
1. History

In 1676, Johannes Schmidt (cited in Bernard, 1885), writing in Latin, described the case of a patient who lost the ability to read as a consequence of a severe stroke. Schmidt feared that his patient, Nicolas Cambier, would die; however, Cambier gradually regained consciousness. According to Bernard, Schmidt observed language problems in his patient, along with partial paralysis affecting only the right side of his body. Initially, Cambier spoke only in "murmurings" and could not express his thoughts. He had word-substitution problems and experienced great difficulty expressing his wishes. Soon after the initial stroke, Cambier began to have severe seizures. However, much to the surprise of his family and doctor, he gradually regained his health and was left with only one problem: he could not read any letters or words. Cambier's symptoms were peculiar in that he had not lost the ability to write or spell. He simply could not identify any of the letters or words he wrote.

1877 Adolph Kussmaul (German) noted that the ability to read might be lost although sight, intellect and speech were unaffected. He invented the term “word blind”, and thought of reading problems as a kind of receptive aphasia. As further cases were reported, two types of word blindness were defined: a. patients could not read but they could write. b. they could neither read nor write. Post mortem examinations of the brains of these patients revealed lesions, softenings or haemorrhages in the occipito parietal region of the left cerebral hemisphere.

1884 and 1887, German ophthalmologist Rudolph Berlin described a group of patients who had normal visual acuity but great difficulty in reading because of cerebral disease as “dyslexic”. At autopsy, he discovered an area of degeneration in the parietal area of the brains of these patients. Berlin saw dyslexia as a member of the family of aphasias.
1895 James Hinshelwood (ophthalmologist, Glasgow), published in *The Lancet* on “Word Blindness and Visual Memory”. In 1917 he published a most influential monograph *Congenital Word Blindness*. Hinshelwood asserted that the primary disability was in visual memory for words and letters, and described symptoms including letter reversals, and difficulties with spelling and reading comprehension.

The 1895 publication prompted Dr Pringle Morgan, a school doctor and GP to describe the case of Percy, a 14 y.o. schoolboy. bright, healthy, and of good family, good at arithmetic, but apparently unable to read and write. Orally, he could more than hold his own in class, but when asked to write became confused. “Carefully winding the string round the peg” became “Calfly winder the strung rond the pag.” Morgan concluded that this was a case of word blindness. (1896).

1887 a school medical officer in Bradford, Dr James Kerr drew attention to several healthy children who presented a picture similar to Percy. Hinshelwood continued to collect cases of what had now come to be called “congenital word blindness”. 1900 and 1917 respectively, he published papers *Letter-Word- and mind-blindness*, and *Congenital Word Blindness*. He (& Morgan) attributed the condition to lack of development of the left angular gyrus. Further cases were reported and in 1907. S. Stephenson reported six cases of word blindness affecting 3 generations of one family, and suggested that a genetic factor was involved. In 1910, J.H. Fisher suggested that the condition might be due to brain damage, and suggested birth injury might be a predisposing factor. Hinshelwood also reported several cases in one family.

Even in Hinshelwood’s day, there was confusion of terminology and definition. He noted the injustice of confusing the “word-blind” with the “mentally defective”. “It is a great injustice to the children affected with the pure type of congenital word blindness, a strictly local affection, to be placed in the same category as others suffering from generalised cerebral defects, as the former can be successfully dealt with, while the latter are practically irremediable.” (pp.93-94)

Samuel T Orton was one of the pioneers of neurological research into word blindness and published his findings in 1925, 1928 and 1937. He was interested in the relationship of left-handedness to brain laterality, and particularly mixed-handedness, which he called “motor intergrading”. He noted frequent reversals, motor clumsiness, some difficulty in understanding spoken language in the absence of deafness, and some difficulty in using language. Because the tendency to reverse and transpose letters was so marked, he invented the term “strephosymbolia” (twisted symbols). He believed the condition was caused by the failure to establish hemispheric dominance in the brain.

Orton was one of the most influential neurologists in this area, but his concepts about brain function, while insightful, were probably too simple to be correct in the light of the last 20 years of brain research. Orton later worked with psychologist and educator Anna Gillingham to develop an educational intervention that pioneered the use of simultaneous multisensory instruction.
Both Hinshelwood and Orton advocated educational methods for dealing with dyslexia (or developmental alexia as Orton would have called it). Both advocated multisensory methods for retraining. Orton stated that the disorder “is explainable as a variant in the hemispheres rather than as a pathological condition and, as a corollary of the latter view, that proper methods of retraining, if started early enough may be expected to overcome the difficulty.” (Archives of Neurology and Psychiatry, 1925 p.602.).

A lot of later work has built on the work of Orton. Norman Geschwind in 1982 wrote, “in his original observations, [Orton] pointed out the frequency of delay in the acquisition of speech in dyslexic children, thus laying the groundwork for the important concept that dyslexia appears on a foundation of delay in the development of the entire system devoted to language.” (Why Orton was right. Annals of Dyslexia 32, 13-30.) Orton also noted the high frequency of concomitant left-handedness, normal visual perception, clumsiness, stuttering, history of delay in the acquisition and use of spoken language, and that dyslexia tends to be familial. Orton pointed out (and Geschwind concurred) that these factors are probably not causal, but concomitant, and may in fact be the results of processes that underlie dyslexia.


2. Concept of SLD

3. Definitions

1. 1968 Word Federation of Neurologists Research Group on Dyslexia and World Illiteracy (Dallas, Tx) Specific Developmental Dyslexia: A disorder manifested by difficulty in learning to read despite conventional instruction, adequate intelligence, and socio-cultural opportunity. It is dependent upon fundamental cognitive disabilities which are frequently of constitutional origin.
Others have argued that dyslexia falls into two or three categories, viz, with and without brain damage. Rabinovich in 1968 argued that there were people with disorders in ability to deal with letters and words as symbols, apparently reflecting disturbed neural organisation. There were also those with clear-cut neurological deficits and those with symptoms similar to those with clear-cut neurological deficits, thus presupposing eg prenatal toxicity, birth trauma or anoxia, encephalitic or head injury. The third group would be those who had reading difficulties consequent upon environmental deprivation. Unfortunately, neurological dysfunction is not always easy to determine, and its determination often depends upon the clinical judgment of the diagnostician. TTS Ingram in 1964 suggested that there are many cases where evidence of actual brain damage is not available, but patients have syndromes of “minimal brain dysfunction”. McDonald Critchley (1964) advised against diagnosing brain damage from such syndromes, because such diagnosis ignores the factor of inheritance, and the fact that frequently it is impossible to locate any actual brain damage in such patients. It is still occasionally done, although the preferred diagnosis these days would be ADHD. In addition, “the plasticity of the nervous system in the young might be expected to compensate for the effect of any circumscribed lesion of very early appearance.” (Developmental Dyslexia, 2nd edition 1970).

2. Joint Committee on Learning Disabilities (1981)

Learning disabilities is a generic term that refers to a heterogeneous group of disorders manifested by significant difficulties in the acquisition and use of listening, speaking, reading, writing, reasoning, or mathematical abilities.

These disorders are intrinsic to the individual, presumed to be due to central nervous system dysfunction, and may occur across the lifespan.

Problems in self-regulatory behaviours, social perception and social interaction may exist with learning disabilities, but do not themselves constitute a learning disability.

Although learning disabilities may occur concomitantly with other handicapping conditions (for example, sensory impairment, mental retardation, serious emotional disturbance) or with extrinsic influences (such as cultural differences, insufficient or inappropriate instruction), they are not the result of those conditions or influences.

(National Joint Committee on Learning Disabilities, 1981.)

3. Federal Register (USA 1977) (PL 94-142)

...children who have a disorder in one or more of the basic psychological processes involved in understanding or in using language, spoken or written, which disorder may manifest itself in imperfect ability to listen, think, speak, read, write, spell, or do mathematical calculations. Such disorders include conditions such as perceptual handicaps, brain injury, minimal brain dysfunction, dyslexia, and developmental aphasia. Such term does not include children who have learning problems which are primarily the result of visual, hearing, or motor handicaps, of mental retardation, of emotional disturbance, or environmental, cultural or economic disadvantage.
4. Who defines LD?

   **i. UK:** Specific learning difficulties (dyslexia). This is defined to include mathematics.

   **ii. USA:** 48 states define LD. 2 serve LD students through non categorical programs. 22 states and DC use the Federal Register definition as it is. 26 states have added to or amended the definition. Overall, however, these definitions all include:
   - failure to achieve
   - psychological process
   - exclusionary
   - significant discrepancy
   - etiological* (44 states) (Encyc. Spec. Ed. 926)

(* history of brain injury; motor coordination problems; slow speech/language development; immature social and emotional development; hyperactivity/hypactivity; frequent illness/absenteeism; surgery at early age; infant problems in feeding, sleeping, temper, crying, prenatal/natal difficulties, low birth weight; premature birth.)

   **iii. Australia (NSW)**

Education Act 1990 as amended:

20 Assistance to government school children with special needs

(1) The Minister may provide or arrange special or additional assistance for government school children with special needs, such as:

(a) children with disabilities, or

(a¹) children with significant learning difficulties,

(2) Any such special or additional assistance may include the provision of financial assistance, facilities, courses of study, staff, staff training or distance education.

(4) For the purposes of subsection (1) (a¹), a child has a significant learning difficulty if a qualified teacher or other qualified education professional is of the opinion that the child is not, regardless of the cause, performing in the basic educational areas of reading, writing, spelling and mathematics in accordance with the child’s peer age group and stage of learning.

5. Defining the Deficit: problems with discrepancy definitions.

All the above definitions seem to imply a discrepancy between the person’s ability and their achievement which cannot readily be explained in terms of environmental, social or physical factors. Most instruments commonly used in defining a person as LD yield disproportionate numbers of minority group placements in the category. (e.g. RA 2 yrs behind CA; RA 2 years behind MA etc). Current trends tend to use more informal assessments, curriculum-based assessments, and even computer-based assessments (as low as 5 yrs old in the case of the Cognitive
Profiling System (UK 1994). But these lack standardisation in most cases, making justification for classification difficult.

a. Reasons for difficulties in defining
   i. LD = any kind of learning problem.
   ii. no single characteristic or syndrome is typical of all LDs.
   iii. each child has their own learning style/pattern. Their LD behavioural symptoms will therefore depend on how they happen to attempt to deal with the problem as they experience it.
   iv. some observable symptoms may be the result of visual or hearing impairments, intellectual disability, emotional disturbances, social maladjustment, health problems, cultural differences, family problems, or poor instruction.
   v. multiple handicaps may obscure a LD.

b. Problems with discrepancy definitions (See eg. Stanovich, K.E., 1991 Discrepancy definitions of reading disability: Has intelligence led us astray? RRQ 26,1 7-29)

Points out that there is now evidence that the acquisition of literacy fosters the development of the very cognitive skills that are required by aptitude measures. This weakens the distinction between aptitude and achievement. He suggests the use of a more educationally relevant measure such as listening comprehension. P.G. Aaron suggested this two years before (Dyslexia and Hyperlexia). There are, however, problems with this measure of discrepancy, too, particularly as the person gets older.

Overwhelming evidence now implicates phonological processing in the dyslexic pattern of performance. But to focus solely on that, as Aaron suggests we might, is to ignore the possibility of orthographic/visual processing deficits. Several investigations have suggested that phonological processing is necessary, but not sufficient for attaining the advanced level of word recognition needed for efficient reading. (See Stanovich’s article).

Note that the deficits for the garden variety poor reader extend into several cognitive domains (e.g. vocabulary, language development, which may be causally related to deficits in comprehension) whereas in the dyslexic they are domain-specific (phonological processing).
Stanovich concludes that the alternatives are either to work on developing “a principled method for measuring discrepancy from listening comprehension or some other verbal aptitude indicator”, or to abandon discrepancy completely and resort to measuring decoding skill alone.

6. Symptomatic definitions:  Bangor

- Left-right confusions
- difficulty with polysyllables
- difficulty with subtraction
- difficulty with tables
- difficulty remembering months forwards and backwards
- difficulty remembering digits forwards and backwards
  - b/d confusions
  - familial incidence

7. Typical difficulties

Kinds of difficulty

- Decoding (critical)
  - Phonemic awareness
- Automatic word recognition
- Fluency (rapid naming of visually presented items)

8. Invariant Symptoms

Invariant symptoms of dyslexia

- Slow reading
- Errors in oral reading (especially function words)
- Spelling errors
- Incorrect use of suffixes (grammar)
- Excessive reliance on context

9. Variant Symptoms

Variant symptoms of Dyslexia

- Reversals
- Neurological soft signs

(P.G. Aaron, 1989, 1994)

10. Subtypes of Dyslexia (LD)

AUDITORY AND VISUAL SUBTYPES IN READING: Research Evidence.

1960-1965 Joseph Wepman. 1964 De Quiros. 1965 Myklebust (all used linguistic tests).

Evidence from Neurology
Marcel Kinsbourne & E.K. Warrington 1963 British Journal of Psychiatry
investigated WISC discrepancy of 20+
Performance IQ higher: impaired receptive/expressive language, phonetically inappropriate spelling errors.

Verbal IQ higher: impaired finger differentiation, maths, L-R sequencing, visuo-motor instructional ability. Letter confusions, reversals.


Dysphonetic dyslexics: auditory/linguistic difficulty.

tendency to bypass phonology & read gestalts.

Dyseidetic dyslexic: mixes up similar shapes.

Mixed: usually severely handicapped.

Mattis, S. French, J. Rapin, E. Developmental Medicine and Child Neurology, 1975: 3 patterns:

Primary Language disorder = auditory/linguistic (28%)

Visual-spatial-perceptual. (14%).

Articulatory/graphomotor/dyscoordination. (48%) (replicated in 1978)

Pirozzolo, Francis J. (Chief, Texas medical Centre Neuropsychology service)

confirms these groupings:

a. auditory-linguistic

poor verbal fluency, anemia, agrammatism.

b. poor visual perception, impaired spatial and ocular-motor function.

Fried and others (1981) used event-related potentials to see whether there were differences in brain function between identified dyslexics and normals. There were. Also, processing auditory information was different in dyslexics, but dyseidetic dyslexics were closer to normals than dysphonetic dyslexics.

"The dysphonetics may not have a fully developed capacity to process auditory information in the normal manner."

Computerised Brain Tomography has shown differences in brain structures of groups of dyslexics (1978).

Brain Electrical Activity Mapping. Frank Duffy and others (1980) were attempting to predict rather than to do post-hoc classifications. They discovered differences in speech-linked areas of the brain with possible underactivation of the frontal systems in dyslexics. Later studies have identified more specific posterior regions.
"The visual cortex and thus the visual system would be a logical candidate for investigation with respect to the nature and perhaps etiology of dyslexia."

Pirozzolo and Rayner (1979) write of auditory dyslexics,
"they suffer from pathologically slow transmission of linguistic information within the left hemisphere, and thus auditory-linguistic dyslexia can be said to resemble an aphasic disorder."

Shafrir & Siegel (1994) JLD:
Reading disability, Arithmetic Disability and Reading & Arithmetic disability.

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<tr>
<th>Types of Learning Disabilities</th>
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<td>(Shafrir,U. &amp; Siegel, L.S. (1994)J.L.D. 27(2) )</td>
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<tr>
<td><strong>Reading Disability</strong></td>
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<tr>
<td>Reading, spelling, vocabulary, phonological processing, short term memory</td>
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<tr>
<td><strong>Arithmetic Disability</strong></td>
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<tr>
<td>Visual-spatial skills, Memory for numbers</td>
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<tr>
<td><strong>Reading &amp; Arithmetic Disability</strong></td>
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The double-deficit hypothesis is a prominent current theoretical model of developmental dyslexia, which predicts three subtypes: phonological, rate, and phonological-rate impaired readers.

Subtyping of children with developmental dyslexia via bootstrap aggregated clustering and the gap statistic: comparison with the double-deficit hypothesis. “Among the participants with developmental dyslexia, there was evidence of a phonological-deficit cluster, a rapid-naming cluster, and a cluster showing both depressed phonological processing and rapid naming.”

13. Incidence:
US Dept Edn 1992: 50% all students in special education in public schools have LD.
National Institutes of Health: 75-80% of special education students identified with LD have their basic deficits in language and reading
National Centre for State Courts and Educational Testing Service 1977: 50% all juvenile delinquents tested were found to have undetected LD.
Hazelden Foundation, Minn.1992: up to 60% adolescents in treatment for substance abuse had LD

Prof Margaret Prior (LaTrobe University, *Australian Temperament Study*): 16% of children at grade 1 were more than one standard deviation below the mean in word reading. By grade 2, one third of them had “recovered”, leaving 10% with ongoing difficulties that did not resolve by Grade 4.

Miles 2004 8947 10 y.o. 3% (severe) +6% to varying degrees (excluding children of low ability) *(Electronic Journal of Research in Educational Psychology 2(2),5–12)*


14. **Other types of LD: dysgraphia, dyscalculia, dyspraxia**

**Dyspraxia**: coordination difficulties are specifically a result of problems with praxis (choosing, planning, sequencing and then executing movement). Verbal Dyspraxia is an articulation disorder that can affect speech sound production and oral non-speech movements. Children with dyspraxia (oral or verbal) have difficulty planning, initiating and executing speech sounds and non-speech movements. It is a motor programming problem, not a muscular weakness.

**Dyscalculia**: Dyscalculia is a broad term for severe difficulties in maths. It includes all types of maths problems ranging from inability to understand the meaning of numbers to inability to apply maths principles to solve problems.
# Difficulty imagining a mental number line
# Particular difficulty with subtraction
# Difficulty using finger counting (slow, inaccurate, unable to immediately recognise finger configurations)
# Difficulty decomposing numbers (e.g. recognizing that 10 is made up of 4 and 6)
# Difficulty understanding place value
# Trouble learning and understanding reasoning methods and multi-step calculation procedures (Anna Wilson, University of Auckland)

**Dysgraphia**: Dysgraphia is a neurological disorder characterized by writing disabilities. Specifically, the disorder causes a person's writing to be distorted or incorrect. In children, the disorder generally emerges when they are first introduced to writing. They make inappropriately sized and spaced letters, or write wrong or misspelled words, despite thorough instruction. (National Institute of Neurological Disorders and Stroke.)

**Paul R. Whiting. President, SPELD NSW**