

Chapter eight

Dyslexia

*In which the reader is invited to become sceptical,
look into hard and soft science and
consider whether dyslexia is benign or malignant.*

It was 'M', many years ago, who started the scales falling from my eyes. Although diagnosed as 'severely dyslexic' at an internationally renowned dyslexia centre his progress in the weekly adult literacy class was rapid and solid. 'M' travelled faster than most.

After a few weeks he suddenly remarked 'It's very odd!'

'What's odd?' I asked.

'You're the first teacher I've ever had who really expects progress and the odd thing is – that's exactly what we're getting!'

And then, 'You don't believe I've got dyslexia any longer, do you?'

Well yes, it did seem odd that progress was so suddenly (and so easily) attainable after such a long history of consistent and effortful failure. And no, 'M' was clearly not dyslexic, if we take dyslexia to mean an innate neurological disability specifically disabling the learning and management of literacy.

What was going on?

Over the years since 'M' left tuition (for university and a degree) I have come to believe that dyslexia is much underestimated. I believe it is a malign and depressing diagnosis of a condition which probably doesn't exist. I do not believe I am alone in this opinion, indeed I know I am not, but it is an opinion which needs to be defended, or explained, nonetheless, in a world in which dyslexia seems to be unquestioningly accepted everywhere and by everyone. This defence, or explanation, is what this chapter will be about.

The muddle of definition:

The term '*dyslexia*' is used, by most of us, very casually. We pick the word up lazily; without thought. We use it without properly defining it, or we define it in terms so broad as to be next to pointless. We apply the term even when we are perfectly well aware that we have no clear definition of it, or satisfactory explanation for it. When we discuss dyslexia it can be correspondingly difficult to see what, precisely, we are discussing, if anything. The word is commonly used to mean nothing more scientifically exact than a difficulty with written language which we do not understand, or perhaps do not care to understand. We tend to use the term to denote a problem with reading &/or writing &/or spelling (and sometimes much more besides) which appears to be inexplicable - especially where there appears to be a discrepancy with what we'd otherwise expect from a particular person. We find the discrepancy so peculiar, so personally threatening, so deeply and intimately offensive, that we are driven to believe, almost to hope, that there must be something constitutionally wrong with the victim; that the cause must be a specific neurological deficit, beyond blame, safely located among all the other medical conditions beginning with 'dys-'. Are we, as we so often do in other contexts, blaming the victim in order to pass the buck?

There is an established, and very rewarding, dyslexia industry. There is considerable academic and commercial vested interest. There seem to be as many aetiologies for (causes for or origins of) dyslexia as there are researchers into it, give or take, and as many wonderfully special assessment methods, remedial schemes, dedicated schools and distinguished gurus as the market will carry. There are breathtaking illogicalities, inconsistencies and outrageous assumptions throughout the scientific literature and beyond. The media cheerfully mangle and distort. Fantastically various definitions and explanations tumble around each other. Weird and colourful creatures appear fleetingly through the muddied waters - are they fish, fowl or beast? Mostly, they rapidly disappear again. But none of this seems to *bother* us nearly enough.

A few quotes from around the literature on dyslexia will illustrate this muddle:

The construct of learning disabilities has historically been difficult to define. (Fletcher 2003)

... the history of dyslexia is littered with theories that were once widely supported but now lie abandoned on the scrap heap ... it is vital that we should continue to treat *everything* as questionable and to regard nothing as beyond dispute. Certainty is for tele-evangelists, not scientific researchers or teachers. Ellis *et al* (1997 pp. 13-14) (their emphasis)

The concept of dyslexia has had a confused, cart-before-the-horse history... Stanovich (1991 p. 22)

Definitions of dyslexia are notoriously varied and no single definition of dyslexia has succeeded in gaining a scientific acceptance which even approaches unanimity... Each researcher or clinician becomes attached to his or her own definition in a manner which is reminiscent of Humpty Dumpty in Lewis Carroll's *Through The Looking Glass* – 'When I use a word ... it means just what I choose it to mean.' Definitions ... soon become muddled when the researcher or clinician is confronted with a variety of adult cases exhibiting highly heterogenous profiles. Beaton *et al* (1997 p.2)

and:

The diversity of theories concerning the biological underpinnings of dyslexia is impressive... It is clear there is some way to go before any consensus is reached regarding the biological basis of dyslexia ... (ibid. pp. 4 – 5)

Students had individual clusters of the cognitive weaknesses usually associated with dyslexia, alongside clear strengths in some cases.... They were also accompanied by widely varying individual configurations of literacy and other difficulties, so much so that the students themselves wondered if they were experiencing the same syndrome. The identification of dyslexia could not by itself predict the individual configurations, and the question of whether or not there was one distinctive syndrome became less important than the issue of learning to describe one's particular situation to a world largely ignorant of these matters, eg 'I am dyslexic and for me this means that I literally cannot write my own name, but I can read quite well and I am now using a word processor.' Herrington (1995 pp. 6 – 7)

..the research literature provides no support for the notion that we need a scientific concept of dyslexia separate from other, more neutral, theoretical terms such as reading disabled, poor reader, less-skilled, etc. Yes, there is such a thing as dyslexia if by dyslexia we mean poor reading. But if this is what we mean, it appears that the term dyslexia no longer does the conceptual work that we thought it did. Indeed, whatever conceptual work the term is doing appears to be misleading. Stanovich (1994 p. 588)

Over a decade ago ... there was little evidence that poor readers of high and low IQ differed importantly in the primary processing mechanisms that were the cause of their reading failure. A further decade's worth of empirical work on this issue has still failed to produce such evidence. Stanovich & Stanovich (1997 p.3)

'One of the fascinations of dyslexia for researchers is that, whatever one's interest in human behaviour and performance, children with dyslexia will obligingly show interesting abnormalities in precisely that behaviour.' Nicolson & Fawcett (1999 p. 156)

Writers often allow dyslexia a scientifically improper scope. Whatever symptoms or deficits they find, however disparate or infrequent, are claimed as indicative of dyslexia. Everything is subsumed. Try this translation of the immediately above:

‘One of the fascinations of Foot and Mouth Disease (FMD) for veterinarians is that, whatever one’s interest in bovine behaviour and performance, cattle with FMD will obligingly show interesting abnormalities in precisely that behaviour’.

If it were possible to say such a thing about FMD (it is not) one would be able to say, categorically, that FMD is either a collection of many syndromes which we have yet to distinguish from each other, or it is not a syndrome at all. No syndrome, however obliging, will show any and every symptom for which we look. We would know for certain, if this appeared to be so, that we were as yet too ignorant to say anything important about aetiology, effects or remediation. We would know that, as yet, we had no identified or understood syndrome to say such things about. We would treat our patient symptom by symptom, empirically, but be obliged to refrain from any diagnosis more precise than, say, ‘sickly cow’. To do otherwise would be hubris, not science. It would also block progress towards understanding, not advance it.

What is dyslexia?

Definitions of developmental dyslexia are many and various, as Beaton *et al* observe. Some are so broad as to be almost meaningless, some are confused and imprecise, some say next to nothing. There is no consensus. A few examples will demonstrate this. The World Federation of Neurologists’ definition from 1968 was, for a couple of decades, the definition most widely quoted and some consensus gelled around it during that time. The WHO also defined thus in 1993. It goes like this:

Dyslexia is a disorder manifested by difficulty in learning to read despite conventional instruction, adequate intelligence and sociocultural opportunity. It is dependent upon fundamental cognitive disabilities which are frequently of constitutional origin.

This may, though, be translated as:

‘Dyslexia is a difficulty with reading which may only be diagnosed if there are no other obvious causes to hand (such as poor schooling, poor parenting, low IQ or social disadvantage). It is caused by there being something wrong with the brain (well, very often, anyway).’

This uncertain definition is simply defining the syndrome as an odd difficulty with reading, given an otherwise apparently normal educational and social history. It almost amounts to a discrepancy definition (of which more later) and makes it impossible for a child from a socially deprived background to be ‘dyslexic’ at all.

The Dyslexia Institute (1989) defines ‘specific learning difficulty’ (which many use as synonymous with ‘dyslexia’) as follows:

Specific learning difficulties can be defined as organising or learning deficiencies which restrict the student’s competencies in information processing, in motor skills and working memory, so causing limitations in some or all of the skills of speech, reading, spelling, writing, essay writing, numeracy and behaviour.

This is so broad, invoking so many discrete and distinct cognitive domains, as to mean very little in fact. Are we to expect a ‘dyslexic’ to be defective in motor skills as well as information processing (whatever that means) *and* memory? Are we to believe that a ‘dyslexic’ with behavioural problems has these as a result of innate ‘organising and learning deficiencies’ (and what are these anyway and how will we recognise them?). If ‘dyslexics’ do not exhibit behavioural problems perhaps, or are not defective in, say, memory or motor skills, are they then no longer ‘dyslexic’ despite continuing poor literacy skills and difficulty in learning literacy?

The British Dyslexia Association (BDA 1989) defines thus:

Dyslexia can be defined as a specific difficulty in learning, constitutional in origin, in one or more areas of reading, spelling and written language which may be accompanied by difficulty in number work. It is particularly related to mastering and using written language (alphabetic, numerical & musical notation) although often affecting oral language

to some degree.

This is precise, in that it specifies that the syndrome is related to written symbols which represent language (or music and possibly number) and that it is definitely constitutional in origin. As we will see, this simple assertion (that there is such a specific difficulty) remains doubtful.

Moray House (1993) defines thus:

Specific learning difficulties can be identified as distinctive patterns of difficulties, relating to the processing of information, within a continuum from very mild to extremely severe, which result in restrictions in literacy development and discrepancies in performances within the curriculum. Reid (1994 p. 3)

This definition very properly, as with the one above, restricts its scope to literacy, which is more than many do. (*dys* [Greek] means difficult, abnormal, impaired and *lexikos* [also Greek] means pertaining to words. Let us not forget this.) It relates the difficulties, though, to defective information processing. This sounds satisfyingly scientific and would, perhaps, be a satisfactory explanation for a defective computer. However, in today's climate of deep ignorance as to what, exactly, our brains consider 'information' to be, what they actually use as 'information', and just exactly what they are doing when they 'process' it, the phrase 'information processing' denotes too vague and ill-understood a concept to be of much psychological value, at least as yet. We are certainly not in any position to assess it in any very meaningful way, particularly not in a neurologically meaningful way. (and see Chalmers 1996, Reilly 1985 & Smith in Olson *et al* 1985) This definition amounts to declaring that specific learning difficulties are difficulties in learning which vary from very mild to very severe, and that these difficulties are related to a deficit in a process of which we have extremely small understanding. The patterns of difficulty are allegedly 'distinctive', but in an unspecified way.

I will be discussing the use of the diagnosis as an alibi explanation, and as one which very conveniently, and comfortingly, locates the blame for literacy failure entirely within the victim's central nervous system. Occasionally even the internationally recognised expert lets this one out of the bag as in Miles (1988) quoted in Pumfrey & Reason (1991 p. 69):

For him [Miles] the term dyslexia assists parents and the child to make sense of occurrences they know to exist. They know the child has difficulty with reading and spelling; they need explanations which remove the sense of self-blame.

Pumfrey and Reason compare this with the 'it's my hormones' explanation of obesity. It is comforting, perhaps, as it absolves from responsibility, but this explanation entirely fails to understand, or make any attempt to solve, the real problem which, as we all strongly suspect, has nothing to do with hormones. An elephant in the room, in fact.

And then sometimes researchers let another out of the bag as when Cooke says (2001 p. 49):

Miles (1995) has questioned whether there can be a single definition of dyslexia; she suggests instead that different people, and different groups, will want a definition to suit their own requirements. This is clearly correct ...

This is, of course, giving up without a blush. If we are all to select our own, personal definition to suit our own particular agenda, then we may as well communicate in grunts for all the sense we will make. 'Dyslexia' has been researched for over a century now. It is astonishing, and considerably revealing, that such confusion still exists and such woolly remarks are still accepted in apparently serious, peer-reviewed, scientific journals.

The Shorter Oxford English Dictionary (1993) probably does as well as any with: 'A developmental disorder marked by an extreme difficulty in reading or in understanding written language; word blindness.' This definition recognises, incidentally, that dyslexia, very strictly speaking, relates solely to reading. The corresponding difficulty with writing or spelling should, properly, be termed 'dysgraphia'. However, we don't want to get in so deep we can't get out again so I will adopt the widespread habit of taking 'dyslexia' to refer to all literacy skills. (I will not adopt the habit of taking it to refer to any failure in any skill or characteristic as in, for example, 'emotional dyslexia' or

'economically dyslexic' (both from BBC Radio 4). Quaintly, I will assume that words have meaning and will try to use them accordingly.) When I write 'dyslexia' I will mean developmental dyslexia which I will here define as '*a difficulty in acquiring or managing literacy skills which is caused by an innate neurological deficit of some kind*'.

Frith (1999) has written a fascinating article on precisely this subject: 'Paradoxes in the definition of dyslexia.' In it she accepts that 'The first of these paradoxes concerns the lack of agreement about the very definition of dyslexia' (ibid. p. 193). She proposes an interesting map on which, she suggests, researchers may inscribe suggested causes and effects such as to build up a multi-layered overview of dyslexia and thereby inch nearer to describing and defining it. She suggests the map should include possible biological causes (brain abnormalities), possible cognitive deficits and possible behavioural problems – all of these related throughout to environmental factors. Tellingly, in an article published in December 1999, about a century since research into 'word blindness' began, she writes that 'In unknown territory this map is largely white, but it will soon be filled in by intrepid explorers.' (ibid. p. 193). It is highly instructive to read this article which makes it very clear how far we really are from either consensus or understanding.

The fact is, as you can see, that none of us appears to have very much idea what 'dyslexia' might be. Regrettably, this does not stop us from making extremely detailed and definite assertions about 'dyslexia' and taking sometimes far-reaching action on the basis of these assertions. In major popular texts on the subject one may read statements like this:

The fact that no exact definition [of dyslexia] has yet been produced is of little consequence...THERE IS ANOTHER QUITE DISTINCT GROUP who have difficulty with reading yet are very able in other ways... for convenience we refer to them as being DYSLEXIC or having DYSLEXIA. Parents, teachers and others understand these words and find them to be an easy form of verbal shorthand to describe the children with whom we are concerned. Doyle (1997 p. 82. his emphases.)

This will not, of course, do. To employ resonant neuropsychological terms for which we have no agreed definition and about which we remain thoroughly ignorant as if they have highly specific and well-understood meaning is spurious. It is improper. It is arrogant and it is fraudulent. We may not legitimately use terminology which appears, particularly to the general public, to be scientifically precise as 'an easy form of verbal shorthand' to mean almost anything almost anyone (eg. 'parents, teachers and others') wants to intuit. We may not properly diagnose subtle, but possibly disabling, neurological deficits on the basis of such imperfect knowledge, asserting that the fact that we understand them not a whit is 'of little consequence'. A diagnosis may have very real consequences; not all will necessarily be favourable. 'Diagnosis' is a grown-up word for a grown-up activity. It demands a proper understanding, but it also demands a proper humility, most particularly in the confessed absence of understanding.

Why all the fuss?

What does it matter whether there really is such a neurological deficit, so long as people who need tuition get tuition? I believe it matters very much, for several reasons. Firstly, much thinking about dyslexia is almost wilfully sloppy and sloppy science never did anyone any good, very particularly the subjects of it. Many appear willing to make the diagnosis, rather fewer are qualified so to do. (We are, after all, discussing neuropathology here!) Many diagnoses stand on small, highly controversial and rather subjectively assessed, evidence. And then, people given a diagnosis of a neurological deficit may find such a label at the least disconcerting, at worst devastating. ('That was another big shock, finding out you're disabled!' cried one student. [Whitehouse 1995 p. 21]) And then, what about those who don't achieve the label? Are they simply (and publicly) to be designated as stupid? And then, we don't appear able to see over or around dyslexia; once the diagnosis has been invoked we seek no other explanations for presenting phenomena. Simpler alternative, much more everyday, scientifically duller, less sexy (and much less lucrative) explanations are very much less assiduously sought once a diagnosis of 'dyslexia' has been made. And finally, and crucially, in the face of such a diagnosis we appear to act differently - we seem to see a need for deficit-focussed, repetitive, tightly controlled and limited practice (e.g. Lee 2002), and we seem abruptly to experience considerably depressed expectations (Kerr 2001 a & b and see notes to this chapter). Suddenly we are content with poorer results - slower and less accurate outcomes more laboriously produced. We may become more sympathetic, but we

also become less demanding. We aim lower. We teach mechanics and detail rather than purpose and flair, rules rather than writing. This is inevitable once we have attributed a student's problems to a single, conceptually simple (albeit imperfectly understood), innate and unalterable cause. This is classic soil in which to grow learned helplessness - and assuredly not only in the student.

Perhaps we should step back and think about what it is, and isn't, that we are actually discussing. Dyslexia raises hackles and passions. People, as you may already have begun to suspect, become highly partisan. Blood pressures, as you may have observed, rise. Tempers easily fray. It behoves me, therefore, to make precisely clear what I am saying and, probably even more importantly, what I am not saying. For reasons which will become clear, I believe dyslexia is very important, a deadly serious problem, albeit a virtual one.

Firstly, what do I mean by dyslexia? There are two kinds of dyslexia – acquired and developmental. (*Developmental* dyslexia is what everyone really means when they simply say 'dyslexia', a habit I shall maintain here.)

Acquired dyslexia is rare, at least in 'pure' form. It is thoroughly unpleasant, but perfectly understandable in terms of cognitive psychology and so is not scientifically controversial. Acquired dyslexia is the result of trauma to the brain occurring after literacy has been learned by it. Some accident (a blow to the head, or perhaps a stroke) results in damage to the part of the brain which had learned literacy skills. Depending on the degree of damage, the skills will be correspondingly lost. The same applies to speech, of course. Many stroke victims have their speech centres damaged and show varying degrees of loss of the power of speech. This is horrible but makes perfect cognitive sense – if you damage the part of the brain which has learned to be responsible for such and such a skill then that skill will be correspondingly damaged. How could it be otherwise?

Developmental dyslexia is an utterly different animal. Here, there is assumed to be an *innate* neurological deficit of some kind. (If there is no neurological deficit behind dyslexia it is, of course, simply a surprising difficulty with literacy, not a 'dys' at all, and back to square one we must all go.) In *developmental* dyslexia the neurological deficit is presumed to be innate, or pre-wired - to have been present since conception or at least since birth. Some claim that the deficit is written into the genes (eg Cardon *et al* 1994, Fisher *et al* 1999, Olson 2004) but others claim that the deficit is the result of damage to the foetus during gestation, for example biochemical trauma *in utero* (eg Geschwind and Galaburda 1987). At any rate, developmental dyslexia is presumed to be a defect or affliction, present from birth or the very earliest childhood, of those parts of the brain which will one day, when the time comes, be expected to learn the skills of literacy. It is, in this view, an innate defect, irremediably hard wired into the brain which is innately pre-wired to learn literacy only in a particular location or locations. These ideas are, biologically speaking, extraordinarily unlikely, as we shall see later. They are also peculiarly pessimistic.

Secondly, what inferences may righteously be taken from a stance of scepticism towards this neurological deficit explanation for surprising difficulty with literacy? One inference that may *not* properly be taken is that the holder of a sceptical opinion towards dyslexia is also sceptical that there are large numbers of people with literacy difficulties. Clearly, and to the certain knowledge of this author, there are many such, and equally clearly many experience rather peculiar difficulties. To doubt that there is a neurological fault in their brains is not the same as denying that they have a problem. Further, and by exactly the same token, the inference that a dyslexia sceptic must think that special educational provision is unnecessary for people with such a surprising difficulty with literacy may also not properly be made. Clearly, people with special difficulties need particular assistance; they should better, perhaps, have practical help without a diagnosis of an incomprehensible, improbable and everlasting neurological defect having to be 'diagnosed' first.

This is because, thirdly, a diagnosis of an innate defect in the brain may cause *learned helplessness* in student and tutor alike (Kerr 1999, 2001a, 2001b). And this is because such a diagnosis is a *maladaptive attribution* – an attribution which disempowers and disadvantages. The literacy difficulty is, by its own definition, being attributed to an innate deficit within the student, which cannot be 'cured' and which can barely be overcome. Extraordinary measures and Herculean efforts will be necessary if it is to be, at any rate. The condition, it has logically to be presumed, will forever make the learning of literacy much slower, less certain, more improbable and more difficult than it would be for a 'normal' person. To accept this is to descend into the damp and fetid cellars of educational pessimism

where learned helplessness grows like a fungus, consuming pleasure, motivation, enthusiasm, confidence, curiosity, engagement, stamina, expectation and performance. This fungus will, of course, infect everyone - family, teacher and taught alike.

Who is 'dyslexic'?

In this section we will consider the main research-based scientific theories concerning the origins and manifestations of dyslexia. First, and fundamental, we consider sample selection.

The most basic aspect of research into any particular group's characteristics is the selection of the sample to be studied. In order to study a particular group of people (for example diabetics, vicars, redheads or management consultants) the characteristics by which membership of the group is awarded must be defined as closely as possible. Criteria would, ideally, include all group members and exclude all non-members. If the study were to be of management consultants, for example, it would not be sufficient simply to include everyone who came to work on a particular train dressed in a dark suit. Such a sample might include several management consultants, but it might also contain dentists, advertising executives, I.T. consultants, white goods salesmen, up-market pickpockets, domestic science teachers, junior ministers and many others. Results of research on such a sample would not be dependably valid in respect of management consultants. It would not have been carried out solely among people known to be management consultants. We would not dependably reach a true sample of management consultants using this blunt selection process. Our research results will not be defensible unless we apply better, tighter sample selection criteria.

Thus it is, of course, with research into 'dyslexics'. Before any meaningful examination of 'dyslexics' is carried out, the criteria for selection must be made clear. Who is 'dyslexic' and who is 'normal'?

Assessment for dyslexia for the purposes of scientific research almost universally continues to regard the *intelligence/achievement discrepancy* criterion as the single pathognomic indicator of dyslexia and researchers either use it themselves, or rely for diagnosis on educational psychologists who use it. (A pathognomic sign is one which, even occurring alone, is sufficient absolutely to indicate a particular syndrome.) The discrepancy criterion is elucidated as follows: The IQ of a person is measured, and then their performance at some literacy skill or skills - reading, for example. The decision is made to consider someone as 'dyslexic' where there is a discrepancy of more than such and such an amount between the performance expected from a person with their particular measured IQ, and their actual performance in administered tests. The discrepancy is often expressed as a *reading age discrepancy* (a discrepancy between their expected reading age according to IQ and their reading age measured by actual reading performance).

Considering a particular discrepancy between measured IQ and performance on norm-referenced literacy tasks to be pathognomic for 'dyslexia' is problematic on two counts. The first is the increasingly frequent finding that a population defined as dyslexic by an IQ/achievement discrepancy criterion does not, in the event, differ reliably or importantly from the general population (Fletcher 2003, Fletcher 2004, Fletcher, Denton & Francis 2005, Fletcher *et al* 2005, Miles & Miles 1999, Samuelsson *et al* 1999, Siegel & Himel 1998, Stanovich & Stanovich 1997, Stanovich 2000, Stanovich 2005, Stuebing *et al* 2002). The discrepancy criterion is, to put it plainly, no longer held to indicate 'dyslexia' or any other neurological learning difficulty reliably, or even at all.

IQ/achievement discrepancy certainly indicates the existence of a problem (it is a discrepancy after all) but the measure says nothing useful about its aetiology (its origin or cause). Most frontline research, even recent research, and most consequent theorising on the nature or remediation of 'dyslexia', however, rests on this definition of the sample population as distinct, different and dyslexic by application of this discrepancy criterion, and by this criterion alone. (eg Brooks & Weeks 1998, Fisher *et al* 1999, Hanley 1997, Hogben 1997, Hynd *et al* 1995, Nicolson & Fawcett 1999 and many, many, many more. Some are coy - Shaywitz's widely read book (2005) states clearly that the discrepancy criterion is no longer valid as a diagnostic tool, but lets slip that the research the book is largely based upon is the Connecticut study, from a few years back. The sample population for that study was identified as 'dyslexic' by exactly that discrepancy criterion.)

Such studies, and such conclusions, may thus be fundamentally, even fatally, flawed. Many subsequent studies rest their arguments entirely upon, or take as their starting point, the apparent results and conclusions from these earlier studies, almost all of them based upon the discrepancy criterion. Where sample selection is invalid, then so are results and so are conclusions and so is almost all we think we know about 'dyslexia'. This is fundamental, so let us look carefully and sceptically at the evidence and the debate. How valid is the discrepancy criterion as a selection tool for 'dyslexia'?

Samuelsson *et al* (1999 p. 94-95) state that '... the use of IQ achievement discrepancy definitions does not constitute a valid approach to define sub-groups of poor readers.' Stanovich (1991 p. 22) says that 'Defining dyslexia by reference to discrepancies from IQ is an untenable procedure.' Siegel & Himel say (1998 p.91) that '... the use of an IQ-based discrepancy definition of dyslexia is invalid on variables closely related to the reading process.' and on p. 102 that '...the use of IQ to define dyslexia (or reading disabilities) seems fatally flawed because of the confounding with SES [socio-economic status] and age.' Adams (1990 p. 59) says that 'Whereas IQ & general cognitive skills seem not to have much bearing on early reading achievement, early reading failures seem to result in a progressive diminution in IQ scores and general cognitive skills.' Stanovich & Stanovich (1997 p.6) write that 'In the largest longitudinal growth-curve investigation ever conducted, Francis *et al* (1996) reported that IQ was completely useless for predicting future reading growth among poor readers.' Miles & Miles (1999 p. 114) say that 'It is perhaps strange that this notion of a discrepancy definition survived as long as it did.'

A single issue of the journal *Dyslexia* considers this subject. In it various authors dispute the validity of the IQ/achievement discrepancy criterion as diagnostic tool. (Miles 1996, Share 1996, Stanovich 1996, Tunmer & Chapman 1996, and see Cotton *et al* 2005.) Stuebing *et al* (2002) have produced a highly regarded meta-analysis of research in this area, clearly showing that the discrepancy criterion should no longer be regarded as valid, and it should no longer be used as a tool for the diagnosis of dyslexia. If this is true today then it was also true yesterday, of course.

Keith Stanovich seems frustrated. He writes that

The persistence of the discrepancy concept in LD [learning difficulties] signals that the field is not yet ready to put itself on a scientific footing and that it will continue to operate on the borders of pseudoscience... The field suffers greatly from its tendency to base practice on concepts and psychometric technologies that have been superseded by subsequent scientific advance. I am referring here to the field's persistence in linking the definition of learning disability to the concept of aptitude-achievement discrepancy and identifying aptitude with intelligence test performance. (Stanovich 2005 p. 103)

Fletcher (2004) observes that 'Recent empirical synthesis and consensus reports share the common finding that IQ is ineffective in the identification of LD [learning disability] ...' and that 'It is widely recognised that the presence of IQ-discrepancy ... does not mean that the student has a neurobiological disorder.' He says that 'The clear consensus ... is to abandon the ability-achievement discrepancy model.' (And see Fletcher, Denton & Francis 2005 & Fletcher, Francis, Morris & Lyon_2005 reporting 'serious psychometric problems' causing low reliability and validity of aptitude-achievement discrepancies as indicative of learning disability - it is not possible accurately, well, or in some cases at all to measure that which we have claimed to measure).

When reading literature claiming that 'dyslexics' exhibit this or that symptom, or behaviour, it obviously behoves us to ascertain how the sample of 'dyslexics' was arrived at – how were they diagnosed. If it was by the discrepancy model of diagnosis, as is almost always the case, the findings are thereby rendered invalid and should, properly, be ignored. They seldom are. Indeed, such findings regularly march cheerfully on, underpinning and 'validating' later work, in bibliography after bibliography. The 'borders of pseudoscience' indeed.

Two other observations are relevant in this context. One is the disassociation between IQ (whatever it might, or might not, be) and reading. They do not correlate well. IQ does not predict reading ability at all well. Reading is well learned by people with high IQs but it is also well learned by those with low IQs. Reading is, in fact, a rather low-level skill and it does not demand high intelligence (Stanovich 2000). (By 'reading' right here I mean decoding to meaning. How well you subsequently manage information you read does, of course, depend at least partly on your intelligence. The actual reading

does not.) Also, it should here be noted that reading well tends to increase IQ whereas reading poorly tends to cause IQ to fall, over time. This is presumed to be because we gain so much of our understanding and knowledge, as well as vocabulary (that with which we formally think) from text, one way or another, and are exposed to so much of our culture, and the wider world thereby. (Adams 1990, Siegel & Himel 1998 and Stanovich 1991).

A second, and perhaps equally fundamental, difficulty lies in the concept of IQ itself. What is it? Some writers (e.g. Turner 1997) claim to measure a multitude of different IQs in a multitude of different cognitive domains with great precision, though even Turner finds himself saying that:

It has often been said that the best indicator of dyslexia in young children is the performance of the father on a reading test. As 80 per cent of cases may be identified in this way, it would compare favourably with more elaborate screening exercises! (ibid. p. 224)

Gipps and Murphy are more circumspect. They say (1994 p. 71) that '... IQ tests are biased in favour of individuals from the dominant culture who designed the tests; in the UK this means those from a white, male, Anglo-Saxon background and, in addition, middle class.' They also say (ibid.p.74) that it is '... impossible to devise tests which do not depend heavily on knowledge which is culture-dependent.' and (ibid. p.90) 'The aura of scientific objectivity surrounding IQ tests meant that there was little attempt to question them.' As Stanovich (1991 p.9) says – '... one would be hard pressed to find a concept more controversial than intelligence in all of psychology.' Adams (1990 p. 59) claims that '... IQ is only weakly and non-specifically related to achievement in the early grades.' Siegel & Himel say (1998 p. 91) that

...the IQ measure is problematic because of several aspects of the IQ test ... IQ is strongly related to socioeconomic status ...IQ is actually a measure of the type of knowledge that is dependent, to a large but unknown degree, on the environmental experiences of the child.

Stanovich (1991 p. 10) writes similarly that

... most psychometricians, developmental psychologists and educational psychologists long ago gave up the belief that IQ scores measured potential in any valid sense ... an IQ test is not properly interpreted as a measure of a person's potential.

Educational psychologists and researchers into dyslexia, notwithstanding all of the above, still regularly use IQ and IQ/achievement discrepancy tests, to make exactly such 'measurements', and to diagnose 'dyslexia' on the strength of it, or at least continue to use data relating to sample populations selected in this manner (e.g. OFSTED 1999, Shaywitz 2005).

And, as an aside in passing, there is a workable alternative to using the discrepancy criterion. In response to the understanding that an IQ/achievement discrepancy indicates nothing particularly useful, and certainly nothing 'diagnostic', a different approach has been developed in the USA. It is known as *RTI (Response To Instruction)* (See Fletcher 2005, Fletcher, Francis, Morris & Lyon 2005, Fletcher, Denton & Francis 2005 & Kovaleski 2004). RTI avoids psychometric testing (in particular IQ) and focuses instead on a student's response, in terms of learning, to instruction received. This has the advantage of relating to an individual's progress or otherwise as it happens (rather than after failure has already occurred), thus being child-centred, immediate and relatively easily operationalised in the real world. It is based directly on practice, so leads directly to appropriate teaching responses. Indeed, as its title indicates, it focuses, quite deliberately, on instruction as well as response. Where there appears to be a problem, educational consideration is immediately drawn to the teaching offered to the student as an individual as well as to his or her learning.

In the words of Fletcher, Denton & Francis (2005 p. 545) 'Hybrid models combining low achievement and response to instruction most clearly capture the LD [learning disabilities] construct and have the most direct relation to instruction'. (In my opinion it is unfortunate that the RTI tool should be used to continue the search for disabilities, so perpetuating the deficit approach and its attendant maladaptive

attributions, but it is also my opinion that RTI does not actually have to be thus. The tool could simply be deployed to find, and address, any learning difficulty in any subject. I imagine that many of the better teachers subvert it exactly thus, in real classrooms.)

Some writers use a different discrepancy to diagnose dyslexia - that between chronological (actual) age and 'reading age' - usually demanding a discrepancy of two or more years (e.g. Williams & O'Donovan 2006). However, such a discrepancy is merely a sign. It is just a symptom. It says nothing, in itself, about the underlying cause or nature of those influences which might be causing it. It is like observing that at any one time a certain percentage, say 10%, of people are likely to be lame and then going on to claim that this indicates that 10% of us has a sprained ankle. The one fact does not at all reliably indicate the other. Thus it is with 'dyslexia'. If someone has a chronological age / reading age discrepancy it tells you only that they read poorly for their age. It tells you nothing of why this is so. We may not properly deduce any aetiology from the discrepancy itself. It is 'diagnostic' of nothing whatsoever other than an unfortunate and as yet unexplained discrepancy.

Those few researchers who attempt to diagnose dyslexia without reference to a discrepancy criterion are beginning to use performance related signs which they claim are pathognomic. There are many tests of literacy abilities to choose between, including single word reading, spelling, pseudoword reading, irregular word reading, homophone/real word choice, homonym choice, phoneme manipulation, rapid automatic naming and reading speed - all mentioned, for a single example, in Paracchini *et al* (2007). They go on to write that 'Unfortunately, there is not universal agreement on which precise ascertainment criteria and psychometric tests should be applied, and different research groups typically use a specific selection of them, often dependent upon the language of the population under study' (ibid. p. 59). There is no consensus, as you can see, as to which test, or which battery of tests, enables a 'diagnosis' reliably to be made. Researchers literally pick and mix. 'Performance deficits' selected by the researcher are said, by the researcher but without evidence (as there is none we can rely on), to be typical of and pathognomic for dyslexia. Researchers differ widely as to which tests are appropriate and what their results may indicate. Such highly subjective criteria for sample selection and such individual interpretation of findings will, however, not stop considerable reliability of diagnosis, validity of result and comparability of conclusion being claimed.

A further means of 'diagnosis' of dyslexia and selection of sample 'dyslexics' is simply to throw in the sponge, deploy the *'bell curve'* of reading ability and define those in, say, the lowest 10% as 'dyslexic' (e.g. Olson 2006, Paracchini *et al* 2007). As you will know, the *normal distribution curve*, or bell curve, is the curve which can be plotted for any attribute which is *normally distributed* across a population (height is the usual example). The curve looks like a bell, hence the name. Reading ability is normally distributed. If you plot reading ability across the population you get the familiar bell shaped curve. It is easy to select, say, the bottom 10% of such a population from their results on reading tests and consequent place on the curve. As Paracchini *et al* write 'RD [reading disability] represents the lower tail of a normal distribution of reading ability found in the general population' (ibid. 2007 p. 59). Kate Nation (2006 p. 2) reaches the same over-extended classification when she writes about '... individuals who are at the low end of distribution - individuals who are reading disabled'. Olson further claims that 'the positive consequence of the bell curve in reading research is that it allows us to apply powerful statistical methods in our genetic analysis of dyslexia and individual differences that depend on normal distributions ...' (Olson 2006 p. 3).

It may be useful, in certain limited circumstances and in certain rather broad but limited ways, to identify and analyse those in the lowest 10% of reading ability. However, it is not legitimate to claim that simply because they all find themselves in this bottom 10% they must all share any particular characteristic, let alone all suffer from the same syndrome, without further evidence that this is so. We have no evidence as to why these poor readers are in this group. We can guess, though, that there will be many and very various reasons for their poor reading. All we can properly say from contemplation of the bell curve is that they all seem to be poor readers. It is improper to claim more than this on this evidence - especially to claim that membership of the poor readers group *per se* indicates possession of a neurological deficit - indicates that all these people suffer from dyslexia. We cannot say this with any certainty whatsoever - the reasons for inclusion in this group will be numerous and various. These poor readers do not constitute a group which is reliably homogeneous. For this reason sophisticated statistical and/or genetic analyses and conclusions in respect of 'dyslexia' are not appropriate, however tempting the wonderful mathematical potentials of the bell curve and the statistical marvels of normal distribution.

A frivolous example to illustrate this general point: Suppose we set up a driving test whereby a thousand randomly selected people are asked to drive a car across rural Wales between two points 50 miles apart. We measure their performance. (Time taken, number of bumps recorded, frequency of road rage incidents etc.) The results will probably approximate to a bell curve of normal distribution of whatever we have decided to define as 'driving ability'. Would this statistical fact mean we can consider that the worst 10% of drivers all share the same characteristics, though? Are they all poor drivers for the same reason? Of course not. Some may have been drunk, or high, others may have been partially sighted, others may have been teenage males charged with testosterone, others again may have been elderly and very cautious, some may have driven for years while some may only just have passed their test, some will only just have got off the plane from Australia, some will have been rendered hopelessly nervous by the knowledge that their driving was being tested, for some the route will be familiar while for others it will be completely novel. And on and on. There will be a plethora of reasons for their poor performance, and regarding these drivers as a homogeneous group with a single 'syndrome' (dysautomobilia?) will not be valid. Nor will it be particularly useful. It will not reliably reveal much of interest, either to science or to the department of transport. Our findings will not enable us to apply sophisticated analyses to make generally useful policy decisions, in fact, nor to reach any particularly valid conclusions about the drivers themselves.

Olson says that

the problematic consequence for the diagnosis of dyslexia is that in spite of the frequent and varying citations in the literature about the percentage of children who have dyslexia, dyslexia does not exist as a discrete diagnostic category that is distinctly separate from the normal population distribution. Statements about the percent of children or adults with dyslexia are based on arbitrary cut points on the low ability tail of the normal distribution, and this is true even if some sort of IQ-reading-discrepancy criterion is employed. (Olson 2006 p. 3).

As we have seen, there are major issues with all of this, and inevitably also with conclusions drawn from research using such non-specific means of selecting populations of 'dyslexics'.

Since there are still no standard, generally agreed pathognomic signs of dyslexia, or tests for it, partly as a result of there being no standardised 'dyslexics' and partly because of the ongoing and acute lack of consensus as to the real nature of dyslexia, all such diagnoses, and all consequent sample selections, are highly questionable. So, of course, is all the science based upon the study of such sample populations and so are all the conclusions allegedly reached thereby.

In short: Studies on 'dyslexics' all experience the same, absolutely fundamental problem, namely the all-important selection of their sample. The question 'who is dyslexic?' has yet to be answered.

The 'gene for dyslexia':

Saint Augustine reputedly wrote 'For so it is, Oh Lord my God; I measure it, but what it is that I measure I do not know.' This quote should illuminate the following discussion on the genetics of literacy. I don't understand genetic discourse well but I insist on my right, and my duty, to remain sceptical while watching this space with a great deal of interest. Literacy attainment, or otherwise, is a very complex social and cognitive issue. Many ill-understood influences powerfully (and variably) affect it. Individual case characteristics and whole-population findings do not necessarily relate at all well. In the words of Steve Ramm "Splitting complex phenomena into genetic and environmental components is just about impossible and, of course, each will feed back upon the other." (personal communication September 2007). This aspect of our debate is, regrettably, much more complicated than it looks at first.

I find the genetic 'proof' of dyslexia the most difficult to deal with. It is difficult for two reasons, first because it seems to me, as a biologically trained person (I am a veterinary surgeon in 'real life'), to run directly counter to common sense, but second because it is a subject which is, perforce, mediated (and understood) through fancy mathematics as genes exert their effects by interacting among each other and with their environment in extremely complex ways we do not yet fully understand (to say the

least). I have immediately to confess an inability to follow the mathematical arguments intelligently, or indeed at all. I am, therefore, not able to challenge ‘proofs’ in the mathematical arena. There are nonetheless relevant, if captious, observations which may perfectly properly be made by the sceptical but mathematically inept, such as myself. There are many papers in which genetic effects on reading, and on dyslexia, are claimed (e.g. Cardon *et al* 1994 [corrected 1995], DeFries 1997, Fisher *et al* 1999, Olson 2006, Paracchini *et al* 2007, Schumacher *et al* 2007, Williams & O’Donovan 2006) and it will be very interesting to see what the map of the human genome eventually tells us. The genetic argument is an important strand in the debate, as it seems to be saying extraordinary and potentially considerable things, and should suffer some critical analysis. Here, I describe some of the work done and display its findings in such a way, I hope, as to allow you to deduce your own responses as well as to read mine.

Every one of us is made up of zillions of cells all containing, and all controlled by, an identical set of 23 chromosomes which make us, genetically, who we are. Each chromosome consists of an immense chain of genes. Each gene is made from a complex protein called *DNA* (deoxyribonucleic acid) – the famous double helix. All DNA is made from a small number of amino-acids. The order in which these amino acids appear on a gene varies between genes and is the *genetic code* (a bit like an alphabet where each amino acid is a letter). Each gene is a unique code, its amino acids arranged in a unique order (a bit like a set of words). Our DNA is unique to us in that the arrangement of amino acids on my genes and genes on my chromosomes is mine and mine alone. Hence its forensic value - it identifies me uniquely. The process of ‘reading’ amino acid and gene sequences is mechanised today and is relatively easily done. The human genome is already being deciphered and its alteration to order may be within reach. Scarily, it might, one day, be possible to reconstruct me (maybe even create an improved me) from a sample of my DNA. More interestingly, it may be possible to reconstruct extinct creatures from bits and pieces found here and there. The dodo might yet live again.

Our genes are responsible for our inherited characteristics. Every gene encodes information (as a particular ordering of amino acids) and a gene may issue instructions within a cell by causing the cell to encode a protein with amino acids ordered in its own image. However, each gene also interacts with other genes, with our own physiology, with our own characteristics and with our environment to exert its effect. It’s a fiendishly complicated area of knowledge and is very much still in its infancy. We are our genes (up to a point) and we are our history and our environment (up to a point) - the problem in any particular instance is, up to which point? For any aspect of ourselves, in this context, the question remains - nature or nurture? The answer is very seldom clear.

We need, of course, loudly to note that finding a gene, or a *QTL* (*Quantitative Trait Locus*), which appears to have some effect, in some circumstances, on an attribute or ability (reading, for example) is assuredly not the same as finding ‘the gene for reading’. (A *QTL*, incidentally, is often rather broader than a gene. It is a section of a chromosome which appears to have an effect on something. It may be as small a section as a single gene, but it might be a section of chromosome many genes wide.) We, and our media, are often appallingly sloppy in our use of terminology and we often say things which are frankly ridiculous as a result. The discovery of ‘the gene for dyslexia’ is often hailed, for example, even in supposedly ‘good’ newspapers. We must keep our wits lively about us unless we are to be led down the seductive paths of prejudice and simplification towards nonsense.

Fisher *et al* (1999), for a preliminary example, claim that between five and ten per cent of schoolchildren are affected by dyslexia (but define their ‘dyslexics’ by IQ/achievement discrepancy). Within the sample thus selected they claim to have found a *QTL* on chromosome 6. This *QTL* ‘... affects both phonological and orthographic skills and is not specific to phonemic awareness.’ (Fisher *et al* 1999 p. 146). And ‘... this locus affects both phonological coding and orthographic coding.’ (ibid. p. 152). (NB. These were tested by non-word and irregular word reading. Both probably ‘test’ for many things other than, and perhaps in many cases much more powerful than, linguistic codings.) They suggest that this *QTL* is ‘... involved in an underlying mechanism that is common to the development of both types of skills.’ (ibid. p. 155) In other words, Fisher *et al* claim to have found a *QTL* which supports both reading and spelling skills. They do, though, also say that ‘... there may be substantial phenotypic variability among subjects designated as affected and there is some dispute about the nature of the core deficit.’ (ibid. p. 146) and ‘...any genetic basis for this disorder [*dyslexia*] is likely to be complex.’ (ibid. p. 146). There is, in other words, plenty still to debate, and the appropriate attitude remains sceptical but open-minded interest.

Olson (2004), studying sets of identical (MZ or monozygotic) and non-identical (DZ or dizygotic) but same gender twins, concluded that approximately half of the reading difficulty he observed was genetically determined. He makes the claim that 'in spite of the high heritabilities for group deficits in phoneme awareness and phonological decoding these deficits can be substantially remediated and even normalised.' (ibid. p.120). (Shaywitz (2005) makes the same claim and even shows scans of apparent changes in gross brain function patterns to show this apparently happening before our very eyes. It is unclear, at least to me, why an innate, hardwired deficit so effectively prevents learning under 'natural' circumstances but allows it to 'normalise' so readily under others. This is an extraordinary claim. Brain scans are also tools of highly debatable value in such a context - and see later.)

Olson found group deficits due to genes at these rates in phoneme awareness (.72), phonological decoding (.71) and orthographic coding (.67). This last is extraordinary in that it seems to indicate a genetic effect on spelling - a 'gene for spelling' in media-speak! Finally, I would wish to note, in respect of this work, that orthographic coding is utterly different from phonological decoding or phoneme awareness, presently widely regarded as the 'core deficit' in 'dyslexia'. Orthographic coding and phonological coding, as we have seen, are utterly different cognitive acts, taking place in utterly different cognitive domains and utterly different anatomical sites. Wherever, and however, we look we seem to find genetic 'explanations' for 'dyslexia'. It is difficult to see what is being found, or measured, and extreme caution in interpretation, even acceptance, of apparent findings is absolutely appropriate.

DeFries (1997) offers an interesting review of dyslexia studies among MZ and DZ twins. He reports that Stevenson *et al* (1987) studied such twin pairs in which at least one member of each showed reading or spelling backwardness (measured as a reading age/chronological age discrepancy) or reading or spelling retardation (measured as IQ/achievement discrepancy). They claimed that 'whereas genetic factors may be important as a cause of reading disability at younger ages, spelling difficulties appear to be more heritable than reading deficits at 13 years of age.' (DeFries 1997 p. 21). They claim, in fact, that spelling is '... the most clearly genetically influenced literacy skill.' (Stevenson *et al* 1987 p. 243, quoted in DeFries).

Wadsworth *et al* (1989) and DeFries *et al* (1991) (both reviewed in DeFries 1997) produce data which they claim supports the conclusion that '... spelling may be less susceptible than reading to environmental influences.' (DeFries 1997 p. 22).

DeFries (1997) also examined twin pairs, at Colorado University. In fact these were the same twin pairs as Olson studied (Olson 2003). DeFries chose a sample of 145 same-gender DZ twins and 195 MZ identical twins. At least one of each pair was diagnosed as 'dyslexic' (by the IQ/achievement discrepancy criterion). His data suggested that '... reading disability is partly a consequence of heritable influences' (ibid. p. 22). This was shown by the apparently more frequently (though not invariably) shared disability among MZ than DZ twins. The data also suggested that there is a greater influence on spelling from their genes as they age and less genetic influence on reading, or at least word recognition. '... spelling deficits are more heritable than word-recognition difficulties in older children ... spelling may emerge as the most genetically influenced literacy skill during early adolescence.' (ibid. pp. 22-23).

What on Earth is going on? These studies appear to indicate (though not always to a great degree of significance, consistency or power and frequently using dubiously selected sample populations) that there is at least one QTL which is at least somewhat, and at least sometimes, affecting phonological, but especially orthographic, coding (coding text to sound, but also, and particularly, to letter patterns – a function of spelling ability). They seem to indicate that the patterns of affliction among identical twins often (but not always) show that spelling performance in particular is at least partly genetically determined. Are we claiming that there is a gene, or QTL, for spelling? How could that possibly be?

This issue raises absolutely fundamental biological questions. Biologically speaking, there can be no 'genes for literacy' *per se* (let alone spelling!). If there cannot be genes specifically for literacy, then neither can there be genes specific to 'dyslexia'. A biologist can assert this on two grounds: First, there has been nowhere near enough evolutionary time, and second, there has been nowhere near enough evolutionary pressure. The first literate acts of our species were probably clay tablet invoices and receipts dating from about six thousand years ago (Manguel 1996, Rayner & Pollatsek 1989). In evolutionary terms this is an eyeblink. Throughout history, literacy has been both vanishingly rare and

absolutely irrelevant to survival or reproduction until extremely recently, if then. It is only in the last 150 years, indeed, that a majority in Britain have become literate and many are not particularly fluently literate even now (Carey *et al* 1997). In global terms it is debateable whether a majority is fluently literate today, and it is also thoroughly debateable how important literacy is to survival or reproduction (survival of the person or the genes) for most people even now.

For a skill or aptitude to become encoded onto genes two conditions are required. Firstly, there must be immense amounts of time (in the order of one hundred thousand years) and secondly that aptitude must provide a clear competitive advantage to individuals, but across the whole population, to force the process of natural selection. If the aptitude is to survive selectively it must matter to survival and reproduction, it must be biologically important, must convey a distinct and biological advantage, and the 'selection pressure' against which the aptitude is advantageous must apply to a majority of the population for a majority of the time. We have had some ninety four thousand years too few and, for the general population, far too little selection pressure for the encoding of any genes specific to literacy. Too few of us have been doing it for too short a time and it has been too unimportant to survival or reproduction. It would make about as much sense, biologically speaking, to propose genes specific for driving ability.

There is, and can be, no 'gene for' literacy ability, or dyslexia, *per se*. It is, though, feasible to imagine a gene or some genes affecting a more general aptitude (detailed sight, for example) which is also, in some unspecific way, necessary to, or supportive of, literacy. To say this, of course, is to say a very fundamental thing about dyslexia. It is to say that *dyslexia, if it exists at all, cannot exist as pure literacy failure*. Dyslexia, if it exists at all, must always, consistently, be accompanied by a fundamental deficit or deficits in a more general aptitude or aptitudes which also, but only coincidentally, relate to the acquisition and management of literacy. The 'dyslexic' will display a relative failure in some general, fundamental, neurological aptitude. He (or she) will do this consistently. In other words, the 'dyslexic' who is perfectly 'normal' in all other respects is mythical. He (or she) must, logically, have some appreciable cognitive deficit. The search for such consistent cognitive deficits accompanying 'dyslexia' continues, with wildly various results and no consensus. A plethora of deficits and abnormalities have been proposed by researchers in recent years. They include phonological awareness deficit, magnocellular system deficit, anomalous lateralisation, abnormal symmetry of the planum temporale, weak automatization, poor motor coordination and a plethora of other neurological abnormalities. None has been proved and all are disputed. There is no consensus.

Many studies, incidentally, (and see Paracchini *et al* 2007) speculate on the actual biological effect whichever QTL they claim affects reading ability may have. What actual effect in the brain may the mutation in 'their' QTL be having? Many describe an effect on the migration of neurones during the brain's development. Known neurological syndromes are mentioned in this context, especially lissencephaly, double cortex syndrome and periventricular nodular heteropia. However, these are gross abnormalities detectable, for example, by scanning. They clearly cause many neurological symptoms, frequently, for example, epileptic seizures. They also cause cognitive deficits. It is unsurprising that, in many cases, literacy difficulties may also be seen. In such syndromes there is gross brain damage and multiple deficits, only one of which is 'dyslexia'. Such gross abnormalities, even when they involve, say, reading difficulty, are not 'dyslexia' as we must properly consider it. Such cases are not the classical understanding of 'dyslexia' which has to involve an innate literacy difficulty found in people without obviously gross lesion or abnormality. A person suffering from lissencephaly, for example, is to be described as a person suffering from lissencephaly (which may, among many other gross effects, also affect their literacy abilities) but not as a 'dyslexic'.

The genetic findings, whatever they really are, amount to an admittedly disturbing aspect of the debate, for those who find dyslexia unconvincing in more general terms. I believe I can properly accept and re-assert a 'general conclusion that a genetic underpinning to 'dyslexia' is far from established' (Steve Ramm, personal communication September 2007). I am not fully qualified to follow detailed genetic argument, and neither, quite probably, are you. We are both required, notwithstanding, to cling fiercely to common sense and scepticism - it is the proper approach to all knowledge.

I believe it is early days yet. Astonishing things, with strange implications, are suggested within this field. Something is being measured by this work (though not always to a high level of significance or very consistently, not always demonstrating much genetic power, not always supported by other research, and seldom carried out in appropriately defined sample populations). What, exactly, is being

measured is not at all clear, as is sometimes admitted even by researchers in the field. Whatever it is, it cannot be 'the gene for dyslexia', or 'the gene for spelling', or 'the gene for literacy'. And finally, understanding the most appropriate way, or ways, all this work is to be interpreted remains very unclear, to say the least. What (and how much) does any of all this mean? Some are prepared to assert very extraordinary things on the basis of rather small and tentative findings, many are more cautious. (Grigorenko *et al* (2006 p. 119) say, for example, that '... we might be amazed to know how far from understanding the genetic bases of reading we actually are'. Schumacher *et al* (2007 p. 294) write that "to date no specific cognitive processes are known to be influenced by the proposed susceptibility genes".) Let us remember the wise words of St Augustine: 'For so it is, Oh Lord my God; I measure it, but what it is that I measure I do not know'.

We should also, because we have our wits still about us, briefly but firmly recall the two issues of carts and horses and affect, both of huge but studiously unrecognised importance. First the unsolved issue of horse and cart in much literacy research – when we discuss the relationship between a skill and an ability, what is cause and what is effect? It is not always clear (to say the least) whether an apparent disability is caused by an apparent skill deficit or whether the apparent deficit is caused by the apparent disability. Put another way, is my skill at orthographic coding, say, the cause of my facility with literacy or has my facility in literacy resulted in a high level of skill in decoding text? Secondly, the consistently unremarked effect of affect on performance: The effect of emotional response and attitude on task performance is often enormous but it is usually unknown, often practically unknowable and almost invariably completely ignored for the purposes of research. Looking the other way is understandable (it makes research and interpretation so much simpler!) but it is also quite unjustified and undoubtedly skews findings and conclusions drastically, often probably fatally. This unconscious researcher bias is only occasionally recognised, as in an interesting article in a special issue of the *Journal of Research in Reading* given over entirely to reading and genetics (Conlon *et al* 2006). It remains, otherwise, elephantine but invisible.

Phonological awareness:

This is the ability to locate, separate and distinguish for what they are the sounds in language. (And see the more detailed discussions in chapter three.) Various aspects of the detailed sounds of language are assessed by researchers - syllables, onset, rime, phonemes, consonants, vowel sounds. (Non-word reading or phoneme manipulations are common tests, for example.) The hottest candidate as I write for the 'core deficit' in dyslexia is phonological awareness. 'Dyslexics' (almost always still IQ/achievement defined) are considered to be less phonologically aware. There is indeed evidence that 'dyslexics' (or at least people with poor literacy skills) have relatively poor phonological skills. (Goswami 1997, Snowling 1995, Snowling & Nation 1997 *but* see also Ellis, McDougall & Monk 1999, Scholes 1998, Thompson 1999) There is also, though, the finding that receiving training in literacy (in an alphabetic language like ours) rapidly improves phonological awareness. This is an important carts and horses issue; it is well aired in Goswami & Bryant 1990 pp. 4-27. (And see Adams 1990, Perfetti & Xhang 1995, Smith 2004, Taft 1991, Thompson 1999.)

Spoken language is inchoate and indistinct. To the fluent listener all (usually) seems clear enough but physical analysis of spoken language shows it to be amazingly messy and gappy. Word boundaries disappear, along with much else; sounds slur into each other or vanish altogether. Listeners obviously have, in fact, to work quite hard to make sense of it all, extrapolating from the minimal sounds offered and mindful of the context in which the speech is probably immersed. Given this phenomenal fuzziness of spoken language it is not surprising that those who do not read or write well, or often, also have poor phonological segmentation skills, nor that the latter improve rapidly with targeted tuition, nor that such improvements support subsequent spelling performance. There is a certain circularity here: learning literacy (in English) causes phonological awareness, something only rather good spellers have.

However, the evidence does not indicate that a 'deficit' in phonological awareness skills is causative of poor literacy skills, merely that it tends to co-exist with them (see chapter three and Castles & Coltheart 2004). Poor phonological awareness does predict literacy performance but this, of course, is not the same as proof of causation, and there are also other skills which predict literacy performance, most obviously letter awareness.

The skill of letter awareness is very much less pursued in the research world but is also powerfully correlated with later reading success (Barlow-Brown and Connelly 2002, Blaiklock 2004, Gallagher *et al* 2000). 'A causal relationship between phonological awareness and reading ability has not directly been established ... performance on phonological tasks may tap into letter-based, rather than purely phonemic, representations' (Whitney and Cornelissen 2005 p. 274). And: 'The National Reading Panel's meta-analysis ... found ... that phonemic awareness instruction using letters helped children learn to read and write, but that phonemic awareness instruction without letters did not help children to learn to read and write' (Besser *et al* 2004 p. 17). And: 'We propose that preliterate phonological encodings do not include a level of representation corresponding to phonemes ... Rather, reading acquisition itself creates a phonemic representation, via linkages of graphemes to groups of phonetic features ... the phonemic encoding depends on a linkage to orthography' (Whitney and Cornelissen 2005 p. 289). And general linguistic ability itself, as you would absolutely expect, correlates with reading ability in a similar manner to letter and phonological awareness (Nation and Snowling 2004).

And this rather obvious theoretical observation is supported by research. The brain is plastic, even in adulthood (Gilman & Newman 1996, Springer & Deutsch 1997). It is changed, physically and procedurally, as a result of learning this, rather than that (Gaser & Schlaug 2003, a whole issue of the *International Journal of Psychology* vol. 39 (2004), Maguire *et al* 2000, Schlaug 2001). Castro-Caldas *et al* (1998), for example, found that, at the very least, '... certain aspects of the ability to deal with phonetic units of speech are not acquired spontaneously but are a result of learning to read.' and that '... learning the written form of language interacts with the function of oral language ... learning to read and write during childhood influences the functional organisation of the adult human brain.' (both quotes *ibid.* p. 1053) How could it be otherwise? (And see chapter three and Adams 1990 p. 69.)

To summarise an argument more fully rehearsed in chapter three and which reverberates around the literacy world: It has not been shown that phonological awareness actually causes literacy skills; that 'the core deficit' in 'dyslexia' really is a deficit in phonological awareness. This awareness may simply (and probably does) correlate very closely with other influences, one immediately obvious candidate being *letter awareness*. When letter awareness is examined it is found to be at least as good a predictor of reading skill as is phonological awareness. The positive effect of letter awareness on reading skill would fit well with the obvious fact that text is a visual signal and must, at least initially, be appreciated visually rather than phonologically. Text is implacably silent. We do not 'hear' it – we see it. It must be managed visually, at least to begin with. How could it be otherwise?

The magnocellular deficit theory:

This is an interesting proposal for the 'core deficit' in dyslexia. Our perceptual systems seem to be twofold - we have parvocell and magnocell systems bringing in data from the senses. The visual system is the one in which magno- and parvocells have been studied to date, but there may be similar dual systems in other modalities (for example the auditory system). A parvocell ('little cell') system delivers very detailed and exact data to the mind, presenting it with clear, complete information. The trouble is that it's slow - it takes time for the perception to reach the mind. Thus, perhaps, the magnocell ('big cell') system, which is much quicker. It won't deliver so fine-grained a perception, so complete a picture, but what it does give, it gives quickly. This can be an important advantage. Your magnocell system will, for example, tell you in good time that a sizeable object is flying through the air towards your head. Your parvocell system will, rather later, tell you that it's about 10 cm across, irregular in shape and made of rather nice grey granite with pretty patterns of reflective crystals on its surface. Horses for courses.

The speed of the magnocell system may have other, collateral advantages. When we read, our eyes stop and read, then snap along the line, then stop and read, then snap further along in the fixation/saccade routine we saw in chapter four. We need to be able to see, fixate and focus in detail, very rapidly and accurately if we are to do this quickly but competently. It has been suggested that it is the magnocell system which enables this. It has therefore been suggested that a deficit in the visual magnocell system might be a cause of reading difficulty in 'dyslexics'. (eg Eden *et al* 1996, Hogben 1997, Stein & Talcott 1999) There is some debate among those favouring this explanation as to whether the deficit is innate, perhaps genetic, and present from birth or whether it might be the result of insufficient training of the eyes below about the age of seven, at which age it is thought the system becomes more fixed and less trainable. It should be noted that some research disputes the theory altogether (e.g. Goulandris *et al*

1998, Johannes *et al* 1996). It should also be noted that this theory involves only the visual system and cannot explain a phonological awareness deficit. 'Dyslexics', it should be further noted, are IQ/achievement discrepancy defined in these studies.

Neurology:

Reading large amounts of research on the extremely varied neurological aetiologies proposed for dyslexia can be like looking in on Bedlam. Extraordinarily multifarious claims reverberate against each other; findings loudly compete. Everyone hopes and believes they hold the Holy Grail. The onlooker is spun this way, then that. It leaves the unwary disoriented, even queasy. On a bad day you wonder if your eyes still point in the same direction. Such reading demands that both feet be kept consciously and conscientiously applied to the terra firma of common sense. The following is but a taste ...

The doyen of writers on the subject of the neurology behind dyslexia is Albert Galaburda. Geschwind and he (1987) proposed a general, overarching theory of 'anomalous cerebral dominance'. They suggested that this arose *in utero* as a result of a testosterone imbalance. Their theory, which can only be described as grand, claimed that the brain's normal asymmetry was disturbed and that this caused a vast array of disorders including dyslexia, other language disorders, autism and disorders of the immune system. Evidence is rather small, wildly mixed and the theory remains warmly disputed (e.g. Bryden *et al* 1994). Problems of measurement and definition bedevil the elucidation of the theory and findings frequently absolutely contradict previous findings. 'Dyslexics' are also defined by the IQ/achievement discrepancy throughout.

Galaburda, and others, claim diagnostic abnormalities and even outright lesions are detectable at post-mortem examination of the brains of 'dyslexics' (only eight were examined, all defined by IQ/achievement discrepancy) and some laboratory rats and mice (Galaburda *et al* 1985, Galaburda (ed) 1993, Galaburda *et al* 1994, Galaburda 1999). On the whole, Galaburda and his co-workers appear to believe that dyslexia is not a genetically determined defect, but that it is induced *in utero* or in the neonatal period. The main, but not only, contender as aetiology is hormonal imbalance, particularly testosterone imbalance. Microscopically revealed 'abnormalities' are reported from a plethora of sites in the mid and fore brains, involving, for example, '... areas of the brain concerned with perceptual processing, as well as those involved in cognitive and meta-cognitive tasks' and '... at least the visual and auditory pathways.' (Galaburda 1999 p. 183). A recurring claim is that the planum temporale, found in the left upper temporal lobe and responsible for much language management, is 'abnormally' symmetrical in 'dyslexics'. How abnormal such abnormalities really are, if at all, is very uncertain indeed and so is how, if at all, they relate to observed abilities or behaviours (Pumfrey & Reason 1991).

'Abnormalities' are reported among 'dyslexics' in numerous living brain structures and behaviours using brain scan techniques. Hynd *et al* (1995), for example, found the genu of the corpus callosum significantly smaller in 'dyslexic' children. Duara *et al* (1991) found another area in the corpus callosum, the splenium, to be larger in 'dyslexics' than 'normals'. Larsen *et al* (1990) found 'abnormal' symmetry in the planum temporale in 'dyslexic' adults, 70 per cent of whom showed symmetry compared to only thirty per cent in 'normals'. (Why the figure is not one hundred per cent is unclear. Nor was it shown that any of the apparent 'abnormalities' in any of these studies actually related to any difference in performance or behaviour.)

Brain scans appear to show exciting, if crude, differences between 'dyslexics' and 'normals'. A recent popular science text on dyslexia, for example, relies heavily upon them (Shaywitz 2005). However, the normal but literate brain will differ from the normal but less literate brain in important functional ways which will show up on a brain scan. This is absolutely unremarkable - they have had radically different experiences and we know that brain architecture is profoundly affected by experience. (Robertson 1999.) (And remember Castro-Caldas 1998, Gaser & Schlaug 2003, Maguire *et al* 2000, Schlaug 2001, those large straws-in-the-wind.) As we have seen, Adams (1990) Siegel & Himel (1998) and Stanovich (1991), for example, all write that reading well, and often, enhances IQ while reading poorly and little does the reverse. Remember also the *Matthew effect*, of which more later. We need not, in fact, invoke neuropathology to explain these apparently neuropathological findings. There are other more parsimonious and credible explanations, of which more later.

It is also important to point out that scans, although they produce wonderful (and marvellously

expensive) pictures cannot really tell us about the detailed procedures within so subtle an organ as the brain. Scans are excellent for gross anatomy and visualising lesions and materially assist in medical diagnosis thereby. Their value in cognitive psychology, however, is much smaller. They may rather generally point to broadly interesting ideas on occasion, but they cannot be expected to do better than that. We cannot expect a scan, or even a series of scans, to show us the procedures which we suspect go on in our heads. These remain invisible – we have yet to film mental processes such as literacy, or love!

It is not at all clear, anyway, how far such weights-and-measures approaches to an organ as outrageously complicated, subtle and individually variable as the brain actually take us. Carts and horses are almost certainly also involved. Sample sizes, particularly when for post-mortem examinations, have been very small (or composed of rats or mice) and ‘dyslexics’ defined by the discredited IQ/achievement discrepancy criterion.

Two Sheffield researchers claim, on the basis of educational, intelligence and adapted clinical assessment of ‘dyslexics’ (discrepancy defined) to have demonstrated severe defects ‘... across the entire cerebellum.’ (Nicolson & Fawcett 1999 p. 166). They claim, flatly, that ‘Dyslexia is genetic in origin ...’ (ibid. p.155) and that it is ‘... characterised as problems in skills automatism’ (ibid. p. 171). Their battery of tests for screening adults for dyslexia includes a test of postural stability which consists of ‘... a calibrated push in the back.’ (Nicolson & Fawcett 1997 p. 78). They claim that ‘dyslexics’ showed ‘... severe and persistent problems’ with literacy but also with ‘... balance and motor skill’ (Nicolson & Fawcett 1999 p. 156). Many celebrated ‘dyslexics’ would dispute this finding (not least, perhaps, Jackie Stewart, three times formula one racing world champion and a crack clay pigeon shot) as many researchers have. This research spawned a ‘therapy’ to ‘cure’ ‘dyslexia’ (and more) by improving motor coordination (the DDAT treatment - Dyslexia, Dyspraxia, Attention Deficit Treatment). Many claim this therapy has proved, on dispassionate examination, to be an expensive chimera, as with so many other cures which have come and gone (Nicolson & Reynolds 2003, McPhillips 2003, Rack 2003, Rack *et al* 2007, Reynolds *et al* 2003, Reynolds & Nicolson 2007, Richards *et al* 2003, Singleton & Stuart 2003, Stein 2003, Snowling & Hulme 2003).

And, in the context of disputed findings, perhaps the infamous ‘*publication bias*’ effect ought to be mentioned. This bias refers to the very human desire to read, and publish, findings rather than non-findings. A large number of studies do not find what others have purported to; they do not support other work. In some cases these studies may be published, but often they are so boring (they may have found absolutely nothing of significance) that they are either not submitted at all or are not published. Editors of journals are human and they operate in a marketplace. They naturally tend to seek the stirringly conclusive rather than the enervatingly inconclusive. It is interesting to publish an article with an exciting new claim - for example perhaps that ‘dyslexia’ is caused by a previously unconsidered influence such as the amount of daylight we experienced in our first year of life. The subsequent research that finds that this isn’t really the case, that there is no relationship between early exposure to sunlight and later literacy ability, is dull reading by contrast. It may never see the light of day, and the original claim will stand, by default, and be referenced in other writings. It may become accepted as ‘truth’, at least for a time. (A. N. Other *et al* have shown that ...) The precise size and nature of this publication bias is not clear, of course, but it exists, may be large (perhaps especially in a field like dyslexia) and considerably affects what we consider to be ‘reality’. We might take less as read if we were able, in fact, to read more. (And a recent study claims that over 50% of studies produce findings which are later found to have been wrong! Oh well ...)

To say that this theoretical area - the neurology, or neuropathology, behind dyslexia - is unresolved is an understatement. No theory has reached anything approaching consensus and theories which have been cross-examined through research are frequently not supported, or not strongly supported. Scepticism and patience remain proper attitudes towards all this research.

Scotopic sensitivity or the Meares-Irlen syndrome:

I once met a lady who wore plain, very pale yellow spectacles when reading. She told me that she was dyslexic and was convinced she could not read any but the simplest of texts without them (they were not lenses but absolutely plain glass). With her spectacles on, she read even academic texts absolutely fluently. She had tried many different colours, all of which helped about equally, she said. She had

plumped for yellow simply because she liked yellow. Irlen lenses (which are plain but colour tinted spectacles), or coloured overlays (which are clear but colour tinted plastic sheets) sometimes, as in this case, have instant and stunning effects on the ease of reading. Sometimes the effect is small and sometimes there is no effect at all. (Irlen 1991, Tyrell *et al* 1995.) A useful, short review of research is Whiteley & Smith (2001). Some writers assert that it is 'dyslexics' who are helped by these lenses, or overlays. However, writing in the same volume of the Journal of Research in Reading as Whiteley & Smith, Wilkins *et al* (2001) report finding that around half of 'normal' schoolchildren in their three samples experienced reading as easier, and did it better, through coloured overlays; some individuals improved by over thirty per cent. They found that 'A substantial proportion of children reported symptoms of visual stress...' (ibid. p. 50) and it was particularly these children who improved most, and most reliably, when using their preferred colour overlay. Symptoms of 'visual stress' included letter movement, text blurring and uncomfortable brightness. Almost a third of those who noticed improvement were still voluntarily using their overlay at the end of the school year, eight months after being introduced to it.

The effect is most gratifying, and when it works, it really works. Nobody knows why it should. Wilkins *et al* (2001) speculate that as 'visual stress' is reportedly more common among migraine and epilepsy sufferers they may all be due to a 'hyperexcitable visual cortex'. Scotopic sensitivity syndrome (or Meares-Irlen syndrome) is a syndrome of the visual system. It is not specific to literacy though capable, apparently, of dramatically affecting it. For 'dyslexia' to have any meaning it must be a syndrome which is specific to literacy - not a syndrome relating to sight in general, for example. The sometime success of Irlen lenses or coloured overlays at alleviating reading difficulty is clearly significant, when it is, but leaves the dyslexia debate approximately where it was before they came along. (Kriss & Evans, 2005, Singleton & Trotter 2005).

Robinson and Conway (1988, unpublished) reported significant improvement in subjects using Irlen lenses in a number of word length, basic academic subjects, reading comprehension, reading accuracy, but not rate of reading. Adler and Atwood (1987) evaluated the results of Irlen lenses on 23 remedial high school students and a matched control group. Significant improvement for the experimental group was noted for time needed to locate words on a printed page, timed reading scores, length of time for sustained reading, and span of focus, as well as other perceptual tasks. Additionally, seven of the 23 experimental found employment, but none of the control group was employed by the end of the semester. In contrast, Winters (1987) was unable to find differences in his study. Winters gave 15 elementary school children four minutes to locate and circle 68 examples of the letter "b" on three pages, each page of which contained 600 random letters in 20 lines of

Figure 8.1 The 'swirl effect' – as reported in the Guardian newspaper 17th October 1995.

The 'swirl effect':

Not so many years ago the 'swirl' and 'see-saw' effects were widely reported and regarded as pathognomic for 'dyslexia'. Oddly, they are not commonly reported any more. The story went like this.

For some 'dyslexics' text appears to swirl so violently about as to be unreadable. Some report a 'see-saw' effect, where lines of text move wildly up and down such that nothing can be made of it. These appear to be genuinely experienced effects, once regarded as typical, even diagnostic, of dyslexia so let us have a look. For a swirl effect, for example, to be actual, one of only two things can be happening – either the visual system is distinguishing text from all other visual stimuli in order specifically to swirl only text around or to the unfortunate person everything swirls about. This latter is not reported and it would, of course, hardly be possible to live if it did. How can we otherwise explain the swirling of print? Could it be an *affectively* mediated effect? Is so much anxious stress associated with the intensely personal demands of print that it comes to feel as if it swirls, or jumps about, or is in some way inherently unreadable? It cannot, in an otherwise normally sighted individual, really be doing so. Is this an unconscious self-defence, transferring responsibility from the individual to his physiology? (and see Martin 1989 p. 53 reporting that when he has to play the piano in front of his teacher he sweats and freezes, and the notes seem to jump around on the page before him). Or Johnston: 'In my view the effect of anxiety in reading difficulty cannot be over-estimated.' (1985 p. 167).

The profound effect of affect (e.g. anxiety, or stress) on literacy has been described in the previous chapter. In their report on 'Dyslexia, literacy and psychological assessment' the British Psychological Society (1999) asserts that

Emotional difficulties can be associated with dyslexia. Whilst these affective responses are not the causes, but rather the consequences, of dyslexia they may contribute to, and exacerbate, learning difficulties in a complex and incremental way. (p. 45).

They produce no evidence for their assertion that such damaging negative affect is a consequence rather than the cause of the 'dyslexia'. They refer to Pumfrey and Reason (1991) who do not, in fact, make this assertion. Pumfrey and Reason produce evidence for both possibilities and are at pains to point out the equivocal nature of the evidence. They assert that 'dyslexics' usually show considerable, sometimes dramatic affective disturbance related to their learning difficulties, while concluding that whether affect is horse or cart has yet to be elucidated. Everyday observation, as well as intuition, tends, in fact, to back the horse. It has been my experience (and see chapter 7) that emotions and feelings are intimately involved in odd literacy difficulties and that they have usually been more cause than consequence of said difficulties.

The word-processor by-pass:

This phenomenon is commonly observed but never appropriately remarked. You will probably have heard or read something like this more than once, for yourself: 'I am dyslexic and for me this means I literally cannot write my own name *but I can read quite well and I am now using the word processor.*' (Herrington 1995 p.7 my emphasis). It is astonishing how often 'dyslexics' are said to enjoy a straightforwardly usual degree of success on the word processor while experiencing ongoing difficulties with a pen. However, assembling language and spellings for word processing and for handwriting are, of course, exactly the same cognitive process; the only different is the different hardware through which the act is mediated. Word processing involves precisely the same cognitive literacy domains as handwriting does; it is simply the eventual choice of expressive tool which differs. In such a case the diagnosis must be at fault - the 'dyslexic' who makes ordinarily good progress on the keyboard does so because there is no such corrosive personal history or expectation of failure associated with the keyboard as there so manifestly is with pen and paper. With luck nobody has told him that he will also be 'dyslexic' on the keyboard, so he isn't. The change of technologies has by-passed all that affective baggage. And, of course, if he is 'normal' on the keyboard, he is 'normal' full stop.

I will recount an anecdote which may be interesting in this context: I know a man who stammers. He is a highly intelligent, very pleasant and witty man, but he stammers so badly that he finds it almost impossible at times to make himself understood. It can take him a minute or two to get through a single sentence. However, this man is a very talented amateur actor; he regularly takes the leading role in the annual pantomime put on by the high quality local theatre group. He is given the lead because he is outstandingly entertaining not only when delivering his lines as the King of Llanastraw, or whatever he is this year, but also, and particularly, at the uproarious final performance, striding to the edge of the

stage and ad-libbing with the audience to stunning effect. He invariably delivers a tour de force, sometimes at length - and he never stammers at all on stage. The show can take anything up to an hour longer than usual because of his interactions with his audience, who love every bawdy minute of it. In his ridiculous role, swathed in a stupendous costume, he commands the theatre, declaiming with perfect fluency and responding devastatingly and swiftly to the heckling rising from the seats below him. In front of exactly the same people come to worship him at the famous post-performance party a few minutes later he will be stammer-bound. Does becoming a king (as he demonstrably does) by-pass whatever trauma, whatever complicated language-associated affect, so profoundly interferes with his speech when he is merely himself?

Cognitive or learning styles:

The neurological deficit explanation of 'dyslexia' is intermittently questioned quite critically. Some researchers seek to replace a medical deficit aetiological explanation with one of different, and differently appropriate, cognitive styles. They claim that we all have radically different minds, with inborn differences of behaviours and aptitudes, rather than that some of us have apparently abnormal brains. Herrington (2001), for example, reviews the field in the context of remediation of adult 'dyslexics' in ABE. She specifically rejects the 'medical disability' view in favour of a '... different thinking/learning style' (ibid. p. 13), while still considering that '... all intractable difficulties with aspects of literacy involve some elements and degree of dyslexia.' (ibid. p.17). The 'learning styles' approach asserts that different people have radically different underlying cognitive styles. They approach learning, in particular, in radically different ways. The further claim is usually made - explicitly or implicitly - that these different styles are innate, are hardwired into the brain by nature (as opposed to learned during nurture). It is important to remember that these claims remain controversial and unproven, of which more a little later.

It is often alleged, frequently without rigorous evidence and on the basis of only a case or two, that 'dyslexics' have special attributes perhaps in compensation for their special difficulties. This is a lovely, warm idea. Unfortunately it is probably only that. Herrington's review of the literature concludes that the case for 'dyslexics' being unusually blessed with particular aptitudes (such as enhanced visuo-spatial skills) to extraordinary degrees is weak. Her own analysis of 'dyslexic' ABE students' subjective reports, however, leads her to suggest that they nonetheless approach literacy from unusual perceptual angles. Some, she claims, say they experience, or need to experience, meaning in written or spoken language visually and graphically - as picture or at least metaphor - before understanding it. They report that they are obliged to 'switch modalities' to find meaning. They also report that their difficulties with literacy are specifically related to the difficulty they experience trying to grasp, or express, meaning in the inexorably linear and sequential medium of text. Memorisation is often anecdotally reported as particularly problematic for 'dyslexics'. Herrington claims 'dyslexic' ABE students often report that their efforts to memorise demand visualisation. These students report that they find it difficult to memorise ideas linguistically but can memorise them as visual images. Even more controversially, perhaps, she asserts that 'dyslexics' may experience time in unusual ways, sometimes as 'a total blank' or 'as if it is a separate dimension' (ibid. p. 22).

The thesis that some people have radically different cognitive styles from the majority of the population, and that these are innate, remains highly speculative (Coffield *et al* 2004 is a useful and appropriately critical review). Many schemes for assessing learning style are aggressively marketed and much money is made from this presently fashionable theory. Sadly, the science is correspondingly selectively reported and truth obscured. The crucial educational question, seldom properly addressed, is to what extent these different learning styles really exist and genuinely relate to underlying and innate cognitive styles, and to what extent they may have been adopted, induced, learned or taught.

To what degree is a 'learning style' natural and to what degree may it be learned? This difficult and inconvenient, but educationally fundamental, question is not well understood and is generally, but absolutely improperly, ignored. Many students indeed appear to have strong preferences for learning in particular ways - for example many students seek to master literacy purely phonically, regarding literacy solely as a matter of 'sounding out'. A learning style advocate will conclude that such a student has an innate bias towards learning through phonic attack; that they experience and learn best through sounds. A sceptic will want to know whether this bias is really innate or whether it has been learned (Johnson 1985). May the student have learned that phonic attack is the only way to do it? Have

repeated patterns of success and failure, and insistent teaching approaches, taught him his 'style'? Indeed, Johnston & Allington (1996 p. 999) quote Barr as saying that

... whereas more able readers show little trace of instructional method past the second grade, less able readers appear to learn narrowly what they are taught; indeed they tend to show quite marked effects of their instructional focus.

A fundamentally important educational dilemma immediately arises from all this. If the learning style a student appears to exhibit is genuinely innate, that is to say that if his brain really is hardwired to operate in that manner, then his learning had best be done using this style. However, if the student has merely learned to use this style then he is missing out on, and must urgently be taught, different styles. Instead of reinforcing a single, learned cognitive style and thereby restricting our student's repertoire we should encourage him to learn and use a variety of approaches, as we do ourselves. If a style really is innate, then we should teach to it. If, however, it is a learned style we should do precisely the opposite.

It has been my experience that students invariably (and I mean invariably) benefit from deliberate exposure to learning approaches at variance to their apparent 'learning style' - by learning, and learning to deploy, a more varied range of approaches to their literacy (see notes to chapters four, five and seven). The student, for example, who employs only phonological attack, the student who doggedly 'sounds out', needs practice in visual approaches. It has been my experience that absolutely all such students do indeed learn, willingly deploy and benefit from, such different 'styles'; an observation which leads me to conclude that their apparent 'learning style' was itself learned rather than innate. Fluent literates in the real world do not rely on a single approach to literacy, a single 'literate style', why should a student?

So, it remains unclear whether there really are significant, and significantly different, learners who consistently rely on radically different learning styles based on innate cognitive styles. At an immediate, educationally practical level the recognition and exploitation, or management, of various cognitive and learning styles may (or, as we have just seen, may not) be good practice. It remains questionable whether variation in cognitive style will ever offer a satisfactory alternative explanation for 'dyslexia' or much in the way of genuine pedagogical insight.

Is there any alternative to 'dyslexia'?

The question everyone asks when faced with scepticism about dyslexia is "Well, what is it, then?" To which the opening reply must be that there is no single "it" for it to be. There are so many other, more prosaic explanations for peculiar difficulty with literacy, each more likely than a highly selective miswiring of the brain. (And 'Occam's razor' springs to mind.) A professor at London University's Royal Veterinary College forty years ago used to cry, in exasperation, 'Common things are common!' - and so they are. He meant to drive home that a clinician has no right to diagnose the rare and esoteric until the everyday has been considered and eliminated. What is sauce for the clinician is sauce for the educationalist (and everyone else too). I am about to run through a mass of quotes, each of which, I hope, illuminates a particular reason for literacy failure. This is the stuff of everyday; much less scientifically exciting (and more probable) than a 'dys'. For exactly that reason, these, or other, explanations are much more legitimate early proposals in our quest for a 'diagnosis'. This list of possible causes is, incidentally, certainly not definitive, you will have many revealing quotes and further ideas of your own.

'Reading and writing are not just cognitive activities - feelings run through them.' (Barton 1994 p. 48) & 'Her reading problem was mostly the fear that she really couldn't learn to read and the shame she would feel if this proved to be so.' (Holt 1982 p. 37) & 'While I was at school I was educated to feel shame and worthlessness, to feel doubt in my own abilities and self-hatred. I was educated to feel small and worthless.' (Miles & Varma 1995 p. 65) & 'It is their intelligent response to a perceived problem that appears to prevent them from developing normal reading behaviours.' (Johnston 1985 p.165) & 'Bog off! I ain't reading that babby stuff!' (Martin 1989 p. 1) & 'The more I thought I couldn't do it the worse I went!' & 'You couldn't do it anyway so you didn't try.' (Newsham 1988 pp. 15 & 16) & [you learn to] '... do something else, that's to deceive ... mainly yourself.' (Mellor 1988 p. 42) & [the poor readers] '... got beat more than anyone ... they got more stupid by every day.' (in Goleman *et al*

1984 p. 46) & 'there was a terror campaign waged against me to get me to spell properly.' (Miles & Varma 1995 p. 65) & [all quotes from a variety of ABE students] 'The teacher was only interested in those who were bright.' (this one, sadly, comes up all the time) but also 'There was over forty children in her class - she had no chance.' & 'I never had stories read to me.' & 'There wasn't a book in the house.' & 'Where we lived, in the country, there was no kids - I just mucked about in the mud.' and my favourite, from an African-American brought up around Alabama, 'I went to school on the days it rained.' And there are, of course, many more where they came from - complexity & wisdom enough without recourse to neuropathology. (and see Arnold 1994, Stanovich 1986, Tizard 1993, Tizard & Hughes 1984, Wixson & Lipson 1996.)

The Matthew effect:

In a long and rewarding article Keith Stanovich (1986) writes that '... the cognitive consequences of the acquisition of literacy may be profound' and that '... the knowledge base of less skilled readers may be less developed because of their lack of reading practice' (both quotes from p. 374). Learning (and using) literacy may make for greater cognitive ability and increase our ability to catch, and hold, 'information' including literacy itself. Using literacy reinforces literacy. On page 380 Stanovich also says that '... a strong bootstrapping mechanism that causes major individual differences in the development of reading skill is the volume of reading experience'. Those who are good at something will do more of it; those who do more of something get better at it and do more of it... Especially is this so with literacy. The good reader in, say, middle school, reads several millions of words a year, whereas the poor reader reads only a few thousand (and probably hates every one). We all know what practice makes. This is the 'Matthew effect', an extremely important, but often overlooked, factor in literacy acquisition and maintenance, as well, perhaps, as in much more general cognition.

Stanovich attributes the invention of the term 'Matthew effect' to a science teacher called Merton, who took the name from the gospel according to St. Matthew XXV: 29, 'For unto every one that hath shall be given, and he shall have abundance; but from him that hath not shall be taken away even that which he hath'. In other words, and this applies to literacy in spades, the more you do the better you get and the more you can, and probably will, do. The rich get richer while the poor get poorer. The simple fact that less literate people read a great deal less than more literate people makes it more difficult for them to progress. It may even skew their cognition in ways which may make it more difficult again. It will lower their measured IQ. The practice of literacy delivers better literacy and easier literacy which, in turn, delivers more practice – how could it be otherwise?

We do occasionally see cases of apparently odd difficulty in acquiring and using literacy. On any but the most blinkered, partial or superficial view it is obvious that there must be, and are, many different reasons behind these cases. There is no space here to do more than hint at a few of these reasons and indeed there has been scandalously little research done in the field of non-neurologically induced literacy difficulties. We can all agree that every individual with literacy difficulties will have a particular history in respect of literacy and the learning thereof. However, it has been my experience that most of these histories have many things in common. They often include some or all of the following (which is certainly not intended to be an exhaustive list):

- Relatively little (sometimes no) literacy activity in the home, and/or relatively little, and relatively undemanding, linguistic activity there (e.g.. Hart & Risley 1995). This means that he (our student) will reach the school world with more ground to make up before he slots in with the rules of the game there and the rather particular linguistic skills he suddenly needs.

- Very early failure in school. This may be due to linguistic unpreparedness, as mentioned above, or that all- important spring from the starting blocks may be slower for more interpersonal, familial, environmental or cultural reasons, occurring within the school or, more probably, outside it. This early failure will be officially noticed, after a while, although our lad will already have felt its icy draught and the first twinges of dread.

- Both he and his helpers (or, as they may appear to him, his inquisitors) may find his failure bewildering, intimidating and frustrating. Everyone's feelings may become very intense, if largely unexpressed. The little victim may exaggerate parental and teachers' emotions and opinions - he will certainly be acutely aware of them. He is intensely aware of the importance of his failure and

understands that it says large things about him. He internalises the general anxiety and may translate it into opprobrium.

- Either the educational system begins to sideline him, claiming him as uneducable and allocating him his place at that table at the back, or remedial action begins. This latter may be at least somewhat inappropriate and only intermittently successful. Ground continues to be lost, at any rate. Literacy is experienced as impossibly difficult, incomprehensible, intimidating and humiliating.

- Confidence in literacy, and ability, fades. Literacy becomes an unpredictable menace, a source of pain and dread. There is no pleasure to be had from it. Strategic straws which occasionally deliver the right answer are grasped and doggedly adopted, which often means that ridiculous things are written or read. Strategies which once succeeded are hugged and deployed stubbornly in every circumstance. Their occasional success reinforces their clutch but their intermittent failure entrenches incomprehension and anxiety. He becomes (in literacy terms at least) highly risk-averse, further draining motivation, engagement and the ability to learn or perform.

- At a later stage of his school career, in secondary school, he may be subject to further remediation, to 'special needs' provision. He feels (probably correctly) that it is all too late. Since he is 'special', 'special' methods are used. These may inculcate rigid and restrictive approaches to literacy, and inappropriate strategies. They may seem to our student to be odd, simplistic and repetitive, even infantile, further depressing his confidence and motivation. They seem to fit with the opinion he feels everyone, including all his most 'significant others', must have of his abilities. He feels (and is) labelled, publicly and privately. He resigns himself to being 'thick' and departs from education as soon as he is able. He will not lightly return.

Is dyslexia benign?

I have recently been sent a mailing among which was an advert for an adult dyslexia organisation. This took the form of a bookmark with their website address on it and a cartoon at one end. This depicted a tiny man, the corners of his mouth turned down in utter misery; his eyes wide open in sorry bewilderment, his fingers hopelessly clutching the window ledge in front of him. The window he peered so desolately out of was that of a prison cell. The bars of the window were made of oversized pencils, tall and bulky, completely dwarfing the demoralised, defeated, and presumably dyslexic prisoner. Escape looked remote and improbable - on the evidence of the cartoon impossible. The message was clear. This is how it is to be dyslexic! This is industrial strength learned helplessness, being powerfully transmitted.

In chapters six and seven we looked into affect. We saw how important attributions can be and how they can contaminate the unconscious; that maladaptive attributions can induce learned helplessness and that they can do this in teacher or student or both. In educational contexts a maladaptive attribution is likely to be the attribution of failure (or difficulty) to innate and irremediable causes, to something we cannot do much, or anything, about. When we diagnose 'dyslexia' we are attributing failure to a deficit, to an unfortunate kink in the brain. Dyslexia is innate (it's the victim's problem, it's their hardware that's at fault). Dyslexia is also mysterious and irremediable (we have no idea how, if at all, we could repair this hardware; we're stuck with the fault).

Attributing literacy difficulty to such a debilitating cause disempowers, and learned helplessness is exactly such disempowerment. Occasionally it is consciously apparent as in 'I was very unhappy. I was told I'd never be able to read or write - I was told this by an educational psychologist.' (Open learning project p. 37). Or as one unfortunate 'dyslexic' remarked '... our minds are wired differently ... the result is we've ended up with muddle in our minds.' (Miles & Varma 1994 p. 58.) Sometimes helplessness is also carefully and consciously transmitted to the hapless student as in a widely distributed book purporting to enable 'tutors in adult, further and higher education' to diagnose dyslexia:

Dyslexia is a disability or specific learning difficulty which needs to be identified and clarified with the student. This is not because of some desire to label students but because students need to understand that their difficulties will not go away with tuition, practice, hard work etc. (Klein 2003 p. 82.)

A student is quoted in another book widely disseminated among teachers of literacy to adults (Lee 2002 p.22). This student, Clive, writes a poem called 'Dyslexia' and it contains these lines:

Only myself to blame
A life sentence
My heart sobs at the news
I am a prisoner for a crime I have not committed
Will I ever be a free man?

It seems likely that the unfortunate Clive has already concluded that the answer is probably 'No'. Indeed, after being told that he has a neurological deficit, a permanent and irremediable disability, how could it be otherwise?

As A. A. Gill says, in the Times of August 12th 1998,

'Dyslexia now has a cachet. It has become fashionable. But there is a real danger here. By pinning this label to all their children parents are giving them a ready made excuse to fail.'

Is a diagnosis of dyslexia, then, always and necessarily, a neutral or benign thing? Does it not also have a subtle but powerfully malign effect all round? Learned helplessness, or disempowerment, associated with maladaptive attributions, among ABE students is well documented (Charnley & Jones 1981, Du Vivier 1992, Levine 1986, Mace 1979, Wallis 1995). Learned helplessness among ABE tutors faced with 'dyslexic' students has also been documented (Kerr 1999 & 2001(b) and see notes to this chapter). Given the clear maladaptive attribution that a diagnosis of an innate and irremediable neurological handicap, or disability, inevitably is how could it be otherwise? (Bar-Tal 1984, Chan 1994 & 1996, Fang 1996, Johnston 1985, Muthukrishna & Borkowski 1995, Peterson *et al* 1993, Stanovich 1986.) Maladaptive attributions engender learned helplessness. Dyslexia, whatever else it may or may not be, is a maladaptive attribution *par excellence*. It will, inevitably, engender learned helplessness. How could it be otherwise?

And it is precisely the inevitability of learned helplessness that renders 'dyslexia' so dangerous; so damaging to its victims. To reiterate from three important sources: '... teachers who believe that ... lack of ability is a stable state will produce a debilitating environment' (Fang 1995 p. 51). 'Dyslexia' is, by definition, a stable state. 'The attribution of failure to a cause for which there is little hope of a cure is profoundly unmotivating' (Johnston 1985 p. 170). There is, by definition, no hope of a 'cure' for 'dyslexia'. We may alleviate symptoms but we cannot locate, or alter, their neurological cause. 'The belief that most learning problems can be blamed upon the student militates strongly against the student's best interests' (Westwood 1995 p. 21). 'Dyslexia' is, by definition, absolutely the student's problem. It is due to a mysterious deficit which belongs entirely to the student. To put all this into plain language, to call a spade a spade, we would have to say that the 'dyslexic' student's difficulty with literacy is solely due to there being something wrong with his own brain. We cannot agree precisely what is wrong and we cannot fix it. The affliction is permanent. We know that this information will deflate and demotivate, but there it is.

Adding to their sense of difference, doom and deficit, many 'dyslexics' are removed from the mainstream for special treatment. Having 'diagnosed' this impenetrable deficit, having marked these people out as radically different in some as yet mysterious cognitive way, we do not, of course, expect our tried and tested teaching and learning methods to work. If we claim that these people are neurologically 'special' it is plain they will need special handling. Methods laboriously refined over the years and successfully used in the mainstream, on which we have consensus and which we clearly understand, will not do. In the words of Jenny Lee 'Very few basic skills courses are appropriate for dyslexic students' (2002 p. 6).

'Dyslexic' teaching involves a highly particular attitude and methodology. There is a distinctive flavour to the methods routinely used with 'dyslexics'. Students are encouraged to remember their special difference at all times. They are told they can learn material if it is presented in a highly structured manner. Their learning, they are led to believe, demands far more repetition than 'normal' people's would (dyslexia remedial schemes overtly demand massive over-learning).

Remedial approaches also commonly entail thinking overtly about the formal detail of language and literacy – the ‘rules’ - the ‘dyslexic’ mind not being trusted to learn directly, unconsciously, as yours and mine did (see notes to chapter 6). Strange things like vowel digraphs, consonant blends, even heterophonic homographs and heterographic homophones, things the rest of us learned (if at all) only after the event, only once we were already fluent, are deliberately foregrounded. Everything is drenched in esoteric terminology, and obscure rules; learning becomes technical and highbrow, compellingly redolent of school and its least comprehensible lessons. Literacy, especially spelling, is approached through phonics and as a rule-based activity - ‘G usually says /j/ when the next letter is E, I or Y.’ (Hornby & Shears 1980) or ‘Words of one syllable ending in F, L or S double that letter after a short vowel.’ (Pollock 1980) or ‘In one syllable words with a short vowel sound we must double the last consonant before adding an ending beginning with a vowel.’ (Allan 1977) or ‘We need to keep the first vowel short when we add *ing*, so we must prevent double vowel power by adding an extra consonant wall (Lee 2002). Grammar, and its ‘rules’, are explicitly taught and are similarly jargon-dense. There is huge demand to memorise the inherently unattractive and unmemorable. Nothing can ever be done without strenuous, and conscious, thought. Accuracy in detail is particularly demanded and stress is considerable as a result. Learning becomes a grinding, drill-based, rule-based affair. There is small room for creativity and meaning. Any literacy skill being learned becomes itself the goal of the learning; the purpose of literacy becomes deployment of its skills rather than the realisation of meaning or art. Literacy becomes a matter of encoding or decoding. Tiny details are endlessly rehearsed. Literacy is ingrained as painful quandary rather than as liberating tool.

Tuition designed for ‘dyslexics’ is usually aggressively (and overtly) prescriptive, technically complex, busy, stern and inquisitorial. It demands constant, fraught intellectualisation. Believers buy into this; sceptics blench. Control is entirely in the hands of ‘experts’; the student is absolutely disempowered. There is an explicit assumption that the mountain is peculiarly high and the road peculiarly rocky. They will be.

A diagnosis of dyslexia may thus function at several different levels and as several different things. On the political level it may be power; once the diagnosis is given there may be privileged access to funding and educational doors may be forced open. On the personal level it may be an alibi explanation, sparing blushes and providing a socially acceptable protection against disgrace and shame. On quite another level, though, it will act as a potent, but unremarked, handicap in the learning of literacy, inducing and entrenching learned helplessness all round. And developmental dyslexia remains controversial and may yet prove to be non-existent. It may even be all four of these different things at one and the same time.

Dyslexia is politically very powerful today. ‘... the media have accepted (why I wonder?) that the case is proven.’ (Martin 1989 p. 19.) The teacher of adult literacy works in an environment where public, management and student all accept the widespread reality of dyslexia. The teacher is obliged (by law, in the UK) to appear to accept it, and to act upon a ‘diagnosis’, even though many find it unconvincing. And dyslexia seems so emollient. ‘Anything so convenient must be right.’ (Galbraith 1962) Dyslexia is nothing if not convenient; it blames the victim, which is such a comfort to everyone else.

‘The most important purpose of education may be the inculcation ... of a deep, even raucous, scepticism.’ (Galbraith 1962). In part because it is so suspiciously convenient, but also because it may be a subtly but profoundly damaging diagnosis of a condition which may itself be illusory, developmental dyslexia surely merits Galbraith’s ‘deep, even raucous, scepticism.’

What should we do?

Notwithstanding all the appropriately sceptical above, of course, literacy teachers are commonly faced with students with officially sanctioned diagnoses of dyslexia, sometimes of severe dyslexia. What do you do, if you are unpersuaded? You have three choices: you may challenge the diagnosis, reinforce it or ignore it. What do I do? I am thoroughly unpersuaded, as you have seen, but even I cannot say with absolute certainty that dyslexia does not exist. We all remain too ignorant as yet for dogmatism. For this reason, and also because the diagnosis (at one level and at this moment) may be helpful to the student (it’s certainly better than being regarded as unintelligent), I do not recommend a direct challenge to the diagnosis. Neither, though, do I recommend it be accepted – this will reinforce the disability fantasy (which I believe dyslexia to be) and will, to the exact degree that this fantasy is

accepted, induce learned helplessness. I am therefore driven to the third way; the 'Mmmm...' approach. When told a student is 'dyslexic' I say 'Mmmm...' and then teach as if the diagnosis had never been made; I treat the student as completely 'normal'. I dismiss dyslexia from my own mind and soon the student will feel it fade from his too. Dyslexia, it has been my experience, eventually withers into forgotten insignificance. It is at this point that educational progress can really begin.

A conclusion, of sorts:

If we could run time forward, what might we see? Cruising the electronic archives in, say, the year 2050, we may read that a gruelling educational controversy rumbled on for many years each side of the millennium in respect of what we called 'dyslexia' at that time. Thankfully, we will note that we have been able to resolve this issue or, as it turned out, these issues. We will see that a lot of what seemed to be evidence for one thing was really evidence for many others. We will read that the hoary and mystifying syndrome we once called 'dyslexia' turned out to be a mix of various social influences, whose reality we were reluctant to admit, and a few neurological deficits, none of them specifically related to literacy. We will see that part of the problem was that literacy was regarded as so important that passionate and sometimes inappropriate pressure was unwittingly applied to early learners in the days before the real nature and purpose of education was fully understood. We will note that many people, in those days, developed affective defences against, and thereby sometimes extraordinary responses to, literacy, often to the process of education itself. It will pain us to read that many people were considerably intellectually reduced as a result. As well as this, though, we will read that we understand better those few neurological syndromes, which, although they are not specific to it, may secondarily affect literacy.

'Dyslexia' will have joined the many psychological ideas which seem faintly embarrassing now, but which once seemed so straightforwardly commonsensical. It will lie, mouldy and neglected in the attic of psychological history, alongside the homunculus, the phrenology head, the ink blot and behaviourism. If we should ever catch sight of it there, among the invigorating but wrong ideas of previous times, we will recognise it as just another casualty of dogged scepticism and the scientific method.