CHAPTER

73

Vascular Aphasia Syndromes

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73.1 INTRODUCTION

The study of aphasia and its associated lesions in the 19th century by Dax (1936), Broca (1861, 1865), Wernicke (1874, 1881) and others revealed new insights about the neural organization of language. Perhaps the most reliable finding was that patients with language impairment typically had damage to the left hemisphere. Damage to the more anterior parts of the brain, particularly the left posterior inferior frontal gyrus (IFG), was often found in those whose spoken output was limited or poorly articulated (Broca, 1865); damage to the more posterior regions in the left temporal lobe (in the absence of damage to frontal regions) was found in those whose spoken output was well-articulated but meaningless (Wernicke, 1881). These early observations established that language functions are localized in the left cerebral hemisphere and provided the groundwork for Lichtheim’s model of aphasia, later adapted by Geschwind (1965). Geschwind put together early observations, along with subsequent reports of behavior and associated lesions (based largely on autopsy), to characterize the collection of frequently co-occurring language characteristics that result from damage to particular areas of the brain. With the advent of computerized tomography (CT) technology in the early 1970s, it was confirmed that classic aphasic syndromes correlated with particular vascular territories (Damasio & Geschwind, 1984).

In this chapter, the vascular aphasia syndromes are described. It is emphasized that the various characteristics frequently co-occur because they all depend on areas of the brain supplied by the same artery (or branch of an artery). If only part of the territory is damaged, then only a subset of the characteristics is present. That is, the characteristics are dissociable. Many of the vascular aphasia syndromes are associated with damage to nonlanguage functions that also depend on brain tissue supplied by the same arterial branch. For example, Broca’s aphasia is associated with left arm weakness or spasticity, because left arm function (like the speech and language functions affected in Broca’s aphasia) depends on territory supplied by the superior division of the left middle cerebral artery (MCA).

Therefore, one can see from the outset that these syndromes are not theoretically coherent syndromes in the sense that they have a single underlying functional basis; rather, they have a shared anatomical basis. After describing the vascular syndromes, we discuss how they can be understood within more current concepts of language and aphasia, and whether they have any usefulness in aphasia research, management of stroke, or rehabilitation of aphasia.

73.2 CLASSIC APHASIA CATEGORIZATION: VASCULAR SYNDROMES

73.2.1 Broca’s Aphasia

Broca’s aphasia is characterized by nonfluent, telegraphic, poorly articulated verbal output in which meaning is conveyed by content or information-carrying words, such as nouns and verbs; however, nouns are named more accurately than verbs (Berndt, Mitchum, Haendiges, & Sandson, 1997; Miceli, Silveri, Villa, & Caramazza, 1984). Morphological inflections (e.g., past tense, plurals) and function words (e.g., articles, conjunctions, prepositions, auxiliary verbs,
pronouns) are often omitted or incorrectly produced, and word order may be incorrect. Thus, speech production is characterized as agrammatic. Although it is widely agreed that individuals with Broca’s aphasia are nonfluent, fluency is a multidimensional construct. “Nonfluency” may be associated with diminished number of words per minute, reduced phrase length, impaired melody, disrupted articular agility, and/or agrammatic sentence production (Goodglass & Kaplan, 1983). The factors that affect speech fluency may vary from one individual to the next. Also, individuals with aphasia may have preserved islands of fluency, particularly for rote and overlearned speech, in otherwise nonfluent novel speech output. Because speech fluency can be defined by a complex set of features, it may be difficult to judge. Experienced speech-language pathologists analyzed the content and fluency ratings on the Western Aphasia Battery for 20 individuals with aphasia. Inter-rater reliability ratings for fluency were poor using published criteria (Trupe, 1984).

It is important to note that the severity of language impairment can vary across individuals with Broca’s aphasia. In its mildest form, Broca’s aphasia presents with reduced phrase length with agrammatic sentence production and relatively retained language comprehension, although there may be deficits in comprehension of multistep commands or sentences with complex syntax (e.g., The flowers that Bob gave to Susan were roses). In more severe presentations, there is reduction of all speech output, with speech being limited to one or more recurrent utterances and automatic sequences (Goodglass, Kaplan, & Barresi, 2001). Repetition is usually halting and dysfluent. Reading comprehension may parallel auditory comprehension. The mechanics of written expression are compromised because patients must write with the left, nondominant hand, because of frequently co-occurring right hemiparesis. Spelling is often impaired, and written output is agrammatic.

Broca’s aphasia, as originally described by Paul Broca, was attributed to lesions of the posterior half of the left third frontal convolution (IFG; Brodmann area 44). This area became known as Broca’s area and, along with surrounding areas in the posterior frontal lobe, is supplied by the superior division of the left MCA. Subsequently, neuroanatomists and aphasiologists challenged the causative association between this circumscribed lesion site and the nature of the aphasia reported by Broca. Mohr et al. (1978) reported that a more widespread region must be damaged to result in the constellation of impairments seen in the syndrome of Broca’s aphasia as originally described. Damage to Broca’s area alone, according to Mohr, results in only a subset of the symptoms, most notably impaired motor speech (Keller, Crow, Foundas, Amunts, & Roberts, 2009). Furthermore, advances in neuroimaging, permitting studies of neural activity in normal adults during cognitive/language tasks, have led away from classic structure/function relationships to the role of brain networks and regions in the performance of cognitive/language tasks. Increasingly, Broca’s area is tied to a variety of language functions (Davis et al., 2008; Grodzinsky & Amunts, 2006), functions that might be critical to language such as verbal working memory (Rogalsky & Hickok, 2011), and even nonlinguistic functions, such as visual searching and visual spatial cognition (Grodzinsky & Amunts, 2006). Many studies distinguish between functions of pars opercularis and pars triangularis (which are distinct cytoarchitectural fields that comprise Broca’s area but have a great deal of anatomical overlap across individuals; Grodzinsky & Amunts, 2006). In individuals with stroke involving Broca’s area, there is often concomitant right hemiparesis or monoparesis of the arm (and right facial weakness) due to ischemia of the nearby precentral gyrus.

73.2.2 Wernicke’s Aphasia

Wernicke’s aphasia is characterized by fluent, effortless, but relatively meaningless, spontaneous speech and repetition, and impaired comprehension at the word, sentence, and discourse levels. Spoken language may be limited to jargon comprising either real words or neologisms (nonwords such as “klimorata”), or a combination of the two. In milder forms, paraphasic errors are present as well as intermittent coherent “social” phrases, such as, “yes, that’s right.” Melodic contour of spoken language is often preserved, initially giving listeners the impression that output is intact. Comprehension may be severely compromised and impaired phonological analysis is thought to underlie the comprehension impairment in Wernicke’s aphasia (Robson, Keidel, Lambon Ralph, & Sage, 2012). Repetition is generally similar to spontaneous speech—fluent jargon. These deficits have been attributed to impaired inhibition of lexical activation, so that the person cannot select the appropriate word, sound, or meaning from competing linguistic units that are also activated (Blumstein & Milberg, 2000). Although such an underlying impairment would account for many of the observed language deficits, it could not easily account for all cases. For example, there have been some reported cases of Wernicke’s aphasia with relatively preserved or relatively impaired categories of words, such as animals or tools (Hillis & Caramazza, 1991a), or selectively impaired nouns relative to verbs (Zingesser & Berndt, 1990). Selective impairment or preservation of particular semantic categories of words indicates a deficit at the level of accessing lexical semantics (Hillis & Caramazza, 1991a). Reading...
comprehension and written expression are typically impaired in a very similar pattern as the impairment in auditory comprehension and verbal expression, respectively, although some individuals may be able to read aloud and spell to dictation regular words that they fail to understand (Hillis & Caramazza, 1991a). In contrast to those with Broca’s aphasia, individuals with Wernicke’s aphasia are typically unaware of their errors; they may have only a shallow awareness that they have some kind of difficulty communicating.

This collection of deficits is usually caused by neural dysfunction in regions supplied by the inferior division of the left MCA, including Wernicke’s area (most of Brodmann area 22, in the posterior, superior temporal gyrus), and often inferior parietal cortex (angular and supramarginal gyri) and inferior and middle temporal gyri. Because the Meyer’s loop of the optic radiations runs through the temporal cortex, there is often a concomitant right homonymous superior quadrantanopsia. The fact that there is individual variability in the cerebral vasculature and the areas supplied by particular arteries can account for occasional dissociations between the typical deficits in Wernicke’s aphasia (i.e., only some of the usual territory is affected by stroke in some cases, such that only some of the symptoms occur) as well as occasional anomalous lesion sites in patients with Wernicke’s aphasia.

73.2.3 Global Aphasia

Global aphasia refers to a profound impairment of all modalities of receptive and expressive language. Individuals with global aphasia typically present with marked impairments of comprehension of single words, sentences, and conversations, as well as severely limited spoken output. Spontaneous verbal output may be restricted to single words, nonwords, or undifferentiated phonation and some individuals’ speech only consists of perseverative utterances (e.g., “no, no”). In addition, reading and writing are typically profoundly compromised. Because of the severity of language impairment, communication partners sometimes need to anticipate the communicative intentions of individuals with global aphasia or rely primarily on gestures or drawing. In most cases, both Broca’s area and Wernicke’s area are damaged (Mazzocchi & Vignolo, 1979) or functionally compromised (Hillis et al., 2004) because of occlusion or stenosis of the proximal MCA (affecting both the inferior and superior divisions) or the internal carotid artery (ICA).

73.2.4 Conduction Aphasia

Conduction aphasia is usually defined as a language impairment characterized by relatively fluent, although paraphasic, spontaneous speech, intact auditory comprehension, and disproportionately impaired speech repetition. Secondary features include reading impairments, variable writing difficulties, and ideomotor apraxia (Benson et al., 1973). Individuals with conduction aphasia display well-articulated responses that are phonemically similar to target words and repetitive self-corrections resulting in increasingly closer approximations to targets. This phenomenon is termed “conduit d’approche” (Goodglass, 1992). Traditionally, conduction aphasia is thought to be caused by a lesion in the arcuate fasciculus, a white matter tract that runs between Broca’s and Wernicke’s areas, and thus is considered a disconnection syndrome because a lesion in the arcuate fasciculus is assumed to interrupt communication between the sensory and motor modules of the classically defined speech-language system (Geschwind, 1965). This hypothesis has been challenged. More recent evidence shows that conduction aphasia is not the result of damage only to the arcuate fasciculus. Although damage to the arcuate fasciculus is reported be present in the setting of conduction aphasia in some contemporary accounts (Geldmacher, Quigg, & Elias, 2007; Yamada et al., 2007; Zhang et al., 2010), it is not reported in others. In fact, cortical lesions alone may produce conduction aphasia (Anderson et al., 1999; Quigg, Geldmacher, & Elias, 2006), indicating that damage to the arcuate fasciculus is not a pre-requisite condition for conduction aphasia. Furthermore, lesions of the arcuate fasciculus do not always cause conduction aphasia (Epstein-Peterson, Vasconcellos-Faria, Mori, Hillis, & Tsapkini, 2012; Selnès, van Zijl, Barker, Hillis, & Mori, 2002). Substantial anatomical evidence suggests that conduction aphasia is caused by damage to the left superior temporal gyrus and/or the left supramarginal gyrus (Axer, von Keyserlingk, Berks, & von Keyserlingk, 2001; Baldo & Dronkers, 2006) due to occlusion of a branch of the inferior division of the left MCA.

73.2.5 Anomic Aphasia

Impairment of word retrieval is the primary feature of anomic aphasia with relatively well-preserved language function in other realms. Individuals with anomic aphasia may use circumlocutions for targets and display protracted pauses in verbal output and use of fillers (e.g., “What is that called?”; “You know what I mean”), resulting in empty or low-content verbal output. Anomia is present in other aphasia syndromes and may be the residual language symptom as individuals recover language abilities. Consequently, anomic aphasia is the least reliably localized aphasia in chronic stroke. However, acute, isolated anomia is most often
associated with small areas of ischemia in left inferior temporal cortex or thalamus (Hillis et al., 2006).

73.2.6 Transcortical Aphasias

Three transcortical aphasias have been characterized: transcortical motor (TCM); transcortical sensory (TCS); and mixed transcortical (MTC). The transcortical aphasias are distinguished by intact repetition ability. TCM aphasia shares many characteristics of Broca’s aphasia but has the distinctive feature of fluent, grammatical repetition. This vascular syndrome is caused by lesions just anterior or superior to (surrounding) Broca’s area (Freedman, Alexander, & Naeser, 1984), often caused by occlusion of the anterior cerebral artery (ACA) (Masdeu, Schoene, & Funkenstein, 1979; Rubens, 1976) or “watershed” areas between the ACA and the MCA. TCS aphasia is similar to Wernicke’s aphasia, except for the presence of accurate repetition. TCS aphasia is usually attributed to lesions in areas surrounding Wernicke’s area, in the watershed territories between the MCA and posterior cerebral artery (PCA), or the PCA territory (Alexander, Hiltbrunner, & Fischer, 1989). Induction of transient TCS aphasia during cortical mapping of individuals with seizure disorders is associated with multiple sites along the posterior superior and middle temporal gyri in classical Wernicke’s area. Electrical interference mapping studies indicate that sparing of Wernicke’s area is not a necessary condition for TCS aphasia (Boatman et al., 2000). MTC aphasia is analogous to global aphasia, with reduced or absent spontaneous speech, severely impaired language comprehension, and preserved repetition with consequent echolalia (Albert, Goodglass, Helm, Rubens, & Alexander, 1981). Lesion sites include both anterior and posterior left hemisphere cortical association areas, sparing the perisylvian language core and producing an “isolation of the speech area” (Rapcsak, Krupp, Rubens, & Reim, 1990).

73.2.7 Subcortical Aphasias

Subcortical aphasias can be classified into three groups: striato-capsular aphasia; thalamic aphasia; and aphasia associated with white matter paraventricular lesions (Kuljic-Obрадовић, 2003). Preservation of repetition is common to all three subtypes, although there are features specific to each subtype. Striato-capsular aphasia and aphasia associated with paraventricular lesions are characterized by impairment of fluency, semantic paraphasias, and generally preserved comprehension. Thalamic aphasia is characterized by impaired comprehension and naming with fluent output containing predominantly semantic paraphasias (Benson & Ardila, 1995; Demonet, 1997). The role of subcortical structures in language remains controversial and different mechanisms have been offered to explain how such subcortical lesions can lead to aphasia. Some ascribe a direct role to subcortical areas in language function (Cappa, Cavallotti, Guidotti, Papagno, & Vignolo, 1983; Damasio, Damasio, Rizzo, Varney, & Gersh, 1982). Others contend that subcortical lesions lead to aphasia through diaschisis—dysfunction of a remote area of cortex caused by impaired input from the subcortical region (Perani, Vallar, Cappa, Messa, & Fazio, 1987; von Monakow, 1914). For example, unilateral lesions of the ventromedial thalamic nucleus produce a major ipsilateral metabolic depression in the cortex and adjacent thalamic nuclei, bilateral metabolic reduction in the basal ganglia, and relatively minor effects in the contralateral cortex (Girault, Savaki, Desban, Glowinski, & Besson, 1985). Language impairments in nontalamic subcortical infarcts have been attributed to hypoperfusion of cortical structures caused by stenosis or occlusion of a large cerebral vessel responsible at the same time as the subcortical infarct (Hillis et al., 2002).

73.2.8 Variability of Vascular Syndromes

These classic aphasia classifications have also been reviewed in detail by Damasio (1992), Goodglass (1993), and Hillis (2007). The early accounts of vascular syndromes used the lesion method in which abnormal behavior is documented in the context of brain pathology, and localization of normal function is extrapolated by assuming that the lesioned area was responsible for whatever function was impaired at the time of assessment (Benson, 1994). It is important to note that individual variability in the shape of the brain as well as the patterns of sulci and gyri renders only approximate localization of cytoarchitectural fields, so even lesions that appear to be in the identical area may not affect identical functions (Amunts et al., 1999; Rademacher, Caviness, Steinmetz, & Galaburda, 1993). Furthermore, human vasculature varies considerably, more so in disease states, so that blockade of a particular branch does not reliably affect the identical areas (Caplan & Van Gijn, 2012). Not surprisingly, studies have variably confirmed the relationship between vascular territories and the vascular syndromes (Gavrilescu & Kase, 1995; Kumral, Bayulkem, Eyyapan, & Yunten, 2002). Furthermore, recovery from aphasia varies substantially among patients (Hochstenback, den Otter, & Mulder, 2003; Lazar, Speizer, Festa, Krakauer, & Marshall, 2008). Depending on the time of evaluation, a person with damage to an entire vascular territory might present...
with the entire vascular syndrome, a partial syndrome, or none of it (if the person recovered completely). One study showed that the vascular syndromes correspond to vascular territories more reliably in the acute than in the chronic stages of stroke (Ochfeld et al., 2010). In acute stroke, language impairments reflect the entire dysfunctional tissue (both infarcted and hypoperfused tissue) not always visible on structural imaging.

Beginning in the 1980s, developments in functional neuroimaging, including PET, functional MRI (fMRI), and magnetoencephalography, expanded understanding of the functional neuroanatomy of language. Safe, noninvasive imaging of the brain reveals that areas in both hemispheres of the brain are activated specifically during language tasks, although the left hemisphere shows more activation in the majority of neurologically normal adults (Binder, 1997; Crinion, Lambon Ralph, Warburton, Howard, & Wise, 2003; Fridriksson & Morrow, 2005; Petersen, Fox, Posner, Mintun, & Raichle, 1988), and that more distant areas of the cortex, such as inferior and anterior temporal cortex (Wise, 2003) and the basal ganglia and thalamus (Kraut et al., 2002), are also activated during language tasks.

### 73.3 VASCULAR SYNDROMES AND CONTEMPORARY PARADIGMS

How do the vascular syndromes fit with more contemporary frameworks of the functional neuroanatomy of language that postulate a dual stream of language processing (Hickok & Poeppel, 2000, 2004, 2007)? The brain is assumed to compute a transformation between thought and an acoustic signal (Schwartz, Faseyitan, Kim, & Coslett, 2012), and it executes parallel processing to synthesize input via interconnected neural networks (Ueno et al., 2011). In the neuroanatomical model of speech processing, proposed by Hickok and Poeppel (2007), speech perception involves auditory-responsive areas in the superior temporal gyrus bilaterally, left more so than right. The processing system then diverges into two streams. The ventral stream is a sound-meaning interface responsible for processing speech signals for comprehension. In the dorsal stream, acoustic speech signals are translated into articulatory representations essential for speech development and production involving auditory-motor integration. These streams are also thought to be bidirectional; the ventral stream mediates the relationship between sound and meaning for perception and production, and the dorsal system can also map motor speech representations onto auditory speech representations (Hickok & Poeppel, 2004, 2007).

When a map of the vascular territories is superimposed onto this neuroanatomical model of speech processing (Figure 73.1), one can see that the dorsal stream is supplied by the superior division of the left MCA, and the ventral stream is supplied largely by the inferior division of the left MCA. Therefore, it is not surprising that individuals with the vascular syndrome of Broca’s aphasia typically have deficits that can be attributed to disruption of the dorsal stream: the articulatory network or sensorimotor interface. Likewise, it is not surprising that individuals with the vascular syndrome of Wernicke’s aphasia have deficits that can be attributed to the lexical interface and/or combinatorial network to map sound onto meaning. The repetition deficit in conduction aphasia is attributed to damage in the Sylvian parietal–temporal (Spt) area, which is located within the Sylvian fissure at the parietal–temporal boundary, reflecting disruption of the dorsal stream route (Hickok & Poeppel, 2004). This hypothesis is supported by fMRI data that show that the region of maximal overlap in lesion distribution in 14 conduction aphasics includes area Spt (Buchsbaum et al., 2011). This is consistent with recent evidence that suggests that conduction aphasia is caused by damage to the left superior temporal gyrus and/or the left supramarginal gyrus (Axer et al., 2001; Baldo & Dronkers, 2006), both of which are anatomical regions associated with the dorsal stream (Schwartz et al., 2012).

Further support is found in studies using computational models to synthesize aphasia. Conduction aphasia is produced after damage to the dorsal pathway in a neuroanatomically constrained computational dual dorsal-ventral pathway computational model (Ueno et al., 2011). Other relevant work includes voxelwise lesion behavior mapping to investigate the association of language impairments and dorsal-ventral streams. Although these studies do not typically associate lesions in particular voxels with specific aphasia syndromes, findings are that deficits in naming and repetition are associated with the dorsal stream (Hanley, Kay, & Edwards, 2002; Ueno et al., 2011) and deficits in comprehension are associated with the ventral pathway (Hillis & Caramazza, 1991b). Finally, it should not be surprising that unilateral vascular syndromes do not typically disrupt the conceptual system, which is bilaterally and widely represented in this model. Rather, conceptual meaning is disrupted in neurodegenerative disease, such as semantic variant primary progressive aphasia.

### 73.4 COGNITIVE PROCESSES UNDERLYING APHASIA

What is lost by characterizing an individual as an exemplar of a particular vascular syndrome? As noted, each vascular syndrome is a collection of frequently
co-occurring impairments to functions that depend on an area of brain supplied by a particular blood vessel. In the past, these deficits were characterized as impairments to particular tasks (e.g., sentence repetition). However, it is now understood that each language task comprises a number of cognitive representations and processes that might depend on different areas of the brain. For example, some of the controversy regarding the anatomical correlates of conduction aphasia may have arisen by characterizing patients as having impaired sentence repetition, rather than identifying what cognitive processes underlying sentence repetition were impaired in individual patients. Cognitive models of language processing include both a semantic and nonlexical phonological mechanism for word and sentence repetition (Hanley et al., 2002). Evidence for this model is found in case reports of performance on language tasks in individuals with neurologic impairments (Hanley, Dell, Kay, & Baron, 2004; Hillis & Caramazza, 1991b). More recently, in a study of sentence recall in healthy adults and an individual with aphasia, phonological information was judged to contribute to sentence recall performance as a complement to semantic and conceptual information (Schweppie, Rumer, Bormann, & Martin, 2011). Some patients may have a disrupted phonological short-term storage system, whereas others may have a disrupted semantic working memory system or disrupted "central executive" component of working memory as the cause of impaired sentence repetition. Each of these impairments might be associated with different lesion sites. Error rates on syllable and pseudoword repetition tasks were increased with inhibitory transcranial magnetic stimulation (TMS) to the posterior part of the superior temporal sulcus and temporoparietal junction in normal volunteers, implicating the role of the dorsal pathway. Differences in error rates were not seen during sentence repetition tasks, suggesting that the ventral pathway may have been recruited for sentence repetition (Murakami, Kell, Restile, Ugawa, & Siemann, 2013).

73.5 POTENTIAL USEFULNESS OF VASCULAR SYNDROMES

73.5.1 Aphasia Research

Grouping patients for research assumes that individuals in the group are homogeneous with respect to something of theoretical interest. The vascular syndromes are frequently co-occurring symptoms.
Individuals with Broca’s aphasia often have both apraxia of speech and more difficulty naming verbs than nouns (Miceli et al., 1984); they also often have weakness of the right arm. These symptoms may all stem from damage to the posterior frontal lobe, supplied by the left superior division MCA. But they do not have a common underlying cause, and they dissociate in individuals. It has been argued that because only a subset of deficits in a vascular syndrome is likely to be present in an individual (depending on the portion of the vascular territory affected by stroke, the degree of recovery, individual variation in anatomy), the syndrome approach has limited usefulness for aphasia research. For example, patients in the group “Broca’s aphasia” might each have different disruptions to cognitive processes underlying articulation and/or sentence production (Caramazza & Badecker, 1989), all due to lesions in areas supplied by the superior division of the left MCA. Alternatives include single subject designs (Caramazza & Coltheart, 2006) and case series analysis (Schwartz & Dell, 2010), or grouping by cognitive impairment (DeLeon et al., 2007). However, classification by vascular syndrome remains the standard basis for describing groups of participants in aphasia research.

### 73.5.2 Treatment of Stroke

Furthermore, grouping patients by affected vascular territory may be quite useful for a stroke neurologist. For example, grouping by vascular aphasia syndromes allowed the discovery that inferior division MCA strokes (associated with Wernicke’s aphasia) were more likely due to cardioembolism (Bogousslavsky, Van Melle, & Regli, 1989), and superior division MCA strokes (associated with Broca’s aphasia) were more likely due to carotid dissection and carotid occlusion (Trupe et al., 2013). Because it is often essential to initiate treatment before the lesion is seen on imaging, a good idea of the volume and location of the affected territory is useful in planning intervention. Furthermore, acute presentations of vascular aphasia syndromes provide the stroke neurologist with information about the likely areas of brain that are dysfunctional (either infarcted or hypoperfused). When there is mismatch between the vascular aphasia syndrome and the structural lesion seen on diffusion-weighted imaging (DWI; which is very sensitive to ischemic tissue even early in stroke), the stroke neurologist knows there is brain tissue that is hypoperfused and at risk for further ischemia. This “diffusion-clinical mismatch” is frequently a prompt for intervention (Reineck, Agarwal, & Hillis, 2005). To illustrate, the patient whose scans are shown in Figure 73.2 had Wernicke’s aphasia at onset but only a tiny lesion on DWI in the insula that could not account for his deficits. His “diffusion-clinical mismatch” prompted further imaging with perfusion-weighted imaging (PWI) the same day and intervention to reperfuse Wernicke’s area. His Wernicke’s aphasia resolved by day 3 with restored blood flow to Wernicke’s area.

### 73.5.3 Aphasia Treatment

Is it useful to group patients by vascular aphasia syndrome for treatment of their aphasia? Certainly, there have been treatments described for Broca’s aphasia, Wernicke’s aphasia, and so on (Carlomagno, Zulian, Razzano, DeMercurio, & Marini, 2013; Conklyn, Novak, Boissy, Bethoux, & Chemali, 2012; Helm-Estabrooks, Fitzpatrick, & Barresi, 1982; Helm-Estabrooks & Ramsberger, 1986). Treatment on the basis of syndrome classification alone would obviously neglect important individual differences known to exist in each of the aphasia syndromes as well as
personal preferences of individual patients, vital aspects of therapy consistent with evidence-based practice. In fact, most of these treatments are therapies for particular deficits within each syndrome. For example, in a case series study including individuals with Broca’s, Wernicke’s, and anomic aphasia, a cueing hierarchy was developed to treat anomia due to impaired access to word meaning or word form in all participants (Best et al., 2013). It would be at least as rational in most cases to describe the intervention as a therapy for “constructing a sentence planning frame” (rather than Broca’s aphasia) or “mapping lexical representations to meaning” (rather than Wernicke’s aphasia). Therapy for specific aphasia syndromes may be optimized by considering the respective roles of the ventral and dorsal streams. For example, the ventral pathway could be leveraged by encouraging patients to process the meaning of a target word during a repetition task in the treatment of conduction aphasia (Ueno & Lambon Ralph, 2013).

However, characterizing patients as exemplars of a vascular aphasia syndrome might be useful for neuro-modulatory treatments that are, by hypothesis, directed to particular sites of the brain. Aphasia treatment is just beginning to take advantage of targeted brain-based interventions, such as transcranial direct current stimulation (tDCS) and TMS designed to facilitate synaptic plasticity (Mottaghy, Sparing, & Topper, 2006; Schlaug, Renga, & Nair, 2008). Most studies have selected patients for treatment on the basis of vascular syndrome (Vines, Norton, & Schlaug, 2011) and have carefully targeted particular areas of the brain based on fMRI (Dmochowski et al., 2013; Fridriksson, Richardson, Baker, & Rorden, 2011) or predetermined regions. However, it is yet to be shown whether the localization of these treatments or the site of lesion/vascular syndrome of patients receiving these treatments (in conjunction with behavioral therapies) is critical to their effectiveness.

73.6 CONCLUSION

The value of classifying aphasias as vascular syndromes continues to be deliberated as the field of aphasiology evolves. The usefulness of classification depends on the goal. Characterizing someone as having a particular vascular syndrome communicates: (i) a set of likely symptoms; (ii) the area of the brain most likely to be affected (at least if the individual has not shown substantial change in either the lesion or the language symptoms since onset); and (iii) the arterial branch most likely to be involved if the cause of aphasia is ischemic stroke. Advances in neuroimaging have allowed refinements in characterizing lesions, so we can now identify areas of the brain associated with impairments in specific cognitive functions underlying language. We are no longer limited to describing the symptoms associated with a vascular territory. New theories of language processing and production complement long-established classification systems, and these novel and emerging perspectives will certainly translate to changes in the conducting of research and treatment of stroke.

Acknowledgments

This publication was made possible by NIH grants R01 DC 05375 and R01 DC 03681 from NIDCD. We gratefully acknowledge this support.

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