SPECIAL ISSUE

SPECIFIC LANGUAGE IMPAIRMENT IS NOT SPECIFIC TO LANGUAGE: THE PROCEDURAL DEFICIT HYPOTHESIS

Michael T. Ullman and Elizabeth I. Pierpont

(Departments of Neuroscience, Linguistics, Psychology and Neurology Georgetown University, USA)

ABSTRACT

Specific Language Impairment (SLI) has been explained by two broad classes of hypotheses, which posit either a deficit specific to grammar, or a non-linguistic processing impairment. Here we advance an alternative perspective. According to the Procedural Deficit Hypothesis (PDH), SLI can be largely explained by the abnormal development of brain structures that constitute the procedural memory system. This system, which is composed of a network of interconnected structures rooted in frontal/basal-ganglia circuits, subserves the learning and execution of motor and cognitive skills. Crucially, recent evidence also implicates this system in important aspects of grammar. The PDH posits that a significant proportion of individuals with SLI suffer from abnormalities of this brain network, leading to impairments of the linguistic and non-linguistic functions that depend on it. In contrast, functions such as lexical and declarative memory, which depend on other brain structures, are expected to remain largely spared. Evidence from an in-depth retrospective examination of the literature is presented. It is argued that the data support the predictions of the PDH, and particularly implicate Broca's area within frontal cortex, and the caudate nucleus within the basal ganglia. Finally, broader implications are discussed, and predictions for future research are presented. It is argued that the PDH forms the basis of a novel and potentially productive perspective on SLI.

Key words: Specific Language Impairment (SLI), procedural memory, declarative memory, language grammar, lexical memory, syntax, morphology, phonology, working memory, mental imagery, motor function, temporal processing, compensation, basal ganglia, caudate nucleus, Broca's area, fMRI, MRI, ERP

INTRODUCTION

Specific Language Impairment (SLI) is generally defined as a developmental disorder of language in the absence of frank neurological damage, hearing deficits, severe environmental deprivation, or mental retardation (for diagnostic definitions and prevalence of SLI, see Bishop, 1992; Leonard, 1998; Tomblin et al., 1997). Other terms have also been used to label such children, including developmental dysphasia, language impairment, language learning disability, developmental language disorder, delayed speech and deviant language (Leonard, 1998; Ahmed et al., 2001). Several factors have complicated attempts to provide a unified theory of SLI, or even of subgroups of SLI. First, despite the standard use of exclusionary criteria to diagnose SLI, the disorder is clearly not limited to language. Rather, the linguistic impairments co-occur with a number of non-linguistic deficits, including impairments of motor skills and working memory, and with other disorders, such as Attention Deficit Hyperactivity Disorder (Hill, 2001; Leonard, 1998; Tirosh and Cohen, 1998). Second, even though SLI must be a consequence of some sort of neural dysfunction, the neural correlates of the disorder have been largely ignored. This potentially valuable information could provide important constraints on explanatory accounts of the disorder. Third, SLI is a classification that is quite heterogeneous (Leonard, 1998; Stromswold, 2000). Surveys document variation within and across subgroups in the particular aspects of language that are affected and in the types of co-occurring non-linguistic deficits, as well as in the severity with which these linguistic and non-linguistic deficits are found (Aram and Nation, 1975; Miller, 1996; Rapin and Allen, 1988; Stark and Tallal, 1981). Although some previous research has focused on apparently distinct SLI sub-types (Bishop, 2000; Gopnik and Crago, 1991; van der Lely et al., 1998), most studies have paid little attention to variation across individuals with *similar* types of deficits. However, the nature of this type of variability may be important, and may even provide clues to the nature of SLI itself.

Two broad competing theoretical perspectives have attempted to explain SLI. One perspective posits that people with SLI, or at least certain subgroups of individuals with the disorder, suffer from a deficit or delay that is specific to the domain of language, specifically to grammar – that is, to the mental capacity that underlies the rule-governed combination of words into complex structures. This viewpoint has been espoused in numerous flavors, many of which identify particular grammatical operations, mechanisms, or types of knowledge that are problematic. For example, it has been proposed that children with SLI have a selective impairment in establishing structural relationships such as agreement (Clahsen, 1989) or specifier head-relations (Rice and Oetting, 1993). Alternatively, it has been posited that at least certain people with

developmental language impairments may be missing linguistic features (Gopnik and Crago, 1991). Another view is based on the observation that normal children pass through a period in the development of language during which they fail to consistently mark tense in main clauses which require it (Wexler, 1994). According to this "extended optional infinitive" account, children with SLI remain in this stage for a much longer period than normal children, with their language deficits reflecting an incomplete specification of the obligatory tense markings that are normally represented in grammar (Rice et al., 1995).

Some grammar-deficit hypotheses posit that the dysfunction in SLI is quite broad within grammar, rather than being highly specific to a particular grammatical function or operation such as agreement or tense-marking. Thus it has been proposed that a broad range of language impairments may be explained by a deficit that affects the mechanisms underlying the learning and/or computation of implicit grammatical rules (Ullman and Gopnik, 1994). Another account claims that wide-ranging grammatical difficulties can be explained, at least in some children with SLI, by a representational deficit of grammatical relations (van der Lely, 1994; van der Lely et al., 1998).

Grammar-deficit hypotheses have, not surprisingly, been quite successful in accounting for many of the grammatical impairments observed in SLI. However, these hypotheses are also somewhat problematic. Few if any of them – particularly those that posit a highly specific dysfunction – can explain the full range of linguistic deficits, either within a given language or cross-linguistically (Leonard, 1996, 1998). For example, a purely grammatical deficit cannot easily account for the word-finding difficulties often observed in SLI. Even within grammar, such hypotheses may not fully explain the combination of syntactic, morphological and phonological deficits that occur. Moreover, hypotheses positing *only* grammatical deficits cannot account for the non-linguistic difficulties prevalent in SLI.

According to the second broad theoretical perspective, SLI is caused by a non-linguistic processing deficit. Some processing-deficit hypotheses claim that the problems are quite *general* in nature, such as a reduced processing rate, or capacity limitations on processing (Bishop, 1994; Kail, 1994; Leonard et al., 1992b; Norbury et al., 2001). This helps to account for some of the breadth of linguistic *and* non-linguistic impairments observed in SLI. In particular, such hypotheses can explain why children with SLI have difficulties processing verbal and nonverbal stimuli that are rapidly presented or of brief duration, and problems with cognitive tasks such as word retrieval, simultaneous task execution, and phonological discrimination (Leonard, 1998). However, the view that impairments in SLI can be captured by a general processing deficit is also somewhat problematic. First of all, it has been argued that this perspective cannot easily account for certain types of linguistic impairments observed in SLI (Gopnik and Crago, 1991; Rice and Oetting, 1993; Ullman and Gopnik, 1999; van der Lely and Ullman, 2001). In addition, because these hypotheses claim that the deficits are quite general, they have difficulty explaining the apparently selective nature of the non-linguistic impairments (see below, and Leonard, 1998). Finally, a limited processing capacity account does not lend itself well to specific predictions or testable hypotheses, because nearly *any* kind of impairment could potentially be explained by processing limitations or generalized slowing.

Not all processing-deficit hypotheses posit a general deficit. Impairments of a *specific* cognitive or processing mechanism have also been proposed. Some investigators attribute the language impairments in SLI to the dysfunction of phonological working memory (Gathercole and Baddeley, 1990; Montgomery, 1995b), or to an "information processing deficit affecting phonology" (Joanisse and Seidenberg, 1998). Others have argued that the impairments in SLI can be explained by a perceptual or temporal processing impairment, particularly of briefly presented stimuli or rapidly presented sequences of items (Merzenich et al., 1993; Tallal et al., 1993; Tallal and Piercy, 1973b, 1974). On the one hand, these hypotheses can explain certain specific deficits observed in SLI, such as difficulties on tasks involving working memory, phonological processing, or the perception of rapidly presented stimuli. However, it is not clear that *all* children with SLI suffer from these problems (Bishop et al., 1999; Tallal et al., 1991; Tomblin et al., 1995; van der Lely and Howard, 1993). Moreover, such hypotheses cannot easily account for the specific pattern of spared and impaired linguistic and non-linguistic functions in SLI (Hill, 1998; Leonard, 1998; Ullman and Gopnik, 1999; van der Lely and Stollwerck, 1996; van der Lely and Ullman, 2001).

In sum, although previously proposed explanatory hypotheses can individually capture specific aspects of the empirical data, none of them can easily account for either the range or the variation of the particular impaired linguistic and non-linguistic functions found across SLI, and even within SLI subgroups. Indeed, we believe that any purely *functional* account of SLI will have difficulty explaining the variety of impairments that occur even within individuals with SLI. Moreover, few hypotheses have seriously attempted to link the cognitive impairments in SLI to the brain, or to account for the range of neural abnormalities observed in the disorder.

Here we propose that a substantial number of individuals with SLI are afflicted with

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