

Familial aggregation of a developmental language disorder*

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Received March 15, 1990; final revision accepted November 21, 1990

Abstract

Gopnik, M., and Crago, M.B., 1991. Familial aggregation of a developmental disorder. *Cognition*, 39: 1-50.

This paper investigates the etiology of developmental dysphasia and its linguistic properties. Data are presented that suggest that at least some cases of dysphasia are associated with an abnormality in a single dominant gene. The results of a series of tests on a large three-generation family, in which half of the members have dysphasia, are reported. These results show that abstract morphology is impaired in these subjects. It is argued further that the data are consistent with the hypothesis that the dysphasics learn the feature-marked lexical items of language as unanalyzed lexical items. They do not have the underlying capacity to learn language by constructing paradigms.

Introduction

This paper will provide a principled linguistic account of the specific language impairment that has been observed to occur over three generations in 16 out of 30 members of a family. It will be shown that the pattern of the occurrence of the impairment in this family is convergent with other recent reports in the literature describing patterns of familial aggregation (Bloom & Lahey, 1978; Neils & Aram, 1986; Tallal, Ross, & Curtiss, 1989a, 1989b; Tomblin,

*We would like to thank Steve Pinker for his very helpful comments about this work in general and specifically for his editorial assistance on this paper. We should also like to thank Jennifer Gurd and John Marshall for their ongoing consultation and Kevin Dunbar and Bob Bracewell for their help on technical aspects of this work. We also thank Susan Mendelson for her assistance in testing the family and Siobhan Moss for her help in the preparation of this manuscript. This work was supported by grants from the Social Sciences Committee of the Graduate Faculty, McGill University, 943-00-62 and the Social Sciences and Humanities Research Council of Canada, 410-90-1744. Requests for reprints should be sent to M. Gopnik, Department of Linguistics, McGill University, 1001 Sherbrooke St. W., Montreal, Quebec, Canada H3A 1G5.

1989). Furthermore, it will be argued that the data can be accounted for by a hypothesis that an underlying component of the grammar is impaired. It will be shown that alternative hypotheses arguing that the disorder is caused by an impairment in auditory processing, in perceptual saliency, or cognitive processing do not appear to provide a principled account of these data. In order to accomplish this task it will be necessary not only to describe the database upon which this account is founded, but also to discuss some fundamental assumptions about the ways in which such data can be gathered and analyzed. The argument that will be presented is that explanations of language disorders need to be founded not on tallies of "errors" or measures of the subjects' performance on arbitrary tasks, but on data that are determined by our knowledge about the principles underlying language and language acquisition. This paper will provide a description of the family that provides the database; a discussion of the evidence from several different sources that suggests a genetic etiology; the theoretical assumptions underlying the analysis of specific language impairment in general; the results of a detailed analysis of the data from this family; and a discussion of these results and their implication for the development of a linguistically principled account of the disorder.

It is a well-known phenomenon that some children, who appear to be normal in every other respect, are very late in developing language and when they do begin to speak their phonology and their grammar are not normal and do not follow the normal developmental path. The clinical diagnosis requires that this language disorder not be associated with any disorder such as deafness, retardation, autism or any other obvious psychological or physical disorder that could account for this problem (Bloom & Lahey, 1978; Stark, 1980; Wyke, 1978; Zangwill, 1978). Children who conform to this clinical description have been referred to variously as "developmental dysphasics", "specific language impaired" (SLI), or "developmental language impaired". Several people have observed that this diagnosis does not describe a single entity, but is a collective term for a range of abnormal development involving differing aspects of speech and language (Cantwell & Baker, 1987; Leonard, 1987). In this paper, the term "dysphasia" will be used to refer to this heterogeneous population, though in later work it may prove useful to reserve the term "dysphasia" for those cases of impairment that are manifested in familial aggregations and reserve the term SLI for those studies in which the familial aggregation variable is not controlled for. Though speech and language pathologists report that there are many of these children in therapy there is no agreed-upon estimate for the numbers of children thus afflicted. Reports in the literature have described difficulties with grammatical morphemes such as plurals and past tense as one of the most apparent

problems (Cromer, 1978; Crystal, 1987; Crystal, Fletcher, & Garman 1976; Johnston & Schery, 1976; Leonard, 1989; Samples & Lane, 1985; Watkins & Rice, 1989; Wyke, 1978). However, though grammatical morphemes are clearly affected, the precise details of the language impairment are sometimes difficult to ascertain from these reports. Sometimes linguistic errors that occur in the transcripts that are given in the text of the articles are not noted in the list of errors that are cited. Moreover the linguistic significance of the pattern of occurrence of items is often not appreciated. It is reported that these children, though they do not produce past tenses, do produce progressive aspect *ing* (Crystal, 1987; Trantham & Pedersen, 1976). It has also been noted that though they are able to produce the form they do not always use it correctly: "The profile chart is confusing with approximately equal numbers of correct vs. incorrect uses of the auxiliary and *ing*" (Crystal, 1987, p. 117). A more thorough and detailed linguistic analysis of the language of this disordered population would appear to be useful in elucidating the precise nature of the problem. It may be the case that there is a heterogeneous population that manifests diverse linguistic symptoms caused by diverse etiologies. This paper will discuss converging evidence from several different studies that suggests there may be a coherent subset of those diagnosed as manifesting this syndrome in whom the etiology appears to be genetic. In addition it will be argued that the problems with language in these cases of genetic dysphasia can be accounted for by a principled linguistic deficit.

The family¹

There are 30 members in the family that was studied, extending over three generations. Of these, 16 have been diagnosed as dysphasic. They are distributed in the following pattern:

¹This work could not have been accomplished without the openness and spirit of cooperation with which the family embraced this project. In particular, Dr. Gopnik would like to thank the grandmother for her enthusiasm and her help in making all of the arrangements for testing in the various homes and to all of the grandchildren for their warmth and playfulness who helped make the task fun. She should also like to thank the teachers at the Speech and Language Unit of the Lionel Primary School for all of their help and interest. In particular, she should like to thank Frances Graham who provided her with important insights about the development of the children.

F (74)* – M (deceased)				
F (46)* – husband	M – wife	F (43)* – husband	M (40)* – wife	F (38)* – husband
f (17)*	m (20)	f (21)	m (10)	<u>m (17)*</u>
m (16)*	f (18)	f (17)	f (7)*	<u>m (15)*</u>
f (12)*		f (12)*	f (5)*	<u>m (14)*</u>
m (8)*		<u>f (11)*</u>	<u>f (2)?*</u>	<u>f (12)*</u>
		<u>m (7)</u>		<u>m (10)*</u>
				<u>m (8)*</u>
				<u>f (7)*</u>
				<u>m (6)*</u>
				<u>f (6)*</u>

The 16 members of the family that are underlined were diagnosed as dysphasic by the school system and all of them have received some kind of speech-language intervention except the grandmother, for whom such remedial services were not available when she was in school. (The 2-year-old has been only tentatively diagnosed as dysphasic because of her late onset of language.) The 22 members indicated by **bold type and asterisk (*)** supplied data for this study. The spouses have never been diagnosed as having any problem with language and the family reports that they do not have such problems. The age of the subject at the time of testing is given in parentheses. The two youngest members of the family of nine children are fraternal twins.

Symptoms

The dysphasia in this family is pronounced. The dysphasic grandchildren have severe phonological² as well as grammatical problems. The existence of both phonological and grammatical problems is typical of dysphasia (Cantwell & Baker, 1987; Crystal, 1987). The children in this family are almost unintelligible until they are about 7 years old and phonological problems persist into adolescence in certain of the grandchildren. In addition to these phonological problems, which will be discussed only briefly in this paper, there are clear

²An investigation of the exact linguistic nature of these phonological difficulties is now being undertaken with G. Piggott. It would be premature to report on the phonology until the final results of this study are available. The argument in this paper does not depend on the outcome of this phonological research. If the problems in phonology can be shown to be caused by a deficit in the underlying phonological system, as the preliminary data seem to suggest, then it might be able to be demonstrated that the phonological deficit and the grammatical deficit were both caused by an inability to construct general linguistic rules from individual exemplars. This paper does provide data which show that the pattern of grammatical errors cannot be accounted for by any simple deficit in the perception or production at the level of phonetics.

problems with grammar. The grandmother reports that her children were similarly impaired when they were young and the parents report that their older children were as impaired as their younger children, but that both their phonology and their grammar improve as they get older. At first glance, the language that the adults produce seems almost unimpaired, but careful testing reveals that this normality is only apparent. Although they have learned strategies for coping with language, their underlying grammar remains severely impaired. The pragmatic aspects of language seem to be unaffected. The impaired family members use language in the same way as the normals: to tell stories, joke, tease, request, respond, and protest.

The impaired parents had from one to several years of speech and language intervention when they were children. The dysphasic grandchildren have been in a special school for language-disordered children since they were 4 years old. The speech and language pathologists report that they have been instructed on features such as number, gender and aspect routinely in the school. Representative case histories of seven members of the family are reported on by Hurst, Baraitser, Auger, Graham, and Norell (1990). They report that "Hearing and intelligence of all affected members were within the normal range" and that "The unaffected members have no speech or language difficulties and all attended normal schools" (Hurst et al., 1990, p. 354). This family is interesting to study on several grounds. In the first place, it provides us with 16 dysphasic subjects ranging in age from 2 to 74 over three generations. The fact that the grandmother reports that her dysphasic children were just like her grandchildren when they were young gives us at least a presumptive reason to suppose that studying the subjects in this family at various ages can give us insights into the pattern of language learning over time in language-impaired subjects. Moreover, the family also provides us with normal subjects, of about the same age and raised in the same social, educational and linguistic environment as the dysphasics, who can be compared to the impaired family members. This paper will report on the results of several tests that were administered to both dysphasics and normals in this family, and will interpret these results in light of a principled account of dysphasia developed on the basis of independent data, thereby providing an explanatory account of the language of these dysphasics. In addition, we will suggest a learning mechanism that allows these dysphasics to simulate, albeit imperfectly, the function of that part of grammar that they are missing.

Evidence for a genetic etiology

There are several different types of evidence that have been used to argue for genetic etiologies: (1) patterns of familial aggregation in large multigener-

ational families: (2) statistical studies of familial aggregation in large samples; (3) twin studies; and (4) linkage studies of the pattern of bases in the DNA of affected individuals and related normals. Each of these different kinds of evidence addresses a different issue in establishing the genetic hypothesis. Large, multigenerational families in which the same disorder can be seen to recur in several different branches of the family and over several generations may also provide evidence for the genetic hypothesis. There are two other possible explanations for such familial patterns of occurrence: the disorder might be caused by a factor in the environment, either physical or social, or, if the family was selected particularly *because* it exhibited this pattern we might be observing a mere statistical anomaly. If several members of the family are not affected and further if there are no demonstrable differences in either the physical or social environment that distinguish the affected members of the family from the unaffected members then it is more likely that the cause is genetic rather than environmental, though, of course, as yet undiscovered environmental factors cannot be ruled out in principle. The likelihood of the pattern in the family being the result of statistical coincidence declines with the size of the family. Large statistical studies avoid the ascertainment bias inherent in familial case studies. In such studies the individual subjects (probands) are chosen first on the basis of some disorder that they exhibit. They are then matched with other normal probands. After the probands are selected, their family histories are obtained. If it can be shown that relatives of the probands with the disorder in question are more likely to have the disorder than relatives of normal probands matched for environmental factors, then it is probable that there may be an inherited propensity for the disorder. Twin studies are another source of evidence for the genetic hypothesis. If it can be demonstrated that monozygotic twins are more similar with respect to the disorder than dizygotic twins, then the genetic hypothesis is more likely than either environmental or in utero accounts. Linkage studies look directly at the DNA. They can indicate that there is a difference in the sequence of bases on a particular chromosome of the affected subjects as opposed to normals. However, they are rarely able to point to a precise gene that is causing the disorder and it is not always the case that individuals that can be shown to have such a sequence always present with the disorder. The penetrance of the gene may vary with the individual. No single piece of evidence can establish the genetic hypothesis; therefore, what is needed is converging evidence from all of these different kinds of sources. Such evidence is now becoming available with respect to dysphasia.

Family case studies

The pattern of familial aggregation in the family that is reported on in this paper is particularly interesting because it is consistent with the hypothesis of genetic etiology by autosomal dominant transmission. The family is being investigated at the Genetics Clinic of the Hospital for Sick Children in London, and Dr. Hurst of that clinic concludes that "Inheritance in this family is autosomally dominant, and chromosome analysis of one of the affected members was normal ... Its importance seems to be that there is a single gene coding for a pathway which is fundamental for developing intelligible language" (Hurst et al., 1990, p. 354). In addition to the evidence from this family, Samples and Lane (1985) report on six siblings, ranging in age from 11.9 years to 5.2 years, in a single family all of whom have specific language-learning disorders. Brief descriptions of the language of these children are provided and a sample paragraph of the language of one of the children is given. The information given about the language of these children, though limited, is consistent with the deficits that will be discussed in this paper. After analyzing the data they conclude that "a genetic component contributes to the disorder" (p. 25).

Statistical studies

Tallal, Ross, and Curtiss (1989a) studied 112 4-year olds: 62 language impaired and 50 controls. Once the probands were selected the incidence of language problems in their siblings and parents was determined. They concluded that "the results demonstrate highly significant differences in the incidence of reports of positive family histories for first-degree relatives between the families of language-impaired and control children" (p. 172). In their second article (Tallal, Ross, & Curtiss, 1989b), they report that language-impaired mothers are three times more likely to have sons than daughters and that these sons are five times more likely to be language impaired than their daughters. They conclude that "the pattern of familial aggregation, if genetically influenced, would be compatible with autosomal dominant transmission, with greater penetrance through the mothers than through the fathers" (p. 994). Our family does not display this sex bias, nor does Tomblin's (1989) sample discussed below; however, the data from this family may be consistent with the hypothesis of greater penetrance through the mothers because all of the affected females in this family have affected children.

Tomblin reports a similar pattern of familial aggregation in a study of 203 children ranging in age from 7 to 9: 51 language impaired and 152 controls. After showing that "the data from this study do not provide support for the environment as a principal mechanism" he concludes that "the possibility that

one or several paths exist between genetic characteristics, neural development and/or function, and ultimately language learning must be considered as plausible" (p. 293). Both Tallal and Tomblin suggest that it is important that their statistical data be supplemented by detailed descriptions of the language of affected family members: "Studies designed to test, in detail, the communication skills of family members with specific developmental language impairment will be necessary to confirm and extend these findings based on self-report data" (Tallal et al., 1989b, p. 996); "The models in this field are best tested with extended family data in which at least three levels or degrees of family relationship exist and in which direct measures of the trait are obtained on all family members" (Tomblin, 1989, p. 294). This paper will provide such a detailed description of the type and degree of the language impairment in the members of an extended family that is consistent with and complementary to the prior case and statistical studies.

Twins

Borges-Osorio, a geneticist, reports on several aspects of language in three sets of monozygotic twins and concludes that his data show that "genetic factors in the etiology of language is now clearly established, although the details of the gene-environmental interaction in the different categories of such defects are far from being elucidated" (Borges-Osorio & Salzano, 1989, p. 99). Unfortunately, though he does report that they had several different types of language errors, he does not describe them in any detail. Tomblin,³ in a study of the language intervention history of 80 sets of male twins, found that if a twin was reported to be monozygotic then the probability that the other twin was also receiving language intervention was over 80%; if the twin was reported to be dizygotic then the probability dropped to 35%. The findings for the dizygotic twins are consistent with the results for male siblings reported in Tomblin (1989), discussed above. These data and the fact that one fraternal twin in the family has dysphasia and the other does not, argue against the hypothesis that the disorder could be caused by any simple intra-uterine interaction between mother and fetuses.

Language errors

The studies cited above show clearly that the familial aggregation of dysphasia that is seen in this family is not unique. Though these studies report very

³Tomblin has reported the results of this study to us and others in personal communication and is now writing up these findings.

little detail on the actual linguistic deficits, the little that they do report is consistent with the kinds of language problems that are typical in our data. Samples and Lane (1985) report problems with plurals, pronouns and tense. Tomblin⁴ reports that adult dysphasics produced more than 2.5 times the number of errors on grammatical morphemes than the normals and that these "errors" included tense and number.

Environmental explanations

The question has been raised as to what proportion of this aggregation can be attributed to genetic factors and what proportion is environmental. Both Tallal and Tomblin point out that the pattern of statistical evidence mitigates against a purely environmental explanation of the familial aggregation data. If we were to entertain the environmental hypothesis that something in the social or linguistic environment in the families of the affected children caused the disorder, then we would be left with another puzzle: how did the normal children, for example, in the family with nine children, escape these environmental influences, and if the cause is environmental why are only half of the children, as would be predicted by the genetic hypothesis, affected? The environmental hypothesis also has trouble in accounting for the fact that though all of the children are receiving input from impaired parents and impaired siblings some of them seem to be able to ignore this impaired input and develop language at the normal time and in the normal way. Leonard (1987) reviews several different studies that address the question as to whether impaired parental input causes language problems, and he concludes that "The evidence that communicative input factors contribute to language difficulties is weak and certainly not the stuff of which theories of specific language impairment should be made" (p. 10).

Genetic marker

Of course, neither the statistical evidence of familial aggregation, nor the family studies in and of themselves, can *prove* the genetic hypothesis. What is needed is direct evidence that there is a characteristic difference in the genetic structure that distinguishes the normals from the dysphasics. It is to be hoped that a genetic marker will soon be found, but even if such a marker were found studies, like this one, of the exact nature of the underlying linguistic deficit would be necessary in order to have an idea of the precise impair-

⁴Personal letter. June 11, 1990.

ment to language development that was caused by the observed differences in the genetic sequence of bases. Moreover, if it were true that an autosomal-dominant genetic factor results in a selective impairment in a particular part of the grammar, this would be support for a hypothesis that language is made up of several autonomous subparts that may have evolved at different times. (For an interesting discussion of the evolution of language see Pinker & Bloom, 1990.)

Neurological evidence

One model for the genetic etiology of dysphasia would be that the gene in question affected some aspect of the development of the brain and that this abnormal neurological system, in turn, constrained the way in which language could be learned. Plante (1990) reports on the results of MRI studies of four families that each included a specifically language-impaired son. She concludes that "atypical perisylvian asymmetries represent a biological risk factor for developmental language disorder, and this risk factor is transmitted through families" (p. 11). These findings are consistent with the reported atypical asymmetries reported on autopsy of a 7-year-old dysphasic reported in Cohen, Campbell, and Yagmai (1989).

Theoretical assumptions

Explanatory models

Some very fundamentally different models have been proposed to account for dysphasia. One model tries to provide an explanation of dysphasia in terms of auditory processing mechanisms, another seeks to provide an explanation in terms of the perceptual salience hypothesis, a third relates language disorders to underlying cognitive deficits. The first model assumes that the problems with language result from an inability to process the speech signal and therefore, because the child cannot process the sounds, they cannot build the linguistic rules that depend on these distinctions. It has been suggested, for example, that "developmental dysphasia is a direct consequence of defective processing of rapidly changing acoustic information" (Tallal & Piercy, 1978, p. 82). Another suggestion has been that if the surface properties of the grammatical morpheme are not phonologically salient then the underlying paradigm representing these features cannot be constructed by language-impaired children (Leonard, Bortolini, Caselli, McGregor, & Sabbadini, 1990). These accounts avoid positing any abstract underlying cognitive processes that guide the construction of grammars. There are several different sorts of

data that argue against these surface processing hypotheses. Under this hypothesis all similar surface forms of the utterance must be affected in a similar way. Yet, Menyuk (1978) reports that "final singletons or clusters were often omitted, except when they were part of the word stem. For example 'bees' might be repeated as 'bee', but 'nose' was never repeated as 'no'" (p. 147). In addition, it is reported that the acquisition of "s" that marks plural in English and "s" that marks possessive in English show a different pattern of impairment in these children (Johnston & Schery, 1976), yet there is no surface difference between these two forms. Therefore, it cannot be merely the processing of the surface sound that gives them problems. Moreover, although it is reported that language-impaired children have problems in perceiving unstressed grammatical morphemes, they do, nevertheless, sometimes produce both the plural "s" and the past tense "ed". Therefore, they must be able to both process auditorally and articulate these sounds at some level. This paper will provide data that show that the adult dysphasics in this family can and do hear and produce the phonological forms in question. They can perceive and produce words like *books* and *walked*. What they cannot do is represent them in terms of underlying paradigms that would allow them to manipulate the abstract representations in these paradigms. In addition, many of the errors that are reported widely in the literature, such as subject pronoun dropping and the use of three different forms in place of aspect, which will be discussed in detail later in this paper, cannot be accounted for by the surface or auditory processing hypotheses.

This paper will provide an account of dysphasia in terms of an underlying psychological mechanism. The assumption that the linguistic behavior of dysphasics is the result of a coherent set of underlying internalized rules constrains the way in which the data must be characterized. The surface forms are important only insofar as they provide the evidence for the properties of the underlying rules that generate them. Moreover the surface forms as individual items do not have any status in the determination of the underlying rules. It is only by looking at the pattern of interrelationships within the data that insight into the underlying rules can be gained. Therefore the comparison of individual surface forms among individuals who differ significantly is inappropriate. Two data sets that differ in some of their items, even though they may be identical in others, may be produced by very different underlying rules. This follows not from any peculiarity about internalized psychological rules, but from the properties of formal systems in general. Two Turing machines that have very similar output may differ significantly in the rules that produce this output; two machines that differ in many respects in their output may have very similar rules. The degree of similarity of the output is not a reliable indicator of the degree of similarity of the system of rules that

produces this output. Therefore, two items that may appear identical on the surface may be generated by very different rules from very different underlying representations. It is within this framework that the concept of *error* in the language of dysphasics must be understood. Such a viewpoint is at odds with the tendency to use tests that score as errors surface forms that the subject produces which are different from those produced by normals and to count as correct those surface forms that are the same (Hamill & Newcomer, 1982). In the dysphasia literature, it is often the case that the surface forms produced by dysphasics are compared individually with similar forms produced by younger children, and it is considered interesting to note that a particular form produced by a dysphasic child is also produced by a younger child. In fact, it is often the case that there is an explicit item for item comparison between the speech of dysphasics and that of normals matched for MLU.⁵ Steckol and Leonard (1979) for example, look at "the percentage that each of 4 grammatical morphemes was correctly produced in obligatory contexts" (p. 296). The forms that they study are the marked surface forms only, such as regular plural, regular past, and irregular past. They conclude that "language-impaired children are not using grammatical morphemes to the degree seen in the speech of normal children at the same level of mean utterance length" (p. 297). In all of these comparisons some "errors" are found to be similar to those made by the younger children and some are found to be different. If our aim is to produce a principled explanation of dysphasia, then the procedures described above are not sufficient. A linguistically principled explanation must assume that the underlying grammar produces all of the language and not just the marked surface forms that differ from the normal forms; that is, the children do not have two grammars – one that produces all of the "errors" and another normal grammar that produces the correct "forms". Of course, it is clear that there are several different subparts of language, each of which is subsumed by its particular set of rules: movement rules are independent of morphology, though they may interact at some levels. (It will be argued here that dysphasics may be impaired in only one subpart of grammar and have other parts of the grammar intact.) In spontaneous speech, dysphasics produce surface forms that appear to be different from those produced by the normal grammar: *two arena, a cups* and surface forms that appear to be the same as those produced by the normal grammar: *a boy, four goals*. However, it is mistaken to assert that the forms that match the normal forms are correct and those that do not are errors. If

⁵The concept of mean length of utterance is not very useful for equating linguistic behavior past the stage at which the minimal length for the argument structure of a sentence has been established because the length of the sentence can be increased by linguistically very different processes.

the underlying rule is wrong then all of the forms produced by it are wrong with respect to the normal language as a whole, even if they happen to resemble the correct surface forms; and all of the forms are correct with respect to the underlying rules of the dysphasic even if they differ from the forms in the normal language. As we have mentioned above, it has been widely reported that dysphasics make errors on plurals. These same data can be accounted for more insightfully by saying that they do not mark number. Therefore a form like "a boy" is just as incorrect as "a cops" because neither of them are marked for number.

A primary question that has been raised with respect to explanations of dysphasia is whether the underlying rules that are impaired are specific to language or whether it is more accurate to say that dysphasics are cognitively impaired and that their problems with language are a secondary effect of this more general cognitive deficit (Cromer, 1978; Eisenson, 1984). Because those parts of language that are unimpaired in these subjects are at least as complex as those that are impaired, it is not clear that this deficit could be caused by a more general cognitive problem. Moreover, these subjects are reported to be in the normal range in the standard non-verbal tests of intelligence. If we are to entertain the cognitive hypothesis, then it would have to be shown that there was a model in which those aspects of language that are impaired can be characterized by different cognitive processes from those aspects of language that are unimpaired, and further, that that particular part of general cognition was impaired. This may be the case, but there has been no cognitive hypothesis yet proposed that provides such an account. Yet, even if this were to turn out to be the case, it would not falsify the hypothesis that there is a deficit in particular linguistic rules. It would simply subsume the linguistic-deficit hypothesis under a more general cognitive-deficit hypothesis. The possibility of finding such a cognitive explanation, should one exist, necessitates having a detailed linguistic account available.

Several authors recently have argued that, in order to understand language acquisition and acquired and developmental disorders, it is necessary to provide such an account in terms of a coherent theory of language (Clahsen, 1989; Gopnik, 1990a; Grodzinsky, 1986; Lightbown & White, 1987). It is argued that, since the actual language that a subject produces is a consequence of an underlying grammar, any *explanation* of the deficit must provide an account of this underlying grammar. The question remains, however, as to what constitutes an appropriate data set or an appropriate underlying grammar. These are really interrelated questions. It is clearly not the case that there is any independent, a priori criteria that can be used to determine what constitutes significant data in the absence of a hypothesis. The underlying grammar that is hypothesized determines what constitutes the significant

data set. Of course this is an iterative process: the occurring "errors" may lead to the postulation of an underlying grammar, but once those underlying rules and representations have been stipulated, they themselves make predictions about the forms that will and will not occur. Providing a principled linguistic account in terms of an underlying grammar is therefore a risky business: clear predictions about the forms that can occur follow from underlying rules and therefore such hypotheses can be shown to be wrong. (This is not the case with the procedure of tallying errors since such a procedure is not constrained by any clear hypothesis. If a new "error" is found it is simply added to the list.) The most parsimonious hypothesis about the form of the underlying grammar is that it conforms to the constraints that are believed to hold for all *possible languages*. The underlying grammar that is postulated should therefore be based on the kinds of rules and representations that we know hold for languages in general. One consequence of this assumption is that item by item analysis is necessarily inadequate because grammars operate over structures, not single items. A given underlying grammar therefore constrains not only individual forms, but also the way in which these forms interact with other parts of the grammar.

The database

Subjects and procedures

Because the extent of the linguistic deficits in this family was not known, it seemed desirable to test a wide range of linguistic abilities. To this end the tests that were administered to the family in August 1989 were adapted from a battery of tests developed by Paradis (1987) for testing the linguistic competence of acquired aphasics. Some additions and changes had to be made to these tests so that particular aspects of a hypothesis that had been found to account for the language of other dysphasics could be tested. All of the subjects were tested individually, in private, in their own home and were audio-taped. The tests were administered to 20 members of this family, who are indicated by **bold** and * on the family chart in section 1. Thirteen of the dysphasics – the grandmother, three daughters and one son and eight grandchildren – were tested. In addition to the tests' results themselves we have, for three of the dysphasics, two of whom did not participate in the testing, notebooks in which they wrote a story once a week for a whole school year. We therefore have data from 15 of the 16 dysphasics in the family. We also tested seven of the unaffected members of the family. The one unaffected son declined to be tested; therefore the seven normals tested were all grandchildren. The normals in the family serve as an excellent control group for

the dysphasics since they have the same dialect, the same social class and the same upbringing. This paper will concentrate on characterizing the competence of the older subjects because, from the point of view of the theory of language, it is important to understand the final state of linguistic competence that these subjects can achieve and the means by which it is arrived at in order to understand the underlying linguistic deficit. The sample that is reported on in this paper consists of six dysphasics aged 16, 17, 40, 42, 45, and 74, and six normals aged 8, 12, 13, 14, 15, and 17. For ease in presentation, the bar graphs in the results section pair the subjects in order of age. For example, the leftmost bars pair the youngest dysphasic (16) with the youngest normal (8); the rightmost bars pair the results for the oldest dysphasic (74) with the oldest normal (17). These pairings are not meant to indicate that there is any particular similarity between paired members. It is assumed that all of the subjects have achieved more or less adult competency. Given that the mean age of the normals is 13 and the mean age of the dysphasics is 39, it would be more likely that the normals have not achieved adult competency than that the dysphasics have not.

The subject of an earlier study in Montreal (Gopnik, 1990b) was retested with these new tests. Previous analysis of his language had provided detailed evidence supporting a hypothesis that his deficit was in the ability to construct underlying features. After we had tested the family reported on in this paper, the mother of the subject in Montreal reported that her husband's uncle had been seriously language impaired. Even though this report is not adequate evidence of a familial pattern of language impairment, we thought, nevertheless, that it was interesting to ascertain whether the performance of the Montreal subject would be similar to that of the family. If it were, then it would not be unreasonable to suppose that the inferences about his language might be relevant to the family. We also tested his mother and his younger brother, who are normal, in order to have a comparison group from Montreal. The results of these Montreal tests will be reported in square brackets.

The tests

Tests 1 and 2: pointing tasks

Method. The subject was required to act out a command spoken by the experimenter. While the subject was watching, 16 objects – 4 books, 4 crayons, 4 coins and 4 balloons – were placed on a table in front of the subject. They were set out in eight piles randomly arranged on the table. Each pile consisted of either three items of the same kind or a single item. Test 1

consisted of six simple commands of the form "Please touch the books." Because there were eight possible responses and only six commands the subject could not respond by merely guessing. The second test consisted of four more complex commands of the form "Put the crayon on the balloons."

Scoring. A response to a singular word was counted as correct if the subject touched a single object of the right kind; a response to a plural word was counted as correct if the subject pointed to more than one object of the right kind.

Results. Two-tailed *t* tests were performed.

Test 1 – simple commands:

Normals mean = 5.33; dysphasics mean = 5.33; $t(10) = 0$; $p = -$. There is no significant difference.

[Montreal normals mean = 5.5; dysphasics mean = 6]

Figure 1 shows that this test was easy for both the normals and the dysphasics: 9 out of the 12 subjects got a perfect score of 6. Only one dysphasic did not get a perfect score, though 2 normals did not.

Test 2 – complex commands:

Normals mean = 3.83; dysphasics mean = 3.33; $t(10) = .958$; $p = .361$. There is no significant difference.

[Montreal normals mean = 4; dysphasics mean = 3]

Figure 1. *Ability to discriminate s-marked plural forms by pointing.*

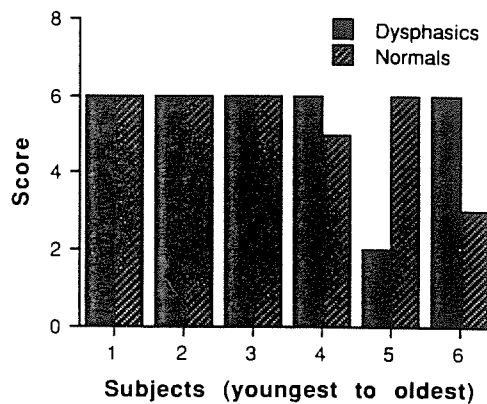


Figure 2. Ability to discriminate s-marked plurals in simple commands.

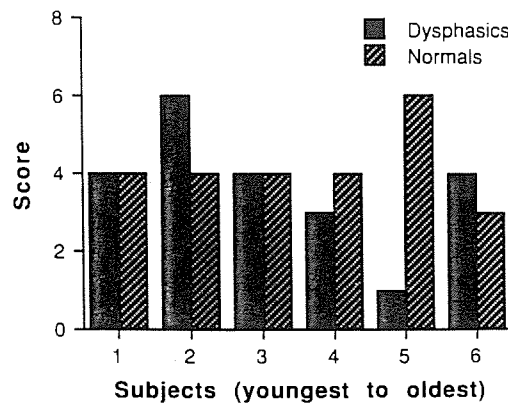


Figure 2 shows that this test was somewhat harder. Only two subjects – one normal and one dysphasic – got a perfect score.

Comments: Tests 1 and 2. There were some unexpected behaviors exhibited by three of the dysphasics. The expected response to “Please touch the books” was that the subject would touch the pile of three books, and to “Please touch the book” was that the subject would touch the single book. All of the normals did this with no hesitation. Three of the adult dysphasics behaved differently. When requested to touch a singular item they asked “Which one?” or touched a single correct item in the pile of three items. When requested to touch a plural noun they touched both the pile of three items and the single item. This had to be scored as a correct answer since the subject had indeed touched a single item in the first instance and more than one item in the second. In some sense their response to “Please touch the books” was more correct than that of the normals since in fact they did touch all of the books. They gave a similar response to the more complex test. In response to the instruction “Put the crayon on the balloons” they added the single balloon to the pile of balloons, took a single crayon from the pile of crayons and put it on the balloons. The dysphasic in Montreal responded in a similar way to these tests; therefore this response is not peculiar to the family, but reflects a more general strategy of dysphasics in performing the task.

Test 3: nonsense plurals

Method. The subject was shown pictures of imaginary animals. Each picture of a single animal was paired with a picture of two or three of the same imaginary creature. The experimenter pointed to the picture showing one of the creatures and said "This is a zoop" and then pointed to the other picture and asked "These are ___?" There were 6 test items – 2 ending with voiceless consonants, 2 ending with voiced consonants and 2 ending with sibilants.

Scoring. A response was counted correct if it had the appropriate plural marking.

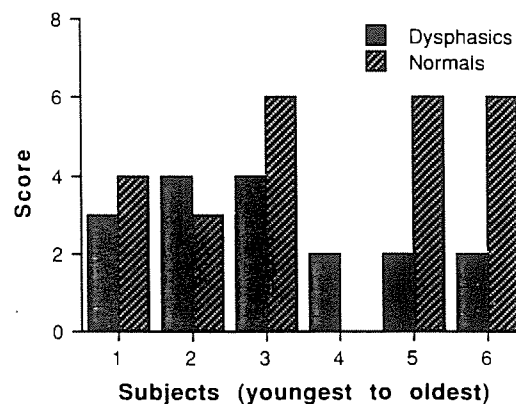
Results. Two-tailed *t* tests were performed. Normals mean = 5; dysphasics mean = 2.83; $t(10) = 3.313$; $p = .0078$. There is a significant difference.

[Montreal normals mean = 6; dysphasics mean = 3]

Figure 3 shows the results of this test by individuals.

Comments. Even when the dysphasics managed to get some items right, they appeared to do so not by using the normal internalized, unconscious set of rules for constructing plurals, but rather by finding a rule and applying it to all cases. One adult subject, when given the first item, paused for a long while repeating it under her breath several times and then whispered to herself "Add an 's'." For the items *sas* and *zash* instead of adding *ez* she

Figure 3. *Ability to pluralize nonsense nouns.*



simply prolonged the sibilant – *sasss*. Another subject when given the first item, “This is a wug. These are ___?”, looked totally confused and laughed nervously and responded, “How should I know?” After some prompting she said “These are wug.” To the next item *zat* she responded “These are zacko.” (The source for this response is unclear, but one might speculate that she perceived that a simple repetition of the word was not the desired response.) When she was given the third item *sas* she smiled broadly, shook her head yes and responded *sasez*, which she repeated several times. She then added *ez* to all of the rest of the items: *zoop* goes to *zoopez*, *tob* goes to *tobez* and *zash* goes to *zashesz*.

Test 4: complex commands

Method. The subject was required to follow a command spoken by the experimenter. Three objects were placed on the table in front of the subject and the subject was then given three three-part commands of the form: “Here are three crayons. Drop the yellow one on the floor, give me the blue one, and pick up the red one.” The subject was instructed to wait until the full command was given before responding.

Scoring. The subject was given one point for each of the commands performed correctly and an additional point if they were performed in the right order.

Results. Two-tailed *t* tests were performed. Normals mean = 11.833; dysphasics mean = 10.667; $t(10) = 1.832$; $p = .097$. There is no significant difference.

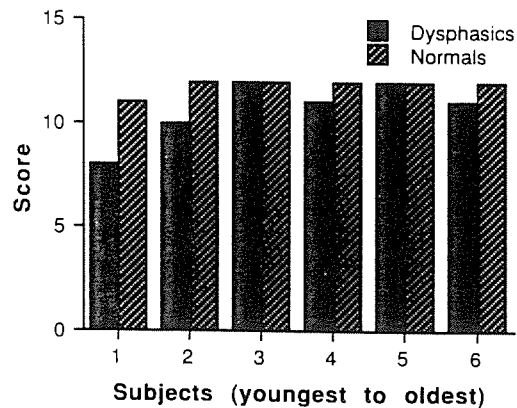
[Montreal normals mean = 11; dysphasics mean = 12]

Figure 4 shows that almost all of the subjects were successful in this test.

Tests 5, 6, 7 and 8: syntactic comprehension

Method. In each of these tests the subject was given a page with pictures and was instructed to point to the picture that best showed the meaning of the sentence. In Test 5, there was a page with four pictures, showing a man washing himself, a woman washing herself, a man washing a boy and a woman washing a girl. The subject was given six sentences that contrasted reflexive pronouns with non-reflexive pronouns – *He washes him*, versus *He washes himself*. In Test 6, there was a page with four pictures, a man holding another man, a man holding a woman, a woman holding a man and a man holding

Figure 4. Ability to perform sequences of commands from auditory memory.



two women. The subject was given four sentences that contrasted pronouns: "He holds him"; "He holds them." In Test 7, there was a page with two pictures: one showing a truck pulling a car and the other showing a car pulling a truck. The subject was given four negative sentences that contrasted active with passive: "The truck does not pull the car"; "The truck is not pulled by the car." In Test 8, there was a page with two pictures: a mother pushing a baby in a carriage and a doctor examining a patient. The subject was given five sentences that contrasted possessives: "Show me the mother's baby" versus "Show me the baby's mother."

Scoring. The subject got a point for touching the appropriate picture.

Results. Two-tailed t tests were performed.

Test 5 – reflexives:

Normals mean = 5.5; dysphasics mean = 5.67; $t(10) = .542$; $p = .599$. There is no significant difference.

[Montreal normals mean = 4; dysphasic mean = 5]

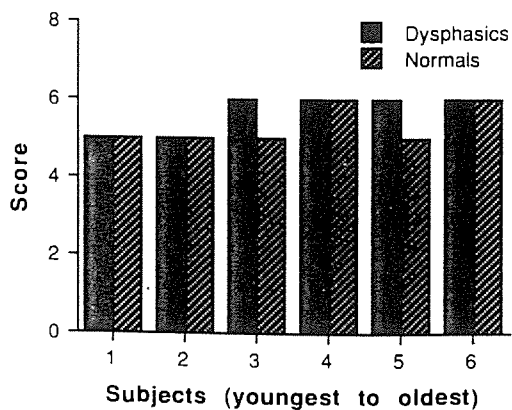
Figures 5–8 show that all the subjects had similar results in these tests.

Test 6 – gender pronouns:

Normals mean = 3.83; dysphasics mean = 3.67; $t(10) = .62$; $p = .549$. There is no significant difference.

[Montreal normals mean = 4; dysphasics mean = 4]

Figure 5. *Comprehension of reflexive forms.*



Test 7 – passives:

Normals mean = 3.33; dysphasics mean = 3; $t(10) = .598$; $p = .563$. There is no significant difference.

[Montreal normals mean = 3; dysphasics mean = 4]

Test 8 – possessives:

Normals mean = 5; dysphasics mean = 4.83; $t(10) = 1$; $p = .34$. There is no significant difference.

[Montreal normals mean = 5; dysphasics mean = 3]

Figure 6. *Comprehension of gender pronouns.*

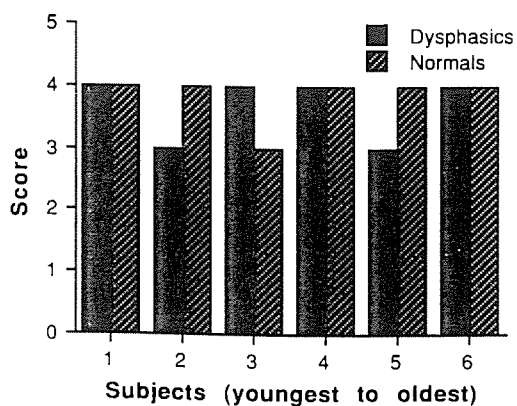
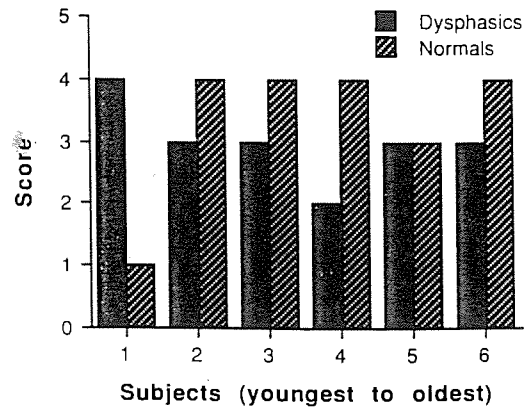




Figure 7. *Comprehension of negative passives.*



In Figure 8, the performance of both dysphasics and normals is near perfect.

Test 9: grammaticality judgements

Method. The subject was instructed "Now you will hear some sentences. Please tell me if the sentence is a correct English sentence. If the sentence is not correct please try to correct it." A practice sentence was given first. Thirty items were given – 9 correct and 21 incorrect. There were errors in number: "The boy eats three cookie"; person: "The boy kiss a pretty girl"; tense: "Yesterday the girl pet a dog"; and aspect: "The little girl is play with her doll."

Figure 8. *Comprehension of possessives.*

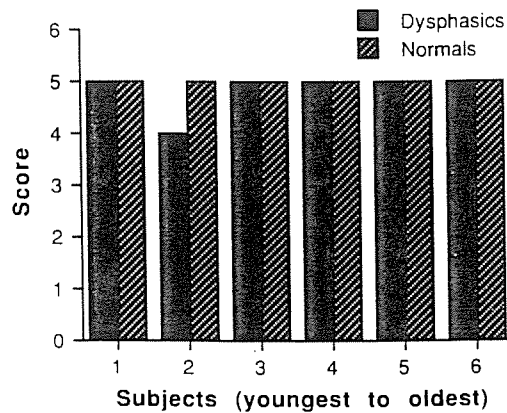
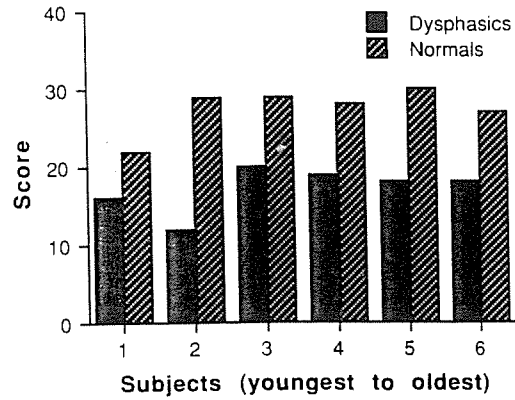


Figure 9. Grammatical judgement of feature errors.



Scoring. The subject got a point for each correct judgement. Scoring the corrections was much more difficult because the dysphasics often changed more than one part of the sentence. We adopted the most generous scoring procedure: the subject was given a point for a correction that resulted in a grammatical sentence whether that correction demonstrated that the subject understood the feature error or not.

Results. Two-tailed *t* tests were performed.
Grammatical judgement:

Normals mean = 27.5; dysphasics mean = 17.17; $t(10) = 6.238$; $p = .00001$.
There is a significant difference.

[Montreal normals mean = 27.5; dysphasics mean = 17]

Note: 17.17 out of 30 in a two-choice task is not significantly different from chance.

Figure 9 shows that the best result of the dysphasics is worse than the worst of the normals.

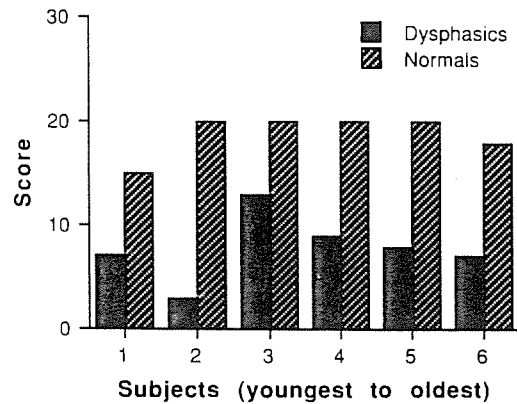
Corrections of the 21 ungrammatical sentences:

Normals mean = 18.33; dysphasics mean = 7.83; $t(10) = 6.196$; $p = .00001$.
There is significant difference.

[Montreal normals mean = 19; dysphasics mean = 7]

Figure 10 shows the striking difference between the ability of the dysphasics and that of the normals to correct feature errors.

Figure 10. Correction of feature errors.



Test 10: derivational morphology

Method. The subject was instructed, "Now you will hear a sentence with a part missing. Please complete the missing part." Two practice sentences were given. There were nine test items. Each consisted of two sentences in which the missing end of the second sentence was derivationally related to the first sentence. "There is a lot of sun. It is very ____." Seven of the test sentences involved derivational rules; two were lexically related: "He has lots of wisdom. He is very ____."

Scoring. The test was scored in two different ways. In the first scoring procedure each item was counted correct if it matched the predicted response. For the item: "I don't like his pride. He is too ____", the response *proud* was counted as correct. In the second scoring procedure the response was counted as correct if it was morphologically related to the stimulus and made sense in the frame given. "I don't like his pride. He is too *prideful*" was scored correct. "You can wash this. It is *dirty*" was scored incorrect.

Results: first scoring. Two-tailed *t* tests were performed. Normals mean = 6.167; dysphasics mean = 2; $t(10) = 4.11$; $p = .0021$. There is a significant difference.

Figures 11 and 12 show the difficulty the dysphasics have in producing derivationally inflected forms. Note in Figure 11 that the two youngest dysphasic subjects did take the test but they did not produce any of the expected responses.

Figure 11. Ability to produce predicted derivational inflections.

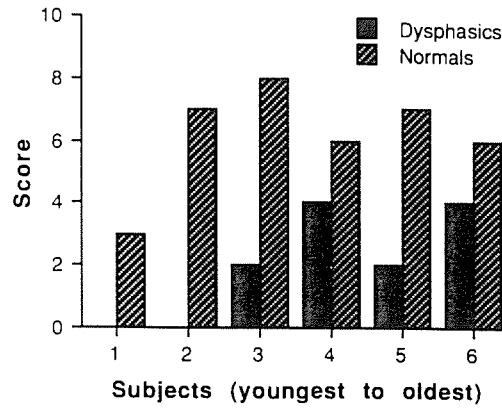
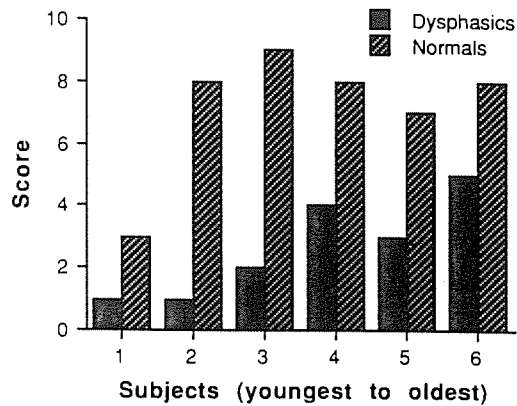


Figure 12. Ability to produce derivational inflections.



Results. Two-tailed *t* tests were performed. Normals mean = 7.17; dysphasics mean = 3; $t(10) = 3.76$; $p = .003$. There is a significant difference.

[Montreal normals mean = 7; dysphasics mean = 2]

Test 11: grammaticality judgements; thematic relations

Method. The subject was instructed, "Now you will hear some sentences. Please tell me again if the sentence is a correct English sentence. If the

sentence is not correct then make it correct." Two practice sentences were given. There were 11 test sentences: 5 incorrect and 6 correct. The incorrect sentences had errors in argument structure: "The girl eats a cookie to the boy"; "The boy puts the book."

Scoring. The subject got a point for each correct judgement. Two strategies were used by both the normals and the dysphasics in the correction task. One strategy was to supply the missing arguments: "The nice girl gives" was corrected by a dysphasic to *The nice girl gives him a ball*. In the other strategy the verb was changed to one that had the given argument structure: "The girl eats a cookie to the boy" was corrected to *The girl gives a cookie to the boy*. The subject got a point for each correction that resulted in a correct argument structure.

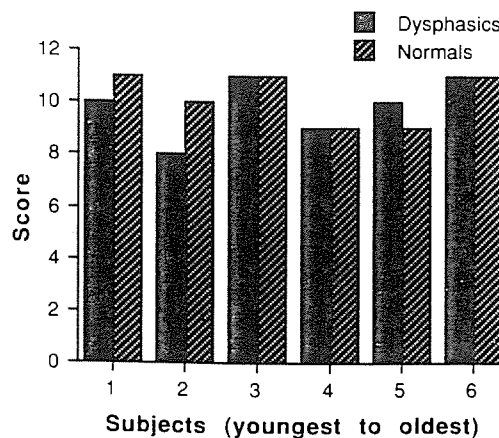
Results. Two-tailed *t* tests were performed.
Grammatical judgement:

Normals mean = 10.17; dysphasics mean = 9.83; $t(10) = .535$; $p = .604$.
There is no significant difference.

[Montreal normals mean = 9; dysphasics mean = 9]

Figure 13 shows similar results for both dysphasics and normals.

Figure 13. *Grammatical judgement of argument structure errors.*

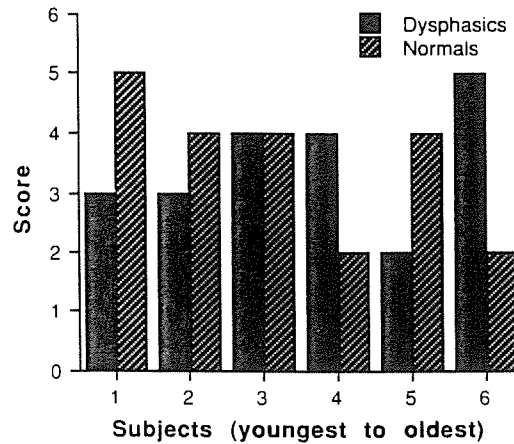


Corrections:

Normals mean = 3.5; dysphasics mean = 3.5; $t(10) = 0$; $p = .$ There is no significant difference.

Figure 14 shows that though there is individual variance, both the normals and the dysphasics range from a perfect score of 5 to a score of 2.

Figure 14. Corrections of argument structure.



Test 12: tense marking

Method. The subject was instructed, "Now you will hear some sentences with a part missing. Please complete the missing part." Two practice sentences were given. There were ten test sentences. They all involved tense change: "Every day he walks eight miles. Yesterday he ____"; "The boy always cries. Right now he ____." Four of the responses required past tense forms, two regular, *kissed* and *walked*; two irregular, *went* and *was*; two required futures, two required present, and two progressive aspect.

Scoring. Two scoring procedures were used. In the first scoring procedure, a response was counted correct only if it matched a predicted response. For the item "Yesterday the girl baked a cake. Tomorrow she ____" the standard, and predicted, response is "will bake a cake". In the second scoring procedure a response was counted correct if it was morphologically related to the stimulus and was correct in the frame given. In the frame above "is gonna bake a cake" was counted correct. In the frame "Every day he walks

eight miles. Yesterday he ____” both responses – “had a rest” because it is not morphologically related to the stimulus, and “walks”, because it is not correct in the frame given – were scored incorrect.

Results: first scoring. Two-tailed *t* tests were performed. Normals mean = 7.83; dysphasics mean = 3.16; $t(10) = 4.058$; $p = .0023$. There is a significant difference.

Results. Two-tailed *t* tests were performed. Normals mean = 9.17; dysphasics mean = 3.83; $t(10) = 4.525$; $p = .001$. There is a significant difference.

[Montreal normals mean = 8.5; dysphasics mean = 5]

Figure 15. Ability to produce predicted tense marking.

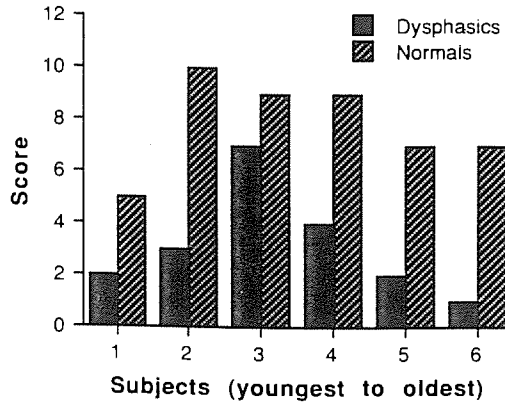
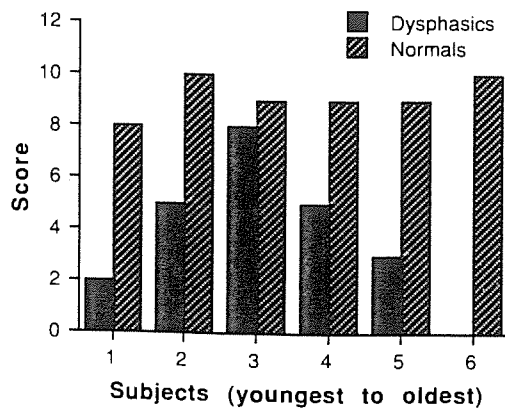


Figure 16. Ability to produce tense marking.



Results. Two-tailed *t* tests were performed. Normals mean = 0; dysphasics mean = 2.5; $t(10) = 3.27$; $p = .008$. There is a significant difference.

The dysphasics often produced unmarked forms of the stimulus verbs in their responses: the normals never did, as shown in Table 1. Although this difference did not obtain significance, the distribution is interesting to note.

The other type of incorrect response was to change the stimulus verb to a semantically related verb. The dysphasics did this much more often than the normals, as shown in Table 2.

Table 1. *Frequency of errors: unmarked verbs*

	Subjects					
Normals	0	0	0	0	0	0
Dysphasics	2	1	3	0	4	5

Table 2. *Frequency of errors: semantically related verbs*

	Subjects					
Normals	2	1	0	0	0	1
Dysphasics	4	5	1	2	0	1

Test 13: listening comprehension

Method. The subject was instructed, "You are going to hear a little story. Listen carefully to the story and then I will ask you some questions about it." The story was three sentences long. There were five questions about things that were explicitly stated in the story.

Scoring. Answers that reflected the subject's knowledge of the contents of the story were scored correct.

Results. Two-tailed *t* tests were performed. Normals mean = 4.67; dysphasics mean = 4.5; $t(10) = .415$; $p = .687$. There is no significant difference.

[Montreal normals mean = 4; dysphasics mean = 5]

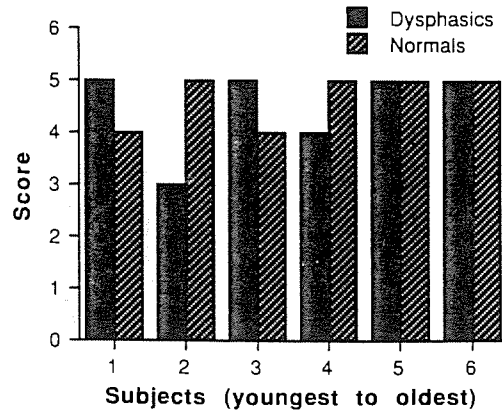
Figure 17. *Listening comprehension.*

Figure 17 shows that there is no clear pattern of difference in listening comprehension between dysphasics and normals.

Test 14: narrative task

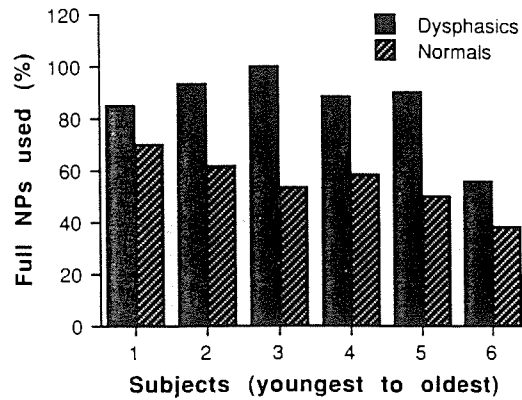
Method. The subjects were shown six cartoons that depicted a narrative. They were instructed, "I am going to show you a set of six pictures. All together the pictures make a little story. Look at the pictures and tell me the story." The page of cartoons was visible to the subject during the story-telling.

Scoring. One of the aspects of story-telling that we were interested in was the use of anaphora. We counted the number of full noun phrases, "the boy" and the number of pronominal noun phrases, "he". Mixed phrases like "his leg" were counted as both pronominal and full. In order to control for the differences in the length of the stories we calculated the percentage of noun phrases in the story that were full noun phrases.

Results. Two-tailed *t* tests were performed. Normals mean = 55.17; dysphasics mean = 91.2; $t(10) = 3.76$; $p = .00001$. There is a significant difference.

Figure 18 shows that all of the dysphasics used significantly more full noun phrases than any of the normals, except for the oldest dysphasic who produced a description of the pictures instead of a narrative (see discussion).

Figure 18. Use of full noun phrases (NPs) in narrative discourse.



Time on verbal response tests

In addition to the scores on the tests we calculated the time that it took for each subject to respond to the tests that required verbal responses: tests 9, 10, 11, 12.

Method. The time interval between the end of the instructions until the end of the subject's responses was calculated for each test. Interruptions were subtracted from the total time. Because one of the dysphasics, in addition to his specific language problems, also had a stutter his data are not included in these statistics. In addition, for the nonsense plurals test some of the sessions were not taped and therefore the times are not available.

Results. Two-tailed *t* tests were performed.

Test 9 – features:

Normals mean (in seconds) = 164.8; dysphasics mean = 242.2; $t(9) = -4.862$; $p = .00009$. There is a significant difference.

Test 12 – tense:

Normals mean = 52; dysphasics mean = 111.6; $t(9) = -4.804$; $p = .001$. There is a significant difference.

Test 10 – derivations:

Normals mean = 51.2; dysphasics mean = 106.8; $t(9) = -3.009$; $p = .016$. There is a significant difference.

Test 11 – argument structure:

Normals mean = 89.667; dysphasics mean = 125.6; $t(9) = -2.765$; $p = .02$. There is no significant difference according to the criteria used in this paper.

Written samples

Method. The notebooks that were kept for the full school year by two of the grandchildren, a 10-year-old boy (T) and an 11-year-old girl (C), were studied. Neither of these children are included in the tests described above. The form of the verbs used and the teacher's corrections of these verbs was studied (for more information about the notebooks, see Discussion). In T's notebook, there were 25 entries with approximate lengths of 35 words. In C's notebook there were 21 entries with approximate lengths of 50 words.

Results. Tables 3 and 4 show the distribution of correctly written verb forms in the two notebooks.

Paired two-tailed t tests were performed comparing the percentage scores for regular verbs versus irregular verbs: $t(11) = -7.94$; mean $x - y = -61.42$; $p = .00001$. There is a significant difference.

The percentage of irregular verbs that were correct was greater for all three measures and for both subjects, T and C, as shown in Figure 19. Figure 19 shows clearly that correct irregular verb forms occur with much greater frequency than regular forms.

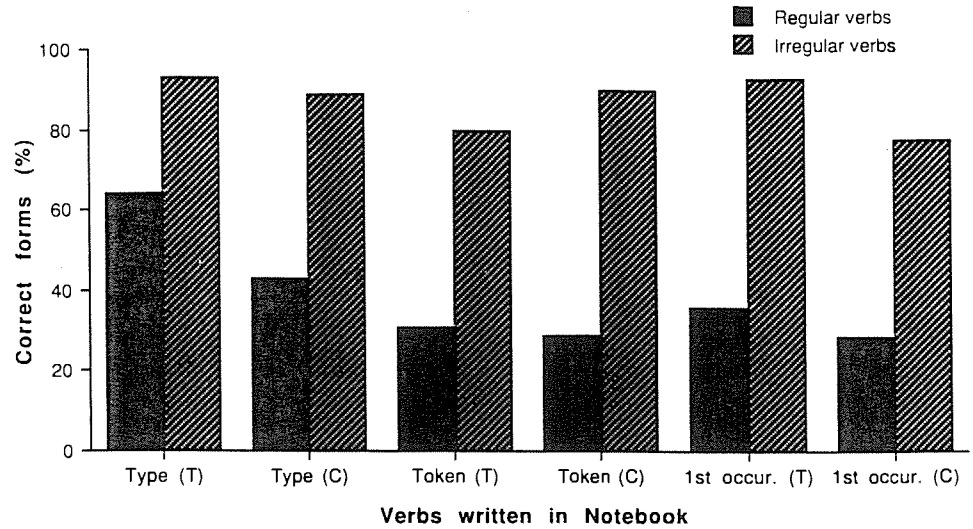
Table 3. *Regular verbs*

Subject	Type	Token	Type correct	Token correct	Correct on 1st occurrence
T	11	48	7 (64%)	15 (31%)	4 (36%)
C	7	17	3 (43%)	5 (29%)	2 (29%)

Table 4. *Irregular verbs*

Subject	Type	Token	Type correct	Token correct	Correct on 1st occurrence
T	14	45	13 (93%)	36 (80%)	13 (93%)
C	9	115	8 (89%)	103 (90%)	7 (78%)

Figure 19. Correctness of written verb forms for T and C.



Discussion

Evidence from other subjects

With respect to developmental dysphasia there has been converging evidence from English (Gopnik, 1990a, 1990b) and German (Clahsen, 1989) that errors characteristic of this population can be accounted for by postulating that a part of the grammar, which for independent theoretical linguistic reasons is considered to be more or less autonomous, is selectively impaired. Leonard et al. (in press), using a somewhat younger population without familial aggregation as a variable, suggest that their data from Italian point to a perceptual saliency explanation rather than a linguistic explanation. There are, at the moment, data that appear to support and data that appear to refute both of these accounts. It may be that the populations are different. It may be that crucial data have not been collected or it may be that the acquisition of morphology is dependent on several different variables. These questions will be elucidated by providing careful linguistic analysis of the data, by making the hypotheses explicit and by detailed interactions among scientists studying these questions. The hypothesis being pursued in this paper is supported by Gopnik (1990a, 1990b), using data from English, and Clahsen (1989), using data from German, who have shown that the language that is seen in some

dysphasics can be accounted for by postulating that one particular level of grammar that represents abstract morphological features is impaired.⁶ It can be shown, as required by such a theory, that all such features, including number, gender, animacy, mass/count, proper names, tense and aspect, are impaired and, furthermore, that all of the grammatical consequences of these features, such as the selection of appropriate determiners, feature-checking for "progressive", and the dropping of subject pronouns before untensed verbs, are also impaired. For example, in normal language, the feature "number" has several consequences: it is marked morphologically on the lexical item; it constrains the choice of determiners; it agrees with the marking on the verb; it agrees with a referring pronoun; and it matches the features of the referent in the world. The data presented in Gopnik (1990b) show that a developmental dysphasic makes errors in each of these domains. We find "three Christmas tree", "a computers", "Superman jump", "Jimmy don't like it, so *they* throw the bowl on the floor", and "the Montreal Forums" (where the subject knows that there is only one Forum). In the case of progressive verbs, it has been proposed for other reasons having to do with normal language (Travis, in press) that "be" and "ing" are both independently generated and each is marked with the feature *progressive*. A feature-matching rule checks to see that both parts of the verb phrase have the feature *progressive*. It follows that if there are no features then no such feature-matching rule can operate and therefore all three forms – "The queen is hiding", "The queen hiding", and "The queen is hide" – should occur. And they do. As we have mentioned above, both the fact that "s"-marked forms are produced and the nature of the pattern of errors in aspect marking are inconsistent with the perceptual saliency hypothesis. Another more complex consequence of the absence of such abstract morphological features is the dropping of pronouns in unallowable contexts: "When they play get points." This does not immediately appear to be connected to features, but theoretical considerations from other normal languages argue that case marking on subject pronouns is triggered by the feature "tense" in the verb phrase and pronouns that are not case marked can be optionally dropped (Guilfoyle, 1984). Therefore, the fact that dysphasics drop subject pronouns follows from the fact that they do not have the feature "tense".

Although some dysphasics are severely impaired with respect to features,

⁶Though Gopnik and Clahsen agree that the deficit is in the feature-marking system, they differ on the precise characterization of the deficit. Gopnik argues that dysphasics cannot mark features in the underlying lexicon; Clahsen argues that though they have the features they cannot construct agreement rules based on these features. The Clahsen hypothesis is consistent with the feature-deficit hypothesis but there are data like the dropping of subject pronouns that can only be accounted for by the more general hypothesis.

other parts of the grammar may be unimpaired. For example, there are cases in which there is no impairment in the ability to produce or understand argument structures, at least in simple linguistic contexts. Such subjects can make reliable judgements about the patterns of nouns that can occur with particular verbs. In spontaneous speech, they do not make errors of this type and in grammatical judgement tests they recognize that the sentence "He puts" is ungrammatical and can make the appropriate corrections. In addition, sentences with syntactic complexity such as "I know how to play basketball" are produced and comprehended by dysphasics. The feature errors occur in every aspect of language: spontaneous speech, writing, grammatical judgement, and repetition. The fact that the same errors occur in all aspects of language argues for the hypothesis that the problem is more likely to be in the underlying grammar than in the auditory processing system. It will be shown in the sections that follow that the test results from the family described above are consistent with predictions that follow from the feature-deficit hypothesis.

Feature errors

Test 9 was designed to test the subjects' ability to recognize errors in feature marking and to demonstrate that they understood the source of the error by correcting the mistake. As the feature-deficit theory would predict, there was a significant difference between the normals and the dysphasics in their ability to judge whether a sentence was correct with respect to features. In fact the performance of the dysphasics in making these judgements was no better than chance. Their ability to correct feature errors was also significantly different than that of the normals. The normals seemed to understand precisely what was wrong with the sentence and specifically corrected the feature error. The dysphasics often changed parts of the sentence that were correct: "Roses grow in the garden" was corrected to "The roses grow in the garden"; "The boy eats three cookie" was changed to "The boys eat four cookie." The dysphasics reported explicitly that they found the feature judgement test very difficult. They made remarks like "I'm not too sure if that's right or wrong." They often repeated the test sentences to themselves and took a long time to consider before they responded. This is reflected in the significant difference between the dysphasics and the normals in the time that it took for them to do the test.

In addition to the general feature test that tested for the full range of features, there were tests and other data that provide additional information about some of the specific features.

Number

Tests 1 and 2 were designed primarily to ascertain the subjects' auditory discrimination for plural marking, as well as their ability to follow instructions. It has been suggested (Tallal, Stark, Kallman & Mellits, 1980) that the widely reported inability of dysphasics to produce plural marking is the result of a deficit in auditory processing that interferes with their ability to process the final "s". There was no significant difference between the normals and the dysphasics on these two tests. These results indicate that the subjects could perceive the difference between "s"-marked forms and unmarked forms and could reliably associate this difference with number. The fact that the performance of the dysphasics on this task was not statistically different from the normals might be interpreted to indicate that they had acquired the rule for plural formation in English. However, these responses are also consistent with the hypothesis, for which more evidence will be cited later, that these subjects have learned feature-marked words as unanalyzed lexical items rather than as the output of morphological rules that operate on the abstract feature "number" in the underlying representations. Under this hypothesis individual "s"-marked forms are, for these dysphasics, simply unanalyzed lexical items that include "numerous" in the specification of their meaning. "Books" therefore is interpreted by them as an unanalyzed lexical item meaning "several reading objects" rather than being generated by the morphological rules from an underlying representation of the form *book + plural*. This interpretation of their underlying grammar provides an explanation for the unexpected behavior reported above. Because these forms are not generated from an underlying rule that uses "s" to mark an abstract generalized distinction between singular and plural these subjects have not internalized the singular/plural contrast. For them the lexical item *books* simply means lots of books and this is reflected in their performance on this test.

In order to test the hypothesis that the dysphasics lexicalize "s"-marked forms and do not generate them from a pluralization rule, their ability to pluralize nonsense words that they had never encountered before was tested (Test 3). There was a significant difference between the dysphasics and the normals on this test. In addition, as described above, the performance and explicit comments of some of the dysphasics suggest that they are referring to explicit rules for solving the problem instead of implicit internalized rules.

Tense

Test results. Test 12 was designed to test the subjects' ability to produce appropriately marked tense. There was a significant difference between the normals and the dysphasics on this test, under both a strong and a weak

criterion of correctness. The normals clearly understood the intention of the test and were able to produce 78% of the predicted responses and 92% correct responses. The dysphasics, on the other hand, did not produce tense-marked verbs in response to the stimulus sentences. They produced only 31% of the predicted answers and 38% correct answers. The answers that were given by the dysphasics were always semantically relevant, but rarely involved a change of tense of the verb. One subject, given the stimulus "Every day he walks eight miles. Yesterday he ____", responded: "had a rest". After being prompted by the experimenter to produce a response that was related to the stimulus word, the subject responded: "walks". The dysphasics had two different strategies of responding to this test. They either produced an unmarked form of the stimulus verb even if an ungrammatical sentence resulted, or they produced a semantically relevant response that resulted in a grammatically correct sentence. What they could not do, and what the normals in the family did with no difficulty, was to comprehend that the point of the test was to manipulate tense marking. The results show that the dysphasics used the same strategy in the test of derivational morphology that will be discussed in a later section of this paper. In addition it should be remembered that all but one of the dysphasics have received extensive language therapy. The two youngest dysphasics that are reported on in this paper have been in a special school for language-disordered children for 12 and 13 years, respectively. The therapists at the school report that they have been instructed every week on tense marking during this time. Yet even with all of this explicit instruction they have not internalized an abstract morphological system that would allow them to produce tense-marked verbs when required to do so.

Written data. An analysis of the written notebooks⁷ of two young dysphasics – T, who was 10 years old, and C, who was 11 – revealed some interesting properties of their use and acquisition of tense marking. Every Monday throughout the term each child wrote a composition about what had happened over the weekend. The first entry is September 12th and the last entry is June 5th. There are 22 entries in C's notebook, with an average length of about 50 words and 25 in T's, with an average length of about 35 words. The difference in length is primarily attributable to the first subject's strategy of constructing the narratives by telling where she went and with whom:

Monday 17th October

On Saturday I went to nanny house with nanny, mummy, John and Carol and we went to Uncle John house with mummy, nanny, John,

⁷The third notebook was not appropriate for this analysis because it included several dictations and did not preserve the teacher's corrections.

Carol and Stewart and we had cup of tea with Uncle John, nanny, John, mummy. Carol and Stewart and we went to nanny's house and we went to my home. On Sunday I went to church with mummy. John and Carol and we came my house and I went to nannys house with mummy.

These notebooks provide the data given in the results section about written tense. Because the stories all concern events of the previous weekend almost all of the verbs should be in the past tense. The teacher corrected the notebooks each week by actually writing the correct forms in the notebook itself. The children had access to these corrections as they wrote the new entries, though it is impossible to know if they took advantage of the overt availability of the corrections in the previous entries. It is clear, however, from the patterns of occurrence of the past tense forms that some learning did take place. These notebooks can give us insights into which past tense forms are known and what the pattern of learning is over the school year. While these notebooks do provide data about tense, they are not useful sources for information about number. In the first place many of the noun phrases are either proper names or other forms that do not receive number marking: "On Saturday I eat my *breakfast*." In those instances where the noun phrase can be marked for number it is difficult to know what the intended referent is and therefore the error in number cannot be established:

On Thursday mum, and sharmaine and me went shopping bought knife and fork and spoon and torch with battery and we comes back home.

[Note the omitted subject pronoun before "bought" which as we have discussed above, argues for the non-occurrence of the feature tense.]

It is clear that the noun phrases are incorrect. It is likely that they bought several knives, forks and spoons and only one torch with one battery. However, such observations are merely inferences and cannot be used as data. Therefore the notebooks have been used as data for tense but not number.

The data given in Figure 19 show that irregular verbs are correct significantly more than regular verbs for both subjects and for both types and tokens. Moreover the dysphasics are more likely to get an irregular verb correct on its first occurrence than a regular verb. This pattern can be elucidated by examining the notebooks in more detail. They reveal that regular past tense verbs are learned in response to the specific corrections that are made by the teacher. However, it is only the specific lexical form, not an underlying rule, that is learned. For example, in the writing sample of the 10-year-old the first entry of the year refers to his activities on the previous weekend:

Monday 12th September

On Saturday I watch T.V. and I watch plastic man and I watch football. On Sunday I had pork and potato and cabbage.

The teacher inserts an "ed" after each occurrence of "watch". The subject gets "watched" right the next time he uses it, but he does not mark past tense on other verbs:

Monday 17th October

On Saturday I got up and I wash my self and I get dress and I eat my breakfast and I watched TV all day and I went to bed. On Sunday I got up and

Again the teacher corrects the past tense by inserting an "ed" after "wash" and "dress" and "get" to "got" and "eat" to "ate". Again he learns these lexical items. Two stories later he writes:

Monday 28th November

On Saturday I got up and I got dressed and I watched Motormouth ... and I ate my dinner.

The pattern throughout the year demonstrates clearly that the subject learns the individual past tense forms as they are corrected by the teacher, but does not generalize these corrections to new verbs.

It is interesting to note that of the 11 regular verbs only four, each of which occurs only once, are correct in their first occurrence: "showed", "asked", "called" and "picked". If we look at the frequencies of these verbs in the present and past (Kuchera & Francis, 1957) we find:

ask, 128; asked, 398; call, 188; called, 401; pick, 55; picked, 78; show, 287; showed 141

For all of these words except "show" the past tense form is more frequent than the present tense form. For the other seven that occur incorrectly in their first occurrence all except one (stop 120, stopped 129)⁸ occur less frequently in their past tense form. These forms occur incorrectly at first and are corrected by the teacher. The three that recur do so correctly.

Irregular verbs behave very differently. There were 15 irregular verbs in the stories. Eleven of the 15 occur correctly throughout. Only one "eat" occurs incorrectly in its first occurrence, is corrected by the teacher and then occurs correctly. The most frequently used irregular verb, "go", occurs 21 times, 13 times as "went" and eight times as "go". However, instead of the learning pattern that we see with the regular verbs, the pattern appears to be

⁸The difference here is small enough perhaps to be not significant and to represent a sampling error.

one of intermittent use. The form "went" occurs correctly eight times before there is an occurrence of "go". The two instances of "be" occur in the present form:

On Saturday my breakfast is beans and bread.

These verbs are not corrected by the teacher, who is presumably interpreting the sentence as a generic statement and not simply a description of a past event. The pattern in the other notebook is very similar as can be seen in the data provided in the results section.

The best explanation for these data is that the past tense verbs are not produced by an underlying productive rule, but rather are simply learned as unanalyzed lexical items. Such an analysis is in accord with proposals already made. Pinker and Prince (1988) propose that strong verbs must be memorized while the past tense of regular verbs can be generated by rule. They argue that the facts of normal language development are clearly on the side of the psychological reality of a dissociation between memorized and rule-generated processes in the acquisition of language in general and verbs in particular. But, as the tense-changing test shows, these dysphasics do not have such rules. Therefore they must learn the regular verbs in the same way as Pinker and Prince propose that normals learn irregular verbs, by memorization. When the teacher corrects a particular verb they can learn the new item by memorizing it, but because this learning never gets encoded into a rule they must learn the next word in the same laborious way. Because regular verbs are not rule generated in these dysphasics, differences in frequencies should have an effect on learning; if the past tense of a verb occurs more frequently than the present it is likely that it will be learned before the present tense form, and vice versa. And this is just what the data show to be the case.

These longitudinal data, coupled with the data from nonsense plurals, support the hypothesis that the forms which the adult dysphasics produce and that appear to be correct from the point of view of feature marking are in fact merely memorized unanalyzed lexical items. They are not rule generated; therefore they cannot be considered to be evidence for the existence of abstract morphological feature marking in the underlying grammar. This is a good example of the argument made at the beginning of this paper that two identical surface forms may in fact be produced by two very different underlying grammars. It is only by understanding the nature of the underlying grammar which produces the surface forms that we can decide whether two surface forms are in fact equivalent. It seems clear, as Pinker and Prince argue, that there are two routes for acquiring words such as plurals and past tense: either you can construct an underlying system of feature marking and then produce these words by rules that operate on these features, which is

done by all normal 3-year-olds, or you can memorize each word as an un-analyzed individual lexical item, which can be done but takes 30 or 40 years to accomplish and then only imperfectly.

Aspect

The written data also provide us with information about aspect. It has been pointed out above that evidence from the structure of normal language requires that *be + progressive* and *ing + progressive* be independently freely generated, and then a feature-checking rule eliminates strings in which only one element is marked *+ progressive*. In the absence of abstract morphological marking there can be no such *+ progressive* marking and therefore no feature-checking rule to ensure that the feature *+ progressive* occurs on both the verb "to be" and the main verb. Therefore all three forms can occur. In C's notebook there are three progressive aspect verbs – one of each kind:

Carol is cry in the church.
and hoping father Christmas came to erve [every] house.
Anne is fighting.

In T's notebook there are eight progressive aspect verbs. In seven of them the "be" does not occur, for example:

I walking down the road.

In the eighth both "be" and "ing" occur:

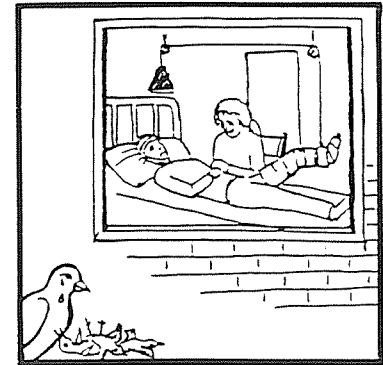
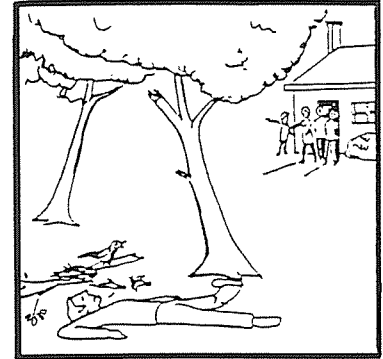
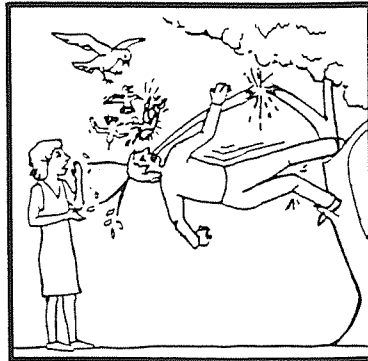
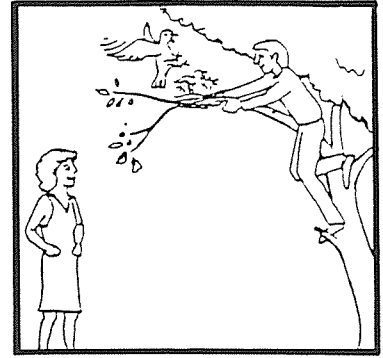
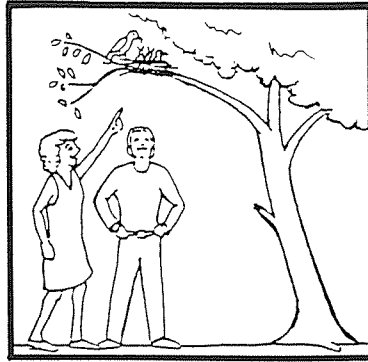
Mum is fighting.

These data, coupled with the data from the features test, confirm the hypothesis that the dysphasics do not have an abstract morphological marker for progressive and therefore cannot have a feature-matching rule.

Anaphoric pronouns

Three of the tests reported above provide information about the subjects' use of pronouns. In Tests 5 and 6 the subjects were required to match a picture to a sentence with pronouns. There was no significant difference on these two tests between the normals and the dysphasics. Furthermore, the results from Test 13 indicate that the ability of the dysphasics to comprehend a brief narrative is not significantly different than the normals. The dysphasics could represent and recall the events depicted in a brief narrative as well as the normals. It should be noted, however, that understanding the stimulus text did not rely in any way on features because all of the intersentential cohesion was accomplished by lexical repetition and not by pronominal reference.

Figure 20. Images used in test to depict a narrative.



Though these results would seem to indicate that the dysphasics can use pronouns and represent narratives, Test 14 reveals a more complex picture. In Test 14 the subjects were shown a page with six cartoons that depicted a narrative (Figure 20) and they were asked to tell the story shown in the pictures.

These tests reveal a very interesting pattern of pronominal reference that supports the hypothesis that pronouns, which are feature marked in the normal grammar, are lexicalized in these grammars. Under this lexicalization hypothesis the entry "he" in the lexicon simply means an unspecified male. If pronouns are represented as lexical items with no feature marking then it would follow that pronoun tests, such as Tests 5 and 6, which require the subject to match a picture to a sentence containing pronouns, would not present a problem because they could be performed on the basis of the meaning of the lexical item alone. However, real anaphora, which requires that features on the pronoun be matched to those on the noun phrase to which it refers, cannot be accomplished if there are no such abstract morphological markers. The narratives that the dysphasics produce in Test 14 show precisely this pattern. All but one of the adult dysphasics – the grandmother – produce stories that include information not explicitly depicted, such as inferred intermediate events and attributed intentional states; therefore, they are clearly constructing a narrative and not merely describing the pictures. The grandmother does not seem to be constructing a narrative based on the pictures, but rather to be describing the individual pictures and using the pronouns as lexical items to refer to the characters in the pictures:

There's a guy gonna climb a tree. He climbs the tree. He falls down and hurts hisself. He's taken to the hospital in an ambulance and he's in a hospital bed.

Each of the six sentences merely describes one of the six pictures. Only one picture in which the neighbors are shown is not described, and there are no inferred actions or intentional states. All of the other stories produced do have these properties of narratives.

However, the intersentential cohesion in these stories is typically accomplished, not by the expected use of pronouns, but rather by the repetition of full noun phrases:

The lady's pointing at the bird and the man is watching her. The man is climbing the tree looking at the birds in their nest. The lady is crying because the man what fall off the tree (pause) and the bird frowd away. The neighbors phone the ambulance because the man fall off the tree. The ambulance come along and put the man into the ambulance to the hospital, and he's got a broken leg in the hospital.

The normals, on the other hand, typically produce stories in which the cohesion is accomplished by pronominal anaphora:

The woman's looking at the birds and she's saying to him, "Oh, they're lovely birds, but they got stuck in the tree." So he says, "Well, I'll get them down then." So he climbs up the tree and the branch breaks. So she's nearly in tears cause she swallowed the wrapping string, and the birds fly off and they fall to the ground cause they can't fly. So he falls on the ground and he breaks his leg. So she runs indoors to call an ambulance with all the neighbors outside the door. The ambulance comes. They get in the van and he's in the hospital, and the birds crying by the window.

The results show that the dysphasics use significantly more full noun phrases than pronouns in constructing narratives. These results are consistent with the hypothesis that the dysphasics do not use pronouns anaphorically, but rather that for the dysphasics pronouns are lexical items that are unmarked for features.

Derivational morphology

The performance of the dysphasics on the derivational morphology test was very similar to their performance on the tense change test, both in terms of their scores relative to that of the normals and also in terms of the strategy that they used to answer the questions. In both of these tests the dysphasics seemed not to be able to understand that the point of the test was to manipulate an underlying grammatical rule, though the normals understood with no hesitation. Even when they were prompted with examples and with specific instructions they seemed unable to perceive that there was an underlying pattern that was governed by a general rule. In this context their strategy of responding semantically was intelligent. The semantic responses that they gave filled the blank in the second sentence in such a way as to produce a syntactically grammatical sentence, and the meaning of the sentence they produced was related to the prior sentence. The fact that the dysphasics behave with respect to derivational morphology in very much the same way as they behave to inflectional features such as tense suggests that inflectional and derivational morphology are deeply similar and have the same or at least closely related psychological representations. This view that derivational and inflectional morphological processes are essentially the same is argued for on purely theoretical grounds by DiSciullo and Williams (1987, p. 69):

Are derivational and inflexional affixes formally different? It appears that they are not – any devices available for one kind of process also seem to be available for the other. The separation of affixes into derivational and inflectional ones

seems entirely a matter of interpretation. It is roughly true that some affixes have more syntactic consequences than others, but it would be best to explain this in terms of the intrinsic properties of the affixes themselves rather than by simply assigning each affix to one of the two groups.

Within this theoretical framework it is to be expected that an inability to do abstract morphology would affect those processes traditionally referred to as derivational in much the same way as those termed inflectional. DiSciullo and Williams conclude their book with a question (p. 110):

If we are correct in our conclusion that syntax and morphology are parallel but independent subcomponents, then we must ask why this is so, why there is not one grand science of the word/phrase. But on this question we do not have a speculation.

It may be that the answer to this question lies in evolutionary processes that gave rise to a gene that controls for the level of abstract morphology.

Argument structure

The task given to the subjects to test their ability to judge and correct errors in argument structure was identical to that which was used for features. Yet in this test there was no significant difference between the normals and the dysphasics in either their ability to make the appropriate judgements or to correct the errors. Therefore the significant difference that was seen between the normals and the dysphasics in judging and correcting errors in the test of abstract morphological features that was identical in structure to the argument structure test cannot be attributed to any difficulty in understanding the experimental task of making grammatical judgements and corrections. The dysphasics do understand the task and can perform as well as the normals when the task itself is within their competence. They can process errors in argument structure, but they cannot recognize errors in feature marking. Other more detailed data have shown that this type of dysphasic has no problem with producing or understanding thematic relations in simple sentences (Gopnik, 1990b).

Test style

The dysphasics and the normals not only differ in their responses to the tests, but even more strikingly in the way they take these tests. In the feature tests the dysphasics puzzle over each example, often repeating the item quietly to themselves. There are long hesitations and qualifiers such as "I'm not very

good at this." They respond as if they are solving a very difficult problem: as if they were asked to make a grammatical judgement in a language they were only somewhat familiar with. The normals, on the other hand, have absolutely no hesitation in responding to these items. In order to quantify this observed difference in test response the time the subjects took to respond was determined. As reported in the results section the dysphasics took significantly longer to do the tasks that involved abstract morphology.⁹ The adult dysphasics report that they find using language very stressful and tiring. They report that they often plan what they are going to say and, when possible, avoid situations in which they must speak.

An explanatory learning model

In the previous sections we have provided data that show that particular abstract morphological rules are not acquired and that learning proceeds lexically. The question is whether this makes sense in terms of a model of how morphology is learned in the course of normal development. It has been argued above that the fact that inflectional and derivational morphology are similarly impaired is consistent with at least one current view of the morphological component. From the point of view of language acquisition it is important to show that the phenomena discussed above are the result of an impairment in a coherent part of an underlying learning mechanism for language. Such an explanation is provided by Pinker (1984) in Chapter 5, which is concerned with the acquisition of inflection. In this chapter, Pinker argues that the data from the way in which inflection is normally acquired require a learning theory that postulates that children are paradigm builders. The model specifies detailed constraints on the way in which such paradigms are constructed and the way in which they are extended. But what is fundamental to this hypothesis is that the child must know that language is likely to have underlying regularities and that these regularities are representable in the form of paradigms. The child must know further that these paradigms are not simply generalizations over observed cases, but that they represent general properties of the morphological system that can be predictive of new cases. It would seem that this knowledge of paradigm building is just what is missing in the dysphasics. They do not know that individual instances must be seen as evidence for the construction of paradigms that encode morpholog-

⁹The precise psychological mechanisms that underlie this delay are not yet clear. Tests are now being devised to investigate this problem.

ical regularities: they appear to have a learning mechanism that sees each word as an independent item that must be learned and entered into a lexicon that specifies its grammatical properties and meaning. The model presented by Pinker for normal acquisition provides an account of morphological learning that can be selectively impaired in such a way that it would produce exactly the phenomena that have been described above.

Conclusion

The aim of this paper was twofold: to provide evidence that some cases of developmental dysphasia may be caused by a genetic disorder and to provide a principled linguistic account of the language in a family that may have such a disorder. Convergent data from case studies, large statistical studies, and twin studies indicate that such a genetic disorder is likely to exist. Neurological data suggest a possible underlying cause for this disorder. The data presented above have been shown to be consistent with a proposal for a linguistically principled account of a possible form of this disorder. This account suggests that the dysphasics do not have the normal language-learning mechanism, described by Pinker (1984), that would allow or, perhaps even compel them, to construct inflectional paradigms on the basis of regularities hypothesized from the observed linguistic evidence. The inability to construct such paradigms would result in a selective impairment of that component of grammar that encodes abstract morphology, while sparing other linguistic abilities such as the determination of argument structures. The data from this family support a model of language learning in these subjects in which the apparent competence exhibited by the adults appears to be a result of a lexical learning strategy rather than the construction of rules that operate on underlying abstract morphological properties. Taken altogether, it is not unreasonable to entertain an interim hypothesis that a single dominant gene controls for those mechanisms that result in a child's ability to construct the paradigms that constitute morphology.

Although the data presented in this paper are not inconsistent with such an interim hypothesis, they are clearly limited. Several unanswered questions remain. With respect to this family alone, the phonological analysis is still incomplete, the analysis of spontaneous speech and the analysis of the data from the younger members of the family is just underway. It may be that their performance on tests is not the same as their spontaneous production. For example, a preliminary analysis of the transcripts does not reveal any major patterns of syntactic errors; however, we do not know yet whether this is the result of the general syntactic competence of the dysphasics or if it is

the result of their avoidance of complex structures. The pattern that we see in this family may be idiosyncratic or may be the result of the peculiarities of English.

It would seem that there are several fundamental questions that are raised, but not fully answered, by the many studies that have been undertaken so far. Among these are: (1) Do developmental language disorders within familial aggregations form a coherent population that differs in significant linguistic ways from other language-impaired populations? (2) If so, what are the particular linguistic properties of the language of these subjects and what sort of underlying grammar must be postulated to account for these properties? (3) How do the linguistic manifestations of this disorder differ across diverse languages? (4) What is the adult state of this disorder and in what way is it achieved? (5) Are these linguistic problems either associated with or caused by auditory, perceptual, or cognitive problems? In order to answer these questions, it is essential that data from other familial aggregations and in other languages be investigated. Many people are working on these questions. For example, Clahsen (1989) in German and Leonard et al. (1990) in Italian provide important data in this direction, although they do not look specifically at family aggregation populations. Tomblin is now undertaking a detailed study of the language of his English-speaking subjects within familial aggregations. Leonard and Tallal, among others, are actively investigating alternative explanatory hypotheses. The data presented here suggest that at least for some populations the feature deficit hypothesis merits further investigation.

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