J. Child. Psychol. Psychiat. Vol. 28, No. 1, pp. 1–8, 1987
Printed in Great Britain.

0021-9630/87 $3.00 + 0.00
Pergamon Journals Ltd.

ANNOTATION

THE CAUSES OF SPECIFIC DEVELOPMENTAL LANGUAGE DISORDER ("DEVELOPMENTAL DYSPHASIA")

D. V. M. BISHOP
University of Manchester, U.K.

A SPECIFIC developmental language disorder is by definition a condition with no obvious cause, since the diagnosis explicitly excludes obvious explanatory factors. How then are we to account for children who fail to develop normal language despite having reasonable non-verbal ability, adequate hearing, no sign of physical or psychiatric disorder, and an unexceptional home background?

Localized brain damage

Children with specific language disorders are sometimes referred to as "developmental dysphasics", suggesting an analogy with dysphasia caused by left hemisphere brain lesions in adults. However, there is seldom any evidence for acquired brain damage in the medical histories of language-disordered children, and studies using more direct techniques to investigate neurological status have yielded contradictory results. Rosenberger & Hier (1980) found no brain lesions on CT scans in 53 learning-disabled children, many of whom had selective verbal impairments and a history of language delay (although abnormal cerebral asymmetry was found in this group). In contrast, Caparulo, Cohen, Rothman, Young, Katz, Shaywitz & Shaywitz (1981) found markedly abnormal CT scans in six out of 16 language-disordered children. Dalby (1977a and b) described abnormal dilation of the temporal horns on pneumoencephalography in 46 out of 87 language-disordered children, with left-sided dilation being especially common. He also found a high rate of atrophic lesions of the cerebellum and brain stem. Such discrepant results might arise from differences in the nature and severity of the disorders studied. Caparulo *et al.* (1981) included several children with associated motor or behavioural impairments, and comprehension problems were a significant feature in at least three of the cases with abnormal scans. Dalby's study is not described in detail but 20% of his sample were below normal or untestable on non-verbal intelligence tests, 20% had epileptic attacks, and some cases were dysarthric. Results will also be influenced by the criteria used for rating abnormality, and the extent to which ratings are blind and objectively specified. Harcherik, Cohen, Ort, Paul, Shaywitz, Volkmar, Rothman & Leckman (1985) used quantitative analysis and blind assessment of CT scans to compare children with neuropsychiatric disorders (including nine cases of developmental language disorder) and a control group. They found no differences in ventricular size or

Requests for reprints to: Dr D. V. M. Bishop, Department of Psychology, University of Manchester, M13 9PL, U.K.

asymmetry nor in brain density between these groups, and concluded that developmental language disorder is seldom associated with CT scan abnormalities unless the child has additional neurological problems.

Lou, Henriksen & Bruhn (1984) suggested that developmental language disorders may result from morphological brain abnormalities not detectable by CT scan. They measured regional cerebral blood flow in eight language-disordered children and reported hypoperfusion of certain brain areas, which they suggested was attributable to an early hypoxic–ischaemic lesion. However, all but one of the language-disordered children had attention deficit disorder and/or visuospatial problems, and no information was given concerning their non-verbal intelligence, leading one to question whether the blood flow abnormality was characteristic of specific language disorder or indicative of more generalized impairment. On visual inspection of their data it is difficult to discriminate the results of language disordered children from those with attention deficit disorder and normal language. Furthermore, it could be that low metabolic activity of language areas is a *consequence* of the inadequate functioning of the brain in language-disordered children, but not related to the cause of that disorder.

Results from electroencephalography will depend on the type of language disorder studied. Electroencephalogram abnormalities, often in the form of spike-waves in the region of the temporal lobes, are frequently found in those rare children who develop severe comprehension problems after a period of normal development (Landau & Kleffner, 1957). Normalization of the EEG by anticonvulsants seldom improves the language disorder (Gascon, Victor, Lombroso & Goodglass, 1973). Electroencephalogram abnormalities can also occur in other types of developmental language disorder (Maccario, Hefferen, Keblusek & Lipinski, 1982), but such cases seem to be uncommon. Waldo, Cohen, Caparulo, Young, Prichard & Shaywitz (1978) found that the incidence of EEG abnormalities in language-disordered children (3/24 with paroxysmal discharges) was no higher than that reported for normal populations (Eeg-Olofsson, 1971).

Given that neurological disease in childhood is very rarely found in children with specific language disorders, if acquired brain damage is responsible, then presumably this must have occurred prenatally or around the time of birth. There is no really satisfactory retrospective study of birth histories of language-disordered children. Pasamanick, Constantinou & Lilienfeld (1956) found no increase in adverse perinatal factors for a group of children receiving speech therapy, but they included various disorders (including stuttering) and it is unclear how many of their cases would have fitted the definition of "specific language disorder". Fundudis, Kolvin & Garside (1979) found a lower mean birth weight and shorter mean gestational age in children with "speech retardation" compared to controls, but their experimental group included many children who were retarded in milestones other than speech.

Studies which adopt the converse approach of following up outcome in children who have survived various perinatal hazards are too numerous to review here. Whilst there have been suggestions that specific language impairment is linked to various perinatal risk factors (e.g. De Hirsch, Jansky & Langford, 1964; Fitzhardinge & Steven, 1972), the vast majority of studies report either global delay, affecting motor, social and non-verbal as well as language development, or no intellectual impairment even after severe perinatal hazards, except for children with major handicapping conditions

(e.g. Ounsted, Moar, Cockburn & Redman, 1984; Stewart, 1984). Having said this, we should note that few studies in this area present their data in a way which is helpful to those interested in the aetiology of specific learning disabilities. Some studies look only at one type of ability at a time, making it impossible to distinguish a specific from more generalized developmental delay. Others give group means for a range of cognitive tests, which can be ambiguous. If a group of children has depressed mean scores on both non-verbal and verbal measures, this could mean that most children are impaired in both areas, or that specific learning disabilities are common, but that some children have a selective verbal impairment whilst others have a selective non-verbal impairment.

In general, studies of children with verifiable lesions do not support the idea that specific developmental language disorders are caused by early localized brain damage. Lesions of the left cerebral hemisphere sustained before one year of age do not result in dysphasia, but rather produce general intellectual impairment affecting both verbal and non-verbal skills (Woods, 1980). Insofar as children with early left hemisphere lesions do have selective verbal impairments, these are unlike the language problems of children with developmental language disorders in both severity and quality (see Dennis & Whitaker, 1977; Woods & Carey, 1979; Bishop, 1981, 1983; Vargha-Khadem, O'Gorman & Watters, 1985).

Auditory–verbal deprivation. I: Home verbal environment

In order to learn language, the child must hear language. Do specific language disorders then result from some deficiency in the quantity or quality of language directed to the child? Studies contrasting the language used by parents of language-impaired children with that of a control group have not found consistent differences (see review by Conti-Ramsden, 1985). Even if one does find an association between parental language and child disorder this does not entail a causal relationship. The parent may adapt his or her language in response to the child's disorder, or it may be that the overlap in genotype and environment between parent and child results in both having a similar verbal deficit. To demonstrate a causal relationship one would need to show that modification of parental speech improves the child's language disorder.

Studies of children in extreme environments indicate that the effects of grossly inadequate parental language can be largely compensated for by adequate verbal experience outside the home. Hearing children of congenitally deaf parents are exposed to severely defective spoken language, yet most of them rapidly develop appropriate language skills once they go to school or nursery (Critchley, 1967; Schiff, 1979). Other children who have been subjected to severe neglect and isolation have made rapid progress in language development when placed in a normal environment (Skuse, 1984). Such good outcome is not universal, but these cases demonstrate that a grossly deficient language environment early in life need not result in permanent verbal impairment.

Auditory–verbal deprivation. II: Recurrent otitis media

A very different type of environmental theory attributes developmental language disorder to deficient auditory stimulation arising from the fluctuating conductive hearing loss associated with secretory otitis media.

Neurophysiological studies with animals have shown that temporary deafness early

in life can affect the development of neuronal interconnections in the auditory system, raising the possibility that a condition such as secretory otitis media could result in long-term impairment of auditory functioning even after the middle ear has returned to normal. Experimental studies on this issue are contradictory and fraught with methodological problems. Verbal deficiencies are found in children with a history of recurrent middle ear disease, but these impairments are small and seldom clinically significant, except where the child has a hearing loss at the time of testing (Paradise, 1981). An increased rate of middle ear disease has been reported in children with specific language disorders, but Bishop & Edmundson (in press, a) suggested that this resulted because secretory otitis media is especially likely to be detected and treated aggressively when occurring in association with language impairment, rather than indicating a genuine increase in the incidence of middle ear disease in language-disordered children. Two recent studies which screened whole populations for ear disease, and so would not be affected by such a bias, failed to find any association between middle ear disease and language disorder (Allen & Robinson, 1984; Fischler, Todd & Feldman, 1985). Rapin (1979) raised the possibility that secretory otitis media might interact with other factors, having particularly adverse consequences for a child who was already vulnerable because of neurological impairment, poor environment or genetic predisposition. Bishop & Edmundson (in press, a) found a positive association between parental report of recurrent middle ear disease and perinatal hazards within a language-disordered population, but no association within a control group, lending tentative support to this view.

Genetic influences

Studies of individuals with chromosomal abnormalities have shown that some types of abnormal karyotype, especially 47 XXX and 47 XXY, are associated with specific delays in the development of language and motor skills (Bender, Fry, Pennington, Puck, Salbenblatt & Robinson, 1983; Netley, 1983). However, investigations of language-disordered children found that although there is an increased incidence of chromosomal disorders, such abnormalities can account for only a small proportion of cases (Garvey & Mutton, 1973; Mutton & Lea, 1980; Friedrich, Dalby, Staehelin-Jensen & Bruun-Petersen, 1982). It may be that an improved sub-classification of language disorders would yield clearer results in genetic studies. Garvey & Mutton (1973) noted that all their cases with chromosome abnormalities had severe expressive difficulties but normal comprehension. The more recent reports did not describe such a clear-cut pattern of disorder, although expressive speech disorders, dyspraxia and clumsiness were frequently mentioned.

Recently, there has been widespread interest in the fragile X syndrome, a physical abnormality of the X chromosome which is only detectable if cells are cultured in a folate-deficient medium. Fragile X syndrome is a common cause of mental retardation, and affected individuals tend to have characteristic speech, language, and social deficiencies (Paul, Cohen, Breg, Watson & Herman, 1984; Largo & Schinzel, 1985). Given that developmental language disorders tend to run in families, and are considerably more common in males than females (see review by Pennington & Smith, 1983), one might ask whether fragile X syndrome could cause language disorders in children who are not intellectually retarded. Cases have indeed been

reported of fragile X syndrome in learning-disabled children of normal intelligence (Hagerman, Kemper & Hudson, 1985). However, these children did not have pure language disorders, but rather a constellation of verbal, motor and attentional problems, typically with mild autistic features. Furthermore, pedigree data from Luchsinger (1970), and unpublished results from our own study of children with specific language disorders, show that it is often the father of a language-disordered child who is affected, whereas fragile X must be transmitted from mother to son.

It seems then that chromosomal abnormalities are unlikely to be involved in the majority of specific language disorders. Nevertheless, the tendency for specific language disorders to run in families suggests we should look for evidence of an inherited predisposition to such disorders, arising from a single major gene or from polygenic influences. Techniques such as pedigree analysis, twin and adoption studies, and linkage analysis have provided insights into the genetic basis of stuttering (Kidd, 1983) and specific reading retardation (Childs & Finucci, 1984), and could be fruitfully applied to specific developmental language disorders (see Ludlow & Cooper, 1983).

How might genetic factors influence language development? One possibility is that the timing of neurological development in foetal life is affected. For example, if neuronal migration did not occur within the normal time span, this could result in the kinds of developmental brain abnormality described by Kemper (1984) in post mortem studies of dyslexic individuals. Alternatively, foetal brain development may proceed along normal lines but subsequent myelination then be delayed, producing a maturational lag in language skills. This kind of theory is attractive because it could explain the finding of non-verbal neurodevelopmental immaturities in language-disordered children (e.g. Johnston, Stark, Mellits & Tallal, 1981) without predicting observable lesions on CT scans. Nuclear magnetic resonance imaging of the brain could detect delayed myelination (Levene, Whitelaw, Dubowitz, Bydder, Steiner, Randell & Young, 1982) but to date has not been used to investigate children with specific developmental delays. At present, the only evidence for such a theory is circumstantial. One prediction is that language-disordered children should follow a normal developmental progression, albeit at a slow rate. In general this is supported by studies which find many similarities between the language skills of language-disordered children and those of younger normally developing children (Leonard, 1979, 1982). Bishop & Edmundson (in press, b) found that many children who present with specific language disorders at four years of age are indistinguishable from normal children on language tests by the age of five-and-a-half. The motor skills of these children mirrored their language development, so that whilst all language-disordered children scored significantly below control levels on a pegboard task at four years, only those children who were still language-disordered at five-and-a-half had poor pegboard scores at that age. This makes sense in terms of a 'maturational lag' model, which regards both language and motor impairments as reflections of neurological immaturity rather than brain damage, and predicts that many children will catch up from such developmental delay.

Nevertheless, there are problems for this type of theory. In particular, there is the question why, if language is merely delayed, do not all children eventually recover? Also, why should a 'maturational lag' affect those areas of the brain concerned with language and motor skills, without apparently involving other regions?

Concluding comments

Current research into the causes of developmental language disorders is hampered by the lack of an agreed classificatory system. Results of investigations into aetiological factors will depend to some extent on the type of child studied. Although progress is being made towards sub-classifying the wide range of problems encompassed within developmental language disorders (Rapin & Allen, 1983; Bishop & Rosenbloom, in press), we are still a long way from a generally acceptable and comprehensive scheme. For the present, what is important is that researchers give clear and detailed descriptions of medical, psychological and linguistic characteristics of children studied, so that we can begin to explain some of the discrepancies between findings of different investigators. Expensive and time-consuming investigations which use sophisticated technology to assess brain function in language-disordered children can often study only a small sample of children. It would be useful therefore if results from different studies could be combined or compared, but all too often descriptions of subjects and methods are insufficiently detailed or objective to allow meaningful comparisons.

A second point is that we should give serious consideration to the possibility that aetiological factors may interact in the causation of developmental language disorders, so that the effect of several factors together is greater than the sum of individual effects. Most research studies concerned with aetiology consider one factor at a time, controlling for any differences in other factors. By using such conventional research designs we minimize the likelihood of discovering interactions between aetiological factors.

Acknowledgement—The author is supported by a Medical Research Council Senior Research Fellowship.

REFERENCES

Allen, D. V. & Robinson, D. O. (1984). Middle ear status and language development in preschool children. *ASHA,* **26,** 33–37.

Bender, B., Fry, E., Pennington, B., Puck, M., Salbenblatt, J. & Robinson, A. (1983). Speech and language development in 41 children with sex chromosome abnormalities. *Pediatrics,* **41,** 262–267.

Bishop, D. V. M. (1981). Plasticity and specificity of language localization in the developing brain. *Developmental Medicine and Child Neurology,* **23,** 251–255.

Bishop, D. V. M. (1983). Linguistic impairment after left hemidecortication for infantile hemiplegia? A reappraisal. *Quarterly Journal of Experimental Psychology,* **35A,** 199–207.

Bishop, D. V. M. & Edmundson, A. (in press, a). Is otitis media a major cause of specific developmental language disorders? *British Journal of Disorders of Communication.*

Bishop, D. V. M. & Edmundson, A. (in press, b). Specific language impairment as a maturational lag: evidence from longitudinal data on language and motor development. *Developmental Medicine and Child Neurology.*

Bishop, D. V. M. & Rosenbloom, L. (in press). Classification and overview of childhood language disorders. In W. Yule, M. Rutter, M. Bax (Eds), *Language development and disorders: Clinics in Developmental Medicine* (double issue). London: SIMP/Blackwell Scientific & Lippincott.

Caparulo, B. K., Cohen, D. J., Rothmann, S. L., Young, J. G., Katz, J. D., Shaywitz, S. E. & Shaywitz, B. A. (1981). Computed tomographic brain scanning in children with developmental neuropsychiatric disorders. *Journal of American Academy of Child Psychiatry,* **20,** 338–357.

Childs, B. & Finucci, J. M. (1984). Genetics, epidemiology, and specific reading disability. In M. Rutter (Ed.), *Developmental neuropsychiatry* (pp. 507–519). Edinburgh: Churchill Livingstone.

Conti-Ramsden, G. (1985). Mothers in dialogue with language-impaired children. *Topics in Language Disorders,* **5,** 58–68.

Critchley, E. (1967). Language development of hearing children in a deaf environment. *Developmental Medicine and Child Neurology,* **9,** 274–280.

Dalby, M. A. (1977a). Aetiological studies in language retarded children. *Neuropaediatrie,* supplement to Vol. **8**, pp. 499–500.

Dalby, M. A. (1977b). Diagnose und Aetiologie der Dysphasie. In W. Elstner & H. Karlstad (Eds), *Dysphasie im Kindesalter* (pp. 30–49). Oslo: Universitetsforlatet.

De Hirsch, K., Jansky, J. J. & Langford, W. S. (1964). The oral language performance of premature children and controls. *Journal of Speech and Hearing Disorders,* **29**, 60–69.

Dennis, M. & Whitaker, H. A. (1977). Hemispheric equipotentiality and language acquisition. In S. J. Segalowitz & F. A. Gruber (Eds), *Language development and neurological theory* (pp. 93–106). New York: Academic Press.

Eeg-Olofsson, O. (1971). The development of the electroencephalogram in normal children and adolescents from the age of 1 through 21 years. *Acta Paediatrica Scandinavica, Supplementum 208.*

Fischler, R. S., Todd, N. W. & Feldman, C. M. (1985). Otitis media and language performance in a cohort of Apache Indian children. *American Journal of Diseases of Children,* **139**, 355–360.

Fitzhardinge, P. M. & Steven, E. M. (1972). The small-for-date infant: II. Neurological and intellectual sequelae. *Pediatrics,* **50**, 50–57.

Friedrich, U., Dalby, M., Staehelin-Jensen, T. & Bruun-Petersen, G. (1982). Chromosomal studies of children with developmental language retardation. *Developmental Medicine and Child Neurology,* **24**, 645–652.

Fundudis, T., Kolvin, I. & Garside, R. F. (1979). *Speech retarded and deaf children: their psychological development.* London: Academic Press.

Garvey, M. & Mutton, D. E. (1973). Sex chromosome aberrations and speech development. *Archives of Disease of Childhood,* **48**, 937–941.

Gascon, G., Victor, D., Lombroso, C. & Goodglass, H. (1973). Language disorder, convulsive disorder, and electroencephalographic abnormalities. *Archives of Neurology,* **28**, 156–162.

Hagerman, R., Kemper, M. & Hudson, M. (1985). Learning disabilities and attentional problems in boys with the fragile X syndrome. *American Journal of Diseases of Children,* **139**, 674-678.

Harcherik, D. F., Cohen, D. J., Ort, S., Paul, R., Shaywitz, B. A., Volkmar, F. R., Rothman, S. L. G. & Leckman, J. F. (1985). Computed tomographic brain scanning in four neuropsychiatric disorders of childhood. *American Journal of Psychiatry,* **142**, 731–734.

Johnston, R. B., Stark, R. E., Mellits, E. D. & Tallal, P. (1981). Neurological status of language-impaired and normal children. *Annals of Neurology,* **10**, 159–163.

Kemper, T. L. (1984). Asymmetrical lesions in dyslexia. In N. Geschwind & A. M. Galaburda (Eds), *Cerebral dominance: the biological foundations* (pp. 75–89). Cambridge, Massachusetts: Harvard University Press.

Kidd, K. K. (1983). Recent progress on the genetics of stuttering. In C. L. Ludlow & J. A. Cooper (Eds), *Genetic aspects of speech and language disorders* (pp. 197–213). New York: Academic Press.

Landau, W. & Kleffner, F. (1957). Syndrome of acquired aphasia with convulsive disorder in children. *Neurology,* **7**, 523–530.

Largo, R. H. & Schinzel, A. (1985). Developmental and behavioural disturbances in 13 boys with fragile X syndrome. *European Journal of Pediatrics,* **143**, 269–275.

Leonard, L. B. (1979). Language impairment in children. *Merrill-Palmer Quarterly,* **25**, 205–232.

Leonard, L. B. (1982). Phonological deficits in children with developmental language impairment. *Brain and Language,* **16**, 73–86.

Levene, M. I., Whitelaw, A., Dubowitz, V., Bydder, G. M., Steiner, R. E., Randell, C. P. & Young, I. R. (1982). Nuclear magnetic resonance imaging of the brain in children. *British Medical Journal,* 285, 774–776.

Lou, H. C., Henriksen, L. & Bruhn, P. (1984). Focal cerebral hypoperfusion in children with dysphasia and/or attention deficit disorder. *Archives of Neurology,* **41**, 825–829.

Luchsinger, R. (1970). Inheritance of speech defects. *Folia Phoniatrica,* **22**, 216–230.

Ludlow, C. L. & Cooper, J. A. (1983). *Genetic aspects of speech and language disorders.* New York: Academic Press.

Maccario, M., Hefferen, S. J., Keblusek, S. J. & Lipinski, K. A. (1982). Developmental dysphasia and electroencephalographic abnormalities. *Developmental Medicine and Child Neurology,* **24**, 141–155.

Mutton, D. E. & Lea, J. (1980). Chromosome studies of children with specific speech and language delay. *Developmental Medicine and Child Neurology,* **22**, 588–594.

Netley, C. (1983). Sex chromosome abnormalities and the development of verbal and nonverbal abilities. In C. L. Ludlow & J. A. Cooper (Eds), *Genetic aspects of speech and language disorders* (pp. 179–195). New York: Academic Press.

Ounsted, M., Moar, V. A., Cockburn, J. & Redman, C. W. G. (1984). Factors associated with the intellectual ability of children born to women with high risk pregnancies. *British Medical Journal,* **288,** 1038–1041.

Paradise, J. L. (1981). Otitis media during early life: how hazardous to development? A critical review of the evidence. *Pediatrics,* **68,** 869–873.

Pasamanick, C. E., Constantinou, F. & Lilienfeld, A. M. (1956). Pregnancy experience and the development of childhood speech disorders: an epidemiologic study of the association with maternal and fetal factors. *American Journal of Diseases of Children,* **91,** 113–118.

Paul, R., Cohen, D. J., Breg, W. R., Watson, M. & Herman, S. (1984). Fragile X syndrome: its relations to speech and language disorders. *Journal of Speech and Hearing Disorders,* **49,** 328–332.

Pennington, B. F. & Smith, S. D. (1983). Genetic influences on learning disabilities and speech and language disorders. *Child Development,* **54,** 369–387.

Rapin, I. (1979). Conductive hearing loss: effects on children's language and scholastic skills. *Annals of Otology, Rhinology and Laryngology,* **88,** supplement 60, pp. 3–12.

Rapin, I. & Allen, D. (1983). Developmental language disorders: nosologic considerations. In U. Kirk (Ed.), *Neuropsychology of language, reading, and spelling* (pp. 155–184). New York: Academic Press.

Rosenberger, P. B. & Hier, D. B. (1980). Cerebral asymmetry and verbal intellectual deficits. *Annals of Neurology,* **8,** 300–304.

Schiff, N. B. (1979). The influence of deviant maternal input on the development of language during the preschool years. *Journal of Speech and Hearing Research,* **22,** 581–603.

Skuse, D. (1984). Extreme deprivation in early childhood—II. Theoretical issues and a comparative review. *Journal of Child Psychology and Psychiatry,* **25,** 543–572.

Stewart, A. (1984). Severe perinatal hazards. In M. Rutter (Ed.) *Developmental neuropsychiatry* (pp. 15–31). Edinburgh: Churchill Livingstone.

Vargha-Khadem, F., O'Gorman, A. M. & Watters, G. V. (1985). Aphasia and handedness in relation to hemispheric side, age at injury, and severity of cerebral lesion during childhood. *Brain,* **108,** 677–696.

Waldo, M. C., Cohen, D. J., Caparulo, B. K., Young, J. G., Prichard, J. W. & Shaywitz, B. A. (1978). EEG profiles of neuropsychiatrically disturbed children. *Journal of the American Academy of Child Psychiatry,* **17,** 656–670.

Woods, B. T. (1980). The restricted effects of right-hemisphere lesions after age one: Wechsler test data. *Neuropsychologia,* **18,** 65–70.

Woods, B. T. & Carey, S. (1978). Language deficits after apparent clinical recovery from childhood aphasia. *Annals of Neurology,* **6,** 405–409.