, 1998; Ramasubbu et al., 1998; Robinson, Bolla-Wilson, Kaplan, Lipsey, & Price, 1986; Robinson, Kubos, et al., 1984; Robinson-Smith et al., 2000; Starkstein et al., 1989; Tang et al., 2002; Wade et al., 1987; Weg & Kulk, 1999). At 2 months poststroke, the incidence ranged from 35% to 61%, although fewer studies assessed the incidence of PSD at this interval (Angelleli et al., 2004; Gainotti, Azzoni, & Marra, 1999; Herrmann et al., 1995; Morris, Robinson, & Raphael, 1990; Morris, Robinson, Raphael, Samuels, et al., 1992; Morris & Robinson, 1995; Palomaki et al., 1999; Sinyor, Amato, et al., 1986; Sinyor, Jacques, et al., 1986). The incidence of PSD 3 months after the stroke ranged from 5% to 63% (Aben et al., 2002, 2003; Andersen et al., 1994; Astrom et al., 1993; Bush, 1999; Carota et al., 2005; Eriksson et al., 2004; Ghika-Schmid et al., 1999; Gottlieb et al., 2002; Hayee et al., 2001; Herrmann et al., 1998; Hosking et al., 2000; Kauhanen et al., 1999, 2000; Kotila et al., 1998, 1999; Singh et al., 2000; Vataja et al., 2004).

PSD may be more common among patients living in a hospital or rehabilitation setting than among those living in the general community.

Risk Factors for PSD

Proposed risk factors for the development of PSD have included age, gender, social support, socioeconomic status, ethnicity, psychiatric history, family history of psychiatric disorder, comorbid medical disease or disability, personality, stroke type, stroke severity, lesion volume, lesion location, and cognition (Eriksson et al., 2004; Kotila et al., 1998, 1999; Morris et al., 1990; Paradiso & Robinson, 1998; Ramasubbu et al., 1998; Robinson, Starr, Kubbs, & Price, 1983; Robinson, Starr, Lipsey, Rao, & Price, 1984, 1985; 1985; Robinson, Starr, Lipsey, et al., 1984; Robinson, Starr, & Price, 1984).

Personal and Social Characteristics

Age. Most of the studies included in this review failed to identify age as a significant risk factor for PSD (Astrom et al., 1993; Fedoroff et al., 1991; Herrmann et al., 1998; Hosking et al., 2000; Morris et al., 1990, 1992, 1995; Nannetti et al., 2005; Niedermaier et al., 2004; Nys et al., 2005; Ramasubbu et al., 1998; Singh et al., 2000; Sinyor, Amato, et al., 1986; Spalletta et al., 2005; Vataja et al., 2004). Although several studies found that older age was associated with the development of depression (Berg et al., 2001; Hayee et al., 2001; Kauhanen et al., 1999, 2000; Kotila et al., 1998, 1999), an equal number of studies showed that younger age was linked to PSD (Carota et al., 2005; Eriksson et al., 2004; Paradiso & Robinson, 1998; Robinson, Starr, et al., 1983, 1985;

Robinson, Starr, Lipsey, et al., 1984; Robinson, Starr, & Price, 1984). Some researchers suggested that among men, younger age may be associated with minor PSD (Morris et al., 1990; Paradiso & Robinson, 1998). The operational definition of *older age* influenced PSD study results; one study found that *old age*, defined as over the age of 55 years, was a risk factor (Berg et al., 2001), while another study showed that *young age*, defined as less than 68, was significantly associated with PSD (Carota et al., 2005). Elderly stroke patients were found to be more likely to become depressed because of other factors such as the presence of one or more comorbidities or the effect of gender.

Gender. Female gender may be a risk factor for PSD. Ten studies in this review found female gender to be associated with an increased risk of PSD (Aben et al., 2002, 2003; Angelleli et al., 2004; Eriksson et al., 2004; Gillen et al., 2001; Hayee et al., 2001; Herrmann et al., 1998; Kotila et al., 1998, 1999; Paradiso & Robinson, 1998; Ramasubbu et al., 1998; Wade et al., 1987), while only one study suggested that men were at higher risk for PSD than women (Morris et al., 1990). In the latter study, men were found to be at risk for minor depression. Thirteen studies (Astrom et al., 1993; Berg et al., 2001; Carota et al., 2005; Fedoroff et al., 1991; Hosking et al., 2000; Kauhanen et al., 1999, 2000; Nannetti et al., 2005; Niedermaier et al., 2004; Singh et al., 2000; Sinyor, Amato, et al., 1986; Spalletta et al., 2005; Vataja et al., 2004; Nys et al., 2005), however, showed that gender was not associated with PSD. The mechanism by which female gender may confer increased risk for PSD is complex. Stroke tends to occur later in life among women than it does in men. Women may be more likely than men to be living alone, be dependent on others for assistance with daily living, or be in poor health before the stroke (Glader et al., 2003; Kotila et al., 1998). In the general population, women are twice as likely as men to experience major depression (APA, 2000); this gender discrepancy may also apply to the depression that occurs after a stroke.

Social support. While research showed that stroke survivors with more social support were thought to be less vulnerable to PSD, definitive evidence is lacking because of the varied definitions of *social support*. Definitions included living alone versus in a nursing home versus with one's family, level of social functioning, number of social contacts, marital or partnership status, and perceived quality of social support. Interpretation of results was difficult for this factor, because some of these definitions of *social support* measured entirely different constructs. While two studies found that living alone after the stroke was associated with PSD (Eriksson et al., 2004; Hayee et al., 2001), five studies found no significant relationship between living situation and PSD (Kotila et al., 1999; Nannetti et al., 2005; Ramasubbu et al., 1998; Vataja et al., 2004; Wade et al., 1987). One study found that living at home 1 month after the stroke was associated with increased risk of PSD at 3 months poststroke

(Singh et al., 2000). One group of researchers reported that a higher level of social functioning was related to a lower risk for PSD (Robinson, Starr, et al., 1983, 1985; Shimoda & Robinson, 1999). All seven studies that assessed the relationship between marital status and PSD reported that no association existed between the two variables (Berg et al., 2001; Fedoroff et al., 1991; Herrmann et al., 1998; Morris et al., 1990, 1992, 1995; Nannetti et al., 2005; Ramasubbu et al., 1998; Singh et al., 2000). One of the studies (Gottlieb et al., 2002) showed that a high level of perceived social support was associated with PSD.

Socioeconomic status. Five studies focused on the possible role of socioeconomic status as a risk factor for PSD (Fedoroff et al., 1991; Morris et al., 1990, 1992, 1995; Paradiso & Robinson, 1998; Ramasubbu et al., 1998; Robinson, Starr, et al., 1983, 1985). Robinson and colleagues consistently included SES in their studies of PSD. In two of their studies, low SES was a significant risk factor for PSD (Paradiso & Robinson, 1998; Robinson, Starr, et al., 1983, 1985), while in three separate studies by this group, there was no association between low socioeconomic status and PSD (Fedoroff et al., 1991; Morris et al., 1990; Ramasubbu et al., 1998).

Ethnicity. There is a paucity of research on ethnicity and PSD. One study (Ramasubbu et al., 1998) reported on the influence of ethnicity, finding that Caucasian race was significantly associated with an increased risk of PSD. This study was conducted in hospitals in New York, Boston, Chicago, and Baltimore, and only 19% of the sample was Caucasian. Information on ethnicity of non-Caucasian participants, however, was not provided. More research is needed to examine the role of ethnicity in PSD.

Disease History

Prior stroke. A history of one or more previous strokes may be a risk factor for PSD. Among the four studies that reported on the role of previous stroke, two found no difference in history of previous stroke between those with and without PSD (Morris et al., 1990, 1992, 1995; Ramasubbu et al., 1998), and two showed an association between history of prior stroke and PSD (Eriksson et al., 2004; Hosking et al., 2000). In their survey of nearly 14,000 individuals 3 months after stroke, Eriksson and colleagues (2004) found a significant correlation between a history of a previous stroke and PSD.

Psychiatric history. The influence of a stroke patient's psychiatric history on the development of PSD presents dilemmas similar to those raised by history of previous stroke. Most researchers excluded stroke patients with a history of depression, but exclusion criteria varied among studies, ranging from the elimination of all patients with any psychiatric history to the exclusion of only those who were depressed at the time of the stroke. Nelson and colleagues (1993) took a unique approach in addressing this limitation by asking patients' significant

others to fill out an assessment of patients' mood and functioning twice: once for patients' premorbid state and once for the present time (2 weeks poststroke). Lack of uniform study populations among studies limits interpretation.

Five of the nine studies that reported on history of depression found a significant relationship between this variable and PSD (Astrom et al., 1993; Fedoroff et al., 1991; Herrmann et al., 1998; House et al., 1990a, 1990b, 1991; Morris et al., 1990, 1992, 1995; Paradiso & Robinson, 1998; Singh et al., 2000; Vataja et al., 2004). The operational definition of *history of depression* may have influenced whether a study found significant results regarding its role as a risk factor for PSD. Astrom and colleagues (1993) defined *history of depression* as prior admission to a psychiatric hospital, and they did not observe an association between psychiatric history and PSD. In contrast, another study defined a *positive history* as previous treatment for depression that could be confirmed by a family member of the patient (Gillen et al., 2001), and they found a significant relationship between a previous depression and the development of PSD.

Family history of psychiatric disorder. Relatively few studies have investigated the role of family history of psychiatric disorder in PSD. One study reported that family psychiatric history was not related to the development of PSD (Fedoroff et al., 1991), but the researchers did not state how the patient's family history was assessed. In contrast, Morris and colleagues (1990, 1992, 1995) assessed stroke patients' family histories with a semistructured psychiatric interview, directly asking about every first-degree relative and verifying results with a relative of the patient. These researchers found a significant relationship between family history of depression or anxiety disorder and PSD.

Comorbid disease. Patients who experience stroke often have comorbid illnesses, the most common being cardiovascular disease (Adams et al., 2003; AHA, 2005). Since heart disease has been linked with an increased incidence of depression (Carney & Freeland, 2003), stroke survivors with heart disease might be at higher risk for PSD. Several studies, however, found no association between comorbid heart disease and PSD (Hayee et al., 2001; Kotila et al., 1998; Nannetti et al., 2005; Tang et al., 2002; Vataja et al., 2004), while only one study showed a significant relationship (Ramasubbu et al., 1998). An insufficient amount of research exists at the present time regarding any possible interaction among stroke, heart disease, and depression.

Clinical Characteristics

Type of stroke. Some studies attempted to distinguish between ischemic stroke and hemorrhagic stroke (Fedoroff et al., 1991; Starkstein et al., 1989) or between ischemic stroke and all other strokes (Kotila et al., 1998; Nannetti et al., 2005; Spalletta et al., 2005), while many researchers limited their samples to ischemic stroke

patients (Aben et al., 2002, 2003; Angelleli et al., 2004; Berg et al., 2001; Carota et al., 2005; Kauhanen et al., 1999, 2000; Niedermaier et al., 2004; Palomaki et al., 1999; Robinson, Kubos, et al., 1984; Robinson, Starr, et al., 1983, 1985; Robinson, Starr, Lipsey, et al., 1984; Robinson, Starr, & Price, 1984; Starkstein, Robinson, & Price, 1988a; Vataja et al., 2004). Stroke subtypes studied included supratentorial versus infratentorial, lacunar (Bush, 1999), cardioembolic (Bush, 1999), and cortical versus subcortical (Astrom et al., 1993; Bolla-Wilson et al., 1989; Nys et al., 2005; Starkstein et al., 1987). Two studies included patients with subarachnoid hemorrhage (Hayee et al., 2001; House et al., 1990a, 1990b, 1991), while other studies specifically excluded patients with subarachnoid hemorrhage, subdural hematoma, or vertebrobasilar stroke (Herrmann et al., 1998; Morris et al., 1990, 1992, 1995; Singh et al., 2000; Tang et al., 2002).

Severity of neurological deficit. The severity of the stroke itself, defined as neurological impairment, may affect the risk of PSD. The majority of studies defined stroke severity as the patient's prognosis based on standardized clinical measures, such as the Scandinavian Stroke Scale (SSS; Scandinavian Stroke Study Group, 1985) and the National Institutes of Health Stroke Scale (NIHSS; Brott et al., 1989). The NIHSS has been shown to be a reliable predictor of patient outcome and has been correlated with infarct volume (Kelly-Hayes, 2004), but this scale was used in only one study in this review (Niedermaier et al., 2004).

Severity of the stroke was strongly associated with PSD,

Every study in this review analyzing stroke severity found increased severity to be a risk factor for PSD (Berg et al., 2001; Hayee et al., 2001; Herrmann et al., 1998; Kauhanen et al., 1999, 2000; Kotila et al., 1998, 1999; Niedermaier et al., 2004; Shimoda & Robinson, 1999; Vataja et al., 2004). Severity of the stroke was strongly associated with PSD when assessed in the initial weeks after stroke, as well as 3 months after stroke. In an open, randomized study of prophylactic antidepressant treatment poststroke (Niedermaier et al., 2004), the only two factors that predicted onset of PSD were nontreatment with antidepressant and degree of stroke severity.

Lesion volume. Nine studies examined lesion volume, as determined on neuroimaging. Eight studies found no significant association between lesion volume and PSD (Astrom et al., 1993; Herrmann et al., 1995; House et al., 1990a, 1990b, 1991; Ramasubbu et al., 1998; Singh et al., 2000; Sinyor, Amato, et al., 1986; Starkstein et al., 1987, 1989). This result contrasts with the finding that stroke severity, as measured by the NIHSS or SSS, was significantly related to PSD in every study that measured stroke severity. Thus, lesion volume and clinical state of

the patient may represent two different variables, with only the clinical severity of the stroke playing a role in PSD.

Lesion location. Research on the role of the stroke location in relation to PSD began with an early study by Robinson and colleagues (Robinson & Price, 1982; Robinson & Szetela, 1981). This research group conducted a subsequent 2-year longitudinal study, the results of which were reported in several articles (Lipsey, Spencer, Rabins, & Robinson, 1986; Parikh, Lipsey, Robinson, & Price, 1987, 1988; Parikh et al., 1990; Robinson, Bolduc, & Price, 1987; Robinson, Bolla-Wilson, et al., 1986; Robinson, Kubos, Starr, Rao, & Price, 1983, 1984; Robinson, Lipsey, Bolla-Wilson et al., 1985; Robinson, Lipsey, & Price, 1985; Robinson, Lipsey, et al., 1986; Robinson, Starr, & Price, 1984; Robinson, Starr, et al., 1983, 1985; Robinson, Starr, Lipsey, et al., 1984; Robinson, Starr, & Price, 1984; Starkstein, Robinson, Berthier, et al., 1988, 1989; Starkstein, Robinson, & Price, 1987, 1988a, 1988b). Findings included an association between anterior left-hemisphere lesion location and PSD, an association between posterior right-hemisphere lesion location and PSD (Robinson, Kubos, et al., 1984), and a correlation between severity of depression and proximity of the lesion to the frontal pole among those with left-hemisphere strokes (Robinson, Kubos, et al., 1983, 1984; Starkstein et al., 1987).

Although several other researchers confirmed an association between PSD and left-hemisphere or anterior lesions (Astrom et al., 1993; Gonzalez-Torrecillas et al., 1995; Nelson et al., 1993; Wade et al., 1987), others did not find any relationship between PSD and the location of stroke (Andersen et al., 1994; House et al., 1990a, 1990b, 1991; Sinyor, Amato, et al., 1986; Sinyor, Jacques, et al., 1986). Some researchers argued that the link between left-hemisphere and anterior lesion location and PSD observed by Robinson and colleagues may have been due to methodological limitations of these early studies (Gainotti et al., 1999; Yamaguchi, Kobayashi, Koide, & Tsunematsu, 1992). One limitation was the use of "arbitrary" anatomical measurements, such as anterior and posterior borders and poles (House, 1987b; Rao, 2000; Singh, Herrmann, & Black, 1998). Several researchers had difficulty determining whether the results were derived from a new cohort of stroke patients or from a group that had already been studied (Hackett, Yapa, Parag, & Anderson, 2005; Rao, 2000). More recent research has failed to find significant associations between PSD and specific lesion locations (Aben et al., 2002; 2003; Berg et al., 2001; Carota et al., 2005; Gillen et al., 2001; Nannetti et al., 2005; Niedermaier et al., 2004; Ramasubbu et al., 1998; Spalletta et al., 2005). A meta-analysis of studies on lesion location and PSD (Carson et al., 2000) found no effect of lesion location, and another meta-analysis (Yu et al., 2004) found only a weak correlation between depression and right-hemisphere lesion.

Researchers who attempted to pool the data from the numerous small studies on lesion location determined that conclusions were prohibited by the systematic exclusion of patients with aphasia and comprehension deficits (Carson et al., 2000; Singh et al., 1998; Yu et al., 2004). Singh and colleagues (1998) noted that the routine exclusion of these stroke patients could contribute to misleading conclusions about the relationship between lesion location and PSD, because a large proportion of these patients are likely to have left-hemisphere and more posterior lesions.

Poststroke Cognitive Impairment

Twenty of the 25 studies that examined the relationship between cognitive impairment and PSD reported a significant correlation between the two variables (Berg et al., 2001; Bolla-Wilson et al., 1989; Carota et al., 2005; Downhill & Robinson, 1994; Fedoroff et al., 1991; Gillen et al., 2001; Gonzalez-Torrecillas et al., 1995; Gottlieb et al., 2002; Herrmann et al., 1998; Hosking et al., 2000; House et al., 1990a, 1990b, 1991; Kauhanen et al., 1999, 2000; Morris et al., 1990, 1992, 1995; Nys et al., 2005; Robinson, Bolla-Wilson, et al., 1986; Robinson, Starr, et al., 1983, 1985; Robinson, Starr, Lipsey, et al., 1984; Robinson, Starr, & Price, 1984; Spalletta et al., 2005; Starkstein et al., 1988a; Vataja et al., 2004; Wade et al., 1987). One of the four studies that failed to find a relationship between PSD and cognitive impairment did find a significant association between aphasia and PSD (Astrom et al., 1993).

Aphasia

Of the eight studies that reported on the role of aphasia in PSD, four studies reported a significant relationship between the two variables (Astrom et al., 1993; Carota et al., 2005; Kauhanen et al., 1999; 2000; Palomaki et al., 1999). These four studies included methods designed to include stroke patients with impaired language. Astrom and colleagues (1993) included perspectives of staff and patients' significant others when assessing for PSD among "dysphasic patients." Palomaki and colleagues (1999) used a speech-and-language pathologist to assess aphasia. Four studies failed to find a significant relationship between aphasia and poststroke depression (Berg et al., 2001; Fedoroff et al., 1991; Morris et al., 1990, 1992, 1995; Nannetti et al., 2005). Fedoroff and colleagues (1991) excluded patients with even moderate cognitive impairment and failed to provide any information on the number of participants excluded. Although Berg and colleagues (2001) reported that aphasia was not related to PSD, they found that impaired verbal function, as assessed by a comprehensive neuropsychological battery, was significantly associated with PSD. In this study, those with severe aphasia, as well as patients who were drowsy or older than 70 years, were excluded. Kauhanen and colleagues (1999, 2000) administered a comprehensive neuropsychological battery to 101 stroke survivors, one-third of whom were aphasic. Seventy percent of

aphasic patients were depressed 3 months poststroke, and 62% were depressed 1 year poststroke. Every PSD study in this review excluded, at a minimum, stroke patients with severe global aphasia, and some studies excluded up to 75% of stroke survivors for reasons relating to language difficulties (Singh et al., 2000). Many have argued that stroke patients with aphasia are likely to be vulnerable to PSD (Spencer et al., 1997; Yu et al., 2004), yet, because they were so frequently excluded from study participation, no conclusions can be made about PSD among aphasic stroke survivors.

Physical Impairment Due to the Stroke

As many as half of all stroke survivors are left with *permanent physical disability* (Bamford, Sandercock, Dennis, Burn, & Warlow, 1991), defined as having difficulty or being dependent on others for the conduct of essential or personally meaningful activities of life (Fried, 2004). Twenty-four of the 30 studies reviewed in this paper that assessed physical disability found a significant association between physical disability and PSD (Aben et al., 2002, 2003; Carota et al., 2005; Eriksson et al., 2004; Fedoroff et al., 1991; Gillen et al., 2001; Gottlieb et al., 2002; Herrmann et al., 1998; Hosking et al., 2000; Kauhanen et al., 1999, 2000; Kotila et al., 1998, 1999; Morris et al., 1990, 1992, 1995; Nannetti et al., 2005; Nys et al., 2005; Paradiso & Robinson, 1998; Ramasubbu et al., 1998; Robinson et al., 1986; Robinson, Starr, et al., 1983, 1985; Robinson, Starr, Lipsey, et al., 1984; Robinson, Starr, & Price, 1984; Shimoda & Robinson, 1999; Singh et al., 2000; Sinyor, Amato, et al., 1986; Spalletta et al., 2005; Vataja et al., 2004; Wade et al., 1987; Weg & Kulk, 1999). The most common assessment of functioning used by PSD researchers is the Barthel Index (Mahoney & Barthel, 1965), a measure of basic activities of daily living. Another commonly administered instrument is the Functional Independence Measure (FIM™), which assesses activities of daily living (Kelly-Hayes, 2004).

Discussion

The research literature on PSD suggests that depression is common among individuals who have suffered a stroke. The incidence of PSD in the acute period (within 3 months) may range from fewer than 10% to more than 50% of stroke patients, depending on when, where, and how these individuals are evaluated for depression. The strongest predictors of PSD were a history of depression, an increased severity of stroke, and poststroke physical or cognitive impairment. Despite the exclusion of many stroke patients with a history of psychiatric disorder and cognitive and physical impairment, these factors still emerged as strong predictors of PSD.

Female gender does not appear to be a risk factor for PSD. Ten studies demonstrated that female gender is associated with PSD, while 13 studies found no relationship between gender and depression. Age does not

appear to be a risk factor for depression in the acute period following a stroke; the majority of studies found no difference in the age of those who did and did not develop PSD. Further research is needed regarding the role of socioeconomic status, ethnicity, social support, prior stroke, comorbid disease, and family history of psychiatric disorder.

The majority of studies found little or no relationship between the location of the stroke and PSD (see Carson et al., 2000, and Yu et al., 2004, for meta-analyses of studies of lesion location). Upon the basis of the available evidence, lesion location is not a risk factor for PSD. No conclusions can be made about the relationship between the type of stroke and risk for PSD because of inconsistent stroke type and subtype definitions used in research. Lesion volume was not related to PSD and should not be used as an indicator of clinical severity of the stroke for the purpose of screening for or identifying PSD. Stroke survivors with aphasia may be at high risk for PSD, and more research is needed in this population.

The strongest predictors of PSD were a history of depression, an increased severity of stroke, and poststroke physical or cognitive impairment.

Limitations

The major limitations of this review were methodological. The definitions of *stroke* and *depression* varied among PSD studies, making it difficult to compare results. PSD research was also characterized by widely varying time intervals between the occurrence of stroke and the assessment for depression, contributing to disparity in the incidence rates of PSD. The systematic exclusion of aphasic stroke patients from research evaluating risk for PSD was another limitation. The decision to include in this review studies done outside the United States may detract from the ability to generalize the findings to stroke patients within the United States. International studies were included in this review because the majority of rigorous research in the field of poststroke depression was conducted in other nations.

When interpreting the results of research in the United States or abroad, cultural influences must be considered. For example, a large portion of PSD research in the United States was conducted in one of two populations: a mostly African American, lower socioeconomic status population from Baltimore, MD (Bolla-Wilson et al., 1989; Downhill & Robinson, 1994; Fedoroff et al., 1991; Lipsey et al., 1986; Morris, Robinson, & Samuels, 1993; Paradiso & Robinson, 1998; Robinson, Starr, et al., 1983, 1985; Robinson, Starr, Lipsey, et al., 1984; Robinson, Starr, & Price, 1984), and a predominantly white, higher socioeconomic status group from Iowa (Robinson et al., 2000; Shimoda & Robinson, 1999). The values, behaviors, and beliefs of

these groups, especially those related to health and illness, may have affected research results. Possibly the most representative sample of stroke patients in the United States was recruited for the Stroke Data Bank Study (Foulkes, Wolf, Price, Mohr, & Hier, 1988), which was initiated in 1978 by the National Institutes of Neurological and Communicative Disorders and Stroke. It included 1,805 stroke patients recruited from major medical centers in Boston, New York, Chicago, and Baltimore.

Another methodological limitation of this review involves the use of computerized database and ancestry searches. Because of the large volume of research on PSD, additional search techniques, such as Internet searching and manual searching of journals, were not used. Alternative methods might have uncovered additional findings.

Clinical Implications

Stroke is an acute and often life-threatening event that represents a new diagnosis and a chronic illness requiring life-long management. Nurses provide direct full-time care for hospitalized stroke patients and their families and therefore are in a unique position to identify some of the subtle symptoms of PSD.

PSD is associated with an increased mortality rate (House et al., 2001; Jorge et al., 2003; Morris, Robinson, Andrzejewski, Samuels, & Price, 1993; for review, see Ranga Rama Krishnan, 2000), independent of age, gender, social class, type of stroke, lesion location, level of social functioning (Morris, Robinson, & Samuels, 1993), ethnicity, education, alcohol consumption, smoking, body mass index, hypertension, or diabetes (Everson et al., 1998). In one study (Jorge et al., 2003), treatment of poststroke patients with an antidepressant reduced poststroke mortality at 9-year follow-up. Therefore, identification and subsequent treatment of PSD should be a priority in the acute poststroke period.

Because stroke patients are particularly vulnerable to emotional, cognitive, and physical impairments that may prevent them from exhibiting depression in the same manner as healthy people (Nelson et al., 1993), an increased level of suspicion is required when caring for them. Risk factors for PSD identified in this review are increased severity of stroke, history of depression, and cognitive or physical impairment. In the acute care setting, nurses can identify stroke patients who may be at high risk for PSD, such as an elderly woman who was treated for major depressive disorder in the past or a man recovering from a stroke that had resulted in hemiparesis and aphasia. Standardized and validated tools to document the severity of neurological impairments are already in use (Kelly-Hayes, 2004); application of these measures to PSD screening could be adapted for clinical nursing practice. Advanced practice nurses working with stroke survivors and their families can institute an integrated plan of care, which can include treating or

preventing depression in high-risk patients (Popovich, Fox, & Burns, 2002; Shoemaker, 2001). Nurses can then coordinate care to maximize resources available to stroke patients upon their discharge from the hospital. Treatment of PSD can prevent or reverse many of its associated negative outcomes (Gonzalez-Torrecillas et al., 1995; Narushima & Robinson, 2003).

Recommendations for Future Studies

Future research should be directed at the prevention and treatment of PSD, focusing on interventions that decrease the high rates of morbidity and mortality associated with this condition. The development of a consensus assessment method for depression would facilitate comparison among PSD studies. Findings from studies with large sample sizes conducted in other nations must be replicated in the United States. Research regarding the role of ethnicity in risk for PSD is needed, because the rate of stroke among African American and Hispanic individuals is approximately twice the rate among Caucasians (Zivin, 2004). Future PSD research should include stroke patients with aphasia as well as patients with a history of depression. These individuals may be at high risk, yet they have been excluded from most current PSD research. Instruments that rely on verbal responses from patients are likely to significantly underestimate the incidence of depression among patients with aphasia. Studying aphasic stroke survivors may present challenges in the diagnosis of depression and will require new research designs.

Depression is common during the first few months after stroke and is associated with significant morbidity and mortality. Risk factors for the development of PSD identified in this review include history of previous depression, increased severity of stroke, and cognitive and physical impairment. Nurses and other healthcare providers can play an important role in preventing, identifying, and treating PSD.

References

- Aben, I., Denollet, J., Lousberg, R., Verhey, F., Wojciechowski, F., & Honig, A. (2002). Personality and vulnerability to depression in stroke patients: A one-year prospective follow-up study. *Stroke*, *33*, 2391-2395.
- Aben, I., Verhey, F., Honig, A., Lodder, J., Lousberg, R., & Maes, M. (2001). Research into the specificity of depression after stroke: A review of an unresolved issue. *Progress in Neuro-Psychopharmacology and Biological Psychiatry*, *25*, 671-689.
- Aben, I., Verhey, F., Strik, J., Lousberg, R., Lodder, J., & Honig, A. (2003). A comparative study into the one year cumulative incidence of depression after stroke and myocardial infarction. *Journal of Neurology, Neurosurgery, and Psychiatry*, *74*, 581-585.
- Adams, R. J., Chimowitz, M. I., Alpert, J. S., Awad, I. A., Cerqueria, M. D., Fayad, P., et al. (2003). Coronary risk evaluation in patients with transient ischemic attack and ischemic stroke: A scientific statement for healthcare professionals from the Stroke Council and the Council on Clinical Cardiology of the American Heart Association/American Stroke Association. *Stroke*, *34*, 2310-2322.
- American Heart Association. (2005). *Heart disease and stroke statistics: 2005 update*. Dallas: Author.

- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text revision). Washington, DC: Author.
- Andersen, G. (1997). Poststroke depression. *European Psychiatry, 12*(Suppl. 3), 255–260.
- Andersen, G., Vestergaard, K., Riis, J. O., & Lauritzen, L. (1994). Incidence of poststroke depression during the first year in a large unselected stroke population determined using a valid standardized rating scale. *Acta Psychiatrica Scandinavica, 90*, 190–195.
- Anderson, C. S., Hackett, M. L., & House, A. O. (2004). Interventions for preventing depression after stroke. *Cochrane Database of Systematic Reviews, 2*, CD003689.
- Angelelli, P., Paolucci, S., Bivona, U., Piccardi, L., Ciurli, P., Cantagallo, A., et al. (2004). Development of neuropsychiatric symptoms in poststroke patients: A cross sectional study. *Acta Psychiatrica Scandinavica, 110*, 55–63.
- Astrom, M., Adolfsson, R., & Asplund, K. (1993). Major depression in stroke patients: A three-year longitudinal study. *Stroke, 24*, 976–982.
- Bamford, J., Sandercock, P. A. G., Dennis, M. S., Burn, J., & Warlow, C. P. (1991). Classification and natural history of clinically identifiable subtypes of cerebral infarction. *Lancet, 337*, 1521–1526.
- Berg, A., Palomaki, H., Lehtihalmes, M., Lonnqvist, J., & Kaste, M. (2001). Poststroke depression in acute phase after stroke. *Cerebrovascular Diseases, 12*, 14–20.
- Bolla-Wilson, K., Robinson, R. G., Starkstein, S. E., Boston, J., & Price, T. R. (1989). Lateralization of dementia of depression in stroke patients. *American Journal of Psychiatry, 146*, 627–34.
- Boland, R. J., & Keller, M. B. (2002). Course and outcome of depression. In I. H. Gotlib & C. L. Hammen (Eds.), *Handbook of depression*. New York: Guilford Press.
- Brott, T., Adams, H. P., Jr., Olinger, C. P., Marler, J. R., Barsan, W. G., Biller, J., et al. (1989). Measurements of acute cerebral infarction: A clinical examination scale. *Stroke, 20*, 864–870.
- Bush, B. (1999). Major life events as risk factors for poststroke depression. *Brain Injury, 13*, 131–137.
- Carney, R. M., & Freeland, K. E. (2003). Depression, mortality, and medical morbidity in patients with coronary heart disease. *Biological Psychiatry, 54*, 241–247.
- Carota, A., Berney, A., Aybek, S., Iaria, G., Staub, F., Ghika-Schmid, F., et al. (2005). A prospective study of predictors of poststroke depression. *Neurology, 64*, 428–433.
- Carson, A. J., MacHale, S., Allen, K., Lawrie, S. M., Dennis, M., House, A., et al. (2000). Depression after stroke and lesion location: A systematic review. *Lancet, 356*, 122–126.
- Chemerinski, E., Robinson, R. G., Arndt, S., & Kosier, J. T. (2001). The effect of remission of poststroke depression on activities of daily living in a double-blind randomized treatment study. *Journal of Nervous and Mental Disease, 189*, 421–425.
- Downhill, J. E., Jr., & Robinson, R. G. (1994). Longitudinal assessment of depression and cognitive impairment following stroke. *Journal of Nervous and Mental Disease, 182*, 425–431.
- Eriksson, M., Asplund, K., Glader, E. L., Norrving, B., Stegmayr, B., Terent, A., et al. (2004). Self-reported depression and use of antidepressants after stroke: A national survey. *Stroke, 35*, 936–941.
- Everson, S. A., Roberts, R. E., Goldberg, D. E., & Kaplan, G. A. (1998). Depressive symptoms and increased risk of stroke mortality over a 29-year period. *Archives of Internal Medicine, 158*, 1133–1138.
- Fedoroff, J., Starkstein, S., Parikh, R., Price, T., & Robinson, R. (1991). Are depressive symptoms nonspecific in patients with acute stroke? *American Journal of Psychiatry, 148*, 1172–1176.
- Foulkes, M. A., Wolf, P. A., Price, T. R., Mohr, J. P., & Hier, D. B. (1988). The Stroke Data Bank Study: Design, methods, and baseline characteristics. *Stroke, 19*, 547–554.
- Fried, L. P. (2004). Epidemiology of aging: Implications of the aging society. In L. Goldman & D. Ausiello (Eds.), *Cecil textbook of medicine* (22nd ed., pp. 100–103). Philadelphia: Saunders.
- Gainotti, G., Azzoni, A., & Marra, C. (1999). Frequency, phenomenology, and anatomical-clinical correlates of major poststroke depression. *British Journal of Psychiatry, 175*, 163–167.
- Ghika-Schmid, F., van Melle, G., Guex, P., & Bogousslavsky, J. (1999). Subjective experience and behavior in acute stroke. *Neurology, 52*, 22–28.
- Gillen, R., Tennen, H., McKee, T. E., Gernert-Dott, P., & Affleck, G. (2001). Depressive symptoms and history of depression predict rehabilitation efficiency in stroke patients. *Archives of Physical Medicine Rehabilitation, 82*, 1645–1649.
- Glader, E. L., Stegmayr, B., Norrving, B., Terent, A., Hulter-Asberg, K., Wester, P. O., et al. (2003). Sex differences in management and outcome after stroke: A Swedish national perspective. *Stroke, 34*, 1970–1975.
- Gonzales-Torrecillas, J., Mendlewicz, J., & Lobo, A. (1995). Effects of early treatment of poststroke depression on neuropsychological rehabilitation. *International Psychogeriatrics, 7*, 547–560.
- Gotlib, I. H., & Hammen, C. L. (2002). *Handbook of depression*. New York: Guilford Press.
- Gottlieb, D., Salagnik, I., Kipnis, M., & Brill, S. (2002). Post stroke depression, first year post stroke, in middle band patients. *International Journal of Geriatric Psychiatry, 17*, 486–487.
- Hackett, M. L., Anderson, C. S., & House, A. O. (2004). Interventions for treating depression after stroke. *Cochrane Database of Systematic Reviews, 3*, CD003437.
- Hackett, M. L., Yapa, C., Parag, V., & Anderson, C. S. (2005). Frequency of depression after stroke: A systematic review of observational studies. *Stroke, 36*, 1330–1340.
- Hayee, M. A., Akhtar, N., Hague, A., & Rabbani, M.G. (2001). Depression after stroke: Analysis of 297 stroke patients. *Bangladesh Medical Research Council Bulletin, 27*, 96–102.
- Herrmann, M., Bartels, C., Schumacher, M., & Wallesch, C. W. (1995). Poststroke depression: Is there a pathoanatomic correlate for depression in the post-acute stage of stroke? *Stroke, 26*, 850–856.
- Herrmann, N., Black, S. E., Lawrence, J., Szekely, C., & Szalai, J. P. (1998). The Sunnybrook Stroke Study: A prospective study of depressive symptoms and functional outcomes. *Stroke, 29*, 618–624.
- Hickey, J. V., & Hock, N. H. (2003). Stroke and other cerebrovascular diseases. In J. V. Hickey (Ed.), *Clinical practice of neurological and neurosurgical nursing* (5th ed., pp. 559–587). Philadelphia: Lippincott, Williams & Wilkins.
- Hosking, S. G., Marsh, N. V., & Friedman, P. J. (2000). Depression at three months poststroke in the elderly: Predictors and indicators of prevalence. *Aging, Neuropsychology, and Cognition, 7*, 205–216.
- House, A. (1987a). Depression after stroke. *British Medical Journal, 294*, 76–78.
- House, A. (1987b). Mood disorders after stroke: A review of the evidence. *International Journal of Geriatric Psychiatry, 2*, 211–221.
- House, A., Dennis, M., Mogridge, L., Warlow, C., Hawton, K., & Jones, L. (1991). Mood disorders in the year after first stroke. *British Journal of Psychiatry, 158*, 83–92.
- House, A., Dennis, M., Warlow, C., Hawton, K., & Molyneux, A. (1990a). Mood disorders and their relation to lesion location: A CT study. *Brain, 113*, 1113–1129.
- House, A., Dennis, M., Warlow, C., Hawton, K., & Molyneux, A. (1990b). The relationship between intellectual impairment and mood disorder in the first year after stroke. *Psychological Medicine, 20*, 805–814.
- House, A., Knapp, P., Bamford, J., & Vail, A. (2001). Mortality at 12 and 24 months after stroke may be associated with depressive symptoms at 1 month. *Stroke, 32*, 696–701.

- Jorge, R. E., Robinson, R. G., Arndt, S., & Starkstein, S. (2003). Mortality and poststroke depression: A placebo-controlled trial of antidepressants. *American Journal of Psychiatry*, *160*, 1823–1829.
- Kauhanen, M. L., Korpelainen, J. T., Hiltunen, P., Brusin, E., Mononen, H., Maatta, R., et al. (1999). Poststroke depression correlates with cognitive impairment and neurological deficits. *Stroke*, *30*, 1875–1880.
- Kauhanen, M. L., Korpelainen, J. T., Hiltunen, P., Nieminen, P., Sotaniemi, K. A., & Myllylä, V. V. (2000). Aphasia, depression, and non-verbal cognitive impairment in ischaemic stroke. *Cerebrovascular Diseases*, *10*, 455–461.
- Kelly-Hayes, M. (2004). Stroke outcome measures. *Journal of Cardiovascular Nursing*, *19*, 301–307.
- Kessler, R. C. (2002). Epidemiology of depression. In I. H. Gotlib & C. L. Hammen (Eds.), *Handbook of Depression*. New York: Guilford Press.
- King, R. B. (1996). Quality of life after stroke. *Stroke*, *27*, 1467–1472.
- Kotila, M., Numminen, H., Waltimo, O., & Kaste, M. (1998). Depression after stroke: Results of the FINNSTROKE Study. *Stroke*, *29*, 368–372.
- Kotila, M., Numminen, H., Waltimo, O., & Kaste, M. (1999). Poststroke depression and functional recovery in a population-based stroke register: The Finnstroke Study. *European Journal of Neurology*, *6*, 309–312.
- Lipsey, J. R., Spencer, W. C., Rabins, P. V., & Robinson, R. G. (1986). Phenomenological comparison of poststroke depression and functional depression. *American Journal of Psychiatry*, *143*, 527–529.
- Mahoney, F. I., & Barthel, D. W. (1965). Functional evaluation: The Barthel Index. *Maryland State Medical Journal*, *14*, 61–65.
- Morris, P. L., & Robinson, R. G. (1995). Personality neuroticism and depression after stroke. *International Journal of Psychiatry in Medicine*, *25*, 93–102.
- Morris, P. L., Robinson, R. G., Andrzejewski, P., Samuels, J., & Price, T. R. (1993). Association of depression with 10-year poststroke mortality. *American Journal of Psychiatry*, *150*, 124–129.
- Morris, P. L., Robinson, R. G., de Carvalho, M. L., Albert, P., Wells, J. C., Samuels, J. F., et al. (1996). Lesion characteristics and depressed mood in the Stroke Data Bank Study. *Journal of Neuropsychiatry and Clinical Neuroscience*, *8*, 153–159.
- Morris, P. L., Robinson, R. G., & Raphael, B. (1990). Prevalence and course of depressive disorders in hospitalized stroke patients. *International Journal of Psychiatry in Medicine*, *20*, 349–364.
- Morris, P. L., Robinson, R. G., Raphael, B., & Hopwood, M. J. (1996). Lesion location and poststroke depression. *Journal of Neuropsychiatry and Clinical Neuroscience*, *8*, 399–403.
- Morris, P. L., Robinson, R. G., Raphael, B., Samuels, J., & Molloy, P. (1992). The relationship between risk factors for affective disorder and poststroke depression in hospitalized stroke patients. *Australian and New Zealand Journal of Psychiatry*, *26*, 208–217.
- Morris, P. L., Robinson, R. G., & Samuels, J. (1993). Depression, introversion and mortality following stroke. *Australian and New Zealand Journal of Psychiatry*, *27*, 443–449.
- Murata, Y., Kimura, M., & Robinson, R. G. (2000). Does cognitive impairment cause poststroke depression? *American Journal of Geriatric Psychiatry*, *8*, 310–317.
- Nannetti, L., Paci, M., Pasquini, J., Lombardi, B., & Taiti, P. G. (2005). Motor and functional recovery in patients with poststroke depression. *Disability and Rehabilitation*, *27*, 170–175.
- Narushima, K., Chan, K. L., Kosier, J. T., & Robinson, R. G. (2003). Does cognitive recovery after treatment of poststroke depression last? A two-year follow-up of cognitive function associated with poststroke depression. *American Journal of Psychiatry*, *160*, 1157–1162.
- Narushima, K., & Robinson, R. G. (2003). The effect of early versus late antidepressant treatment on physical impairment associated with poststroke depression: Is there a time-related therapeutic window? *Journal of Nervous and Mental Disease*, *191*, 645–652.
- National Institute of Mental Health. (2005). The numbers count: Mental disorders in America. Retrieved June 28, 2005, from www.nimh.nih.gov/publicat/numbers.cfm.
- Nelson, L. D., Cicchetti, D., Satz, P., Stern, S., Sowa, M., Cohen, S., et al. (1993). Emotional sequelae of stroke. *Neuropsychology*, *7*, 553–560.
- Niedermaier, N., Bohrer, E., Schulte, K., Schlattmann, P., & Heuser, I. (2004). Prevention and treatment of poststroke depression with mirtazapine in patients with acute stroke. *Journal of Clinical Psychiatry*, *65*, 1619–1623.
- Nys, G. M. S., van Zandvoort, M. J. E., van der Worp, H. B., de Haan, E. H. F., de Kort, P. L. M., & Kappelle, L. J. (2005). Early depressive symptoms after stroke: Neuropsychological correlates and lesion characteristics. *Journal of the Neurological Sciences*, *228*, 27–33.
- Palomaki, H., Kaste, M., Berg, A., Lonnqvist, R., Lonnqvist, J., Lehtihalmes, M., et al. (1999). Prevention of poststroke depression: One-year randomized placebo controlled double blind trial of mianserin with six month follow-up after stroke. *Journal of Neurology, Neurosurgery, and Psychiatry*, *66*, 490–494.
- Paradiso, S., & Robinson, R. (1998). Gender differences in poststroke depression. *Journal of Neuropsychiatry and Clinical Neuroscience*, *10*, 41–47.
- Parikh, R. M., Lipsey, J. R., Robinson, R. G., & Price, T. R. (1987). Two-year longitudinal study of poststroke mood disorders: Dynamic changes in correlates of depression at one and two years. *Stroke*, *18*, 579–584.
- Parikh, R. M., Lipsey, J. R., Robinson, R. G., & Price, T. R. (1988). A two-year longitudinal study of poststroke mood disorders: Prognostic factors related to one and two year outcome. *International Journal of Psychiatry in Medicine*, *18*, 45–56.
- Parikh, R. M., Robinson, R. G., Lipsey, J. R., Starkstein, S. E., Fedoroff, J. P., & Price, T. R. (1990). The impact of poststroke depression on recovery in activities of daily living over a two-year follow-up. *Archives of Neurology*, *47*, 785–789.
- Pasacreta, J. V., Minarik, P. A., & Nield-Anderson, L. (2000). Anxiety and depression. In B. Ferrell & N. Coyle (Eds.), *Textbook of palliative nursing*. New York: Oxford Publishing Company.
- Popovich, J. M., Fox, P. G., & Burns, K. R. (2002). The impact of depression on stroke recovery in the U.S. *International Journal of Psychiatric Nursing Research*, *7*, 842–855.
- Ranga Rama Krishnan, K. (2000). Depression as a contributing factor in cerebrovascular disease. *American Heart Journal*, *140*(Suppl. 4), 70–76.
- Ramasubbu, R., Robinson, R., Flint, A., Kosier, T., & Price, T. R. (1998). Functional impairment associated with acute poststroke depression: The Stroke Data Bank Study. *Journal of Neuropsychiatry and Clinical Neuroscience*, *10*, 26–33.
- Rao, R. (2000). Cerebrovascular disease and late life depression: An age old association revisited. *International Journal of Geriatric Psychiatry*, *15*, 419–433.
- Robinson, R. G., Bolduc, P. L., & Price, T. R. (1987). Two-year longitudinal study of poststroke mood disorders: Diagnosis and outcome at one and two years. *Stroke*, *18*, 837–843.
- Robinson, R. G., Bolla-Wilson, K., Kaplan, E., Lipsey, J. R., & Price, T. R. (1986). Depression influences intellectual impairment in stroke patients. *British Journal of Psychiatry*, *148*, 541–547.
- Robinson, R. G., Kubos, K. L., Starr, L., Rao, K., & Price, T. R. (1984). Mood disorders in stroke patients: Importance of lesion location. *Brain*, *107*, 81–93.
- Robinson, R. G., Kubos, K. L., Starr, L. B., Rao, K., & Price, T. R. (1983). Mood changes in stroke patients: Relationship to lesion location. *Comprehensive Psychiatry*, *24*, 555–566.
- Robinson, R. G., Lipsey, J. R., Bolla-Wilson, K., Bolduc, P. L., Pearlson, G. D., Rao, K., et al. (1985). Mood disorders in left-handed stroke patients. *American Journal of Psychiatry*, *142*, 1424–1429.
- Robinson, R. G., Lipsey, J. R., & Price, T. R. (1985). Diagnosis and clinical management of poststroke depression. *Psychosomatics*, *26*, 769–772, 775–778.
- Robinson, R. G., Lipsey, J. R., Rao, K., & Price, T. R. (1986). Two-year longitudinal study of poststroke mood disorders: Comparison of acute-onset with delayed-onset depression. *American Journal of Psychiatry*, *143*, 1238–1244.

- Robinson, R. G., & Price, T. R. (1982). Poststroke depressive disorders: A follow-up study of 103 patients. *Stroke*, *13*, 635–641.
- Robinson, R. G., Schultz, S. K., Castillo, C., Kopel, T., Kosier, J. T., Newman, R. M., et al. (2000). Nortriptyline versus fluoxetine in the treatment of depression and in short-term recovery after stroke: A placebo-controlled, double-blind study. *American Journal of Psychiatry*, *157*, 351–359.
- Robinson, R. G., Starr, L. B., Kubos, K. L., & Price, T. R. (1983). A two-year longitudinal study of poststroke mood disorders: Findings during the initial evaluation. *Stroke*, *14*, 736–741.
- Robinson, R. G., Starr, L. B., Lipsey, J. R., Rao, K., & Price, T. R. (1984). A two-year longitudinal study of poststroke mood disorders: Dynamic changes in associated variables over the first six months of follow-up. *Stroke*, *15*, 510–517.
- Robinson, R. G., Starr, L. B., Lipsey, J. R., Rao, K., & Price, T. R. (1985). A two-year longitudinal study of poststroke mood disorders: In-hospital prognostic factors associated with six-month outcome. *Journal of Nervous & Mental Disease*, *173*, 221–226.
- Robinson, R. G., Starr, L. B., & Price, T. R. (1984). A two-year longitudinal study of mood disorders following stroke: Prevalence and duration at six months follow-up. *British Journal of Psychiatry*, *144*, 256–262.
- Robinson, R. G., & Szetela, B. (1981). Mood change following left hemispheric brain injury. *Annals of Neurology*, *9*, 447–453.
- Robinson-Smith, G., Johnston, M. V., & Allen, J. (2000). Self-care self-efficacy, quality of life, and depression after stroke. *Archives of Physical Medicine and Rehabilitation*, *81*, 460–464.
- Rouchell, A. M., Pounds, R., & Tierney, J. G. (2002). Depression. In M. G. Wise & J. R. Rundell (Eds.), *The American Psychiatric Publishing textbook of consultation-liaison psychiatry: Psychiatry in the medically ill* (2nd ed.). Washington, D.C.: American Psychiatric Publishing.
- Scandinavian Stroke Study Group. (1985). Multicenter trial of hemodilution in ischemic stroke: Background and study protocol. *Stroke*, *16*, 885–890.
- Shimoda, K., & Robinson, R. G. (1999). The relationship between poststroke depression and lesion location in long-term follow-up. *Biological Psychiatry*, *45*, 187–192.
- Shoemaker, N. (2001). Depression and stroke: Improving patient outcomes with a team approach. *Caring*, *20*, 6–8, 10, 12.
- Singh, A., Black, S. E., Herrmann, N., Leibovitch, F. S., Ebert, P. L., Lawrence, J., et al. (2000). Functional and neuroanatomical correlations in poststroke depression: The Sunnybrook Stroke Study. *Stroke*, *31*, 637–644.
- Singh, A., Hermann, N., & Black, S. E. (1998). The importance of lesion location in poststroke depression: A critical review. *Canadian Journal of Psychiatry*, *43*, 921–927.
- Sinyor, D., Amato, P., Kaloupek, D. G., Becker, R., Goldenberg, M., & Coopersmith, H. (1986). Poststroke depression: Relationships to functional impairment, coping strategies, and rehabilitation outcomes. *Stroke*, *17*, 1102–1107.
- Sinyor, D., Jacques, P., Kaloupek, D. G., Becker, R., Goldenberg, M., & Coopersmith, H. (1986). Poststroke depression and lesion location: An attempted replication. *Brain*, *109*, 537–546.
- Spalletta, G., Ripa, A., & Caltagirone, C. (2005). Symptom profile of DSM-IV major and minor depressive disorders in first-ever stroke patients. *American Journal of Geriatric Psychiatry*, *13*, 108–115.
- Spencer, K. A., Tompkins, C. A., & Schultz, R. (1997). Assessment of depression in patients with brain pathology: The case of stroke. *Psychological Bulletin*, *122*, 132–152.
- Starkstein, S. E., Robinson, R. G., Berthier, M. L., Parikh, R. M., & Price, T. R. (1988). Differential mood changes following basal ganglia versus thalamic lesions. *Archives of Neurology*, *45*, 725–730.
- Starkstein, S. E., Robinson, R. G., Honig, M. A., Parikh, R. M., Joselyn, J., & Price, T. R. (1989). Mood changes after right-hemisphere lesions. *British Journal of Psychiatry*, *155*, 79–85.
- Starkstein, S. E., Robinson, R. G., & Price, T. R. (1987). Comparison of cortical and subcortical lesions in the production of poststroke mood disorders. *Brain*, *110*, 1045–1059.
- Starkstein, S. E., Robinson, R. G., & Price, T. R. (1988a). Comparison of patients with and without poststroke major depression matched for size and location of lesion. *Archives of General Psychiatry*, *45*, 247–252.
- Starkstein, S. E., Robinson, R. G., & Price, T. R. (1988b). Comparison of spontaneously recovered versus nonrecovered patients with poststroke depression. *Stroke*, *9*, 1491–1496.
- Sturm, J. W., Donnan, G. A., Dewey, H. M., Macdonnell, R. A., Gilligan, A. K., Srikanth, V., et al. (2004). Quality of life after stroke: The North East Melbourne Stroke Incidence Study (NEMESIS). *Stroke*, *35*, 2340–2345.
- Tang, W., Ungvari, G. S., Chiu, H. F. K., Sze, K., Woo, J., & Kay, R. (2002). Psychiatric morbidity in first time stroke patients in Hong Kong: A pilot study in a rehabilitation unit. *Australian and New Zealand Journal of Psychiatry*, *36*, 544–549.
- Vataja, R., Leppavuori, A., Pohjasvaara, T., Mantyla, R., Aronen, H. J., Salonen, O., et al. (2004). Poststroke depression and lesion location revisited. *The Journal of Neuropsychiatry and Clinical Neurosciences*, *16*, 156–162.
- Wade, D. T., Legh-Smith, J., & Hewer, R. A. (1987). Depressed mood after stroke: A community study of its frequency. *The British Journal of Psychiatry*, *151*, 200–205.
- Weg, F., & Kulk, D. (1999). Poststroke depression and functional outcome: A cohort study investigating the influence of depression on functional recovery from stroke. *Clinical Rehabilitation*, *13*, 268–272.
- Whyte, E. M., & Mulsant, B. H. (2002). Poststroke depression: Epidemiology, pathophysiology, and biological treatment. *Biological Psychiatry*, *52*, 253–264.
- World Health Organization (1990). *International Classification of Diseases: Revision 10*. Geneva, Switzerland: Author.
- Yamaguchi, S., Kobayashi, S., Koide, H., & Tsunematsu, T. (1992). Longitudinal study of regional cerebral blood flow changes in depression after stroke. *Stroke*, *23*, 1716–1722.
- Yu, L., Liu, C. K., Chen, J. W., Wand, S. Y., Wu, Y. H., & Yu, S. H. (2004). Relationship between poststroke depression and lesion location: A meta-analysis. *Kaohsiung Journal of Medical Sciences*, *20*, 372–380.
- Zivin, J. A. (2004). Approach to cerebrovascular diseases. In L. Goldman & D. Ausiello (Eds.), *Cecil Textbook of Medicine* (22nd ed.; pp. 2280–2287). Philadelphia: Saunders.

Continuing Education Credit

Journal of Neuroscience Nursing is pleased to offer the opportunity to earn neuroscience nursing contact hours for this article online. Go to www.aann.org, and select "Continuing Education." There you can read the article again or go directly to the posttest assessment. The cost is \$15 for each article. You will be asked for a credit card or online payment service number.

The posttest consists of 10 questions based on the article, plus several assessment questions (e.g., How long did it take you to read the article and complete the posttest?). A passing score of 80% (8 of 10 questions correct) on the posttest and completion of the assessment questions yields 1 nursing contact hour for each article.

The American Association of Neuroscience Nurses is accredited as a provider of continuing nursing education by the American Nurses Credentialing Center's Commission on Accreditation.