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## Phonological, Lexical, Syntactic, and Semantic Disorders in Children

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### Brain Measures Common to the Study of Language Disabilities

A variety of procedures is currently used to investigate brain processes underlying language disabilities. These include functional magnetic resonance imaging (fMRI), MRI, positron emission tomography (PET), magnetoencephalography (MEG), and event-related potentials (ERPs). Each procedure, of course, has its strengths and weaknesses. MRI provides information concerning the morphology of brain structures, whereas fMRI monitors hemodynamic processes, such as changes in brain functions reflected during extended periods of language processing. PET operates in a somewhat similar fashion but tracks the flow of radioactive elements injected into the blood to identify areas actively engaged in a language task. MEG can detect small fluctuations in the brain's magnetic field in response to task demands, and ERPs, a portion of the ongoing EEG that is time locked to the onset of a stimulus event, can reflect rapid changes in the brain's encoding and processing of a speech sound, word, or even sentence. All procedures enable investigators to map linguistic and cognitive functions onto brain structures (Fonaryova Key *et al.*, in press).

Although studies of brain processing usually use one of these procedures, it is clear that much is to be gained from using a combination of procedures. For example, the high temporal sensitivity of ERP techniques can provide a means for determining the sequential relationships that exist between the specific areas of brain activation identified through fMRI (Georgiewa *et al.*, 2002). Moreover, convergence in source localization across fMRI, MEG and ERP procedures ensures that solutions are not biased by particular approaches but may reflect different aspects of what occur in the brain in response to stimulus input or task demands (see Hugdahl *et al.*, 1998).

In general, differences are noted in brain responses and structures for different disabilities (Harter *et al.*, 1988a, 1988b), but there are similarities across disabilities as well. Brain differences could relate to general cognitive processing differences (e.g., attention) that may be impaired in some types of disabilities (Holcomb *et al.*, 1986), or brain differences could reflect the involvement of different structures in response to task demands. Generally, brain structure

and functional differences have been thought to be related to poor language function in general (Molfese and Segalowitz, 1988) and to dyslexia in particular (Eckert *et al.*, 2001; Frank and Pavlakis, 2001). Orton (1937) as well as Travis (1931) held the belief that early signs of lateralization serve to identify children at risk for developmental language disorders. More recent investigations continue to indicate that differences in cerebral asymmetry associated with atypical organization of the left hemisphere are a marker for dyslexic children (Heim and Keil, 2004). However, although reports often link hemisphere differences and language disorders, current thinking indicates that the pathology as well as the neurophysiology of developmental language disabilities are a great deal more complex than originally thought and extend well beyond the classically defined language areas of the brain (Eden *et al.*, 1996). For example, some point to the neural circuitry to account for brain organizational differences between impaired and nonimpaired children, as well as between children with different types of language disabilities (Eden *et al.*, 1996; Leonard *et al.*, 2002; Sarkari *et al.*, 2002). For example, dyslexic readers fail to exhibit the usual network of anterior and posterior brain areas over left hemisphere regions, whereas children with attention deficit hyperactivity disorder appear to have an abnormality in the prefrontal and striatal regions.

For the purposes of this present chapter, the review of brain structures and functions involved in language disabilities is limited to autism, developmental dyslexia, Down syndrome, specific language impairment (SLI), and Williams syndrome. Links between brain and behavior in these developmental disabilities are highlighted.

### Autism

Autism is a neurodevelopmental disorder characterized by impairments in language, communication, imagination, and social relations (American Psychiatric Association, 1994). Estimates of occurrence in the general population range from approximately 1 in 200 to 1 in 1000 (Fombonne, 1999). Although nearly 25% of children with autism have essentially normal vocabulary and grammatical abilities (Kjelgaard and Tager-Flusberg, 2001), another 25% may remain mute for their entire lives (Lord and Paul, 1997). Many underlying language problems found in autistic children are believed to be linked to social and emotional deficits. Although the leading causes of autism

remain unknown, the interplay of multiple genes with multiple environmental factors is considered a factor (Akshoomoff *et al.*, 2002).

### **General Brain Imaging Results for Autism**

Most imaging studies of children with autism are carried out with sedated children and thus focus on brain structures rather than functional differences (Rapin and Dunn, 2003). Even so, structural differences noted in autistic populations are often contradictory. The most consistent findings include increased cerebellar hemisphere, parieto-temporal lobe, and total brain volume. Current research findings also show that the size of the amygdala, hippocampus, and corpus callosum may differ from that of normals (see Brambilla *et al.*, 2003, for a review).

### **Social – Brain Difference and Autism**

Neurologically, many of the social aspects of language acquisition (e.g., social orienting, joint attention and responding to emotional states of others) are tied to differences in the medial temporal lobe (amygdala and hippocampal), which is larger in autistic children than in age-matched controls (Brambilla *et al.*, 2003; Sparks *et al.*, 2002). This brain region is thought to be related to performance on deferred imitation tasks – a skill that may be important in language acquisition (Dawson *et al.*, 1998). Further, the increase in amygdala size may have consequences for important skills such as discriminating facial expressions (Adolphs *et al.*, 1995; Whalen *et al.*, 1998) and joint attention (Sparks *et al.*, 2002). Both skills appear to be important for language acquisition and are often impaired or absent in autism.

### **Phonology and Autism**

A shift in the latency of the first positive peak in the ERP (P1) and the following first large negative peak (N1) to speech sounds in typically developing children is believed to result from maturational changes related to synaptogenesis, myelinogenesis, and dendritic pruning, possibly reflecting cortical auditory system maturation (Bruneau *et al.*, 1997; Eggermont, 1988; Houston and McClelland, 1985). Findings with autistic children for these two ERP peaks are mixed: some studies report longer N1 latencies in children with autism (Dunn *et al.*, 1999; Seri *et al.*, 1999), whereas others report shorter N1 latencies with autistic children (Oades *et al.*, 1988) or no differences between autistic and control children (Kemner *et al.*, 1995; Lincoln *et al.*, 1995).

Using MEG, the N1 correlate is the M100 or N1m. Gage *et al.* (2003) found that the M100 shifted in latencies in the left and right hemispheres with age

for typically developing children listening to tones but occurred only in the left hemisphere for autistic children. This neural activation was localized to the supratemporal sites, reflecting activity of the auditory cortex. Overall, children with autism also exhibited delayed M100 latencies compared to controls, indicating a fundamental difference in the auditory processing of autistic children.

### **Semantics and Autism**

When given a semantic (meaning) categorization task, autistic children exhibit no differences in the N400 between deviant and target words, unlike age-matched controls (Dunn *et al.*, 1999). Surprisingly, autistic children's categorizing errors were not higher than controls, indicating that although the autistic children could categorize based on semantics, they could not attend to the global context and could not discern that one ending was more common than another. As a result, their brains appeared not to process out-of-category words as 'deviant.'

Many autistic children have limited word knowledge and limited comprehension of meaning in connected speech (Dunn *et al.*, 1999). Mental words, such as 'think', 'believe,' and 'know,' are rarely part of the autistic child's vocabulary (Happe, 1995), which is speculated to be caused by differences in the limbic system and as reflecting consistent with the problems of these children in processing emotional information (Dawson *et al.*, 1998).

### **Dyslexia**

Developmental dyslexia refers to the abnormal acquisition of reading skills during the normal course of development despite adequate learning and instructional opportunities and normal intelligence. Estimates are that 5–10% of school-age children fail to learn to read normally (Habib, 2000). Dyslexia can exist in isolation, but more commonly it occurs with other disabilities, such as dyscalculia (mathematic skill impairment) and attention deficit disorder, both with and without hyperactivity. Studies of dyslexia usually indicate the involvement of left-hemisphere perisylvian areas during the reading process. The specific areas identified vary somewhat depending on the component of reading being engaged in but overall the extrastriate visual cortex, inferior parietal regions, superior temporal gyrus, and inferior frontal cortex appear to be activated.

When one examines specific skills, visual word form processing is associated with occipital and occipitotemporal sites, whereas reading-relevant phonological processing has been associated with superior temporal, occipitotemporal, and inferior frontal

sites of the left hemisphere. However, there is some variation in the scientific reports. For example, although some studies report a hemisphere asymmetry in the area of the planum temporale related to dyslexia (Frank and Pavlakis, 2001), others report no such effect (Heiervang *et al.*, 2000).

A number of studies have identified brain anatomical differences that distinguish dyslexic from normal brains (see Hynd and Semrud-Clikeman [1989] for an earlier review). For example, Eckert *et al.* (2003), using MRI scans, reported that dyslexics exhibited significantly smaller right anterior lobes of the cerebellum, pars triangularis bilaterally, and brain volume than controls. Correlation analyses showed that these neuroanatomical measurements relate to reading, spelling and language measures of dyslexia (see also Grunling *et al.*, 2004). Although earlier studies report hemisphere differences in the region of the planum temporale between dyslexics and controls, more recent studies investigating the morphology of the perisylvian cortical area in a clinical sample of children failed to find morphological differences at this locale that were associated with the diagnosis of dyslexia (Hiemenz and Hynd, 2000). Scientists have also reported differences between dyslexics and controls in the corpus callosum – the band of fibers connecting the two hemispheres (von Plessen *et al.*, 2002). These researchers reported differences in the posterior midbody/isthmus region that contains interhemispheric fibers from primary and secondary auditory cortices – a finding that converges with other reports of developmental differences during the late childhood years, coinciding with reading skill development.

#### General Brain Imaging Results for Dyslexia

There are general consistencies across phonological, semantic, and syntactic processing in that enhanced activation of the left extrastriate cortex is found when visuospatial, orthographic, phonologic, and semantic processing demands are placed on the dyslexic group (Backes *et al.*, 2002).

Researchers argue that variations in brain processing relate to language and cultural factors – a finding that parallels behavioral investigations of language differences. For example, using fMRI, Siok *et al.* (2004) reported that functional disruption of the left middle frontal gyrus is associated with impaired reading of the Chinese language (a logographic rather than alphabetic writing system). No disruption was found for the left temporoparietal brain regions. Siok *et al.* argue that such differences reflect two deficits during reading: the conversion of the orthography (characters) to syllables, and the mapping of the orthography onto the semantics. Both processes, the

authors argue, are mediated by the left middle frontal gyrus that coordinates and integrates various information about written Chinese characters in verbal and spatial working memory (see also Eckert *et al.*, 2001; Grigorenko, 2001).

#### Phonology and Dyslexia

Dyslexic readers show less activation of both the temporal and the prefrontal cortex during phonologic processing (Backes *et al.*, 2002). Intriguingly, similar areas of lowered activation are seen in other populations with reading problems, reinforcing the notion that inferior frontal and superior temporal brain areas support reading skills (e.g., neurofibromatosis; see Backes *et al.*, 2002).

When magnetic source imaging (MSI) was employed during phonological tasks, Papanicolaou *et al.* (2003) reported consistent brain maps across children that differentiate between dyslexic and nondyslexic children in the left and right posterior temporal regions. Moreover, following reading interventions with the dyslexic children, brain sources shifted from the right to the left hemisphere, indicating that intervention ‘normalizes’ as the child’s brain moves from an ineffective to a more efficient use of brain structures and pathways (Simos *et al.*, 2002; for replication, see Temple *et al.*, 2003).

MEG investigations into the perception of speech cues such as voice onset time (VOT) indicate that children with dyslexia experienced a sharp peak of relative activation in right temporoparietal areas between 300 and 700 milliseconds poststimulus onset, a point markedly later in time (~500 milliseconds) relative to normal readers. This increased late activation in right temporoparietal areas was correlated with reduced performance on phonological processing measures (Breier *et al.*, 2003). Further, there are data indicating an early relation between the perception of speech cues in early infancy and the emergence of reading disorders as late as 8 years of age (Molfese & Molfese, 1985; Molfese, 2000; Molfese *et al.*, 2005; Lyytinen *et al.*, 2003). These studies indicate that infants who go on to develop normal language skills generate ERPs over left frontal and temporal brain regions that discriminate between speech sounds, whereas ERPs collected from infants at risk for developing a reading disorder fail to discriminate between these same sounds.

In phonological related tasks such as rhyming, fMRI differences are found between dyslexic and control children (Corina *et al.*, 2001). During phonological judgment, dyslexics generated more activity than controls in right than left inferior temporal gyrus and in left precentral gyrus (see Georgiewa *et al.* [1999] for replication). During lexical judgment, dyslexics

showed less activation than controls in the bilateral middle frontal gyrus and more activation than controls in the left orbital frontal cortex. In an ERP study paralleling this study, Lovrich *et al.* (1996) reported that rhyme processing produced more pronounced group differences than semantic processing at about 480 milliseconds, with a relatively more negative distribution for the impaired readers at centroparietal sites. By 800 milliseconds, the impaired readers displayed a late positivity that was delayed in latency and that was of larger amplitude at frontal sites than that for the average readers.

When brain activation patterns were studied in dyslexic and nonimpaired children during pseudo-word and real-word reading tasks that required phonologic analysis, differences were noted in posterior brain regions, including parietotemporal sites and sites in the occipitotemporal area. Reading skill overall was positively correlated with the magnitude of activation in the left occipitotemporal region – an area similarly found to discriminate between adult groups of readers and nonreaders (Shaywitz *et al.*, 2002). A similar effect was demonstrated using MEG (Simos *et al.*, 2000).

### Semantics and Dyslexia

During lexical judgment, less activation in bilateral middle frontal gyrus and more activation in left orbital frontal cortex occurred for dyslexic compared to nondyslexic children (Corina *et al.*, 2001). In a related task, in which children read words and pronounceable nonwords, fMRI results detected a hyperactivation of the left inferior frontal gyrus in dyslexic children. ERPs collected from the same children converged with the fMRI findings and showed topographic difference between groups at the left frontal electrodes in a time window of 250–600 milliseconds after stimulus onset. A related study by Molfese *et al.* (in press) reported similar findings, as well as a slower rate of word processing over left hemisphere electrode sites in dyslexic children compared to normal and advanced readers.

### Reading and Dyslexia

Relatively few studies have investigated brain activation when the child is reading continuous text (Backes *et al.*, 2002). One exception is a report by Johnstone *et al.* (1984), who monitored silent and oral reading, noting that reading difficulty affected the central and parietal ERPs of dyslexics but not the controls. In addition, different patterns of asymmetry were found for the two groups in silent compared to oral reading at midtemporal placements.

## Down Syndrome

Down syndrome (DS) is characterized by a number of physical characteristics and learning impairments, as well as IQ scores that may range from 50 to 60. Individuals with DS typically are microcephalic and have cognitive and speech impairments, as well as neuromotor dysfunction. In addition, problems generally occur in language, short-term memory, and task shifting. Typical language problems involve delays in articulation, phonology, vocal imitation, mean length utterance (MLU), verbal comprehension, and expressive syntax. Spontaneous language is often telegraphic, with a drastic reduction in the use of function words: articles, prepositions, and pronouns (Chapman *et al.*, 2002). Language deficits may arise from abnormalities noted within the temporal lobe (Welsh, 2003). Individuals afflicted with DS commonly suffer from a mild to moderate hearing loss (78% of DS children have a hearing loss; Stoel-Gammon, 1997), which may partially account for the delay in phonological processing and poor articulation.

DS occurs in approximately 1 in 800–1,000 live births. Ninety to 95% of cases are caused by a full trisomy of chromosome 21, and 5% result from translocation or mosaicism. Considerable individual variability exists in cognitive development among those afflicted, with the greatest deficits in development observed with full trisomy-21, where specific genes have been associated with brain development, specifically the cerebellum development, and produce Alzheimer-type neuropathology, neuronal cell loss, accelerated aging, and so on (Capone, 2001). Individuals with DS commonly exhibit neuropathology resembling that seen in Alzheimer disease, with some patients showing symptoms beginning as early as age 35 years.

### General Brain Imaging Results for DS

Brains of DS individuals appear to have a characteristic morphologic appearance that includes decreased size and weight, a foreshortening of the anterior–posterior diameter, reduced frontal lobe volume, and flattening of the occiput. The primary cortical gyri may appear wide, whereas secondary gyri are often poorly developed or absent, with shallow sulci and reduced cerebellar and brain stem size (Capone, 2004). MRI studies indicate a volume reduction for the whole brain, with the cerebral cortex, white matter, and cerebellum totaling 18% (Pinter *et al.*, 2001a). Hippocampal dysfunction occurs in DS (Pennington *et al.*, 2003), perhaps because of the reduced size of the hippocampus, as determined by MRI (Pinter *et al.*, 2001b), and the cerebral cortex

has fewer neurons at all cortical layers. In addition, dendritic spines appear longer and thinner than in matched controls (Capone, 2004; Seidl *et al.*, 1997). Studies using MEG indicate atypical cerebral specialization, showing a greater activation of the right hemisphere in DS when compared to normal controls (Welsh, 2002). This greater activation is confirmed by PET studies (Nadel, 2003), indicating that the brain of the DS individual is working harder to process information, although less effectively.

Brain morphology in DS does not differ dramatically from normals throughout the first 6 months of life. Delayed myelination occurring within the cerebral hemispheres, basal ganglia, cerebellum, brain stem, and nerve tracts (fibers linking frontal and temporal lobes) occurs after 6 months (Nadel, 2003; Capone, 2004). Other critical periods of brain development affected by DS include neuronal differentiation, proliferation, and organization. A reduction in neuronal number and density was noted for most brain areas examined, specifically within interneurons and pyramidal neurons. However, this differs on a case-to-case basis and has been hypothesized as a potential explanation for the spectrum of neurodevelopment impairment observed (Capone, 2004).

#### Phonology and DS

Research of neural function indicates that in DS there may be a delay in the development of the auditory system (Nadel, 2003). Phonological delays exhibited in DS cases are often linked to differences in anatomy and central nervous system development in DS. In addition, limits on auditory working memory and hearing may account for deficits observed in phonological processing (Tager-Flusberg, 1999).

#### Semantics and DS

Dichotic listening tasks involving DS children generally result in a left-ear advantage, indicating that these individuals use their right hemisphere to process for speech (Welsh, 2002). On the basis of such findings, Capone (2004) argued that difficulties in semantic processing in DS occur from a reduction in cerebral and cerebellar volume. In addition, the corpus callosum is thinner in the DS brain in the rostral fifth, the area associated with semantic communication. Welsh (2002) speculated that the thinner corpus callosum isolates the two hemispheres from each other, making it more difficult to integrate verbal information.

Vocabulary growth in DS children is delayed increasingly with age (Chapman, 2002). Studies using dichotic listening tasks report a left-ear advantage for DS, indicating that lexical operations are carried out primarily in the right hemisphere, a finding opposite

to that found with normal developing children. In fact, individuals with DS who exhibit the most severe language deficits demonstrate the most atypical ear advantage (Welsh, 2002).

#### Syntax and DS

Children with DS exhibit a delay in syntax production that generally becomes evident with the emergence of two-word utterances, and syntax is often more severely impaired than lexical development (Chapman, 1997). Verbal short-term memory may be affected, limiting the ability to understand syntactic relations. Research on short-term memory points to hippocampal dysfunction in DS children (Pinter *et al.*, 2001a). MRI studies of adults with DS highlight the possibility that reductions in volume size observed in DS may contribute to the development of language and memory deficits. It has been hypothesized that the cause of language deficits observed in children with DS are primarily related to memory and learning and are most associated with deficits observed in the hippocampal region (Nadel, 2003).

#### Specific Language Impairment

It is estimated that approximately 7% of the 5-year-old population is characterized with specific language impairment (SLI), and that SLI is three times more likely in males than females. The basic criteria underlying this disorder include normal intelligence (IQ of 85 or higher), language impairment (language test score of  $-1.25$  sd (standard deviation) or lower), no neurological dysfunctions or structural anomalies, successful completion of a hearing screening, and no impairment in social interactions. Speculations as to causes focus on the biological and environmental issues, but with no resolution. Because of the heterogeneity of the phenotype, it is difficult to study this population as a single unit (Leonard, 1998). As a consequence, results and conclusions resulting from any particular study are limited to the specific subset of SLI under study.

#### Phonology and SLI

A phonological processing delay exists in children with SLI, where the children have a problem distinguishing similar spoken sounds (i.e., /b/ vs. /p/) from one another, as well as show lower accuracy in processing speech sounds at rapid ISI (interstimulus intervals). Improvement occurs with age in SLI children; however, the plateau reached is still below normal levels. ERP patterns of older SLI children in comparison with same-age and younger control children show a correlation in brain wave patterns to that of the younger population in response to auditory tone

presentation. An auditory immaturity hypothesis is indicated as a basis for the delay in phonological processing in SLI. This hypothesis points to the auditory system as the basis for developmental delays found in SLI children (Bishop *et al.*, 2004). In fact, an fMRI study showed that individuals with SLI had less activation in brain regions specific to language processing as well as phonological awareness (Hugdahl *et al.*, 2004). Furthermore, MMN (mismatch negativity), a region of the ERP that is an indicator of stimulus discrimination, indicates a deficit in discrimination of CV (consonant–vowel) syllables that differ in the place of articulation in SLI children (Uwer *et al.*, 2002). Infants as early as 8 weeks of age who are at risk for SLI are already showing MMN delays in their latency response when presented with auditory speech sounds (Friedrich, 2004). These findings indicate that delays in discrimination skills are present from an early stage of development.

### **Semantics and SLI**

Semantic abilities are problematic in SLI. Investigations into the neural substrate of these issues have made some headway in recent years. In particular, the N400 (Kutas and Hillyard, 1980), a large negative component of the ERP that correlates with semantic ability and occurs approximately 400 milliseconds after a stimulus begins, is altered in populations of SLI children, as well as in their parents. This brain component is enhanced in fathers of SLI children compared to controls in response to the unexpected ending of a sentence (Ors *et al.*, 2001). For example, the N400 response is normally larger in response to the last word in the sentence, ‘The train runs on the banana’ than if the final word is ‘track.’ Atypical N400 amplitudes also are found in children with other language deficits (Neville *et al.*, 1993). MEG studies have pinpointed the lateral temporal region as the origin of the N400 response (Simos *et al.*, 1997). Intracortical depth recordings in response to written words point to the medial temporal structures near the hippocampus and amygdala (Smith *et al.*, 1986).

### **William's Syndrome**

William's-Beuren syndrome (WS) results from a rare genetic deficit (about 1 in 20 000 births) caused by a microdeletion on chromosome 7 (Levitin *et al.*, 2003). This genetic etiology present in WS allows researchers to identify developmental abnormalities associated with WS from birth. Characteristics of WS include dysmorphic facial features, mental retardation, and a unique behavioral phenotype (Bellugi *et al.*, 1999,

2000; Levitin *et al.*, 2003). Recently, Mervis and colleagues (Mervis, in press; Mervis *et al.*, 2003) have formulated a cognitive profile for WS by analyzing the relative weaknesses and strengths often associated with the genetic syndrome. Markers for this profile include a very low IQ and weakness in visuospatial construction, as well as strengths in recognition of faces, verbal memory, and language abilities. These findings have been replicated by other researchers (Galaburda *et al.*, 2003).

### **Anatomical Aspects of WS**

MRI studies note anatomical differences in brain morphology in WS that include a bilateral decrease in the dorsal posterior regions in both hemispheres with an increase in the superior temporal gyrus, frontal lobe, and amygdala (Galaburda *et al.*, 2003). Schmitt *et al.* (2001a) recorded MRI images in 20 individuals with WS (age: 19–44 years) compared to 20 age- and gender-matched participants. In WS adults, the midsagittal corpus callosum was reduced in total area, and within the corpus callosum, the isthmus and splenium were disproportionately smaller. However, the frontal lobe and cerebellum were similar in size to those of controls (Schmitt *et al.*, 2001b). The decrease in volume within the corpus callosum and the parietal lobe has led many researchers to speculate that these findings could explain visuospatial weaknesses in this population (Schmitt *et al.*, 2001a; Eckert *et al.*, 2005). Other studies indicate abnormal clustering of neurons in the visual cortex (Lenhoff *et al.*, 1997). In contrast, the language strength predominately found in WS children may be caused, based on the MRI data, by the relatively unimpaired frontal lobe and cerebellum and the enlarged planum temporale (auditory region), particularly in the left hemisphere (Lenhoff *et al.*, 1997; Bellugi *et al.*, 1999, 2000).

### **Semantics and WS**

Studies indicate that WS children are capable of semantic organization, although the onset is often delayed (Mervis and Bertrand, 1997; Mervis, in press). WS children tend to list low-frequency words when asked to complete the task (Mervis, in press). In studies of lexical and semantic processing, unique ERP patterns are recorded from WS children to auditory stimuli during a sentence completion task that includes anomaly words at the end of the sentence (Bellugi *et al.*, 2000). In general, the expected component at N400 associated with anomaly words in WS was more evenly distributed across the scalp, with no hemispheric interaction (Bellugi *et al.*, 2000). This finding is unusual, given the left-hemisphere activation common in typically developing children (Bellugi

*et al.*, 2000). In addition, during the positive peak at 50 milliseconds, WS individuals produced an abnormally large spike. A 'smaller than normal' negative peak at 100 milliseconds and a large positive peak at 200 milliseconds can be seen in the WS population, but not within normal controls (Bellugi *et al.*, 1999).

### Phonology and WS

Little research has examined the neural bases for phonological processing in children with WS. A current study by Fornaryova Key *et al.* (in progress) examined the brain's response to speech syllables (/ba/ and /ga/) in eight children with WS (age: 4.03–4.64 years). The results indicate that the left hemisphere of WS children is engaged in discriminating between different speech sounds, rather than showing the lack of hemisphere differences that Bellugi *et al.* (2000) would predict. In addition, variations in the second large positive ERP component (P2) to speech sounds correlated highly with a range of language and verbal abilities, such as those needed for performance on the Matrices subtest of the K-BIT.

### Syntax and WS

In normal, age-matched controls, ERP responses to nouns, adjectives, and verbs (or open-class words) tend to invoke a N400 peak in the right posterior lobe. Words like articles, conjunctions, and prepositions (or close-class words) elicit an early negativity peak in the anterior portion of the left hemisphere (Bellugi *et al.*, 1999). Using ERPs to open and closed-class word stimuli, WS subjects do not display the typical evoked pattern at the N400 peak for open-class words in the right hemisphere, but instead, a negativity in the left hemisphere was found (see Bellugi *et al.*, 1999). For closed-class words, the typical left-hemisphere pattern found in normal subjects is not found in individuals with WS (Bellugi *et al.*, 1999). These findings indicate that the neural functional organization for syntactic processing is different for individuals with WS, even though results from MRI studies report similar frontal lobe and cerebellum sizes to matched age and sex controls (Bellugi *et al.*, 1999, 2000).

### Summary and Conclusion

Across the five developmental disability areas reviewed here, much is already known about the underlying neural bases for some impaired phonological processes, but exceptionally little is known concerning the neural underpinnings of other deficits involving syntactical processing. At the same time, in areas where some research is available, it is evident that language deficits are not unique to a single syndrome and do not result

from the dysfunction in a single, discrete brain structure. Rather, language disorders are multidimensional and involve neural processes that arise out of complex interactions between multiple cortical brain regions, and neural pathways, as well as from genetic factors whose phenotypic expression is mitigated through dynamic environmental factors. There are, no doubt, other as-yet-unknown factors. Clearly, we are still in the earliest stages of our quest to understand the complex relationships that exist between developmental language disabilities and the brain. Lest we get discouraged, it is important to keep in mind is that we at least have begun that quest.

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## Phonology in the Production of Words

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### Phonological Representations in the Mental Lexicon

How are words represented in the brain? Words have a meaning and a form, and presumably these two aspects of words are represented and processed separately in different areas of the brain (for a recent overview see Indefrey and Levelt, 2004). For instance, each act of speech production is planned in advance and starts with the intention to talk about a specific 'meaning' which is to be conveyed to the interlocutor(s). Therefore, the first step in speech production is called conceptualization (Levelt, 1989). In this phase, the content of an utterance is represented as prelinguistic units or concepts. During the next step, called formalization, concepts become lexicalized, i.e., lexical entries corresponding to the concepts are retrieved. Formalization can be divided into two processes, namely, grammatical encoding and phonological encoding (Levelt *et al.*, 1999). This division is based on empirical data, such as speech errors. Garrett (1975) already observed that there are at least two categories of exchange errors, i.e., word exchanges and segment (phoneme) exchanges. An example of a word exchange is *laboratory in your own computer* (Fromkin, 1971); *laboratory* and *computer* belong to different syntactic phrases, but they are of the same syntactic word class, i.e., nouns. Segment or phoneme exchanges, in contrast, typically result from the same syntactic phrase, but from words of different syntactic word classes, e.g., *our queer dean* (instead of *our dear queen*; an original spoonerism). This pattern of word and segment exchanges can be explained by assuming that word exchanges occur during

grammatical encoding, whereas segment exchanges occur during subsequent phonological encoding. During grammatical encoding the syntactic structure of an utterance is specified including the syntactic word class of an individual word, but not its phonological form. That is why words of the same word class are exchanged, no matter what their phonological make up is. In contrast, during phonological encoding the words of an utterance have already been selected, i.e., their syntactic word class information can no longer influence the planning process, but their phonological form is still to be specified. During this specification segments or phonemes from adjacent words can accidentally become active at the same time, and then they can be exchanged and result in a sound error.

In the meantime, on-line experimental evidence for the division between grammatical and phonological encoding has been obtained. Schriefers *et al.* (1990) asked Dutch participants in the laboratory to name pictures while presenting them with auditory distracter words. When the distracter words were semantically, i.e., categorically, related to the target picture name (e.g., *gieter* 'watering can'), participants were slower to name the picture of a rake (*bark*) compared to an unrelated distracter word (e.g., *bel* 'bell') (see Figures 1–3). However, this happened only when the distracter words were presented slightly before picture onset or simultaneously with the picture onset (see Figure 4). When the distracter words were phonologically related to the picture name (e.g., *harp* 'harp'), however, the naming of *bark* was faster than in the unrelated control condition (see Figures 5–7). However, this effect disappeared when the phonologically related distracter words were presented before picture onset (see Figure 8).

The received account for the semantic interference effect (*bark-gieter*) is that the lexical entry *gieter* does