

How does the brain learn language? Insights from the study of children with and without language impairment

The 1999 Ronnie Mac Keith Lecture

D V M Bishop*, Oxford Study of Children's Communication Impairments, Department of Experimental Psychology, University of Oxford, OX1 3UD, UK.

**Correspondence to author at above address.*

E-mail: dorothy.bishop@psy.ox.ac.uk

Neurobiological studies have generated new ways of thinking about development of brain structure and function. Development involves more than just growth from simple to complex structures. The initial over-abundance of neurons and synaptic connections is subsequently pruned of those that are non-functional. In addition, as behavioural and cognitive functions emerge and become automatized, the underlying brain representations are reorganized. In this paper, I shall argue that these different modes of neurodevelopmental change provide a useful metaphor for examining language acquisition. It will be argued that language acquisition can involve learning to ignore and inhibit irrelevant information, as well as forming new ways of representing complex information economically. Modular organization is not present from the outset, but develops gradually. This analysis suggests a new way of assessing specific language impairment (SLI). There has been much debate as to whether children with SLI lack specific modular components of a language processing system. I propose instead that these children persist in using inefficient ways of representing language. Finally, I consider what we know about the neurobiological basis of such a deficit. There is mounting evidence that children with SLI have subtle structural anomalies affecting the language areas of the brain, which are largely genetically determined. We should not, however, conclude that the language difficulties are immutable.

Experimental studies of animals have increased our understanding of perception, motor control, memory, and learning in the brain. When it comes to language, however, animal models are of little use, and for many years our main source of information was opportunistic, involving documentation of language deficits arising as a consequence of brain lesions. Such studies taught us a great deal about how language is processed in the adult brain, but said nothing about how language was learned. And if we studied children with focal brain injuries, we found that not only were they very rare, but they also had remarkable powers of recovery. In 1962, Basser described an extraordinary series of patients who had undergone radical neurosurgery to remove brain tissue that had been damaged very early in life. These children had infantile hemiplegia associated with intractable seizures and behavioural difficulties, and in most the surgery led to substantial improvements in both seizure control and behaviour. Removal of large areas of the left hemisphere was associated with surprisingly little impairment in language development, even when complete hemidecortication was carried out. Basser proposed that when potential language areas were damaged before language had been learned, then homologous areas in the right hemisphere could take over language functions. Subsequent neurosurgical and brain imaging studies have replicated and extended these findings (e.g. Rasmussen and Milner 1977, Ogden 1988, Müller et al. 1998). Such studies are intriguing and informative for what they can tell us about the plasticity of functional localization in the human brain, but they throw little light on the neural basis of normal language learning.

Over the past two decades, major technical development in the field of brain imaging has enabled studies of neurological correlates of language function in the intact brain.

However, the application of such methods to children has been limited by both ethical and practical considerations. The general consensus is that it is not defensible to apply methods involving radioactive isotopes to children purely for research purposes, so the use of methods such as positron emission tomography (PET) and single photon emission computerized tomography (SPECT) has been limited to clinical applications. Non-invasive methods such as functional magnetic resonance imaging (fMRI) are ethically more acceptable, although there may be concern about subjecting a child to a procedure that is noisy, confining, and uncomfortable. In addition, fMRI requires the subject to keep their head still while performing experimental tasks in the scanner, and this makes it unfeasible with many children.

If this paper were simply a review of brain imaging studies of normal language development, it would be very brief indeed. However, rather than taking that approach, I aim here to consider a range of more indirect sources of evidence. I shall begin by summarizing what we know about brain development from birth to adolescence. Developmental neurobiologists now reject a simple model of development by proliferation, and recognize that the transition from infancy to adulthood entails a great deal of reorganization, which involves radical pruning as well as growth. In addition, evidence from neuroimaging suggests that different brain areas may be implicated when a function, such as language, is being learned, rather than when it is well practised and automatized.

I shall then go on to draw parallels with work on language acquisition, where recent studies suggest that language learning also involves more than a progression from simple to more complex forms of processing. In some respects, language learning is better viewed as a progression from detailed encoding of complex material to more simple and economical forms of representation. For instance, a young child may learn many different words to describe different actions, and only later recognize the underlying commonalities between different verbs that allow one to abstract general grammatical rules that apply to all verbs. Once underlying patterns and regularities are appreciated, the knowledge base can be organized and used more systematically and efficiently, with properties of new elements being deduced rather than painstakingly learned. Furthermore, the types of cognitive processing that are involved in language learning may be different from those which occur once language is fully learned.

In the last section of the paper, I shall turn to consider the case of inadequate language development. We know that focal damage to the language areas of the brain typically leads to functional reorganization, so that language is represented elsewhere. However, such plasticity does not apply universally, and some children have immense difficulty learning language for no obvious reason. Recent research suggests that such difficulties are the consequence, not of acquired neurological damage, but of abnormal neurological development early in prenatal life, leading to a brain that is not optimally organized for language learning. Genes appear to be strongly implicated in this process.

Studies of brain development

ADDITION, SUBTRACTION, AND REORGANIZATION

For many years, neurological development was viewed as a simple process of gradual growth from a simple to a complex

state. However, we now know that the processes involved are far more complex. Developments that occur from conception to childhood may be subdivided into those that are additive, subtractive, or involve reorganization. Additive processes involve proliferation of neurons, development of synaptic connectivity, and myelination. The major subtractive processes are programmed cell death, which is seen prenatally, and synaptic pruning, most of which occurs postnatally. Massive overproduction of synapses in the first 2 years of life is mirrored by an increase in metabolic activity in the brain, which may reach 150% of adult levels in early childhood, before gradually declining (see Chugani and Chugani 1997 for a review). If we were to compare additive processes to the growth of a bush, then subtractive processes resemble a form of topiary, carving a new shape by discarding non-functional connections. However, brain development is more than overproduction followed by cutting back. It is also involves substantial functional reorganization. There are three important ways in which the brain of the young child differs from that of the adult in terms of how focal representations are, how far inhibitory processes are involved, and how automatized brain-mediated behaviours are.

The first point of difference is that functions that are represented focally in the adult brain may be carried out by more diffuse brain regions in the child. Fodor (1983) noted that a brain that operated in a modular fashion, with specific regions subserving dedicated functions, is highly efficient. However, contrary to Fodor's suggestions, modularity in most systems seems to be an emergent property of the developing system, rather than something that is innately prespecified. For instance, stimulation of a region of motor cortex that produces single finger movements in an adult may lead to whole hand or arm movements in the young child. As regards language, converging evidence from several sources suggests that early word learning may involve substantial activation of the right hemisphere, with this role gradually diminishing as more language is learned. Of particular interest in this regard is a recent study by Chiron et al. (1997) on subjects aged from 18 days to 19 years who were imaged because they suffered from transient abnormal symptoms. This study showed a developmental shift from the right to the left hemisphere in the amount of metabolic activity in the plurimodal parieto-temporal area. We cannot yet be certain of the functional consequences of this shift, but it could, as the authors suggest, reflect major involvement of the right hemisphere in language processing in very young children, as was originally proposed by Lenneberg (1967). Lenneberg used data from acquired aphasia in childhood to support his case, arguing that there was an unusually high rate of aphasia after right hemisphere lesions in children compared with adults. However, others argued that the data were flawed by the inclusion of patients who might have had bilateral lesions (Woods and Teuber 1978). A recent study of early language development after unilateral focal brain injury lends further support to Lenneberg's position: at around 13 months of age, children with right-sided lesions had slower receptive language development and poorer gestural communication than those with left-sided lesions (Bates et al. 1997). However, these deficits appear transient and do not occur in older children who sustained early brain damage. Converging evidence of right hemisphere involvement in early language learning comes from an electrophysiological

study by Mills et al. (1997), who measured evoked brain responses (ERPs) as normally developing children listened to familiar words, unfamiliar words, or familiar words played backwards. Each type of stimulus gave a different pattern of ERPs. However, whereas at 13 to 17 months of age, ERP differences between comprehended and unknown words were broadly distributed bilaterally over anterior and posterior regions, by 20 months of age they were restricted to more focal regions of the left hemisphere. These authors argued that their results reflect a reorganization of neural systems mediating language, which become lateralized and more focal as the child develops.

A second way in which the child and adult brains differ is in terms of inhibitory processes. For instance, Müller et al. (1998) found differences between children and adults using PET to measure contralateral brain activation during sequential finger–thumb tapping. Both age groups showed similar levels of activation of the primary and secondary motor cortex on this task, but only the adults showed significant reduction of activity in non-motor temporoparietal cortical areas. Little is understood about how such inhibitory processes are mediated, but their emergence may be related to development of the prefrontal cortex, which has rich connections to other brain regions, and matures relatively late in development (Huttenlocher and Dabholkar 1997).

A third difference between adults and children is in terms of automatization of brain-mediated skills. In cognitive psychology, a distinction is drawn between controlled and automatic processing (Shiffrin and Schneider 1977). When a new skill is first being learned, it consumes attentional resources, and is hard and effortful: this is controlled processing. After sustained practice, however, the skill becomes automatic, and can be carried out while simultaneously performing other tasks: this is automatization. There has been considerable interest in the neurological basis of automatization, with the cerebellum postulated as playing a key role (e.g. Thach 1996). In line with this, Müller et al. (1998) showed substantial activation of ipsilateral cerebellum during finger–thumb tapping in adults, but not in children. Interpretation of this finding must be cautious, given that the participants in this study were all undergoing neurological investigation, often for seizure disorder. Furthermore, the significance of cerebellar activation is controversial, with some researchers proposing decreasing involvement of the cerebellum as skills are learned, and others the opposite. Nevertheless, this imaging study raises the possibility that cerebellar activation may be a useful indicator for studying child–adult differences in automatization of skills.

We can summarize these differences by saying that compared with the brain of the child, representation of function in the adult brain is likely to be more focal, to make greater use of inhibitory processes, and to implicate non-cortical regions concerned with the automatization of skills.

What drives brain development?

Traditionally, the process of brain development has been likened to development of other bodily organs: a maturational process under genetic control. And genes are certainly important. There has been an explosion of knowledge about how specific genes influence brain development, both from animal studies, and from molecular genetic analyses of individuals with rare genetic disorders (see Nowakowski 1987

for examples). However, we now know that the developing brain is uniquely sensitive to environmental inputs. There is evidence that all types of neurodevelopmental change, additive, subtractive, and reorganization, can be influenced by environmental stimulation.

ENVIRONMENTAL INFLUENCES ON ADDITIVE PROCESSES

Greenough et al. (1987) noted that the eminent psychologist, Donald Hebb, first aroused interest in how environmental manipulations might affect brain development, when he compared his home-reared rats with laboratory-reared rats on maze-learning tasks, and demonstrated that the former group was superior. This led to an interest in environmental manipulations that might influence brain development. Greenough et al. (1987) reviewed the literature on this topic and concluded that living conditions can make a substantial difference to brain weight and thickness in the rat, with larger neuronal cell bodies, more glial cells, and greater synaptic connectivity in those from more stimulating environments. Purves (1994) marshalled evidence from the sensory cortex for a direct effect of brain metabolic and electrical activity on growth of the neuropil – the so-called ‘glow and grow’ theory. However, to assume that environmental stimulation invariably affects brain growth is a mistake: Goldman-Rakic et al. (1997), for instance, reviewed studies in which environmental stimulation failed to produce the anticipated acceleration in synaptic growth. We do not yet know why stimulation is sometimes effective and sometimes not, but it may depend on the nature and intensity of the stimulation, and the point in development at which it is applied.

ENVIRONMENTAL INFLUENCES ON SUBTRACTIVE PROCESSES

One of the major advances in our understanding of neurodevelopment over the past two decades has been the realization that processes of synaptic pruning are dependent on environmental input. Most of this work has been concerned with the visual system in experimental animals (see Daw 1995 for a review), though this general principle has been extended to humans to account for phenomena such as persisting binocular deficit arising from distorted visual input in amblyopia. Vicente and Kennedy (1997) suggested that activity-dependent refinement of connections may be summed up by the phrase ‘cells that fire together wire together’ – whereas connections between cells that are not synchronously activated will wither away.

ENVIRONMENTAL INFLUENCES ON FUNCTIONAL SPECIALIZATION

In humans, studies of literacy show how experience can affect development of focal brain representations. We know from studies of patients with acquired dyslexias that focal regions of the brain are implicated in reading. Literacy is a recent human development and it is inconceivable that specialized innate mechanisms have evolved to mediate this skill. Rather, they develop as reading becomes a learned and automatized process. A recent study by Castro-Caldas et al. (1998) provides a particularly striking demonstration of this point. They recruited illiterate and literate adults from a Portuguese community where literacy was not universal. The illiterate group did not have any constitutional lack of reading aptitude; they simply had not received formal education in reading. In other respects they were closely matched to the literate participants. Castro-Caldas et al. found that literacy

affected performance on a task that involved repeating strings of unfamiliar speech sounds (nonsense words). The illiterate adults performed much worse on this task, even though it did not involve any written language. Furthermore, when fMRI was used to compare brain activation during nonsense word repetition, a distinctive area of activation could be seen in the left parieto-temporal region in the literate group, but not in the illiterate group. The authors concluded that the way in which the brain processes spoken language is affected by environmental experiences.

Language development: addition, subtraction, and reorganization

In some respects, parallels can be drawn between the principles underlying neurological development and those involved in language learning. If consideration is restricted to what children say, then learning appears additive, with a straightforward picture of increasing complexity. At the phonological level, children start with a relatively small repertoire of speech sounds, which gradually differentiate. Thus, the young child might collapse the sounds /t/ and /k/, and /d/ and /g/, so that 'cat' becomes 'tat', and 'goat' becomes 'doat'. By the age of around 3 years, however, most children have learned to produce these contrasting sounds distinctively. Vocabulary increases and differentiates: initially, a word such as 'bird' may be used to refer to aeroplanes, birds, and balloons, but gradually each concept acquires its own name, and over time, the child will learn specific terms for penguins, crows, and parrots. In terms of syntax, growth can be represented in a similar way, with simpler units being combined into larger structures as the child gains in competence: what Tomasello (1998) has likened to a 'Lego® construction' view of language learning. In all these respects, language learning does resemble an additive process, with a transition from simple to more complex.

Recently, however, several aspects of language learning have been described that resemble processes of subtraction and reorganization rather than addition. As children grow older, irrelevant information is discarded, and critical information is encoded more economically. We need to be cautious in using brain development as a metaphor for language acquisition: it would be over-simplistic to assume any one-to-one correspondence in structure between neurons and synapses and the mental representations that they encode. Nevertheless, the brain metaphor can be helpful in suggesting new ways of thinking about developing processes in the cognitive domain. This point will be illustrated with examples from phonological discrimination, lexical representations, and syntactic development.

PHONOLOGICAL DISCRIMINATION: INFANCY TO CHILDHOOD

Psychologists have devised an ingenious range of methods for inferring how preverbal children hear sounds. In one such procedure, an infant's rate of sucking is recorded, while repeated sounds are played through a loud speaker. When a novel sound is first encountered, the infant's rate of sucking typically increases, and then gradually habituates and decreases, as the sound becomes familiar. Suppose we play the child a repeated sound, but then slightly alter it. If the sucking rate increases, we can infer the infant has detected the change. If it remains the same, the altered sound is being treated as equivalent to the original sound. This method has

been used to assess the child's ability to discriminate between different speech sounds, and in particular to assess how far discrimination depends on exposure to a specific language. Different languages differ not just in their grammar and vocabulary, but also in the sounds that are used to signal meaning. In French, for instance, 'rue' and 'roux' are perceived as different words, because the contrast between the 'oo' sound is critical in French and changes the meaning. The 'rue' vowel, which is written phonetically as /y/, is produced in the front of the mouth (say 'eee' with lips rounded to achieve this sound). The 'roux' sound, written phonetically as /u/, is produced further back, like English 'oo'. For a French speaker, there is as much perceptual difference between 'rue' and 'roux' as there would be for an English speaker between 'rue' and 'roe'. However, the contrast between /y/ and /u/ is not contrastive in English. We would treat 'rue' and 'roux' as homophones. This is just one example: there are numerous differences between speech sounds that can potentially be used contrastively to signal meaning, and each language uses only a portion of these.

This raises the question of how children come to learn which sounds are important in their own language. Studies on babies' sound discrimination have shown that newborn infants do differentiate between many sound contrasts, including those that are not meaningful in their own language. However, during the first 9 months of life, this ability disappears, and they become sensitive only to those contrasts that are meaningful in the native tongue (see Jusczyk 1997 for a review). This is an important illustration of how learning to be an efficient language user involves learning what to ignore as well as what to attend to. Just as cortical information processing may be more efficient by eliminating non-functional synapses (Changeux and Danchin 1976), speech perception is optimized by eliminating attention to aspects of sounds that do not affect meaning (see also Guenther and Gjaja 1996). If we study how children respond to speech differences as well as how they produce speech, it seems that pruning as well as growth is involved.

LEXICAL REPRESENTATIONS: FROM SYLLABIC CHUNKING TO PHONOLOGICAL AWARENESS

Vocabulary growth in children is not linear. There is a prolonged initial period when the child learns new words at a fairly slow rate, followed by a 'vocabulary spurt' when learning is greatly accelerated. This spurt could be explained by the way words are encoded. At the earliest stages of language learning, children seem to learn whole words, or in some cases, whole phrases, as a kind of 'gestalt'. That is to say, they imitate the sound pattern that they hear, but do relatively little analysis of the internal structure of the word. Young children may not, for instance realize that the words 'bee', 'boo', and 'baa' all begin with the same sound. They may have difficulty in distinguishing 'lots' and 'lost'. There will be awareness that both words contain similar sound features, such as the sibilant noise made by 's', but they will not break words down at the level of individual speech sounds, or phonemes. And this is perhaps less surprising than we might think, because the acoustic signal corresponding to a given phoneme can be very different depending on which sounds precede and follow it (Liberman et al. 1967).

Jusczyk (1994) suggested that children become more aware of subsyllabic units in speech when their vocabulary

reaches such a size as to require some efficient means of storage and retrieval. The problem is similar to that of organizing a dictionary. Once the lexicon extends beyond a handful of words, it becomes difficult to find the word you want unless they are organized by some principled means. In an English dictionary, this is done by alphabetic order. In our 'mental lexicon' of spoken words, it is suggested that organization is by phonological characteristics, so that words that begin with the same sounds are stored together. Jusczyk's suggestion was that the young child has a more haphazard method of organizing material, but develops a new approach to storing new words once some awareness of subsyllabic phonological units develops. Even so, there is no rapid jump from working with whole syllables to working with phonemes. Most preschool children around the age of 4 years have some difficulties in analysing words at the level of the phoneme. They might be able to tell that 'cat' and 'bat' rhyme, but still not appreciate that the middle vowel is the same as in 'ham'. They might also have problems breaking up consonant clusters such as 'kl' or 'pr' into individual sounds, and so would have difficulty in appreciating that there are more sounds in 'clown' than in 'down'. It has been suggested that full phonological awareness, i.e. the ability to analyse syllables into component consonants and vowels, may develop only when the child learns to read, because the alphabet makes the segmental structure of syllables very transparent. In languages that do not have an alphabetic writing system, or in illiterate people, this level of phonological awareness may develop later, or not at all (Morais et al. 1987, Suk Han Ho and Bryant 1997).

This is another example of an aspect of language learning that is not readily described by simple growth. Rather, what we see is an initial state in which the child appears to learn in large chunks (i.e. syllables), with gradual development of a more efficient coding strategy in which words are encoded in terms of a few familiar building blocks (phonemes). Such a strategy makes learning new words much easier, because they can be decomposed and recognized as consisting of familiar elements. Bishop (1997a) illustrated this using the example of the Chinese character shown in Figure 1. If you study this for a few seconds, and then remove the picture and attempt to draw it, you are likely to have extreme difficulty, unless you happen to be familiar with the Chinese script. If you are fluent in Chinese, you are still unlikely to have previously encountered this particular character, because it is rare and archaic. However, you will be able to benefit from the fact that all Chinese characters are made from a basic set of brushstrokes, and your familiarity with these will make it much easier for you to encode and reproduce this novel stimulus. My contention is that the child who attempts to imitate a novel word is confronted with a similar problem. If the novel word can be decomposed into a set of familiar sounds, it is much easier for the child both to repeat and remember it. If not, then the new word is perceived as containing an overwhelming amount of detail that cannot be systematically related to words already in the vocabulary.

SYNTAX: FROM VERB-SPECIFIC CONSTRUCTIONS TO SYNTACTIC CATEGORIES

One of the most enduring problems in child language acquisition is how children learn syntax. Linguists working in the tradition of generative grammar have as their goal the specification of the abstract rules that are, usually unconsciously,

learned by native speakers of a language. We know that it is grammatically acceptable to say 'didn't the man you saw yesterday have a hat?' but not 'didn't the man you saw have yesterday a hat?'. We know that it is Barry who is being tickled in the sentence 'John said Barry was tickling himself', but it is John in the sentence 'John said Barry was tickling him'. Most competent speakers of English would be at a loss to specify the rules they adopt in making such judgements, but implicitly they must have them. Chomsky's famous sentence 'Colourless green ideas sleep furiously' was devised to illustrate that grammatical knowledge is quite separate from meaning: people will readily accept that this sentence is 'well-formed' (in contrast to a sequence such as 'sleep green furiously colourless ideas'), even though it makes no sense (Chomsky 1957, p 15). On the basis of such evidence, he concluded that humans have implicit knowledge of abstract grammatical rules, rather than just learning by imitating the language they hear. Because young children master grammar rapidly and efficiently, without direct instruction and with minimal correction, Chomsky concluded that they have some innate knowledge of grammatical principles. This knowledge was assumed to be of a very general kind, to accommodate the fact that children will learn whichever language they are exposed to (see, for example, Chomsky 1972). Rather than the child working out abstract grammatical rules from language input, language development was seen as a process of selecting, from a range of predetermined possibilities, the rules that applied to the ambient language.

Recently, however, this view of language acquisition has come under attack. It has been argued that there is no evidence that young children have any knowledge of abstract grammatical categories and rules when they first start constructing sentences. Tomasello (2000) argued that at 2 or 3 years of age, children simply learn whole phrases and sentence frames by imitation. The child who says 'daddy hit ball'



Figure 1: Study this Chinese character for a few seconds, and then try to draw it. A person unfamiliar with the Chinese script finds this difficult because they must remember all the visual detail, rather than encoding the visual pattern in terms of a small set of familiar brushstrokes.

has not performed any grammatical analysis of the sentence as a subject–verb–object construction, but merely understands the verb ‘hit’ and has learned a sentence frame X hit Y, where X is the one doing hitting, and Y is the thing that is hit. Tomasello provides two sources of evidence for this proposition. Firstly, naturalistic data show that children’s early sentence constructions are highly verb-specific. The child who says ‘daddy hit ball’ is likely also to say ‘daddy hit teddy’ or ‘me hit ball’, but may not use this kind of construction with other verbs that have been learned, such as ‘cut’ or ‘draw’. Secondly, when children under 3 years of age are taught novel made-up verbs, such as ‘flooze’ by an adult who says ‘Look! floozing’ or ‘the horse is floozing the cow’, they will be able to produce novel utterances incorporating the new verb when encouraged to do so, but they typically restrict themselves to the sentence frames that the adult used. As children grow older, they become aware of abstract similarities between sentence frames based on different verbs, but this is something that develops only after a critical amount of language has been learned.

Clear parallels occur between Tomasello’s account of syntax acquisition, and the account of lexical learning given above. In both domains, the hypothesis is that the child starts learning whole linguistic chunks – of phonological information in the case of lexical learning, and of word strings in the case of syntax. Initially, these are not fully analysed at an abstract level, and the child may be unaware that different words or sentence frames have a similar underlying structure. However, once a critical mass of information has been acquired, patterns can be detected. In the case of the lexicon, analysis of phonological structure of words leads to awareness of common subsyllabic units. In the case of syntax, the child develops implicit awareness that different verbs may be associated with the same type of sentence frames. In both cases, once a more abstract level of representation is achieved, this will lead to more efficient storage and retrieval of linguistic information, and more rapid learning of novel forms. Children can learn to use things like verb endings in one of two ways: either by rote-learning each inflected word (e.g. played, painted, walked) individually, or doing an analysis that allows them to abstract the rule that you form past tense by adding ‘ed’. The evidence is that they start out rote-learning and then learn the more abstract rules. However, there is not a dramatic switch from one form of learning to the other (as some linguistic theories would have us believe). Awareness of abstract rules develops gradually, and proceeds alongside rote learning. Indeed, according to Tomasello (2000), the learning processes adopted by the very young child are not replaced by more abstract rule-governed language acquisition. They remain an essential component of learning in a language that contains many constructions that are highly lexically specific, and are not readily generated by any known grammatical rules (e.g. the ‘let alone’ construction, as in ‘I wouldn’t live in America, let alone New York’, ‘John didn’t like going on holiday, let alone flying in a plane’).

What drives language development?

In terms of brain development, many people see maturation as a biological phenomenon, and the role of environmental input tends to be underestimated. At the psychological level of description, however, quite the opposite can occur. Many people assume that the principal factor driving learning is experi-

ence, and the role of biology is ignored. There is no doubt a great deal of representational change *is* dependent on experience. The studies on perceptual development in infants are a clear example: the language that a child is exposed to will affect how the child perceives sound contrasts. The study by Castro-Caldas et al. (1998), described above, also illustrates the case well. The ability to analyse and manipulate language at the level of phonemes is not something that just develops according to some biological timetable; rather it depends on exposure to an alphabetic writing system. However, when we turn to consider individual differences in language learning, it becomes clear that biology as well as environment affects how language is learned.

When language learning goes awry: the case of specific language impairment

Delayed language development is frequent reason for a parent of a preschool child to consult a paediatrician. Although causal factors such as hearing loss or structural abnormalities of the articulators can be found in some cases, all too often the reason for delay is unknown, especially when the child is developing normally in all other respects. Sometimes the child is just a ‘late bloomer’, who catches up with the peer group, but often the problem persists, leaving the child at a disadvantage when formal education begins. In the past, this kind of difficulty was termed ‘developmental dysphasia’, but now it is more common to speak of specific language impairment (SLI) or developmental language disorder.

There is a large, and contentious, body of research concerned with documenting what it is about language learning that poses such problems for some children. Although SLI is notoriously heterogeneous, in a large proportion of cases the most obvious difficulties are with the formal rule-like aspects of language, i.e. phonology and syntax. Because good models of either language development or underlying neurological development have been lacking, adult-centred approaches in the analysis of developmental disorders have usually been adopted (Bishop 1997b). Thus, the focus has been on identifying a specific stage of language processing that is disrupted, or to search for brain abnormalities in regions where damage is known to lead to aphasia in adults. The overview given above, however, suggests a different way of looking at SLI.

A common approach to analysis of expressive phonological problems in SLI has been to examine individual speech sounds, to see whether difficulties in either producing or perceiving particular phoneme contrasts might be implicated. However, I and my colleagues (Bird et al. 1995) have argued that the problem may not be to do with telling sounds apart, as in seeing that acoustically different sounds are exemplars of the same phoneme. The suggestion is that some children may get stuck at the stage of analysing speech at the level of the syllable, and remain unaware of the subsyllabic units of which it is composed (Bishop 1997a). Just like the non-Chinese person attempting to learn words written in Chinese script, the child will adopt a cumbersome and uneconomic mode of analysis that fails to capitalize on prior knowledge when learning new words, and which makes storage and retrieval of learned words effortful.

There are interesting parallels between this account of phonological impairments and conclusions from studies of syntactic difficulties in SLI, which have also emphasized the

extent to which these children may learn whole words or phrases without awareness that they are built from common elements. Thus, the child may know that the word 'dogs' refers to more than one canine animal, and 'cats' to more than one feline animal, without appreciating that there is a general rule of forming plurals by adding 's' (Gopnik 1990). Clearly, this would make the task of language learning much more difficult. Others have proposed a somewhat different account, arguing that children with SLI do have some implicit awareness of such rules, but have limited processing capacity (Leonard 1998). This means that, instead of being applied automatically and effortlessly, the rule is fragile, and will only be applied when the language system is not heavily burdened with other demands. The suggestion is that the knowledge that is automatic in other children remains effortful to apply in those with SLI.

What is interesting about these accounts of phonology and syntax is that they suggest that the problem is not that bits of the phonology or syntax of the language are missing. Rather, the child becomes stuck in immature modes of processing language, which hamper new learning. Because the same surface behaviour, e.g. repeating and memorizing a new word, or adding a grammatical ending to a word, may be achieved by different means in different children, we need to focus less on what words or sentences children say or understand, and more on the underlying representations they use to encode language. The less efficient processing methods used by children with SLI mean that, even if they appear to have mastered a formal aspect of language, their knowledge may be fragile. This explains why adults who appear to have recovered from SLI can still perform poorly when taxed with specific tests that contain high information load and require them to deal with novel material (Tomblin et al. 1992, Bishop et al. 1996).

EVIDENCE FOR PRENATAL INFLUENCES ON BRAIN DEVELOPMENT IN SLI

At the biological level, SLI poses something of a puzzle, because it contravenes what we know about the robustness and plasticity of language learning. In reviewing studies of environmental influences on language development, Bishop (1997a) concluded that quite large variations in language input have relatively small effects on rate of language learning, or eventual language competence. Children with SLI do not typically have evidence of neurological damage or disease. And, as we have already noted, focal brain damage, even when it impinges on the classical language areas, seldom results in obvious language impairment, unless the damage is bilateral. Thus is it difficult to find any environmental factors that could be responsible for SLI, but nor is there an obvious acquired neurological basis. Increasingly, the evidence is pointing to genetic influences, which would be likely to affect the earliest stages of brain development (Fisher et al. 1998, Stromswold 1998). But this discovery simply raises new questions: how could such genetic influences operate?

In recent years, there have been several brain imaging studies of individuals with SLI. These have provided mounting evidence that SLI is associated with atypical neurological development in two brain regions that are known to be crucial for language functioning, namely Wernicke's area and Broca's area. Several studies of the posterior language areas

reported that children with SLI show reduced or reversed morphological asymmetry of the brain. For instance, Gauger et al. (1997) used magnetic resonance imaging (MRI) to measure the size of the planum temporale in 11 children with SLI and 19 control children, all aged around 9 years. The planum temporale is situated in the lower bank of the Sylvian fissure, and together with the lateral and inferior parts of the superior temporal gyrus, constitutes Wernicke's area, a region critical for language comprehension. The children with SLI were less likely than the control children to have leftward asymmetry of the planum temporale. Although different studies have used different criteria for defining the brain regions of interest, a finding of reduced or reversed morphological brain asymmetry of language areas in the temporal lobe in children with SLI agrees with earlier reports (Cohen et al. 1989, Jernigan et al. 1991, Plante et al. 1991). Nor does the finding seem restricted to clinical populations. Leonard et al. (1996) studied 40 normally developing children using MRI, and found that, among those aged 5 to 9 years, anatomical asymmetry of the horizontal bank of the planum temporale was significantly correlated with success on a test of phonological awareness. For young children, good phonological awareness was associated with greater leftward asymmetry. At older ages (10 to 12 years), this relationship was not preserved, with most children scoring at ceiling on the phonological awareness measure, regardless of degree of cerebral asymmetry.

Recent studies have extended the assessment of brain morphology in SLI to encompass frontal brain regions important for language function. The pars triangularis is located in the inferior frontal gyrus and is a core part of Broca's area. Lesions of this region in the left hemisphere of adults lead to dysfluent aphasia affecting grammar and phonology. Gauger et al. (1997) found that the pars triangularis was smaller in children with SLI compared with control children on the left side only. Furthermore, the SLI group was more likely than control children to have rightward asymmetry for this structure. The demonstration of differences also in the frontal language areas is compatible with a recent report by Clarke and Plante (1998), who imaged the brains of parents of children with SLI and of a control group of adults. Both groups of adults were assessed on a language battery and, as might be expected given the familial nature of SLI, a high proportion showed residual language deficits. Interestingly, parental language level, rather than relatedness to a child with SLI, was correlated with brain morphology. An unusually large number of those with evidence of language impairment had additional sulci in Broca's area.

Studies showing minor structural variations associated with SLI raise questions about the underlying mechanisms leading to atypical neurodevelopment. One theory, popularized by Galaburda et al. (1985), was developed to account for developmental reading difficulties, but is often extended to developmental language disorders more broadly defined. This maintains that such learning difficulties arise as the consequence of abnormalities in prenatal processes of neuronal migration that lead to distortions of the structured columnar organization of cerebral cortex. This account meshes well with evidence of genetic influences on SLI, as we know from animal models that neuronal migration can be influenced by genes (Nowakowski 1987). According to this view, then, genes directly affect prenatal development, so that the brain

of the child with SLI is not optimally organized for language learning from the outset. This account could provide a novel answer to the question of why the usual processes of recovery of function do not operate in children with SLI (cf. Bates 1997). When a brain is damaged, neurons that normally form connections in the damaged area will be unable to locate their targets. It seems likely that the stimulus leads them to seek other sites with which to connect. However, if there is no damage, but rather an unusual arrangement of neurons, or a smaller number of neurons in language-relevant areas, then it may be that connections are made, but in a non-optimal fashion, perhaps with less redundancy that would normally be found.

There is, however, a much more radical explanation for the atypical structure and function of the brains of children with SLI. This maintains that the neurological differences arise postnatally and might be as much consequence as cause of differences in linguistic ability. One such account was proposed by Locke (1994), who suggested that the primary problem in SLI is a genetically determined impairment of social cognition. This meant that the young child is poorly tuned in to communication, and consequently fails to make much progress in learning vocabulary. The net result is that the child fails to achieve the 'critical mass' of vocabulary that is needed to activate left hemisphere brain mechanisms that are concerned with analysing phonological and grammatical structure. Thus, the child with SLI persists in using inefficient right-hemisphere mechanisms for language learning. This is a highly speculative account, which, in effect, characterizes the child with SLI as failing to achieve focalization and reorganization of language, both at the neurological and behavioural level. It offers the intriguing possibility that the grammatical deficits in SLI are not caused by any fundamental deficit in grammar-learning mechanisms, but by a much earlier problem in social communication, which has the consequence that grammatical analysis mechanisms never get properly activated or established.

A related kind of explanation was proposed by Leonard et al. (1996) to explain findings linking phonological awareness with brain lateralization in their normally developing sample of children. They noted that although their study could be interpreted as indicating that children born with marked brain asymmetries have a special talent for phonemic awareness, it is also possible that children who work hard at conquering phonemic awareness develop asymmetry faster than other children do. This would not necessarily rule out a role for genes, but it suggests they exert a more indirect effect on cortical development, by predisposing the child to attend certain aspects of speech signals. This predisposition may lead to increased exposure to certain kinds of stimuli, which affect the development of neural maps for encoding that function. Thus, synchronized neural activity in the left planum temporale, as the child attends to phonologically relevant stimuli, may cause shrinkage of the corresponding area on the right side of the brain. According to this view, asymmetry arises by a process of environmentally induced pruning rather than growth. It is not inconceivable that a similar account could explain the differences in gyral patterns, given that cortical gyration changes dramatically in the first 2 years of life (Martin et al. 1988). However, not all the data supports this type of account. For instance, in SLI, atypical asymmetry is sometimes associated with a reduction rather than an increase in

brain volume. Indeed, the study by Gauger et al. (1997) found the right hemisphere was significantly narrower in those with SLI than the control children. The question of whether brain differences in SLI develop over time or are present from the outset remains open, and is unlikely to be resolved without longitudinal studies.

IMPLICATIONS FOR INTERVENTION

Reports of a genetic basis for SLI tend to be greeted with interest by researchers but dejection by clinicians. Where abnormal brain structure can also be confirmed, the gloom among clinicians deepens. This is because there is often a deep-rooted belief that the effects of genes are immutable, and no amount of remediation can compensate for a biological deficit. It is important, therefore, to put these results into perspective. Data from neuroimaging, far from demonstrating a deterministic biological influence on language, strongly indicate that abnormal brain structure is not the whole explanation for SLI. According to Leonard (1997), brain imaging studies of developmental disorders were embarked on in the hope that they would reveal distinctive 'holes in the brain' for each developmental disability. Nothing could be further from the truth. Investigators were surprised at how difficult it was to find evidence of structural abnormalities in people with major cognitive or behavioural deficits. Furthermore, even when minor brain abnormalities were discovered, they had neither sensitivity nor specificity for a given disorder. For instance, lack of asymmetry of the planum temporale has been statistically associated with schizophrenia and dyslexia, as well as SLI. Furthermore, associations between unusual brain structure and abnormal function, though statistically significant, are far from perfect; many people with developmental disorders have normal brain asymmetry, and many normally functioning control people do not. Rather than assuming that brain anomaly leads deterministically to disorder, it seems more appropriate to regard atypical brain structure as a risk factor that makes it more likely that the child will develop a disorder, but the nature, severity, and persistence of disorders are likely to depend on non-biological factors.

Direct evidence of environmental influences on brain function has been provided by studies showing that extensive training of experimental animals on specific tactile discriminations can not only improve performance, but also alter the size of functional maps in sensory cortex (see Merzenich et al. 1996a for a review). Merzenich et al. (1996b) have applied the same principles to intervention for children with language-learning disabilities. This work is still at an early phase, and somewhat controversial. There is debate as to how far improvements are attributable to specific components of the intervention (i.e. is the critical training auditory or linguistic), how much improvement could be due to placebo or practice effects, and how much generalization there is to everyday communication skills. Nevertheless, the general thrust of this work is to be welcomed, stressing as it does the fact that a neuroscientific approach generates optimism rather than nihilism about possibilities for intervention. A similar point was made by Gauger et al. (1997), who suggested that the child who has a genetically-based neurodevelopmental disorder may require extra environmental stimulation to encourage developing fibres to find appropriate synaptic targets. Intervention research has demonstrated that modifications of

language input to children with SLI can influence both rate of vocabulary acquisition (Ellis Weismer 2000) and mastery of syntax (e.g. Fey et al. 1993). Therefore, intervention is not pointless; rather, we need to devise novel learning environments that go beyond the rather haphazard linguistic experiences that most children encounter. The latter kind of input might be adequate for a brain that is optimally organized for language learning, but not for the child with SLI.

To conclude, we have come a long way from the days when the model of SLI was of a child who simply had something missing – either in terms of neurology or in terms of underlying language processes. Contemporary research suggests we need to look at dynamic processes of brain development, and language learning, if we are to gain a full understanding of SLI.

Accepted for publication 15th November 1999.

References

- Basser LS. (1962) Hemiplegia of early onset and the faculty of speech with special reference to the effects of hemispherectomy. *Brain* **85**: 427–60.
- Bates E. (1997) Origins of language disorders: a comparative approach. *Developmental Neuropsychology* **13**: 447–76.
- Thal D, Turner D, Fenson J, Aram D, Eisele J, Nass R. (1997) From first words to grammar in children with focal brain injury. *Developmental Neuropsychology* **13**: 275–343.
- Bird J, Bishop DVM, Freeman N. (1995) Phonological awareness and literacy development in children with expressive phonological impairments. *Journal of Speech and Hearing Research* **38**: 446–62.
- Bishop DVM. (1997a) *Uncommon Understanding: Development and Disorders of Language Comprehension in Children*. Hove: Psychology Press.
- (1997b) Cognitive neuropsychology and developmental disorders: uncomfortable bedfellows. *Quarterly Journal of Experimental Psychology* **50A**: 899–923.
- North T, Donlan C. (1996) Nonword repetition as a behavioural marker for inherited language impairment: evidence from a twin study. *Journal of Child Psychology and Psychiatry* **37**: 391–403.
- Castro-Caldas A, Petersson KM, Reis A, Stone-Elander S, Ingvar M. (1998) The illiterate brain: learning to read and write during childhood influences the functional organization of the adult brain. *Brain* **121**: 1053–63.
- Changeux JP, Danchin A. (1976) Selective stabilization of developing synapses as a mechanism for the specification of neuronal networks. *Nature* **264**: 705–712.
- Chiron C, Jambaque I, Nabbout R, Lounes R, Syrota A, Dulac O. (1997) The right brain hemisphere is dominant in human infants. *Brain* **120**: 1057–65.
- Chomsky N. (1957) *Syntactic Structures*. The Hague: Mouton. p 15.
- (1972) *Language and Mind (enlarged edition)*. New York: Harcourt Brace Jovanovich.
- Chugani HT, Chugani DC. (1997) Positron emission tomography studies of developing brain. In: Keshavan MS, Murray RM, editors. *Neurodevelopment and Adult Psychopathology*. Cambridge: Cambridge University Press.
- Clark MM, Plante E. (1998) Morphology of the inferior frontal gyrus in developmentally language-disordered adults. *Brain and Language* **61**: 288–303.
- Cohen M, Campbell R, Yaghai F. (1989) Neuropathological abnormalities in developmental dysphasia. *Annals of Neurology* **25**: 567–70.
- Daw NW. (1995) *Visual Development*. New York: Plenum Press.
- Ellis Weismer S. (2000) Intervention for children with developmental language delay. In: Bishop DVM, Leonard LB, editors. *Speech and Language Impairments in Children: Causes, Characteristics, Intervention, and Outcome*. Hove: Psychology Press.
- Fey M, Cleave PL, Long SH, Hughes DL. (1993) Two approaches to the facilitation of grammar in children with language impairment: an experimental evaluation. *Journal of Speech and Hearing Research* **36**: 141–57.
- Fisher SE, Vargha Khadem F, Watkins KE, Monaco AP, Pembrey ME. (1998) Localisation of a gene implicated in a severe speech and language disorder. *Nature Genetics* **18**: 168–70.
- Fodor JA. (1983) *The Modularity of Mind*. Cambridge, MA: Bradford Books.
- Galaburda AM, Sherman GF, Rosen GD, Aboitiz F, Geschwind N. (1985) Developmental dyslexia: four consecutive cases with cortical anomalies. *Annals of Neurology* **18**: 222–33.
- Gauger LM, Lombardino LJ, Leonard CM. (1997) Brain morphology in children with specific language impairment. *Journal of Speech, Language and Hearing Research* **40**: 1272–84.
- Goldman-Rakic PS, Bourgeois J, Rakic P. (1997) Synaptic substrate of cognitive development: life-span analysis of synaptogenesis in the prefrontal cortex of the nonhuman primate. In: Krasnegor NA, Lyon GR, Goldman-Rakic PS, editors. *Development of the Prefrontal Cortex*. Baltimore: Paul H Brookes. p 27–47.
- Gopnik M. (1990) Feature blindness: a case study. *Language Acquisition* **1**: 139–64.
- Greenough WT, Black JE, Wallace CS. (1987) Experience and brain development. *Child Development* **58**: 539–59.
- Guenther FH, Gjaja MN. (1996) The perceptual magnet effect as an emergent property of neural map formation. *Journal of the Acoustical Society of America* **100**: 1111–21.
- Huttenlocher PR, Dabholkar AS. (1997) Developmental anatomy of prefrontal cortex. In: Krasnegor NA, Lyon GR, Goldman-Rakic PS, editors. *Development of the Prefrontal Cortex*. Baltimore: Paul H Brookes. p 69–83.
- Jernigan T, Hesselink JR, Sowell E, Tallal P. (1991) Cerebral structure on magnetic resonance imaging in language- and learning-impaired children. *Archives of Neurology* **48**: 539–45.
- Jusczyk PW. (1994) Infant speech perception and the development of the mental lexicon. In: Goodman JC, Nusbaum HC, editors. *The Development of Speech Perception: The Transition from Speech Sounds to Spoken Words*. Cambridge, MA: MIT Press. p 227–70.
- (1997) *The Discovery of Spoken Language*. Cambridge, MA: MIT Press.
- Lenneberg EH. (1967) *Biological Foundations of Language*. New York: John Wiley.
- Leonard CM. (1997) Language and the prefrontal cortex. In: Krasnegor NA, Lyon GR, Goldman-Rakic PS, editors. *Prefrontal Cortex: Evolution, Development, and Behavioral Neuroscience*. Baltimore: Brookes Publishing. p 141–66.
- Lombardino LJ, Mercado LR, Browd SR, Breier JJ, Agee OF. (1996) Cerebral asymmetry and cognitive development in children: a magnetic resonance imaging study. *Psychological Science* **7**: 89–95.
- Leonard LB. (1998) *Children with Specific Language Impairment*. Cambridge, MA: MIT Press.
- Liberman AM, Cooper FS, Shankweiler D, Studdert-Kennedy M. (1967) Perception of the speech code. *Psychological Review* **74**: 431–61.
- Locke JL. (1994) Gradual emergence of developmental language disorders. *Journal of Speech and Hearing Research* **37**: 608–16.
- Martin E, Kikinis R, Zuerrer M, Boesch C, Briner J, Kewitz G, Kaelin P. (1988) Developmental stages of human brain: an MR study. *Journal of Computer Assisted Tomography* **12**: 917–22.
- Merzenich MM, Wright B, Jenkins W, Xerri C, Byl N, Miller S, Tallal P. (1996a) Cortical plasticity underlying perceptual, motor, and cognitive skill development: implications for neurorehabilitation. *Cold Spring Harbor Symposia on Quantitative Biology* **61**: 1–8.
- Merzenich MM, Jenkins WM, Johnston P, Schreiner C, Miller SL, Tallal P. (1996b) Temporal processing deficits of language-learning impaired children ameliorated by training. *Science* **271**: 77–81.
- Mills DL, Coffey-Corina S, Neville HJ. (1997) Language comprehension and cerebral specialization from 13 to 20 months. *Developmental Neuropsychology* **13**: 397–445.
- Morais J, Alegria J, Content A. (1987) The relationships between segmental analysis and alphabetic literacy: an interactive view. *Cahiers de Psychologie* **7**: 415–38.
- Müller RA, Rothermel RD, Behen ME, Muzik O, Mangner TJ, Chakraborty PK, Chugani HT. (1998) Brain organization of language after early unilateral lesion: a PET study. *Brain and Language* **62**: 422–51.

- Nowakowski RS. (1987) Basic concepts of CNS development. *Child Development* **58**: 568–95.
- Ogden JA. (1988) Language and memory functions after long recovery periods in left-hemispherectomised subjects. *Neuropsychologia* **26**: 645–59.
- Plante E, Swisher L, Vance R, Rapcsak S. (1991) MRI findings in boys with specific language impairment. *Brain and Language* **41**: 52–66.
- Purves D. (1994) *Neural Activity and the Growth of the Brain*. Cambridge: Cambridge University Press.
- Rasmussen T, Milner B. (1977) The role of early left-brain injury in determining lateralization. *Annals of the New York Academy of Science* **299**: 255–69.
- Shiffrin R-M, Schneider W. (1977) Controlled and automatic human information processing: II. Perceptual learning, automatic attending and a general theory. *Psychological Review* **84**: 127–90.
- Stromswold K. (1998) Genetics of spoken language disorders. *Human Biology* **70**: 293–320.
- Suk Han Ho C, Bryant P. (1997) Development of phonological awareness of Chinese children in Hong Kong. *Journal of Psycholinguistic Research* **26**: 109–26.
- Thach WT. (1996) On the specific role of the cerebellum in motor learning and cognition: clues from PET activation and lesion studies in man. *Behavioral and Brain Sciences* **19**: 411–31, 503–527.
- Tomasello M. (1998) Review article and discussion: the return of constructions. *Journal of Child Language* **25**: 431–42.
- (2000) Acquiring syntax is not what you think. In: Bishop DVM, Leonard LB, editors. *Speech and Language Impairments in Children: Causes, Characteristics, Intervention, and Outcome*. Hove: Psychology Press.
- Tomblin JB, Freese PR, Records NL. (1992) Diagnosing specific language impairment in adults for the purpose of pedigree analysis. *Journal of Speech and Hearing Research* **35**: 832–43.
- Vicente AM, Kennedy JL. (1997) The genetics of neurodevelopment and schizophrenia. In: Keshavan MS, Murray RM, editors. *Neurodevelopment and Adult Psychopathology*. Cambridge: Cambridge University Press. p 31–56.
- Woods BT, Teuber HL. (1978) Changing patterns of childhood aphasia. *Annals of Neurology* **3**: 273–80.