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Language as a Stressor in Aphasia

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Abstract

Background—Persons with aphasia often report feeling anxious when using language while communicating. While many patients, caregivers, clinicians and researchers would agree that language may be a stressor for persons with aphasia, systematic empirical studies of stress and/or anxiety in aphasia remain scarce.

Aim—The aim of this paper is to review the existing literature discussing language as a stressor in aphasia, identify key issues, highlight important gaps, and propose a program for future study. In doing so, we hope to underscore the importance of understanding aspects of the emotional aftermath of aphasia, which plays a critical role in the process of recovery and rehabilitation.

Main Contribution—Post stroke emotional dysregulation in persons with chronic aphasia clearly has adverse effects for language performance and prospects of recovery. However, the specific role anxiety might play in aphasia has yet to be determined. As a starting point, we propose to view language in aphasia as a stressor, linked to an emotional state we term “linguistic anxiety.” Specifically, a person with linguistic anxiety is one in whom the deliberate, effortful production of language involves anticipation of an error, with the imminence of linguistic failure serving as the threat. Since anticipation is psychologically linked to anxiety and also plays an

important role in the allostatic system, we suggest that examining physiologic stress responses in persons with aphasia when they are asked to perform a linguistic task would be a productive tool for assessing the potential relation of stress to “linguistic anxiety.”

Conclusion—Exploring the putative relationship between anxiety and language in aphasia, through the study of physiologic stress responses, could establish a platform for investigating language changes in the brain in other clinical populations, such as in individuals with Alzheimer’s disease or persons with post traumatic stress disorder, or even with healthy aging persons, in whom “linguistic anxiety” might be at work when they have trouble finding words.

Introduction

Persons with aphasia often report feeling anxious when using language while communicating. The experience of language as a stressor is described in the excerpt below, written by a woman who acquired aphasia as a young adult:

... the net effect of this behavioral impairment is that the aphasic is especially conscious of and deliberate in language production ... speech is a matter of special concern, anxiety, and labor. The aphasic always has to worry about what to say, how to say it, and how to keep command of that which has already been planned... the aphasic must deliberately choose words and then attend to planning how to form, arrange, and speak them. (Newborn, 1998:196)

By her account, the compromised linguistic skills in aphasia force patients into a state of extreme concern about the clarity and accuracy of their verbal communication, even in the face of the simplest verbal exchange. It could be argued, then, that persons with aphasia are preoccupied with the increased risk of breakdown in communication as a result of the language disorder, which results in a state of “linguistic anxiety.” We contend that the cumulative stress associated with language use that brings about a state of anxiety in (some) persons with aphasia, where anxiety is taken to be “a state of self-preoccupation in which evaluation of one’s (inadequate) capabilities to deal with the threat is prominent. Accompanying this negative affective state is a strong physiological or somatic component... This somatic state may be the physiological substrate of “readiness,” which may underlie a state of preparation to counteract helplessness” (Barlow, 2004:64). While we construe stress and anxiety as two related but independent constructs, we found the use of these terms in the literature to be, at times, inconsistent, vague, and ambiguous. As a result, throughout the paper we made a conscious effort to keep these concepts distinct, to the extent possible.

The notion that aphasia involves anxiety is not new and dates at least as far back as Goldstein (1942; 1948; 1959) and Luria (1970) who maintained that aphasic patients “avoid the frustration, humiliation, anxiety, and catastrophic reaction that may be otherwise induced by the prospect of speaking defectively” (Sapir & Aronson, 1990:504). Consequently, language breakdown in neurologic patients cannot be viewed in isolation from the potential effects anxiety might have on it, a stance which we adopt in this review.

While many patients, caregivers, clinicians and researchers would agree that language may be a stressor that could bring about anxiety in persons with aphasia, systematic empirical studies of anxiety in aphasia remain scarce. The aim of this paper is to review the existing literature, identify key issues, highlight important gaps, and propose a program for future study. In doing so, we hope to underscore the importance of understanding aspects of the emotional aftermath of aphasia, which plays a critical role in the process of recovery and success of rehabilitation (Code, 2010).

Emotional Changes among Stroke Survivors

The study of emotional changes following stroke, such as depression and anxiety, is fraught with methodological issues that make the interpretation of reported findings difficult. There is little agreement among researchers about what symptoms are diagnostically valid for detecting post stroke depression and anxiety (Chemerinski & Levine, 2006). Many rely on definitions provided by the Diagnostic and Statistical Manual of Mental Disorders, 4th edition, text revision (American Psychiatric Association, 2000). However, these criteria have been disputed, since they include signs or symptoms that may be found in a broad range of neurologic disorders that are not necessarily related to depression or anxiety, such as weight loss, diminished appetite, or insomnia, and are, thus, diagnostically questionable (Herrmann & Wallesch, 1993; Chemerinski & Levine, 2006). In addition, some common post stroke neurological signs or symptoms, such as altered facial expressions and problems in motor speech and prosody, could mistakenly be taken to reflect a depressive state (Code & Muller, 1992; Stern, 1999; Code & Herrmann, 2003). Exacerbating the methodological problems, studies rarely use the same assessment batteries, scales, questionnaires, and baseline measures in their experimental studies (Herrmann & Wallesch, 1993; Code & Herrmann, 2003; Robinson, 2006; Frankel, 2008). Furthermore, researchers often do not consider the possible consequences that premorbid personality traits, coping styles, and responses to loss might have for changes in patients' emotional state following stroke and do not control for them in their research design (Cohen, 1998; Greenop, Almeida, Hankey, van Bockxmeer, & Lautenschlager, 2009).

Nonetheless, studies of post stroke depression and anxiety are important in that they consider the emotional wellbeing of stroke survivors, which is as elemental to their human experience as it is for healthy individuals (Code & Herrmann, 2003). An important component of emotional wellbeing is anchored in a person's capacity to engage in human interaction (Code, Hemsley, & Herrmann, 1999; Code & Herrmann, 2003), which is certainly changed in persons with aphasia (Tanner, 2003). Those studies that have explored the consequences of emotional dysregulation in persons with aphasia have mostly focused on post stroke depression (Hemsley & Code, 1996; Code & Herrmann, 2003), with little attention to the incidence and effects of anxiety, the study of which seems to have been neglected in the general stroke population as well (Sagen et al., 2010).

Changes in stroke survivors' emotional states are not uncommon, and may include a wide range of emotional states, such as depression, anxiety, anger, mania, euphoria, psychosis, denial, catastrophic reaction, apathy, irritability, aggression, and pathological laughing/crying (Cohen, 1998; Chemerinski & Levine, 2006; Robinson, 2006). Of these, the most common is depression, defined, according to the Diagnostic and Statistical Manual of Mental Disorders, 4th edition, text revision, as a "mood disorder due to stroke with depressive features for two weeks or more, including at least four of the following depressed mood features: changed appetite/weight, disrupted sleep, decreased interest or pleasure, psychomotor agitation or retardation, poor energy, feelings of worthlessness or inappropriate guilt, concentration problems, and suicidal thoughts" (American Psychiatric Association, 2000). The exact incidence of this clinical state, however, remains controversial, with considerable variability reported, ranging from 5% to 60% (Lipsey, Robinson, Pearlson, Rao, & Price, 1985; Starkstein & Robinson, 1988; House, Dennis, Warlow, Hawton, & Molyneaux, 1990; Kauhanen, et al., 1999; Code & Herrmann, 2003; Hackett, Yapa, Parag & Anderson, 2005; Chemerinski & Levine, 2006; Barker-Collo, 2007; Townend et al., 2007; De Wit et al., 2008; Small & Llano, 2009). A commonly accepted estimate for the development of depression stands at approximately one-third of stroke survivors (Hackett et al., 2005; Hilari, Northcott, Roy, & Marshall, 2010). Post stroke anxiety, defined, according to the Diagnostic and Statistical Manual of Mental Disorders, 4th edition, text revision, as a

“state of excessive worrying present for a period of at least 6 months, accompanied by at least three of the following symptoms: restlessness, irritability, low energy, poor concentration, increased muscle tension, poor sleep”, shows slightly different and less variable figures than those reported for depression, with 14–21% of stroke patients exhibiting post stroke anxiety (Burvill et al., 1995; Astrom, 1996; Leppavouri, Pohjasvaara, Vataja, Kaste, & Erkinjuntti, 2003; Barker-Collo, 2007; Sagen et al., 2010).

In spite of the high likelihood of their occurrence, depression and anxiety are often overlooked and/or misdiagnosed in the acute stage (Bogousslavsky, 2003; Chemerinski & Levine, 2006; Paolucci, 2008; Sagen, Vik, Moum, Morland, Finset, & Dammen, 2009; Sagen et al., 2010) and carry over, untreated, into the chronic phases (Astrom, Adolefsson, & Asplund, 1993; De Wit et al., 2008; Clarke & Currie, 2009; Sagen et al., 2009). Astrom et al. (1993), for example, found a rise in the incidence of post stroke depression over the span of three years from three months post onset through three years post stroke, with a dip at 12 months (25% at the acute stage, 31% at three months, 16% at 12 months, and 29% at 24 months), coincident with diminished life satisfaction and reduced participation in daily activities. However, those patients who suffer depression in the acute stage are not necessarily the same as those who end up experiencing depression chronically (Staub, Carota, Karapanayiotides, Berney, & Bogousslavsky, 2001). Symptoms observed in the acute and chronic stages are somewhat different, possibly because of the distinct pathophysiology associated with each (Small & Llano, 2009). Acutely, patients often show pseudodepressive symptoms, such as emotionalism, catastrophic reaction, pathological crying, anxiety, apathy, and loss of psychic self-activation, often misclassified as depression (Bogousslavsky, 2003), whereas subacutely, patients mostly exhibit signs of minor depression typically manifested as loss of interest or pleasure (Small & Llano, 2009).

Depressed or anxious stroke survivors have been found to suffer adverse effects on their self-agency, volition, identity, personality, drive, motivation, and physical and cognitive functioning (Sharpe et al., 1994; Herrmann, Black, Lawrence, Szekely, & Szalai, 1998; Kauhanen, 1999; Berg, Palomaki, Lehtihalmes, Longvist, & Kaste, 2001; Carota, Rossetti, Karapanayiotides, & Bogousslavsky, 2001; Mukherjee, Levin, & Heller, 2006; Robinson, 2006; Santos, Caeiro, Ferro, Albuquerque, & Figueira, 2006; Sagen, 2010). Impaired cognitive functions include verbal logical thinking, comprehension, nonverbal problem solving, verbal memory, visual memory, attention, executive functions, and psychomotor speed, which seem to persist well into the first year post stroke (Kauhanen et al., 1999; Robinson, 2006). These changes have been attributed to neurological, psychological, and psychosocial factors (Gainotti, 1997; Cohen, 1998; Code & Hermann, 2003; Thomas & Lincoln, 2008), where the post stroke altered emotional state is considered to be either a primary consequence of damage to particular neural networks in the left hemisphere (Starkstein & Robinson, 1988; House et al., 1990; Starkstein et al., 1990; Castillo, Starkstein, Fedoroff, Thomas, & Robinson, 1993; Beblo, Wallesch, & Herrmann, 1999; Herrmann, Bartels, & Wallesch, 1993; Herrmann, Bartels, Schumacher, & Wallesch, 1995; Lauterbach, Jackson, Wilson, Dever, & Kirsh, 1997; Robinson, 2003; 2006), or a secondary response to the need to adjust to a life with disability (Herrmann & Wallesch, 1993; Code & Herrmann, 2003; Robinson, 2006). Patients who fail to adapt have been found to experience difficulties engaging in social interactions, putting them at risk of increased social isolation, stigmatization, and marginalization (Herrmann & Wallesch, 1993; Code & Herrmann, 2003; Mukherjee et al., 2006), impeding their physical, cognitive and functional recovery (Neau et al., 1998; Pohjasvaara, Erkinjuntti, Vataja, & Kaste, 1998; Pohjasvaara, Vataja, Leppavouri, Kaste, & Erkinjuntti, 2001; Chemerinski, Robinson, & Koiser, 2001; Singh et al., 2000; Bogousslavsky, 2003; Chemerinski & Levine, 2006; Masskulpan, Riewthong, Diapratham, & Kiptniratsaikul, 2008; Clarke & Currie, 2009; Gusev & Bogolepova, 2009), and possibly even increasing their risk of mortality (Morris, Robinson, Andrzejewski, Samuels, & Price,

1993; Shimoda & Robinson, 1998; Gainotti, Azzoni, & Marra, 1999; Bogousslavsky, 2003; Linden, Bloomstand, & Skoog, 2007; Townend, Brady, & McLaughlan, 2007a).

An important factor affecting the long-term prognosis of stroke survivors is the co-morbidity of depression with anxiety (Starkstein et al., 1990; Castillo et al., 1993; Shimoda & Robinson, 1998; Leppavuori et al., 2003; Mukherjee et al., 2006). Anxiety has been found to have consequences for the onset, duration, severity, and efficacy of treatment of post stroke depression (Coryell, Zimmermann, & Pfohl, 1985; Coplan & Gorman, 1990; Liebowitz et al., 1990; Shores et al., 1992). However, it has been argued that post stroke depression and anxiety are, in fact, independent of one another and do not share the same etiology (Shimoda & Robinson, 1998). While not precisely mapped as yet, the neural architecture associated with each condition may be different (Starkstein et al., 1990; Castillo et al., 1993). There is also some evidence that anxiety selectively affects activities of daily living (ADL) while depression influences cognitive functioning (Shimoda & Robinson, 1998). However, this observation is questionable since it is based on a study with methodological shortcomings, which the authors themselves list, including use of insufficiently sensitive cognitive assessment tools, bias in population selection (exclusion of patients with severe comprehension problems and inclusion of mostly black participants), and lack of control of patients' pharmacotherapy regimens. Moreover, other researchers have found that depression is associated with low ADL scores (Sinyor et al., 1986; Parikh, Lipsey, Robinson, & Price, 1987; Primeau, 1988; Astrom et al., 1993; Kotila, Numminen, Waltimo, & Kaste, 1998; Neau et al., 1998; Fure, 2006).

Anxiety in Aphasia

The consequences of anxiety for speech or language capacity have typically been discussed in relation to a range of voice and speech disorders, such as developmental stuttering and psychogenic and neurogenic stuttering (for a recent review, see Lundgren, Helm-Estabrooks, & Klein, 2009. See, also, Aronson, 1985; Cox, 1986; Sapir & Aronson, 1990, Peters & Hulstijn, 1984; Weber & Smith, 1990; Caruso, Chodzko-Zajko, Bidinger, & Sommers, 1994; Alm, 2004; Ezrati-Vinacour & Levin, 2004; Doruk et al., 2008; Palasik, Irani, & Goberman, 2009; Ozdemir et al., 2010). A small group of studies examined the consequences of anxiety for motor speech in aphasia and found problems, such as dysphonia, in patients with Broca's aphasia but not Wernicke's aphasia, which have been attributed to psychological stress (Luchsinger & Arnold, 1965; Masdeu, Schoene, & Funkenstein, 1978; Ryalls, 1984; Heesch, Ryalls, & Hagoort, 1988; Sapir & Aronson, 1990). However, the study of anxiety, as it might relate specifically to the language component in aphasia, has received far less attention (Gainotti, 1972; Sapir & Aronson, 1990; Gainotti, 1997; Starkstein, Fedoroff, Price, & Robinson, 1993; Cohen, 1998; Carota et al., 2001). In these few studies, descriptions of how language performance is compromised in the face of anxiety are typically based on imprecise linguistic measures, such as "subjective verbal expression", that provide little insight into what aspects of language might be vulnerable to breakdown in an anxious patient with aphasia (Starkstein et al., 1993; Carota et al., 2001).

Aphasia and Physiologic Stress Responses to Language Use

If persons with aphasia, in fact, experience language as a stressor, with the cumulative effect of stress resulting in an anxious state, then they should perceive language use as a threat, entailing certain behavioral and physiologic responses to situations in which they are required to speak. An intricate subcortical and cortical network is involved in the interpretation and regulation of stress, including the brain stem reticular system, limbic structures, and frontal lobes, with activation of the autonomic nervous system and hypothalamo-pituitary-adrenal axis (HPA) being a characteristic stress response (Abreu et

al., 2009; McEwen & Gianaros, 2010; Seeman, Epel, Gruenewald, Karlamangla, & McEwen, 2010). Allostatic systems (sympathetic, neuroendocrine, and immune systems) modulate the response and adaptation to stress, producing increased arousal (Ursin and Erickson, 2004, McEwen, 2007). Optimal responses of the stress system are critical for wellbeing, successful task performance, and engagement in appropriate social interactions (Chrousos, 2009). The protective benefits of allostasis come with a cost to adaptation, commonly referred to as allostatic load (McEwen & Stellar, 1993; McEwen, 2007), “the wear-and-tear on the body and brain resulting from chronic dysregulation (i.e., over-activity or inactivity) of physiological systems that are normally involved in adaptation to environmental challenge” (McEwen & Gianaros, 2010:194). In the general stroke population, such dysregulation adversely affects motor skills, cognitive function, prognosis, and mortality rate (e.g., Franceshini, Teneconi, Zoppoli, & Barreca, 2001; Christensen, Boysen, & Johannsen, 2004; Marklund, Peltonen, Nilsson, & Olsson, 2004). Researchers have used different biomarkers to measure allostatic load in persons under stressful and stress-free situations to determine their physiologic stress responses, including measures of cortisol in saliva, urine, and blood, epinephrine, norepinephrine, dopamine, and growth factors, as well as measures of cardiovascular function, such as blood pressure (Abreu et al., 2009).

With rare exception, there are virtually no studies that directly examine the physiologic stress responses in persons with aphasia, in relation to their language performance. The one exception that we could find is the series of studies by Laures-Gore and colleagues, which measured salivary cortisol levels in persons with aphasia and in healthy controls in relation to performance on linguistic tasks (Laures, Odell, & Coe, 2003; Laures-Gore, Heim, & Hsu, 2007; Laures-Gore, DuBay, Duff, & Buchanan, in press). Laures-Gore and colleagues (2007) hypothesized that persons with aphasia would show hypercortisolemia in response to the stressfulness of the linguistic demand, stemming from chronic HPA dysregulation. For the linguistic task, the investigators used an adaptation of the Trier Social Stress Test: A 5 minute speech, during which subjects were asked to tell an unfamiliar listener about their occupation, followed by questions. For persons with aphasia, this referred to their job before their stroke; for controls, to their current job or their job before retirement. Five of the fifteen aphasic participants required prompting to complete this task. A nonlinguistic task administered to all participants was the Mirror Drawing Test, in which subjects were asked to trace the picture of a star, where indirect visual contact with their hand was made through a mirror placed in front of their hand. In addition to obtaining physiological measurements of stress, the investigators also measured participants’ self-perceived level of stress at the conclusion of the initial thirty-minute baseline waiting period and then again at the end of each task.

Profiles of cortisol levels sampled throughout the testing period differed between aphasic patients and controls, but differences between the linguistic and the nonlinguistic tests were not statistically significant. On the self-evaluative perception of stress test, a clear difference between the two groups emerged, with persons with aphasia rating the linguistic test as being much more stressful than the non-linguistic task. The differences in cortisol profiles between the two groups, however, did not lend themselves to easy analysis. Laures-Gore et al. (2007) proposed that patients with aphasia consistently experience language as a repetitive stressor and, as a result, suffer from chronic HPA hypoactivity, commonly found in a range of stress related disorders (e.g., Heim, Ehler, & Hellhammer, 2000; Fries, Hesse, Hellhammer, & Hellhammer, 2005). Nonetheless, this explanation needs to be reconciled with evidence cited in Carota et al. (2001), according to which patients with left hemisphere damage show increased autonomic responses which have been claimed to inhibit the arousal system modulated by the right hemisphere, leading to decreased arousal overall.

Decreased arousal in persons with aphasia has consequences for their attentional systems (McNeil, Odell & Tseng, 1991; Tseng, McNeil, & Milenkovic, 1993; McNeil & Pratt, 2001; McNeil, Matthews, Hula, Doyle, & Fossett, 2006; Hula & McNeil, 2008; Murray, 2002; Laures et al., 2003). Problems in attention allocation have been reported for aphasic patients, in terms of slower reaction times and poorer accuracy in dual-task paradigms, where linguistic stimuli are simultaneously presented with other competing input (e.g., Tseng et al., 1993; Murray, Holland, & Beeson, 1997a; 1997b; 1998; Murray, 2002; 2004; Laures, 2005; McNeil et al., 2006; Murray, Keeton, & Karcher, 2006; Hula, McNeil, & Sung, 2007; Hula & McNeil, 2008). It is not implausible to propose that **linguistic anxiety** feeds into this attentional dysfunction, where the patient's hyperfocus on area of worry, i.e., concern about impaired language performance, reduces his/her ability to attend to the language task at hand, a problem continually reinforced by aroused physiologic stress responses. The suppression of these negatively laden thoughts and feelings competes for the same attention resources available for processing task-relevant information, and so might lead to suboptimal performance.

Studies exploring arousal in aphasia have not directly focused on patients' physiologic responses such as galvanic skin response (Heilman, Schwartz, & Watson, 1978), blood pressure, and cortisol levels (Laures et al., 2003) in high stress situations. However, Laures et al. (2003) assumed that increased arousal would be observed with increased task demand. Whether such increased demand is, in fact, stress-related is hard to say, since it is ultimately the brain that determines what is stressful for an individual (McEwen & Gianaros, 2010). It does so by "supporting conscious and unconscious appraisal processes; it determines the health-damaging or health promoting behaviors that result from this appraisal; and it regulates peripheral alldynamic control systems that feed back to the brain to affect functional and structural neuroplasticity" (McEwen & Gianaros 2010:210). Findings from Laures et al. (2003) paint a mixed picture, with mild changes in blood pressure but no cortisol reactivity in persons with aphasia in response to performance on linguistic and nonlinguistic auditory vigilance tasks. To account for this dissociation, the investigators suggested considering the distinct temporal dynamics of the two physiologic systems examined (the immediacy of the cardiovascular reactivity vis-à-vis the delayed reactivity of cortisol) as a source for this difference. They also speculated that the non-threatening nature of the experimental design was insufficiently challenging to arouse the patients. Laures-Gore et al. (2007) made similar arguments, even though the experimental methods used in the (2007) study were designed to increase threat and, so, arousal. Since the patients in this study once again failed to show cortisol reactivity, the investigators conjectured that persons with aphasia are habituated to language tasks, which are frequently administered to them while in treatment. Under such an account, though, patients would not have been expected to show heightened levels of cortisol at the onset of testing. As Laures-Gore et al. (2007) themselves acknowledge, since no other measures of physiologic stress responses were examined in the patients, it is quite possible that effects in other physiologic systems (e.g., blood pressure, skin conductance, heart rate) may have been overlooked.

A potential drawback of this study concerns the imprecision of the language measures selected for analysis. The authors do not report the number or types of errors the patients made while producing the elicited discourse sample discussing their occupation. In determining the possible consequences that anxiety might have for the language produced by persons with aphasia, the well-formedness of their linguistic output should be scored. Such analyses might include measures of communicative effectiveness (Menn, Ramsberger, & Helm-Estabrooks, 1994), lexical productivity, syntactic architecture, and discourse organization (Coelho, 2007; Sherratt, 2007), which take into account the number of elements produced, the appropriateness of the structures chosen, omissions, substitutions, and other

types of errors.ⁱ Such an analysis would have perhaps helped account for why five of the fifteen patients required prompting to complete the task.

In a subsequent study using the Laures-Gore et al. (2007) experimental paradigm, Laures-Gore et al. (in press) made an attempt to address this issue, measuring cortisol reactivity before and after the same discourse task, and correlating cortisol levels with behavioral measures of word productivity (Cherney, Shadden, & Coelho, 1998) and error frequency (Docherty, 1996) in the elicited discourse speech samples. While this attempt is a step in the right direction, the fact that the discourse samples elicited were of different types—narrative vs. conversation guided by open-ended questions—poses a problem for the interpretation of the findings. These discourse types have different textual features with distinct processing demands that call for specific linguistic structures (Ulatowska & Olness, 2007; Berman, 2008), and so might involve word productivity and error frequency which are not comparable to one another. Thus, it is not clear to us what it would mean for a patient to have a low or high index of word productivity or error frequency, if these measures are calculated across these two text types. Despite these methodological quibbles, we note the pioneering work of Laures-Gore and her colleagues in this complicated and important but understudied domain.

Laures-Gore et al. (in press) predicted that lower levels of cortisol would be associated with lesser word productivity, but found the opposite in some of their patients (less than half): a moderate association between word productivity and cortisol levels in persons with aphasia, where increased levels were linked to greater word productivity. They offer several possible explanations, linking cortisol reactivity to task persistence, effort exerted during task performance, or the involvement of psychosocial variables, such as ego, unpredictability, evaluative threat, and lack of control. To this list, we propose adding motivation, which in early studies of aphasia has been linked to anxiety (Shill, 1979), where it has been claimed that appropriate levels of anxiety optimally challenge patients inducing better performance (for related comments, see the section below). At present, these accounts remain speculative in nature, especially given the small sample size on which the observations are based. Nonetheless, this finding underscores the importance of using sensitive language measures in studying the putative relationship between anxiety and language in aphasia.

A potential measure of interest is filled pauses (*ums*), the occurrence of which in neurologically intact individuals has been linked to high anxiety, evaluation, apprehension, and self consciousness, with more pauses produced as a function of greater self-consciousness about speech and focused attention on linguistic output (Christenfeld & Creager, 1996). Extending this account to persons with aphasia, Christenfeld & Creager (1996) have proposed that the higher number of *ums* observed in the speech of Broca's patients, in comparison with Wernicke's patients, is associated with their heightened awareness of their impairment and deliberation in speaking, which disrupts the automaticity of speech production. Measuring changes in the distribution of filled pauses in the speech of anxious persons with aphasia over time can serve as a behavioral marker of stress and/or anxiety, much like Laures-Gore et al.'s (in press) index of word productivity, allowing for long term follow-up of patients' anxiety and so improving their rehabilitative prognosis.ⁱⁱ

ⁱGoral & Kempler (2008), who performed a treatment study examining changes in patterns of verb production in chronic aphasia, used the number of verbs and nouns per text, and the verb-noun ratio as outcome measures. These measures might be particularly useful, since the task they used to elicit discourse samples in their study is similar to the one Laures-Gore et al. (2007) used in that it required the patient to talk about his job before the stroke.

ⁱⁱInterestingly, in a recent study assessing cognitive and emotional functionality in neurologically intact elderly participants over a period of six months, the occurrence of filled pauses ('ums') in phone interviews has been found to be a reliable marker for monitoring changes in mood and executive functions over time (Penard et al., 2009).

Reducing Anxiety in Aphasia

Reducing anxiety in aphasia is of clinical importance, given the presumed link between increased levels of stress and patients' poor adaptation skills (Pachalska, Knapik, Smolak, & Pytel, 1987) and paucity of coping resources (Laures-Gore et al., 2007a; Laures-Gore et al., in press). In early studies of aphasia recovery, level of anxiety has been identified as key to patients' motivation to perform well, and hence was linked to successful rehabilitation (Wepman, 1951; 1953; Luria, 1963; 1970; Darley 1972; 1975; Shill, 1979). Excessive anxiety may delay or even block recovery; lack of anxiety promotes it; and sufficiently heightened anxiety enables it (Shill, 1979). This claim is consistent with the well-established Yerkes-Dodson Law (1908), linking level of performance to level of mental and physiological arousal, following an inverted U-shaped curve, with poorest performance observed at both ends of the arousal curve (lowest and highest). Of course, determining what level of anxiety is ideal for recovery is difficult. Degree of anxiety has been associated with awareness of disorder/disability, expectations of recovery, acceptance and lack thereof from friends and family, type of feedback from the various caregivers, clinicians, and other persons involved in the rehabilitation process (Darley, 1975). Skelly (1975) has identified four main areas of concern that patients typically report, based on data collected from fifty stroke patients: (i) difficulty comprehending incoming speech, especially from professional staff, (ii) observing nonverbal negative reactions from caregivers and clinicians, (iii) being withheld information about interventions, on the assumption that they cannot understand the explanations provided, and (iv) feeling lack of respect of personhood, through intrusive questions or requests to perform certain tasks in treatment. In the current aphasiology literature, the relevance of motivation for restitution of disturbed functions in aphasia continues to be discussed in the context of psychosocial studies investigating social participation (e.g., Dalemans, De Witte, Beurskens, Van Den Heuvel, & Wade, 2010) and patient resilience (Cyr, 2010), and in some pharmacotherapy studies, where changes in patients' motivation have been found in response to the administration of a dopaminergic agent (L-dopa) (Seniow, Litwin, Litwin, Lesiak, & Czlonkowska, 2009).

The potential benefit of reducing anxiety to improve language performance in patients with aphasia has been discussed in a small number of studies exploring the effects of stress reduction on persons with aphasia treated with Complementary Alternative Medicine (CAM) therapies such as acupuncture (Jianfei, Meifang, & Jia, 1988; Zhajun, 1989), hypnosis (Thompson, Hall, & Sison, 1986; Maganiello, 1986), relaxation training (Ince, 1968; Marshall & Watts, 1976; Yesavage & Jacob, 1984; Murray & Ray, 2001), biofeedback (McNeil, Prescott, & Lemme, 1976; Katz, Bhardawaj, & Carstens, 1999), and nature-based therapy (NBT) (reviewed in Lundgren, 2004). Findings from these studies are, for the most part, inconclusive or difficult to interpret, since they lack methodological rigor, are based on small sample size, and include few or no quantitative language/speech and cognitive measures (Laures & Shisler, 2004). More solid results come from mindfulness and relaxation studies, the most frequently used CAM therapy to date (Murray & Kim, 2004; Lundgren, 2006; Marshall & Laures-Gore, 2008), whose benefits for adult neurogenic communication disorders have been found in stroke survivors, patients with traumatic brain injury (TBI), and patients with dementia (Laures & Shisler, 2004; Murray & Kim, 2004). This technique involves maintaining attentional focus on stimulus or muscle activity while inhibiting competing thoughts or sensory input, the result of which is physiologic changes opposite to those associated with stress (Murray & Kim, 2004). Such relaxation treatment has been shown to be efficacious when given in conjunction with other conventional language therapies (Murray & Ray, 2001; Murray & Kim, 2004; Murray, 2008). Murray and Ray (2001), for example, found that a patient with chronic aphasia who received relaxation therapy prior to syntactic stimulation improved on testing in number of grammatical utterances, correct information units (CIUs), and successful utterances produced.

Additional evidence suggesting that reducing anxiety in patients with aphasia might promote their language performance has been found in a small number of pharmacotherapy studies, where the administration of the beta blocking agent propranolol was associated with improved naming in both nonfluent and fluent aphasic patients (Tanaka, Albert, Fujita, Nonaka, & Oka, 2006; Tanaka et al., 2009; Beversdorf et al., 2007). Tanaka et al. (2006) and Tanaka et al. (2009) were interested in determining whether improved language performance might be correlated with measurable changes in the function of the autonomic nervous system. They used an open label design to measure patients' language performance (Boston Naming Test and auditory comprehension), heart rate, and mood, at baseline, before, during, and following treatment, and found that propranolol indeed improved language performance in persons with aphasia, and did so by selectively affecting a physiological expression of anxiety (reduction in heart rate was observed while scores on the visual analog mood scale remained unchanged). Although these findings are limited, at best, for methodological reasons (open label, small sample size, no placebo condition), they suggest that the physiological aspects of autonomic nervous system (ANS) function might be more important to language function in aphasia than has been previously considered. Beversdorf et al. (2007), who also found significant improvement in naming in patients with aphasia after administration of propranolol, linked the observed beta-adrenergic effects to the modulation of signal-to-noise ratio in the cortex (Hasselmo, Linster, Patil, Ma, & Cekic, 1997; Heilman, Nadeau, & Beversdorf, 2003). Future studies with more power, distinguishing peripheral and central autonomic effects in persons with and without aphasia, will be helpful as research develops into the relation of autonomic system function to aphasia.

Targeting patients' anxiety by regulating their ANS responses might result in an increased ability to attend to a given language task. It is also possible that providing patients with attention training could bring about a reduction in their level of anxiety. There is an emerging body of evidence suggesting that in neurologically intact but anxious individuals, attention training away from "threat" alleviates their anxiety (Eldar & Bar-Haim, 2010). How this might be applied to the treatment of persons with aphasia is a project for further development, especially if the source of their anxiety is language itself. Perhaps the incorporation of nonverbal elements into therapy could serve as a means to shift the patients' attention away from that which might be overly threatening. The question of whether drugs targeting attention, e.g., noradrenaline, dopamine, and acetylcholine (ant)agonists (for a recent review, see Sivan et al., 2010) can also be used to reduce anxiety opens yet another avenue for future research. Indeed, the efficacy of different drugs for language performance in aphasia (for a review see, Small & Llano, 2009) might very well have an attention component that is indirectly modulated.

Language as a Stressor in Aphasia

As detailed in this review, post-stroke emotional changes in persons with chronic aphasia clearly have adverse effects for language performance and prospects of recovery. However, the specific role anxiety might play in aphasia has yet to be determined. As a starting point, we propose to view language in aphasia as a stressor, linked to an emotional state we term "linguistic anxiety." Specifically, a person with linguistic anxiety is one in whom the deliberate, effortful production of language involves anticipation of an error, with the imminence of linguistic failure serving as the threat. Such a proposal would not only offer an extension to the earliest speculations about the role of anxiety in aphasia (e.g., Goldstein, 1942; 1948; 1959), it could also be incorporated into a speech production model (e.g., Caramazza, 1997; Levelt, 1992; 2001), where anticipation of error might work in tandem with mechanisms of error detection, repair, and speech planning.

The notion of anticipation of error does not imply that patients would necessarily perceive themselves as stressed and/or anxious prior to completion of a linguistic task. Behavioral measures of stress and/or anxiety need not map onto perceptions of stress and/or anxiety (Laures-Gore et al., in press). Self-ratings of stress and/or anxiety could very well follow a task (as in Laures et al., 2007), or even be denied altogether (as in Tanaka et al., 2009) as a function of a repressive coping style (Myers, 2010). Since anticipation is psychologically linked to anxiety and also plays an important role in the allostatic system (McEwen, & Gianaros, 2010), measuring allostatic load in persons with aphasia when they are asked to perform a linguistic task, might provide a useful tool for assessing the relation of stress to “linguistic anxiety.” Such a study would reflect an eclectic approach to psychological changes observed in aphasia, which ties together post-stroke biochemical brain alterations, psychological coping styles, and reaction to loss (Tanner, 2003).

Implications for Research

From a methodological standpoint, the use of physiologic stress responses in the study of aphasia and language performance might offer a means to identify whether or not a person with aphasia might be experiencing language as a stressor, pointing to the possible presence of linguistic anxiety, without having to rely only on linguistically demanding questionnaires (Laures et al., 2007) or on subjective self reports of stress and/or anxiety, which often provide a misleading picture of a patient’s emotional state. In an extensive review of 146 studies examining autonomic nervous activity in emotions of healthy individuals, Kreibig (2010) found several reliable cardiovascular, electrodermal, and respiratory measures of anxiety. In these studies, anxiety was induced by an anticipatory experimental condition, such as threat of shock or speech preparation, and manifested itself by increased heart rate, higher diastolic blood pressure, increased nonspecific skin conductance rate, higher respiratory rate, and other physiologic measures. These changes reflect “sympathetic activation, vagal deactivation, and a pattern of reciprocal inhibition.” Whether such changes would also be observed in persons with aphasia is uncertain, given that their neural architecture is clearly not equivalent to that of neurologically intact individuals (for a related comment, see Laures-Gore et al., 2007).

A range of physiologic measures could be used to examine physiologic stress responses and language performance in persons with aphasia, to determine which system might be activated in response to an anxiety-inducing stimulus. While the efficacy of cortisol reactivity as an appropriate measure of physiologic stress responses in persons with aphasia is currently being examined (Laures-Gore et al., 2007; Laures-Gore et al., in press), alternative (or additional) biomarkers such as heart rate, skin conductance, or pupillometry might be used to allow for continuous measurement and offer insight into the potential patterns of language-based anxiety peaks.

To induce anxiety in persons with aphasia, the experimental design would have to be sufficiently threatening to generate a stress response sufficiently similar to the stress induced in real-life communicative situations that are perceived as threatening to people with aphasia. Laures-Gore et al. (2007) emphasized the need to include a social-evaluative component in the experimental design, where participants might perceive judgment of their performance as “costly” enough to induce anxiety. They cited previous research where such experimental methods were associated with HPA responsiveness. We would agree that such a component of research design would be relevant, given the literature investigating effects of “stereotype threat” on task performance, where social stigma is used as a means to increase the threat of failing on a task. In such studies, members of a social group are introduced to a domain-specific negative stereotype stigmatizing their group (Steele & Aronson, 1995; Steele, 1997), adversely affecting their task performance for fear of confirming that social stigma (e.g., women’s suboptimal performance on complex math

problems when told that math solving abilities are linked to gender differences). This component of research design increases the stakes of the assigned tasks by influencing a patient's perception of either the relevance of the task to the study, as in "only some of your scores will be important to study, but we will not tell you which ones count"; or the tasks' difficulty, as in "the tasks you will be given will become increasingly difficult."

To determine whether persons with aphasia have linguistic anxiety or are generally anxious, their performance on both linguistic and nonlinguistic tasks should be analyzed. Patients might show variability in terms of levels of anxiety associated with the production of language in a constrained task vs. a naturalistic setting. Thus, it would be appropriate to include in such a study both standardized confrontation tests, such as the Boston Naming Test (Kaplan, Goodglass & Weintraub, 1983), as well as discourse elicitation tasks, such as picture descriptions or narrative production. It would also be useful to include both production and comprehension tasks, so as to minimize the potential effects of body movement on the physiologic stress response, which might be more noticeable in language production. Patients' performance on tasks involving competing stimuli would be helpful in demonstrating the potential consequences of "linguistic anxiety" in aphasia, given the relationship between heightened anxiety and compromised attention.

Interestingly, inducing stress by degrading the quality of the auditory stimulus (Kilborn, 1991; Dick, Bates, & Ferstl, 2003), increasing the speed of stimulus presentation (Miyake, Carpenter, & Just, 1994), and introducing a competing stimulus (Blackwell & Bates, 1995) has been shown to generate transient aphasia-like speech patterns in neurologically unimpaired individuals, suggesting that ANS dysregulation, indeed, interferes with intact language function. If such physiologic changes, directly measured through cardiovascular, electrodermal, and respiratory measures of anxiety (see Kreibig, 2010), mediate language variability in what might be described as a "normal-to-aphasic continuum" (Silkes, McNeil, & Drton, 2004), identifying those physiologic indices could help explain aspects of adaptation and neural reorganization involved in aphasia recovery (Hula & McNeil, 2008).

Clinical Implications

Understanding the impact of emotional changes following stroke in persons with aphasia continues to pose a challenge for persons facilitating aphasia therapy (Code, 2003; 2010). Adequate treatment of patients must involve precise and explicit specification of all therapy components (Byng & Black, 1995; Horton & Byng, 2000), including examination of the potential role anxiety might have in language function in aphasia. While most clinicians would agree that level of anxiety in persons with aphasia is one of the important psychosocial factors linked to patients' quality of life, only few address its management in the context of the therapy they provide, mostly for fear of offering an imbalanced impairment-focused therapy (Dembowski, 2006).

To this end, measuring physiologic stress responses in patients with aphasia might offer an unbiased window into determining the extent to which anxiety needs to be regulated in a patient, if at all, and so enable the tailoring of specific therapeutic interventions to his/her particular needs. For example, measuring blood pressure in patients might help identify cases of "white coat effect", which has been reported in some persons with mild aphasia when placed in a clinical setting controlled by a speech therapist (Sakamoto, Sakamaki, Tani, Sugai, & Kurabayashi, 2002). Such patients might enjoy self-guided sessions (Sakamoto et al., 2002) or group therapy (Kovarsky, Curran, & Zobel Nichols, 2009; Simmons-Mackie & Damico, 2009), where clinician involvement can be kept to a minimum and might be perceived by the patient as less anxiety inducing. While group therapy is not directly designed to reduce anxiety, it might offer patients a supportive and relaxed environment, where they can consistently show psychosocial and speech-language gains

(Elman & Bernstein-Ellis, 1999; Ross, Winslow, Merchant, & Brumfitt, 2006; Elman, 2007; Simons-Mackie et al., 2007). Similarly, patients could be placed in mindfulness or relaxation therapy programs (Murray & Ray, 2001; Murray & Kim, 2004; Murray, 2008) or a pharmacotherapy regimen, as an adjunct to any standard speech-language therapy they might be receiving, to help manage their stress, reduce their anxiety and enhance their rehabilitation outcome. Preliminary evidence suggests that such interventions hold promise, but studies exploring this idea are still few and far between.

Beyond aphasia

Exploring the putative relationship between anxiety and language in aphasia, through the study of physiologic stress responses, could establish a platform for investigating language changes in the brain in other clinical populations, such as in individuals with Alzheimer's disease or persons with post traumatic stress disorder, or even with healthy aging persons, in whom "linguistic anxiety" might be at work when they have trouble finding words. Findings from such studies would fit within the broader psychosocial debates linking anxiety and language function in neurologically intact individuals, including second language learners, public speakers, and stutterers, shedding light on the yet to be determined role that the autonomic nervous system might have for language function in the anxious brain.

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