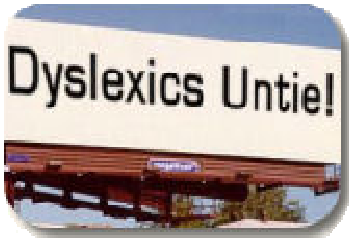
DYSLEXIA



This Section covers the following topics-

Preamble

Some Controversial Issues

- The tendency to label any reading problem as being a case of dyslexia.
- The common practice of incorrectly identifying dyslexia from a WISC/WAIS profile or other cognitive test profiles.
- The early literature focussed on conceptual, theoretical definitions of dyslexia, not on operational, "practical" definitions.
- Defining dyslexia.
- The notion that one particular method or program of instruction has general superiority over all others in the treatment of dyslexia.
- The debate over causation.

Some Fundamental Points

My Clinical Observations

- How Can Dyslexia be Identified?
- Key Tell-Tale Signs
    - Primary Area
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Teaching Students with Dyslexia

A Review of Relevant Research and Literature

- Phase 1: (1895-1976)
- Phase 2: (1977-Present including DSM-5 updates)

References

PREAMBLE

Originally, I had not planned to write a specific section on dyslexia, thinking that it was unnecessary given the comprehension coverage of Learning Disability. The generic term Learning Disability encompasses the conditions known originally by their formal medical/scientific names, e.g., dyslexia, dysgraphia and dyscalculia. In addition, problems with spelling and written expression are also included.

As dyslexia is the most common type of a Learning Disability, the information presented in the **Section-Learning Disability**, is obviously relevant and should be consulted by interested readers.

In the strictly narrow sense, dyslexia refers to severe reading impairment. However, as mentioned above, dyslexia is almost always associated with other problems with literacy skills and also with some problems in mathematics. As the term Learning Disability embraces all of these related problems, it is an accurate and appropriate "cover-all" term to use.

Continuing confusion and misinformation about such fundamental issues as definition, characteristics, causation and treatment have led me to write a separate section on dyslexia.

My special interest in dyslexia resulted in Doctoral Research at Monash University. I completed my studies in 1976, with the presentation of a thesis entitled "A Study of the Perceptual Bases of Specific Reading Disability". The term dyslexia was not in favour at this time with "specific reading disability" being the most common, less contentious synonym. During the 1960s and 1970s, very few people in Australia were formally studying dyslexia. This was surprising as considerable interest was being generated in the field, hence the establishment of SPELD (Specific Learning Difficulties Association) and AREA (Australian Remedial Teachers' Association) now known as LDA (Learning Difficulties Australia). Being a young teacher and psychologist, I had the privilege of having close associations with both organizations during their pioneer years. To highlight the lack of empirical research in the field at this time, my thesis had to be marked in the USA. For a very brief summary of my research findings see below.

As a special education teacher, educational psychologist and later an Associate Professor of Psychology and Special Education at Monash University, I continued to be very interested in dyslexia. In the Faculty of Education, I lectured in reading, reading disability, learning disability and dyslexia and other related areas. As Director of the Krongold Centre for Exceptional Children and Adolescents, I was responsible for the supervision of the clinical practica of students studying special education and educational and clinical psychology.

Many of the children and adolescents referred to the Krongold Centre were diagnosed as having dyslexia. Since leaving Monash University, I have continued to have a private practice and many of my clients have been diagnosed with dyslexia.

Since the mid 1960s, I have assessed over 3,500 individuals, approximately 1,500 or more of whom were students with dyslexia. In many cases, I have actually followed the young student through to senior school and even to University and beyond.

On a personal note, I have found that my surname not only indicates my profession (much to the mirth of observant kids), but is also a very quick test of dyslexia-a small group of people mispronounce/mispell Sykes as SKYES!

SOME CONTROVERSIAL ISSUES

Most of the controversy surrounding dyslexia revolves around the following issues.

1. The tendency to label any reading problem as being a case of dyslexia.

Originally, the term dyslexia was reserved for a severe and chronic impairment in reading. The term covered a small percentage of the population-approximately 5%. To fit the diagnostic criteria, the individual had to satisfy the following-

- Average or above general intellectual ability
- No identifiable factor that could explain the academic underachievement. This included the following-intellectual disability, early bouts of middle-ear infection, language delay, sensory impairment, health problems including specific illnesses, bilingualism, socio-economic disadvantage, behavioural or personality problems, prolonged absenteeism, inappropriate or poor instruction, low motivation for learning and attentional deficits.

Fundamentally, dyslexia has been defined by exclusion. That is, no known causal factor or factors can be identified that explain(s) the unexpected academic underachievement. Hence some early writers have referred to it as the "residual" category of reading problems.

In contrast, general reading problems are known to be caused by the following factors, often in combination-

- below average intellectual abilities
- early bouts of middle-ear infections with associated intermittent hearing loss
- speech and language delays
- sensory impairments
- neurological impairment
- prolonged illness
- emotional and behavioural problems
- socio-cultural factors
- bilingualism
- low motivation and poor task commitment
- poor attentional and concentration skills
- premature, poor or inappropriate instruction

The DSM-5 redefines Specific Learning Disorders and includes comments on the two most common types of SLD- Dyslexia and Dyscalculia (problems with maths). Dyslexia is an alternative term used to refer to a pattern of learning difficulties characterised by problems with accurate or fluent word recognition, poor decoding, and poor spelling abilities. If dyslexia is used to specify this particular pattern of difficulties, it is important to specify any additional difficulties that are present, such as difficulties with reading comprehension or math reasoning. The severity of Dyslexia needs to be classified as Mild, Moderate or Severe. (For extensive comments on the DSM-5 reclassification and new definition of Specific Learning Disorders, see Section-**Learning Disability** pp. 9-13).

2. The common practice of incorrectly identifying dyslexia from a WISC/WAIS profile or other cognitive test profiles.

As the literature stresses, this is not a valid procedure. (See the **Learning Disability Section** for more information). The common, "tell-tale" educational signs of dyslexia are unexpected and unexplained academic underachievement in reading, spelling and written expression and frequently also in handwriting and areas of mathematics.

3. The early literature focussed on conceptual, theoretical definitions of dyslexia, not on operational, "practical" definitions.

This made it very difficult for teachers and psychologists to have a workable checklist and hence accurately identify students with dyslexia. Characteristic difficulties including problems processing and retaining directional information were not clearly articulated. (See the **Learning Disability Section** for detailed checklists). Hence, teachers and psychologists were not given a guide to the key "observable" difficulties necessary to make an accurate diagnosis of dyslexia.

4. The notion that one particular method or program of instruction has general superiority over all others in the treatment of dyslexia.

There is no royal road to the successful treatment of dyslexia. Despite many extravagant claims, no independent, empirical evidence exist that shows one method/strategy to have general superiority over another. Of course, this should not be surprising. Students with dyslexia do not form a homogeneous group. Each student's performance profile, both psychologically and educationally, is unique. Obviously, chronological age and gender vary as do a student's positions on the developmental continuum in terms of cognitive ability and reading ability. Dyslexia also varies in severity (severe, moderate, mild). Furthermore, a student with dyslexic is invariably having learning disabilities in a range of areas-**NOT JUST READING**. These areas typically include spelling, written expression, handwriting and aspects of mathematics. Clearly, each individual will present with a unique profile, and hence, will require her/his own special instructional program.

5. The debate over causation.

It has been argued by some professionals that the following difficulties cause dyslexia-

- Difficulties with Phonological Processing
- Deficiencies in Verbal Encoding
- Problems with Short-term Auditory Memory (STAM)
- Working Memory Difficulties identified by WISC and WAIS profiles
- Processing Speed Problems identified by WISC and WAIS profiles

Since my doctoral research in 1976 and my subsequent research and clinical observations, I continue to contend that problems processing directional information play a very significant causal role in learning disabilities, including dyslexia.

The important distinction between causation and correlation is frequently ignored in many articles or reports.

Seeking an endogenous (constitutional) explanation of causal factors, early researchers contended that dyslexia stemmed from one or more of the following-

- Brain Dysfunction
- Genetic Predisposition
- Developmental Delay

The latter two propositions have continued to gain acceptance.
Following some 40 years of professional work with dyslexic students, I contend that there is a very strong familial predisposition in most cases of dyslexia. So often, the father of a student being assessed reveals that he had severe early literacy learning problems. Furthermore, difficulties understanding directional concepts and processing directional information are invariably recalled, e.g., confusion over left and right, including fascinating stories of strategies used to distinguish the right hand/side from the left hand/side (especially when driving), problems reading a street directory and maps, reading timetables and telling the time.

The DSM-5 identifies a Specific Learning Disorder (including Dyslexia) as a neurodevelopmental disorder with a biological origin that is the basis for abnormalities at a cognitive level that are associated with the behavioural signs of the disorder. The biological origin includes an interaction of genetic, epigenetic, and environmental factors which affect the brain's ability to perceive or process verbal or nonverbal information efficiently and accurately.

SOME FUNDAMENTAL POINTS

Twelve fundamental points that need to be understood about dyslexia -

1. dyslexia strictly refers to an impairment in the ability to read
2. dyslexia in its early form is usually severe and chronic
3. dyslexia usually becomes less severe with time and often progresses through the severe, moderate to mild stages with increasing age and appropriate education
4. dyslexia is not any type of reading problem but one that is experienced by a small, specific population of individuals
5. dyslexia is defined by exclusion; it is considered to be a neurodevelopmental disorder with a biological origin
6. dyslexia is identified by a pattern of problems beyond just reading: in most cases, problems are also experienced in other literacy skills including handwriting, spelling and written expression and often problems occur in areas of mathematics
7. dyslexia is the most common type of learning disability
8. dyslexia is more prevalent in males than females
9. dyslexia runs in families: it is genetic and has a neurological base
10. dyslexia, at the psychological level, is considered to be caused by information processing problems, particularly phonological skill deficits, although the precise nature of the antecedent(s) continue to be hotly debated
11. dyslexia can be treated but despite numerous claims, no scientifically established universal "cure" has been identified
12. dyslexia can be treated: individually planned educational strategies based on instruction which is direct, explicit, systematic and sustained, hold the key to treatment

MY CLINICAL OBSERVATIONS

As mentioned above, I estimate that I have assessed over 1,500 students with dyslexia. In addition, I review over 300 psychological reports annually on student's seeking special funding or special examination arrangements. Many of these students have learning disabilities including dyslexia. Hence, I have a very extensive set of clinical and research records on which I have based my observations and comments.

HOW CAN DYSLEXIA BE IDENTIFIED?

In understanding the aetiology of dyslexia, the keen reader is referred to information in the **Learning Disability Section**. Here, it is postulated that Learning Disability (including Dyslexia) can be studied at three levels-

- educational
- psychological
- neurological

The psychological and educational levels will be the main foci below.

What are the key psychological, cognitive or information processing problems?

Essentially, I contend that the basic problems centre on the two aspects of **Directionality**-

- **Sequence**
  - processing and retaining sequential information
- **Orientation**
  - processing and retaining orientational/positional information

In terms of the basic cognitive or information processing weaknesses underpinning dyslexia, the student's cognitive and educational profiles implicate directional uncertainty/confusion. That is, problems processing and retaining auditory and/or visual sequential information and/or processing and retaining positional, visual-spatial information. For instance, these problems can be observed in activities such as-

- Serial learning (the rote learning and retention of basic factual, sequential information-days in the week, months in the year, seasons in the year, birth date, telephone number, address, letters in the alphabet, number sequences and facts and more advanced information including rules and principles).

- Processing and retaining verbal sequential information (remembering verbal instructions, retaining number and letter sequences, retaining word patterns and spelling patterns and retaining mathematical facts and processes).
- Processing and retaining visual-spatial, positional information (knowledge of left and right, directional and positional information-letters and numerals, telling the time, mathematical signs, common fractions, etc.)

- Productivity rate (speed of output, especially involving sequential and directional information).

Such difficulties are observable in cognitive profiles and responses on psychological tests such as the WISC/WAIS and educational test profiles as well as the student's responses in reading, spelling, written expression, handwriting and mathematics.

What are the key "surface" educational problems?

Educational signs (clearly seen in a young student's workbooks and also in educational test profiles) can occur in different areas of the curriculum. These signs change with the age of the student and their place on the learning continuum. Typically, a student will show more severe signs during the early school years, moderate signs during the middle school years and in most cases, mild to only subtle signs during the final school years and beyond. These signs are observable in the following areas of the curriculum-

- handwriting
- reading
- spelling
- written expression
- mathematics
- other curriculum areas, e.g.,
    - Geography
    - Graphic Communication

A very detailed account of these signs is provided in several places on this DVD, including the following-

- **Learning Readiness**-checklists are provided.
- **Early Learning Essentials 1: Directionality**-checklists are provided.
- **Learning Disability**- checklists are provided.
- Relevant information is also provided in the separate subject areas-
    - **Reading**
    - **Spelling**
    - **Written Expression**
    - **Mathematics**

KEY "TELL-TALE" SIGNS

Very briefly, key **EARLY,** common "tell-tale" signs of a Learning Disability (including Dyslexia) include the following. With Dyslexia, the primary area is reading. However, as indicated above, secondary literacy and numeracy areas are also implicated.

PRIMARY AREA

Reading

- Slow development of phonological skills
- Faulty knowledge of the alphabet
- Slow development of phonic skills
- Slow acquisition and development of basic word recognition and word-attack skills
- Oral reading errors-reversals, substitutions and omissions-especially involving common, small words
- Slow development of reading vocabulary and reading comprehension skills
- Strong dislike of reading

SECONDARY AREAS

Handwriting

- Faulty pencil grip
- Incorrect letter formation-letter reversals, especially b/d, p/q, j, s, z
- Poorly aligned and spaced writing
- Messy writing
- Difficulty progressing from print to a cursive style

Spelling

- Slow acquisition and development of basic phonological and phonic skills
- Letter-sound confusions
- Letter-name, letter-sound confusions
- Letter reversals
- Sequencing problems-order of letters in words
- Severe difficulty spelling common, phonetically irregular words

Mathematics

- Reversing numerals
- Difficulty establishing a one-to-one correspondence
- Problems counting forwards
- Severe difficulty counting backwards
- Slow recognition of number words
- Difficulty spelling number words
- Slow development of automatic number facts-basic bonds
- Very slow learning multiplication "tables"
- Major problems with the basic algorithms-vertical "sums"

- Major problems with common fractions
- Great difficulty "reading" an analogue clock

It must be stressed that many young students experience some of the above problems. The critical issue in the diagnosis of a Learning Disability/Dyslexia is that these problems are both severe and chronic (i.e., persist beyond Grade 3) and are not attributable to known causal factors.

As a final and positive note, it should be stressed that dyslexic students usually develop at least functional reading skills. Of course, many students become satisfactory readers and experience success with their VCE and University studies.

In most cases, however, spelling, handwriting and written expression difficulties persist throughout life. Other persistent problems involve studying, taking notes, time management including both planning and organisation.

Many adults who experienced dyslexia in their early years report continuing problems including remembering sequential information (e.g., telephone numbers, car registration numbers, bank account pin numbers, significant dates, identifying left and right, port and starboard, reading a street directory and maps and understanding compass directions, finding a parked car, subtle speech-production difficulties, mental maths problems and calculation difficulties. Generally speaking, reading is slow and difficult and not well liked. Lapses in reading (reversing or mispronouncing simple words), handwriting (letter and numeral reversals) and spelling are not uncommon.

In addition, many dyslexic students display unique, very creative and imaginative techniques to aid learning and retention. As adults, they frequently have advanced talents in certain vocational areas including-planning and design, visual and performing arts, computer and technical fields.

TEACHING STUDENTS WITH DYSLEXIA

Teaching students with dyslexia is very controversial. Essentially, the controversy centres on the debate over the orientation of treatment/intervention.
The instructional orientation to adopt with dyslexic students is perhaps the most contentious issue in the broad field of Special Education.

Basically, three approaches are adopted-

- Skill Training
- Process Training
- Alternative Therapies

The **Skill Training** approach focusses on the academic deficit- e.g., reading. Hence, the sub-skills of reading are assessed and deficit areas taught/remediated-
eg.,

- Phonological skills
- Phonic skills
- Word recognition skills
- Word-attack skills

Basically, **Skill Training** involves modifying existing educational approaches and giving specific, intense and sustained instruction in underdeveloped areas of reading.

In **Process Training**, psychological or neurological processes assumed to underlie reading, are the focus of training-
e.g.,

- Visual training
- Auditory training
- Perceptual training
- Memory training
- Verbal training

Alternative Therapies are usually advocated by professionals working in "non-educational" fields and include the following-

- coloured overlays and lenses
- dietary control
- movement and exercise
- applied kinesiology
- physical manipulation
- neuro-linguistic programs

Despite many extravagant claims, especially for certain **Alternative Therapies**, no strong, independent empirical evidence exists to support the use of **Process Training** or **Alternative Therapies**.

Fortunately, the majority of informed literature continues to emphasise that modified educational programs hold the key to the treatment of dyslexia.

If **Process Training** and/or **Alternative Therapies** is/are employed, inevitably **Skill Training** is also required.

The critical issue then is -how essential and relevant is/are **Process Training and/or Alternative Therapies**?

Skill Training or Focussed Educational Instruction

Dyslexia can be successfully treated. However, it is essential that teachers have a knowledge of the fundamental features of dyslexia and associated difficulties. While some characteristics are not directly relevant to instruction (e.g., some underlying processing deficits), awareness of these gives the teacher an understanding of the nature of difficulties associated with dyslexia and, hence, a rationale for planning and implementing an appropriate instructional program.

Obviously, in Skill Training, the teacher requires an-

- excellent knowledge of early literacy learning
- excellent knowledge of early literacy teaching strategies

Hopefully, most primary school teachers have had training in these basic areas. If not, such information is readily available. The interested reader can find such information on this DVD, especially the following **Sections**-

- **Reading**
- **Spelling**

An individually planned program is required for each student with dyslexia; however, each program usually shares common elements. Fundamentally, the task is to modify existing educational programs. Program variation depends on such variables as the student's age, their stage of learning, their learning style and rate of learning and their position on the reading development continuum.

To obtain such information, there is a need for both formal and informal assessment using psychological and educational tests.

Formal psychological and educational testing provides general standards of achievement with some insights into important parameters such as processing strengths and weaknesses, learning style, learning rate and instructional needs.

Informal assessment involves direct observation of the student's educational knowledge and skills in handwriting, reading, spelling, written expression and mathematics. Invariably, these observations provide the most useful instructional information.

In planning an instructional program, the following dimensions are paramount-

- Individual
- Direct
- Explicit
- Systematic
- Sustained

Relevant learning essentials to consider include the following-

- Time on task, time to learn
- Multimodal orientation-e.g., visual, auditory, motoric
- Reinforced practice
- Encouragement and positive feedback
- Parental support

TEACHING LITERACY SKILLS

Literacy learning has two early stages or phases-

- the pre-literacy stage
- the early literacy stage

The Pre-literacy Stage

Relevant skills/abilities include the following-

- general language skills
- phonological skills
- visual discrimination and retention skills

Relevant information is available on this DVD in the following Sections-

- **Learning Readiness**
- **Early Learning Essentials 1: Directionality**
- **Early Learning Essentials 2: Language Development and Concept Formation**
- **Reading**
- **Learning Disability**

General language abilities include receptive and expressive language skills. Such aural and oral language skills are essential pre-requisites for written language including reading, spelling and written expression. Of course, such language skills continue to be critically important in the ongoing development of reading, especially the higher-order skills of reading vocabulary and reading comprehension.

The relevant phonological skills and visual processing skills are well defined in the checklists and other areas in the above **Sections** on this DVD.

At the pre-school level, the emphasis is basically on the **pre-literacy skills** mentioned above. However, in some cases, it will be appropriate to introduce advanced pre-school students to **early literacy skills** including-

- more advanced phonological skills
- knowledge of the alphabet
- early phonic skills
- early word recognition skills
- prose reading skills

The Early Literacy Stage

Obviously, the formative stages of reading occur in the early primary school years. The development and acquisition of word recognition skills are of paramount importance for students.

Reading

In the initial teaching of reading, the Whole Language Approach and Skill Centred Approaches are usually taught in combination. See **Reading Section**, for detailed notes on these approaches.

Once a student begins to experience difficulties, including a student identified with dyslexia, experts recommend the implementation of a modified, individually planned educational program.

Such a program is usually based on the following components-

- Relevant visual and auditory processing skills
- Phonological skills
- Phonic skills
- Word recognition and word-attack skills
- Prose reading skills
- Reading comprehension

Specific information on the following skills is also available on this DVD.

Phonological Skills

During the early years of childhood these skills are acquired and develop very naturally in most children. Phonological skills, including the ability to hear, identify, discriminate, analyse and integrate speech sounds (phonemes) are usually well developed before a child learns to read and spell. For some children, however, explicit, formal instruction is needed for the acquisition of phonological skills.
There is growing support for the contention that dyslexia stems from a deficit in phonological processing or difficulty in recognising that spoken words are comprised of discrete phonemes (for example, that the word "dog" is composed of the sounds d/o/g). See the **Reading Section** for a very comprehensive coverage of phonological skills.

Phonic Skills
Following the earlier development of phonological skills, phonic skills are acquired and involve the association of speech sounds (phonemes) with the visual letters (graphemes). Because the alphabet is "man-made", we must explicitly learn the grapheme-phoneme relationships. The lack of a one-to-one correspondence between sounds and letters in the English language causes many young readers considerable confusion and difficulties. See the **Reading Section** for further information on phonic skills.

Word Recognition Skills
These are the essential, critical skills in reading. Through repeated practice, the aim of instruction is to develop automaticity, i.e., the instant recognition of words. It is widely accepted that the core deficit in dyslexia is learning to read at the word level. See the **Reading Section** for additional information on word recognition skills.

Prose Reading Skills
To strengthen word recognition skills and to increase reading fluency, oral and silent reading is important. Listening to a child read or participating in paired-reading activities, is an excellent way of providing "natural" reading practice. Repeated oral reading, especially of favourite books, is very beneficial. Paired-reading activities involve the teacher or parent reading together with a child.

This method provides opportunities to-

- listen to a child reading
- encourage reading by praise and positive comments
- identify reading errors/miscues
- practise reading in context
- ensure that motivation is high through wise book selections

I recommend that parents buy two copies of selected readers. This enables the paired-reading to occur while parent and child read the same book "side by side" or when they reading each book in other locations, for instance-opposite each other at the kitchen table or when the child is in bed. It also gives a very positive message to the child that their readers are valued and important.

For further information on these reading strategies see the **Parenting Section**.

Reading Comprehension
While this skill is ultimately the fundamental purpose of reading, during the early primary school years, reading comprehension takes a secondary role to the most critical early skill of word recognition.

Information on Reading Comprehension can be found in the **Reading Section**.

Spelling

As mentioned above, most students with dyslexia eventually reach a satisfactory standard of reading, although they usually continue to be slow and reluctant readers. However, most students with dyslexia will experience problems with spelling and written expression throughout life.

The information presented above on Reading forms an essential foundation for a knowledge of teaching spelling.

Reading involves the recognition of the written word while spelling involves the retention and recall of the written word. Recognition is a much easier cognitive process than retention and recall. Hence, spelling (retaining and recalling the written word) is extremely difficult for most students with dyslexia.

Essentially, the major problem in dyslexic students is spelling common, essential, phonetically irregular words.

To aid and strengthen the retention and recall of written words, two approaches are recommended in combination-

- Drill and repeated practice-involving flashcards
- Understanding and insight-involving rules and patterns

In other words, learning to spell requires a flexible and functional combination of automaticity and awareness.

Importantly, patience is needed by both teachers and parents in assisting dyslexic students learn to spell.

From my experience, I consider that teachers need to plan and implement a spelling program far more thoughtfully for dyslexic students and other underachieving students. For instance-

- the "traditional" list of words to be learned and practised at home each week needs to be carefully selected.

- the list needs to be short and relevant-with difficult, but common, essential words (e.g., Dolch Words, 200 Magic Words and Oxford Words) forming the basics with some interesting/personal/topical/thematic words also included.

- the common, essential words need to be revised regularly.

- an error analysis provides an excellent insight into the nature of the student's spelling problems (e.g., are the errors mainly "acceptable" phonetic representations, rather bizarre visual approximations or a combination of both?).

- words should be revised and tested in two ways- spelling words separately and in short pieces of dictation.

- parents need to play an important supportive role.

Teachers should be aware that students with dyslexia, by definition, have average or above intellectual abilities, and therefore many are very capable of learning spelling rules and identifying patterns. Obviously, such rules need to be introduced judiciously. Hence, by say Grades 3 or 4, most students can develop what experts have called "a set for diversity"- i.e., that letters can make different sounds depending of their position in words.

Helpful information on spelling can be found in the **Spelling Section** with some information for parents included in the **Parenting Section**.

The basic word lists can also be found in the **Spelling Section.**

A REVIEW OF RELEVANT RESEARCH AND LITERATURE

PHASE 1 (1895 to 1976)

To gain some insight into the early phase of research into dyslexia involving definitions, characteristics and diagnostic criteria, the reader may find the following review of literature that I conducted for my doctoral research in 1976 helpful as a starting point.

The following information has been taken from my Doctoral Dissertation entitled- A Study of the Perceptual Bases of Specific Reading Disability, Monash University, 1976

A Brief Summary of My Doctoral Research Findings

For my study, carefully selected students with specific reading disability were matched with normal readers. Each group comprised 50 male students, all of whom were in Grade 2. To fully explore the perceptual abilities of the students, a wide range of perceptual tests was administered. It was necessary to supplement the available tests with some tests specifically developed by myself. I continue to use some of these tests and they are presented on this site-
Early Learning Essentials 1: Directionality (Appendix).

Briefly, my study revealed pronounced deficiencies in the disabled readers in the following areas-

- processing and retaining visual sequences and visual orientation
- processing and integrating auditory sequences

A classification of disabled readers was undertaken by means of a cluster analysis. Results indicated that, in terms of perceptual characteristics, specific reading disability is a heterogeneous disorder, not a homogeneous disorder as often implied in the literature.

Four groups of disabled readers were identified. All four groups were shown to be deficient in-

- retention of visual sequences and visual orientation, and,
- auditory integration involving the processing of sequential information. (Today, this would be referred to as phonological processing difficulties)

Each of the four groups was also shown to possess a different array of perceptual difficulties.

Overall, the results indicated that the students with a specific reading disability possessed a general directional confusion or uncertainty processing both visual and auditory information.

This important finding that students with specific reading disability/dyslexia experience problems processing directional information has been constantly confirmed in my subsequent research and clinical practice. Hence, my continuing special interest in the concept of directionality in much of my writings in this DVD. Mention has been made of the many **Sections** where directionality is addressed.

Today, as the following review of literature indicates, phonological processing problems have been elevated by many researchers to be the primary causal factor in dyslexia.

I often wonder what

- Frank (1935, 1936)
- Creak (1936)
- Schilder (1944)
- Kass (1962)
- Bruce (1964)
- Wepman (1968)
- Ingram, Mason and Blackburn (1970)
- Naidoo (1978)

and other earlier researchers would think of this so called "recent" development. Interestingly, I rarely see their names mentioned in the references/bibliographies of contemporary investigators of dyslexia!

A GENERAL HISTORICAL REVIEW

The history of clinically recognised reading difficulties in children of assumed normal intelligence can be traced back to the mid 1890s when Kerr (1896), a British school physician, reported briefly on the subject of aphasic conditions in children. Prompted by a short note written by Hinshelwood (1895) on the topic of acquired word-blindness, Morgan (1896) attributed the disorder to a congenital defect in the brain, and likened it to alexia, a condition he assumed was caused by injury to the cerebral cortex.

These early reports triggered off a wave of interest in the subject and articles were written by a number of investigators (Fisher, 1905; Hinshelwood, 1900, 1904, 1907; Nettleship, 1901; Stephenson, 1904; Thomas, 1905). The most prolific early essayist on the topic was Hinshelwood, a British ophthalmologist. In 1900, he published an article entitled "Congenital Word-Blindness" and gave a detailed description of two cases from his own practice and noted how closely the symptoms paralleled those in cases where the defect was caused by brain damage. He hypothesised a defect in the angular and supramarginal gyri in the left side of the brain in right-handed people.

In 1917, Hinshelwood presented his collected findings from thirty-one cases of congenital word-blindness and summarised the work of other authors to that date. This first systematic description of the subject detailed aspects of aetiology, symptomatology and treatment. Some of his cases experienced difficulties in recognising whole words but could identify letters; others had problems in identifying single letters. Some, but not all, failed to recognise written figures. It was also noted that the condition occurred more frequently in boys than in girls.

Hinshelwood attributed this "grave and rare" disorder to a congenital defect of development in the special area of the brain subserving the visual memory of words and letters. He contended that the abnormality within this circumscribed cerebral area could be due to either disease, injury at birth or faulty development. He favoured the last interpretation and concurred with Thomas (1905) that in many cases there was evidence of hereditary transmission.

While Hinshelwood's monograph is quite comprehensive, he did not analyse the type of reading errors most commonly made or comment on the frequent history of associated retarded speech development or on mixed laterality of eye, hand and foot, an increased tendency to left-handedness and ambidexterity noted earlier by McCready (1910a, 1910b).

McCready, a pediatrician, regarded reading difficulties as part of a more global syndrome of aphasia or language disability. He originally considered that most aphasias were due to ontogenetic causes, a biological variation or a stigma of degeneration, similar to that in such cases as colour-blindness and that only rarely was the cause one with definite pathology. However, in 1926, he asserted that often an aphasic state may be a result of brain damage at birth.

The next significant phase in the investigation of congenital word-blindness began with the work of Orton, an American neuropathologist. In 1925, he described the cases of fifteen children who were retarded in reading and showed certain similarities in the errors which they made in reading. Two of these cases fitted Hinshelwood’s description of congenital word-blindness and the others seemed to form a continuous grading between this extreme and the normal. He noted the extent to which these children, in their reading and writing, tended to reverse the orientation of letters and the order of letters in words and whole words.

Orton was also impressed by the frequency with which he found his patients to be ambidextrous or left-handed or to show conflicting laterality of eye and hand. To account for these observations and to bring the whole syndrome into greater consonance with advancing neurological views concerning cerebral localisation, Orton (1943) hypothesised a physiological deviation due to a failure or delay in acquiring unilateral cerebral dominance. He did not consider the condition to be due to a lesion in the brain or to an agenesis in a localised area.

It should be stressed that Orton was interested in the broad area of specific language disability. He believed that developmental disorders such as word-deafness, delayed speech, stuttering, reading, writing and spelling disabilities and abnormal clumsiness were often found together in individual patients and emphasised their common aetiological and clinical features. He, like others, noted the importance of heredity in the genesis of specific language disability.

After a period of relative neglect, specific reading disability once more attracted the attention of medically trained investigators especially Hallgren (1950) and Hermann (1959) in Scandinavia, Critchley (1964, 1970), Ingram (1969, 1971) and Rutter (1969) in Britain and Birch (1962), Rabinovitch (1962) and Thompson (1969) in America.

Psychologists and educators have naturally tended to pursue lines of enquiry different from those adopted in medical research. In essence, more attention has been directed to environmental and educational factors that inhibit the learning process than to aspects of cerebral pathology. The reason for these different approaches is obvious; of necessity medical research has been focussed mainly on aetiology, while psycho-educational research has been primarily concerned with educational and psychological causes and treatment.

Many psychologists and educators have been inclined to deny the existence of a single specific factor as the cause of a substantial proportion of reading disabilities in school children (Burt, 1937; Gates, 1927; Malmquist, 1958; Monroe and Backus, 1937; Morris, 1966; Robinson, 1946; Schonell, 1948; Vernon, 1957). Such authors contend that the causes of reading difficulties are usually numerous, complex and interrelated. Furthermore, reading ability is viewed as a continuum from the advanced readers through the normal to the extremely backward and illiterate.
The causes of extreme retardation are thus considered to be of the same nature as those of milder degrees of backwardness, namely, physical handicaps, psycho-dynamic disturbances, experiential deprivation, poor or inappropriate instruction, lack of motivation and intellectual defects. Another objection raised by some writers relates to the inability to differentiate specific reading disability from other forms of reading retardation (Bond and Tinker, 1967; Harris, 1956; Malmquist, 1958).

More recently, however, there has been a growing tendency, especially among clinical and educational psychologists (McLeod, 1966; Mason, 1967; Money, 1962, 1966; Johnson and Myklebust, 1967; Naidoo, 1972; Shankweiler, 1964a; Vernon, 1965, 1971; Yule, 1973) to re-affirm the views of physicians and neurologists that, within the large population of children with reading disability, the children with specific reading disability constitute a distinct clinical entity which can be differentiated from other types of reading disability on the basis of several signs, including its severity and high resistance to remedial teaching, the excessive and persistent reversals in reading and writing, certain types of spelling errors and various accompanying visual and auditory perceptual deficits - and all despite their having at least normal intelligence.

In the late 1960s and 1970s, interest in specific reading disability was considerable, with numerous books and journal articles being devoted to this subject. Furthermore, a special research group of the World Federation of Neurology was formed in order to achieve an international focus on the problem. Professional interest was accompanied by a heightening concern of parents for their children who were failing to master basic reading skills. Associations such as the Orton Society and the Association for Children with Learning Disabilities in the United States of America, the Dyslexia Institute in England, the Canadian Association for Children with Learning Difficulties, the Wordblind Institute in Denmark, and the Specific Learning Disabilities Association (SPELD) in Australia being established to assist children with reading and learning disabilities.

DEFINITIONS, TERMINOLOGY AND SYMPTOMATOLOGY

The early papers concerned with specific reading disability dealt mainly with severe cases of the condition and tended to provide an aetiological explanation of the disability. Hinshelwood defined congenital word-blindness as a

> congenital defect occurring in children with otherwise normal and undamaged brains characterised by a difficulty in learning to read so great that it is manifestly due to a pathological condition, and where the attempts to teach the child by the ordinary methods have completely failed. (Hinshelwood, 1917, p.40).

He stressed that genuine cases must possess two essential features, namely, gravity of the defect and purity of symptoms. Hinshelwood was adamant about the need to restrict the term "congenital word-blindness" to a very narrow and specific group of children. In fact, he urged that the term "congential dyslexia" should be reserved for those commonly occurring slight degrees of reading disability.

In spite of much apparent evidence to the contrary, the terms "congenital word-blindness", as originally defined and "specific reading disability", as presently understood, are not synonymous terms. The latter term, being more generic, would presumably encompass the children originally defined as having the more severe "congenital word-blindness" as well as those possessing the milder, more frequently occurring "congenital dyslexia".

Orton (1925) considered that reading disability formed a graded series in severity and that only occasionally was a child unable to attain a fair facility in reading. He disliked the term "congenital word-blindness" because of its association with the acquired condition and the implications of brain damage. He offered the term "strephosymbolia" (twisted symbols) as a descriptive name for that group of children who showed unusual difficulty in learning to read. By attributing the reading disability to a variant in the physiological development of the brain rather than describing it as a pathological condition, Orton (1937), like Hinshelwood, considered the condition to be reversible, provided that proper methods of instruction were commenced early enough.

Some definitions, however, stress both aetiology and symptomatology. For example, Rabinovitch stated that in developmental dyslexia:

> Capacity to learn to read is impaired without definite brain damage being suggested in the case history or upon neurological examination. The defect is in the ability to deal with letters and words as symbols, with the resultant diminished ability to integrate the meaningfulness of written material. The problem appears to reflect a basic disturbed pattern of neurological organization. Because the cause is biological or endogenous, these cases are diagnosed as primary reading retardation. (Rabinovitch, 1962, p.74).

The most widely used approach among physicians is diagnosis by exclusion: essentially a differential diagnosis, it relies on ruling out other explanations of the child's inability to read. This commonly accepted medical approach has provided useful definitions of specific reading disability (Critchley, 1968, 1970; Eisenberg, 1966; Hermann, 1959; Myklebust and Johnson, 1962; Skydsgaard, 1942).

An example of this type of definition has been proposed by the Research Group on Developmental Dyslexia of the World Federation of Neurology. Specific developmental dyslexia was defined as follows:

> 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. (Critchley, 1970, p.ll).

In spite of its usefulness, defining or diagnosing specific reading disability by exclusion has the disadvantage of identifying too few children. It excludes from diagnostic consideration the critical point that specific reading disability may co-exist with any or a combination of contributing factors or be aggravated by them (Boder, 1971).

However, the condition can only be identified with a high degree of certainty if it exists in isolation. Otherwise, it is likely to be impossible to disentangle it completely from other causes of reading retardation (Reid, 1969).

When specific terminology, as opposed to definitions, is examined it is found that a large number of different terms has been used as synonyms for specific reading disability, a term first reported by Orton (1928) and subsequently adopted by a number of writers including Bakker and Satz (1970), Doehring (1968), Eisenberg (1966), Eustis (1947), Schilder (1944), Silver and Hagin (1960), and Wolfe (1941).

The disparate terminology employed reflects the numerous conceptions concerning its aetiology and the lack of knowledge regarding its nature. Table 1 lists some of the terms that have been used by investigators.

It is frequently claimed that specific reading disability can be recognised by a number of signs and symptoms (Critchley, 1964, 1970; Gallagher, 1962; Hallgren, 1950; Hermann, 1959; Money, 1962; Orton, 1937; Rutter and Yule, 1975; Shankweiler, 1964a; Vernon, 1962). However, it is usually admitted that no one sign is, by itself, pathognomonic or definitive (Hermann, 1959). Several signs may appear either in a retarded reader not suffering from specific reading disability, or as a transient stage in a normal reader. The aggregate of signs forms a "variable syndrome", the distinguishing feature being the clustering of several signs together.

As Myklebust and Johnson have reported:

> It should not be construed that all facets of this syndrome of childhood dyslexia necessarily will be present in a given child. Rather, when taken as a group, these symptoms characterize children having this type of reading disorder. (Myklebust and Johnson, 1962, p.18) .

Money defended this rather vague and indefinite description of the condition:

> It is not at all rare in psychological medicine, nor in other branches of medicine, that a disease should have no unique identifying sign, that uniqueness being in the pattern of signs that appear in contiguity. Out of context, each sign might also be encountered in other diseases, or, in different intensities, in the healthy. Specific dyslexia is no exception in this respect. (Money, 1962, p.16).

Rabinovitch (1959) has stated this same position succinctly when he attributed to the dyslexic child a characteristic pattern, with much variability from individual to individual. Hence, specific reading disability should not be regarded as a unitary phenomenon. It is necessarily a rather broad concept which refers to a number of types of reading disability which may or may not be related in aetiology and in the underlying disturbance of function (Shankweiler, 1964b).

TABLE 1

SOME TERMS THAT HAVE BEEN USED AS SYNONYMS FOR SPECIFIC READING DISABILITY

Author	Date	Terminology
Morgan, W.	1896	Congenital Word-Blindness
Claiborne, J.	1906	Congenital Symbol Amblyopia
Jackson, E.	1906	Developmental Alexia
Witmer, L.	1907	Amnesia Visualis Verbalis
Rutherfurd, W.	1909	Congenital Dyslexia
Wallin, J.	1921	Dyslexia
Apert, E.	1924	Congenital Familial Dyslexia
Orton, S.	1925	Strephosymbolia
Frank, H.	1936	Word Blindness
Skydsgaard, H.	1942	Constitutional Dyslexia
Schilder, P.	1944	Congenital Alexia
Hallgren, B.	1950	Specific Dyslexia
De Hirsch, K.	1952	Gestalt Blindness
Rabinovitch, R. et al.	1954	Primary Reading Retardation
Drew, A.	1956	Familial Dyslexia
Malmquist, E.	1958	Special Reading Disabilities
Gallagher, J.	1960	Specific Language Disability
Ingram, T.	1960	Specific Developmental Dyslexia
Critchley, M.	1962	Developmental Dyslexia
Kass, C.	1962	Severe Reading Disability
Money, J.	1962	Reading Disability
Myklebust & Johnson	1962	Childhood Dyslexia
Rutter M. et al.	1970	Specific Reading Retardation
Symes & Rapaport	1972	Unexpected Reading Failure

The phenomena which have been frequently mentioned characterising specific reading disability can be classified into two exclusive categories: oral reading errors and non-reading characteristics which have been observed to occur in conjunction with the reading errors.

Although a multitude of factors has been reported to co-exist with specific reading disability, the only factor which is common to all cases is marked reading disability. Clinical investigators have commented on the characteristic nature of the oral reading errors (Critchley, 1970; Frank, 1936; Hermann, 1959; Ingram, 1960; Money, 1962; Orton, 1925, 1928; Saunders, 1962; Schilder, 1944; Shankweiler, 1964a).

Some researchers (Critchley, 1970; Ingram, 1960; Orton, 1937) have indicated that certain types of errors in children's oral reading are symptomatic of specific reading disability. Such workers affirm that these disabled readers tend to be distinguished by the errors they make both in the orientation of letters and the incorrect sequencing of both letters and words: they appear to make an excessive number of mistakes in dealing with the spatial patterns of graphic symbols, so that they disorient them by transposing either the position of letters or their sequence.

A slight modification of this position has been adopted by Money (1962) and Eisenberg (1966) who asserted that the dyslexic student is not unique in making reversal and translocation errors, but is conspicuous in making a large number of them over a lengthy period of time. Hermann was of the same opinion:

> It should be made clear, ... that there are no types of error so characteristic that the diagnosis of word-blindness can be based on them alone. The mistakes made by beginners in school resemble the difficulties of the word-blind reader, but whereas the normal subject surmounts them within a certain period of time, these mistakes persist in the word-blind. It is indeed striking how the word-blind continue to commit these "primitive" errors (rotations and reversals) for many years and even indefinitely, these errors thus giving the performance of the word-blind subject a definite qualitative character.
> (Hermann, 1959, p.43).

Other investigators (Hallgren, 1950; Malmquist, 1958) have been unable to obtain any confirmation of the assertion that there exists a specific, clearly separable group among retarded readers who, especially in the process of acquiring reading skills, make reading errors which differ in kind from those made by other groups of children with reading difficulties or by normal children. Consequently, these authors contend that the difference between diverse groups of readers with varying degrees of reading skill should be seen more in terms of frequency than type of error.

The accompanying defects or signs which have been linked with specific reading disability range widely in accord with the researcher's interests, aetiological preconceptions, theoretical biases and methodological approach.

It is common for specific reading disability to be associated with other developmental disorders and a variety of minimal, or soft, neurological signs pointing to a parietal lobe dysfunction (Cohn, 1964; Critchley, 1970) or neurophysiological immaturity (De Hirsch, 1952; Bender, 1963; Satz and Sparrow, 1970).

Certain patterns of sensory-motor, language, perceptual and cognitive defects, as revealed by psychological test profiles, have been found to be associated with specific reading disability.

These patterns are viewed as important corroborative evidence of specific reading disability by neurologists, pediatricians and psychologists and are often relied on by educators in planning compensatory or remedial programmes. The defects found to co-exist with specific reading disability include the following: spelling disorders (Critchley, 1968; Myklebust and Johnson, 1962); writing difficulties (Orton, 1925; Hermann, 1959); speech and language disorders (Ingram, 1971; Rawson, 1968); other symbol defects (Hermann, 1959; Rabinovitch, 1968); dyscalculia (Myklebust and Johnson, 1962); clumsiness and motor inco-ordination (Bakwin and Bakwin, 1966; Waites, 1968); anomalies of cerebral dominance (Eustis, 1947; Silver and Hagin, 1964); finger agnosia (Kinsbourne and Warrington, 1966); perceptual disorders (Ingram, 1960; Rabinovitch, 1968); memory defects (Myklebust and Johnson, 1962); directional confusion (Money, 1962; Rutter and Yule, 1973).

Reid (1969), after reviewing the many different individual "clinical signs" that have been used collectively by writers to indicate a positive diagnosis of dyslexia, suggested the following four-fold classification:

(a) perceptual difficulties (for example, spatial difficulties, visuo-motor disorders);

(b) anomalies in development (for example, laterality, speech, motor control);

(c) circumstantial evidence (such as familial incidence of similar reading difficulties);

(d) the precise nature and severity of the reading and spelling difficulties.

DIAGNOSTIC CRITERIA

The differential diagnosis of specific reading disability does not rest on any one criterion. Often the extent of reading retardation, the presence of accompanying signs and the lack of obvious causal explanations are taken to indicate a positive diagnosis.

General Criteria of Specific Reading Disability

A study of the writings of different physicians shows wide disagreement with respect to their diagnostic criteria of specific reading disability.

Hinshelwood (1917) based his diagnosis on certain points, in particular the gravity and purity of the condition together with its unresponsiveness to ordinary methods of instruction.

Hallgren (1950) employed the following criteria for a positive diagnosis of "specific dyslexia":

(a) difficulties in learning to read and write;

(b) ability in reading and writing during the first years at school definitely below the average of the class;

(c) a definite discrepancy between proficiency in reading and writing and in other school subjects;

(d) a definite discrepancy between ability in reading and writing and the child's general ability.

Hallgren admitted the unsatisfactory nature of this form of identification and concluded:

> I have not found it possible to define the syndrome of specific dyslexia more exactly. In my series, there is therefore no sharp borderline between the group of cases of specific dyslexia and the group of other cases. (Hallgren, 1950, p.8).

Hallgren failed to specify the extent of the discrepancy necessary to qualify as a disabled reader. As he did not have access to standard reading tests for his diagnosis of specific reading disability, he gleaned information from parents concerning possible reading and writing disabilities which their children may have had.

Hermann (1959) enumerated four characteristics which support the retention of developmental dyslexia as a single clinical category distinct from mere backwardness in reading:

(a) there is a definite discrepancy between the level of reading skill and other evidence of intellectual development;

(b) additional cases are often found in members of the same family;

(c) the defective performance in reading and writing is marked by rotation and reversal of letters and words and confusion and disfigurement of letters;

(d) the difficulties in reading and writing do not respond readily to remedial treatment and may persist for many years or even to adulthood in spite of good oral language development and high motivation to learn.

Critchley (1964) based his arguments for the existence of developmental dyslexia on four criteria: persistence into adulthood, the peculiar and specific nature of the errors in reading and writing, the familial incidence of the defect, and the frequent association with other symbol defects. As Reid (1969) has stated, the first of these is diagnostic only in retrospect and the fourth leaves room for considerable variation.

Rabinovitch (1968) delineated the syndrome of specific reading disability in considerable detail which included the following features:

(a) reading retardation;
(b) impairment in spelling and arithmetic;
(c) defects in visual and auditory functions;
(d) broader language deficits;
(e) specific concept-symbolization deficiency in orientation;
(f) neurological deficit.

Although there is some common ground in the writings of investigators, it is clearly apparent that different criteria are stressed by the various research workers seeking a positive definition of specific reading disability. Such ambiguity and inconsistency concerning the factors listed as characterising specific reading disability have obviously played a causal role in hindering an understanding of its nature and treatment.

Criteria of Reading Retardation

Unfortunately, no consistent criterion of reading retardation has been utilised in studies of specific reading disability and in certain instances no attempt has been made to measure or report the extent of reading retardation.

Hinshelwood (1917) provided very little evidence of the extent of the children's reading retardation other than mentioning a general inability to recognise letters or read simple and common words.

Orton (1925) did, however, base his assessment of reading retardation on objective criteria. These included the number of reversals in reading, the ratio of mirror-reading time to reading time of normally oriented text, the level of reading performance compared with expectation for mental age and competence in other subjects. However, he failed to report with any detail the measures used, the performance level indicative of reading retardation or the results obtained.

Although later researchers have tended to be more explicit in defining retardation, there is little consensus concerning what actually constitutes reading retardation.

Investigators have used either a fixed or variable criterion of reading retardation. However, the actual level set as indicating a significant disability has varied considerably. Authors who have set a particular fixed measure of retardation have selected either IQ or MA as an indicator of reading potential (Doehring, 1968; Ingram et al., 1970; Mason, 1967; Zigmond, 1966) or chronological or grade age (Boder, 1971; Naidoo, 1972; Rutter et al., 1970; Wolf, 1967). Recognising the weaknesses in defining reading retardation in terms of a fixed discrepancy between reading age and some measure of reading capacity, several writers have used a variable criterion in the form of either a sliding scale (Goyen, 1969; Kass, 1962; Rabinovitch, 1968) or a RQ (Clark, 1971; Lovell et al., 1964b).

PREVALENCE OF SPECIFIC READING DISABILITY

While the exact prevalence of specific reading disability amongst the school population is unknown, it is most frequently taken to be between four and ten per cent (Critchley, 1970; Rutter and Yule, 1973) although actual estimates have ranged from 0.05 per cent (Thomas, 1905) to thirty-five per cent (Walcutt, 1961). This wide variability in the estimation of incidence would seem to arise from differences in definitions and diagnostic criteria.

Considerable controversy has raged over the reported incidence of the disability. For example, Hinshelwood (1917) believed Thomas's (1905) estimation to be an exaggeration of the frequency and suggested that these were not cases of congenital word-blindness as he understood the term. He did not attempt to estimate its frequency but stated that it was "moderately rare". He continued:

> The truth is that this great divergence of opinion as to the frequency of the condition is simply due to the fact that some later writers have extended the term congenital word-blindness to include slight degrees of defect in the visual word centre, while the earlier writers had reserved it only for those grave cases which could be regarded as pathological. (Hinshelwood, 1917, pp. 80-81).

Obviously, subsequent writers have, likewise, not restricted their attention to the rather rare, grave and pure cases that Hinshelwood studied and reported. By using a less stringent definition, these researchers have included other types of disabled readers in their study populations and hence have reported a higher incidence of the disability. Table 2 lists the frequency estimates of specific reading disability reported by a number of investigators.

TABLE 2

REPORTED INCIDENCE OF SPECIFIC READING DISABILITY

Author	Date	Country	Frequency (%)
Thomas, C.	1905	England	0.05
Illing, E.	1929	Germany	0.07
Makita, K.	1968	Japan	0.98
Matejcek, Z.	1968	Czechoslovakia	2
Rabinovitch, R.	1968	U.S.A.	2-3
Orton, S.	1928	U.S.A.	2-4
Ronne, H.	1937	Denmark	2-4
Tarnopol & Tarnopol.	1976	U. S.A.	3
Hagger, D.	1970	Australia	3-5
McLeod, J.	1966	Australia	3.25
Rutter, M. et al.	1970	Isle of Wight	3.7
Wallin, J.	1921	U.S.A.	4.5
Walsh, R.	1976	Australia	5
Critchley, M.	1970	England	5-10
Bender, L.	1963	U.S.A.	5-15
Childs, S.	1959	U.S.A.	5-25
Silver, A. & Hagin, R.	1960	U.S.A.	5-25
Malmquist, E.	1958	Sweden	8.5
Hallgren, B.	1950	Sweden	10
Hermann, K.	1959	Denmark	10
Sinclair, A.	1948	Scotland	10
Thompson, L.	1969	U.S.A.	10
Tamm, A.	1924	Sweden	11
White, J. & White, M.	1972	Australia	18.7
Walcutt, C.	1961	U.S.A.	20-35

Critchley (1964) commented that the greatest frequency of developmental dyslexia was in English-speaking and Scandinavian countries and noted that the disorder was found to a lesser degree in other countries including Germany, Italy, Russia and Rumania. Cases of specific reading disability have also been reported in Chinese (Kline and Lee, 1969), Spanish (Strong, 1972, 1973) and Polish (Markiewicz and Zakrzewska, 1973) children. Other writers have referred to the universal nature of the disorder (Matejcek, 1968; McLeod, 1972; Rawson, 1972). Of the many reasons offered to explain the differences in the reputed incidence of the disability, some authors suggest that the variant levels of complexities of different languages play an important role (Claiborne, 1906; Hermann, 1959; Ingram, 1971; Maruyama, 1958; Matejcek, 1971).

Other factors which may account for the differences in cross-national estimates of specific reading disability have been discussed in Section 2.34 of Chapter 2. Makita (1968) commented that out of 9,195 school children in Japan, slightly less than one per-cent were reported by their teachers as having a reading disability. He attributed this extremely low incidence of dyslexia primarily to the regular relationship between Japanese Kana script and pronunciation. He concluded that reading disability is "more of a philological than a neuropsychiatric problem." (p.613).

Certain authors, however, warn that the findings of this study could be due to certain socio-cultural factors, which include the absence of information on diagnostic services in Japan, a general lack of experience with reading disability and hence an insensitivity to the problem and the traditional tendency amongst Orientals to "save face" (Critchley, 1970; Leong, 1972). Despite these observations, Makita's article emphasises the importance of phonetic irregularities and ambiguities in the formation of reading disability.

Most writers report that significantly more boys than girls suffer from specific reading disability (Bannatyne, 1971; Hinshelwood, 1917; Ingram et al., 1970; Klasen, 1972; Orton, 1943; Rutter et al., 1970; Wallin, 1921). A few authors, however, were unable to find a marked sex difference (Hallgren, 1950; Jastak, 1934; Malmquist, 1958). Amongst other conclusions, Hallgren (1950) stated that "... the calculations thus indicate that the sex distribution of specific dyslexia in the normal population does not differ appreciably from the normal sex distribution." (p.185).

In a group of children with "special reading disabilities", Malmquist (1958) found that although the number of boys was greater that that of the girls, the sex difference was not statistically significant.

Critchley (1970) summarised the findings of fifteen authors and although their estimates varied, the sex-incidence of specific reading disability appears to be approximately four males to one female. He strongly favoured a genetic interpretation of these findings.

Some writers see nothing of fundamental importance in this finding, suggesting that, for various socio-cultural reasons, boys with reading difficulties more frequently come to the attention of the investigators than do girls similarly affected (Goodacre, 1968; Hallgren, 1950).

Other authors have advanced more explicit explanations. These include the psycho-dynamic meaning of reading which may be different for the two sexes (Blanchard, 1946), the possible detrimental effect on boys of a sequence of female infant school teachers (Rutter et al., 1970;Wyatt, 1966) and boys' greater resistance to school and discipline (Pringle et al., 1966).

Wyatt (1966) lists other reasons that have been advanced to explain the different sex distribution of the disability. They include: (a) basal reader materials are less motivating and less satisfying to boys than to girls; (b) the school environment at the primary level is more frustrating to boys than to girls; (c) boys are less motivated to read because they identify reading as a feminine activity.

A number of authors have commented on the existence of a biologically-determined, developmental difference, between the sexes. Boys have been shown to develop physically at a slower rate than girls from before birth to adulthood (Flory, 1935; Tanner, 1960, 1968). It has also been suggested that the biological immaturity of males leads to greater vulnerability to various forms of stress and that the reading retardation constitutes a stress response resulting from an interaction between biophysical immaturity and the social and psychological demands of society on the child (Bentzen, 1963).

According to McCarthy (1954) and Terman and Tyler (1954), one of the most consistent findings to emerge from the data gathered on the acquisition of language skills in children is the slower development in boys of almost all aspects of language, including reading. Furthermore, there is a greater prevalence of males among the speech and linguistic defective populations (Eisenson, 1958; Ingram, 1959; Myklebust and Boshes, 1960; Rutter and Yule, 1973; Travis, 1957).

As Shankweiler (1964b) stated, research on the sex differences in the maturation of language functions (Kimura, 1963) and sensory development (Criscuolo, 1968; Ghent, 1961) provides momentum for the closer comparative examination of dyslexic boys and girls.

AETIOLOGY OF SPECIFIC READING DISABILITY

Implicit in a current popular opinion of specific reading disability is the assumption that the disorder is due to central nervous system dysfunction (Clements, 1969; Critchley, 1970; Rabinovitch, 1968; Satz and Sparrow, 1970; Tec, 1971). The inadequate operation of the central nervous system may be the result of structural damage or functional delay, which in turn may be caused by one or more factors including injury, trauma, disease, maturational delay or genetic factors (Critchley, 1962; McGrady 1968; Myklebust and Johnson, 1962). Each of these hypothesised aetiologies has been advanced as a cause of specific reading disability.

Brain Damage

It has long been known that the acquisition of reading ability and actual reading ability may be impaired by brain damage. In fact, the recognition of congenital word-blindness was the direct outcome and result of the previously gained knowledge of the symptoms of brain pathology in adult patients (Critchley, 1970). Kussmaul (1877) is usually acknowledged as being the first investigator to isolate a loss of the ability to read in adults. He termed this disability "word-blindness".

The term "dyslexia", first suggested as an alternative term for "word-blindness" by Berlin (1887), was defined by Bateman (1890, in Critchley, 1964) as "... a form of verbal amnesia in which the patient has lost the memory of the conventional meaning of graphic symbols." (p.1). Dejerine (1892, in Critchley, 1970) attributed this syndrome to a lesion in the medial and inferior portions of the left occipital lobe and also surmised that a destruction of the fibres connecting the two occipital lobes was a significant feature.

It is not surprising that children with congenital word-blindness were suspected of having lesions similar to those described in adults who had lost the ability to read. The possibility that some structural defect in the brain could inhibit learning to read was mentioned in the pioneer work of Claiborne (1906), Fisher (1910), Morgan (1896), Thomas (1905) and Wallin (1921). However, not one of these researchers or, as far as can be ascertained, any subsequent proponents of this theory have presented any direct evidence for brain lesions in cases of developmental dyslexia.

Renewed interest in the possibility that neurological defects underlie reading disability was aroused by investigators who were studying the learning difficulties of children known to be suffering from brain damage. For example, Lord (1937) showed that many of the features described in specific reading disability were also to be observed in children suffering from cerebral palsy.

During the following decade Strauss and associates (1942, 1947) and Gesell and Amatruda (1947) studied children with minimal brain injury who displayed combinations of various irregularities of learning, psychological function, behavioural disorders and neurological abnormalities.

Strauss and Lehtinen (1947) commented that the failure or partial failure to master reading processes is an experience shared by a large number of brain-injured children. They held that disturbances in perception and behaviour caused by brain injury result in difficulties in academic learning.

Gesell and Amatruda contended that:

> In many instances the symptoms of minimal injury are so benign that they escape attention... The type which expresses itself in speech difficulties, poorly defined unilateral dominance, and in delayed integration may later result in serious difficulty in the acquisition of reading. (Gesell and Amatruda, 1947, pp.247-248).

As Ingram (1969) stated, the findings that a high proportion of children who suffer from cerebral palsy and other manifestations of brain damage are slow to speak and have difficulty in learning to read have given further rise to speculation as to the causes of reading difficulties in children.

Weir and Anderson (1958) found neurological evidence of brain damage in approximately seventy-five percentage of 181 children with problems of school failure, especially in reading, speech and personality characteristics. Cohn (1961) compared a group of forty-six children having reading and writing problems with 130 randomly chosen normal children aged seven to ten years. He found striking differences, favouring the normal readers, in right-left orientation, double simultaneous tactile stimulation, the patellar reflex, the Babinski sign, motor co-ordination, the mechanics of speech and EEG findings. Cohn considered that the reading difficulties in this group were not isolated phenomena dependent on a specific lesion but the result of a generalised brain dysfunction which was responsible for a slow rate of maturation.

Myklebust and Johnson (1962) reported positive evidence of brain dysfunction in approximately seventy-five per cent of children with dyslexia whom they studied. Prechtl and Stemmer (1962) investigated a group of fifty hyperkinetic children from nine to twelve years of age who showed choreiform movements. It was found that ninety per cent of these children with suspected minimal brain damage had trouble in reading. It was suggested that these defects in their neural functioning may also lead to a lag in the development of cerebral dominance and to a delay in the development of complex functions, such as reading.

Zigmond (1966) conducted neurological tests on dyslexic and normal children aged nine to twelve years. It was found that a significantly greater proportion of dyslexic children were classified as being deficient in neurological integrity, the implication being that there is more likelihood of finding positive neurological signs in a child who is not achieving in reading despite intellectual, sensory and emotional integrity than in a normal reader.

0ther investigators including Clements (1969), Crosby and Liston (1969), Rabinovitch (1968), Shankweiler (1964a) and Silver and Hagin (1964) have reported evidence of positive neurological signs in dyslexic children. This particular view is supported by studies which have shown a high incidence of pre-natal and para-natal complications in the history of children with reading problems.

The presence of maternal and foetal factors has been implied by the studies of Eustis (1947), Fisher (1910), Kinsbourne and Warrington (1963), Lyle (1970) and Prechtl (1962) and these in conjunction with motor, neurological and learning deficits strongly suggest neurological dysfunction.

In an extensive search of hospital records, Kawi and Pasamanick (1958) showed that out of a series of 205 children with reading retardation, there had been gestational complications in a number of cases. For instance, preeclampsia, bleeding and hypertension were indicated in 16.6 per cent, while in a control group of normal readers, maternal incidents of this kind occurred in only 1.5 per cent. From these findings they suggested that severe brain damage leads to stillbirth, abortion and neo-natal death. In a descending gradient, lesser traumas can result in cerebral palsy, epilepsy and behavioural disorders, whilst the most benign form of brain damage is followed by faulty speech and developmental dyslexia. They concluded "... it would appear that a certain proportion of reading disorders might be added to the continuum of reproductive casuality." (p.1423).

Certain writers have sounded a cautionary note regarding the occurrence of organic defects in dyslexic children. Bryant (1964) held that the dyslexic child does not necessarily show gross defects on either neurological or pediatric examination. Hermann (1959) emphasised that in congenital word-blindness no abnormality in the brain can be demonstrated through observation with the naked eye or through microscopic examination. Rabinovitch (1962) contended that dyslexia may be present without definite brain damage being indicated in the case-history or upon neurological examination.

In 1970, Ingram et al. published a study of eighty-two children, sixty-six of whom they considered to suffer from "specific developmental dyslexia". An attempt was made to classify them according to the likelihood of their suffering from brain damage. An investigation of their developmental histories and detailed neurological examinations revealed abnormal signs in only twenty-two per cent of the cases. While confirming to some extent previously reported findings, their study also clearly established that specific reading disability can be present without any clinical evidence of brain abnormality. Obviously, the assumption that specific reading disability is necessarily associated with organic brain defect is unfounded.

It is difficult to determine to what extent neurogenic dysfunction represents actual damage to the central nervous system in the absence of gross neurological deficits. The conclusion that dyslexic children suffer from brain injury or have minimal brain damage is still made partially on a presumptive basis according to negative diagnosis, or diagnosis by exclusion.

Findings in clinical populations (that is, severely retarded readers referred to a reading clinic) tend to be more positive while findings in an unsolicited survey population usually remain negative. This is not surprising since the referred clinic population probably samples the extreme end of the distribution of reading disabilities and presumably includes cases of acquired dyslexia which are found much more frequently than in a normal sample. In addition, the types of problems studied at any particular clinic reflect the services provided and the factors influencing referral to it and these will tend to bias any attempt to examine associations between different variables.

Critchley (1970) asserted that most neurologists would be reluctant to envisage any focal brain lesion - dysplastic, traumatic or otherwise - in developmental dyslexia despite the fact that cases of acquired alexia result from cerebral damage. After reviewing the evidence he believed that the theory of minimal brain damage is unconvincing for three reasons: it conflicts with the factor of inheritance; it ignores the notion of plasticity of developing nervous systems; it is unsupported by documented evidence of structural damage or alterations.

However, it should be acknowledged that many investigators, including Critchley (1964) and Gooddy (1967), would emphatically exclude a child with evidence of brain damage from the category of specific reading disability, although, of course, brain damage and dyslexia may occur together incidentally in an individual case. Such children possess another variant of reading disability referred to as "symptomatic dyslexia" (Critchley, 1970) or reading retardation secondary to brain injury (Eisenberg, 1966; Geschwind, 1962; Keeney, 1968; Rabinovitch, 1962).

In summary, the evidence from studies investigating the presence of "hard" or "soft" neurological signs of brain damage amongst dyslexic children has been contradictory or ambiguous. As Benton (1975) has recently stated, "... a neurological basis for developmental dyslexia has not been established, the empirical evidence bearing on the question being both inconsistent and circumstantial." (p.37).

Maturational Delay

The concept of immature functioning of the brain as a causal explanation of developmental dyslexia has a long heritage. Controversies, however, occur in explaining these immaturities, their origin and their relationship to reading retardation. This notion can be found in the writings of several authors. Hinshelwood (1895) postulated a lag in the maturational development of the angular gyrus region in the left cerebral hemisphere as the physiological mechanism in congenital word-blindness. Apert (1924, in Orton, 1925) reporting on a case of "congenital familial dyslexia", believed it to be caused by a developmental delay of functional, not anatomic, origin. Pick (1924, in Orton, 1925) listed language difficulties including congenital word-blindness and stated emphatically that these disorders were due to developmental delays, not brain lesions.

More than any other research worker, Orton (1925) was responsible for espousing the concept of developmental delay and the suspected underlying defective physiological mechanisms. He viewed language as an evolutionary human function associated with the development of a hierarchy of complex integrations in the central nervous system and culminating in the unilateral control by one of the two brain hemispheres (cerebral dominance). He hypothesised that retardation in acquiring reading was caused by some physiological variation in this natural process of growth and development.

Obviously influenced by Orton's work, Schilder (1944) and Bender (1958) considered developmental dyslexia to be a manifestation of a congenital disturbance in the gestalt function with the resulting outcomes: right-left confusion, a tendency to reverse words, difficulty in recognising single letters and an inability to dissect the spoken word into its constituent sounds and to combine the sounds of a word. This disturbance was believed to be the product of immature and poorly differentiated neurogenic patterns. Bender (1963) regarded a maturational lag to be based on the concept of functional areas of the brain and of personality developing according to a recognised pattern. A maturational lag signifies a slow differentiation within this pattern. "It does not indicate a structural defect, deficiency, or loss. There is not necessarily a limitation in the potentialities and at variable levels maturation may tend to accelerate, but often unevenly." (p.30). She suggested that these cases are understandable in terms of "embryonic plasticity", that is, as yet unformed, but capable of being formed, being impressionable and responsive to patterning and possessing the potentialities of patterns, as yet, unfixed.
Eustis (1947) stated that "... a slow rate of neuromuscular maturation, by prolonging the normal period of spatial confusion in hand and eye, seems to be the primary cause of specific reading disability." (p.454). In 1954, Rabinovitch and associates concluded that primary reading retardation is due to a developmental dysfunction (primarily involving the parietal and parietal-occipital regions) rather than acquired brain injury.

Drew (1956), a neurologist, elucidated minor neurological abnormalities in three patients whom he considered had familial dyslexia. He indicated that the reading disorder was due to a delayed development of the parietal lobes and reveals itself in a disturbance of gestalt recognition.

In 1959, the Danish neurologist, Hermann, hypothesised that specific reading disability was caused by an underdevelopment of the "directional function". The developmental uncertainty reflected itself in confusion between left and right, finger agnosia and also by certain elementary types of oral reading errors.

Kinsbourne and Warrington (1963) identified, among backward readers and writers, two groups with developmental defects reminiscent of acquired cerebral syndromes in adults, which were referred to as the "Gerstmann" group and the "language retardation" group. These authors emphasised that their comments in no way implied that the child with an analogous developmental syndrome to the acquired syndrome in adults, has a similarly localised cerebral lesion, or any gross cerebral damage at all. However, at the functional level striking similarities between the two syndromes were noted.

The concept of a lag in the maturation of the brain and nervous system continues to gain acceptance as a broad causal explanation for specific reading disability, partly because of the failure of neurological studies to yield evidence of structural brain damage.

Money, for example, has stated that:

> The great majority of reading disability cases will be classifiable not on the basis of brain pathology, but simply as representative of a lag in the functional development of the brain and nervous system that subserves the learning of reading. (Money, 1966,p.34).

Money stressed that a maturational delay does not necessarily have a single cause. It may be the result of several different factors such as heredity, specific neuro-psychological deficit and experiential deprivation.

In 1961, Critchley concluded that the principal neurological deficits associated with specific reading disability point to a parieto-occipital dysfunction and a state of ambilaterality, which imply a certain lack of maturation. He reiterated this point in 1964 stating that "... both cerebral ambilaterality and dyslexia are to be equated with immaturity of cerebral development, is the view most widely held today among neurologists." (p.53). This statement lends confirmation to opinions expressed earlier by Bender (1963), Gesell and Amatruda (1947), Gooddy and Reinhold (1961), Orton (1925), Rabinovitch (1962), Schilder(1944), Zangwill (1962) and many other writers.

Birch (1962) further elaborated the role of a maturational lag in developmental dyslexia. He postulated that reading disability may stem from the inadequate development of appropriate hierarchical organisation of sensory systems. One of the essential features in the development of reading readiness mentioned by Birch is for the visual system to become hierarchically dominant. "Failure for such dominance to occur will result in a pattern of functioning which is inappropriate for the development of reading skill." (p.164). Birch (1962) also conceived of a developmental sequence of perceptual levels beginning with discrimination followed by analysis and then synthesis. He believed that reading disability may result from the inadequate development of the higher and more complex levels of visual perceptual functioning.

Delayed maturation of the ability to perform cross-modal tasks is believed to be the factor responsible for some cases of language disability, including reading disability (Birch and Belmont, 1964; Blank and Bridger, 1966).

De Hirsch et al. (1966) studied reading failure in young children. A large proportion of the pre-school children referred for a variety of oral language defects developed reading, writing and spelling difficulties several years later. The clinical impression gained of these children was one of striking immaturity. In spite of adequate intelligence their performance on a variety of perceptual-motor and language tasks resembled that of younger children.

A study conducted by Ingram et al. (1970) provided further evidence for the view that specific reading disability may be associated with a developmental lag in basic perceptual or motor functions. They found that dyslexic children performed poorly on a range of visual and auditory tasks and on certain motor activities.

Satz and Sparrow (1970) hypothesised that a lag in maturation of the functioning of the left hemisphere underlies specific reading disability. They extended this concept further by specifying a developmental schedule of lateralised functions, proceeding from the lateralisation of gross and fine motor skills to sensory-motor functions and finally to the lateralisation of speech and language. This maturational lag hypothesis makes it possible to integrate the diverse patterns of deficits observed in dyslexic children. It also provides an explanation for some of the contradictory observations made with different populations and at different age levels by integrating them into a developmental schedule.

Of the different hypotheses advanced to explain specific reading disability, several authors continue to believe that the theory of cerebral immaturity warrants the most serious attention (Critchley, 1970; Naidoo, 1972; Rawson, 1971).

Thompson (1969) supported this view and postulated on the possible cause of the "biophysical immaturity." He stated:

> In the majority of cases, reading disability is due to a physiological developmental or maturational lag which in itself may be accompanied by other signs of immaturity... That the developmental lag has an hereditary anlaga [sic], predisposition, or diathesis cannot be denied. That minimal or "soft" insults to the brain from various sources during the perinatal period and early infancy might enhance or even produce the developmental lag cannot be disproved. (Thompson, 1969, p.111).

The developmental lag hypothesis would regard "soft" signs such as inadequate cerebral dominance, spatio-constructional confusion and motor inco-ordination as epiphenomena. Moreover, this theory would explain why it is that these minor signs are rarely demonstrated in older dyslexics. The difficulties in reading persist, however, because the opportunities for learning basic reading skills have usually passed.

Genetic Factors

Many researchers contend that the maturational lag results from an inherited predisposition. A relationship between genetic factors and specific reading disability has long been suggested. Numerous clinical studies have either shown or suggested a familial incidence of reading problems (Critchley, 1970; Drew, 1956; Eustis, 1947; Fisher, 1905; Hinshelwood, 1917; Klasen, 1972; McCready, 1926; McGlannan, 1968; Orton, 1937; Skydsgaard, 1942; Sladen, 1972; Thomas, 1905).

The question of an hereditary tendency in developmental dyslexia was systematically investigated by Hallgren (1950) in an extensive genetic study. He studied 276 cases of specific dyslexia - 116 affected children and 160 secondary cases (siblings and parents of the affected) and found indications of dyslexia in eighty-eight per cent of the families. He concluded that he had established the genetic transmission of this abnormality.

These conclusions, however, may be challenged on several grounds. Hallgren relied more on the history of reading and writing disturbances in selecting this group than upon the contemporary status of these skills. Consequently, many relatively mild and "recovered" cases were included. The absence of explicit quantitative criteria for selection and the extension of the dyslexia concept to embrace mild grades of reading disability also makes these findings difficult to interpret.

The data on Hallgren's secondary cases were for the most part from retrospective interviews. The reliability of retrospective information has been questioned (Robbins, 1963). Furthermore, familial incidence of a given type of behaviour does not necessarily mean that the abnormality is genetically determined. As Owen (1968) and Rutter (1969) have suggested, the family relationships and subsequent social learning within the family may explain common patterns of behaviour.

More convincing support for constitutional factors in reading disability comes from the study of twins. Hermann (1959) presented data showing that in twelve pairs of monozygotic twins there was complete concordance regarding dyslexia. Of the thirty-three pairs of dizygotic twins concordance was found in only eleven pairs. Since all the uniovular twins in these studies showed concordance, the role of heredity as a critical aetiological factor in certain types of learning disabilities can be postulated. As a result of his investigation Hermann reported:

> Of various contributory causes of congenital word blindness, only one factor is found to be invariable, viz. heredity, which must be regarded as the specific pathogenic factor. (Hermann, 1959, p.106).

Critchley (1962) stated that it was unusual to find a dyslexic child who did not have a family with one or more similarly afflicted members. Bryant and Patterson (1962) asserted that, in many cases, probably more than half of their interviews would reveal that other family members have a record of reading disability. Doehring (1968) found that forty per cent of the parents of his dyslexic children had experienced reading problems, as against ten per cent of normal controls.

In a series of studies Owen, Adams, Forrest and Fisher (1968) reported a relationship between familial antecedents and reading disability. These authors, using a range of psychological, neurological and educational measures, showed highly significant familial similarities between educationally handicapped children and their siblings in learning patterns, learning deficits, neurological immaturities and school behaviour.

Newton (1970) found genetic factors in fifty-five per cent of her cases. Rutter et al. (1970) also showed that a family history of reading difficulties, in either siblings or parents, was more prevalent in the children with specific reading retardation than in a control group.

De Hirsh et al. (1966), however, failed to obtain a significant correlation between reading achievement and any familial characteristics in the total sample studied which included "failing readers", "slow starters" and "superior achievers". The authors believed that this finding may have been attributable to a lack of accurate information. Furthermore, as no separate data are provided for the eight "failing readers", the familial incidence of severe reading disability is not ascertainable.

Although a family history of reading disability has been used to argue that reading difficulties are hereditary in origin, other explanations, especially sociological ones, are highly plausible (Ravenette, 1971; Rutter et al., 1970). As Ravenette (1971) has stressed, genetic transmission is transmission of physiological attributes, not psychological: "...whilst it is true that genetic factors underly many aspects of human appearance or behaviour, many more aspects are primarily the reflection of psychological processes." (p.108).

In discussing the notion of the genetic origin of dyslexia, Goldberg and Schiffman (1972) stressed that no chemical or other objective proof that dyslexia is genetically determined has as yet been advanced, nor would a biochemical change in a dominant disorder be expected.

After reviewing the literature, Benton (1975) and Vernon (1971) concluded that there is evidence of a hereditary predisposition in some cases of developmental dyslexia.

It should be noted that it is not always implied that genetic factors are the sole determinants of specific reading disability. For instance, Orton (1937) emphasised the importance of environmental forces on the child. In fact, he argued for the use of the term "developmental", instead of the original term "congenital", because it accounted for both endogenous and exogenous factors. While Orton clearly recognised and accepted the importance of heredity, he felt that the use of the term "congenital" "... tends to overstress the inherent difficulty and to under-emphasize the many environmental factors, both specific - such as methods of teaching - and more general - such as emotional and social forces." (p.69).

CLASSIFICATION OF SPECIFIC READING DISABILITY

It was generally assumed that children with developmental dyslexia constituted a homogenous group (Boder, 1971). However, some investigators have indicated that children with specific reading disability are heterogeneous both aetiologically and clinically (Bannatyne, 1971; Ingram, 1960; Kinsbourne and Warrington, 1966; Klasen, 1972; Wolf, 1967).

Shankweiler (1964b) contended that developmental dyslexia should not be regarded as a unitary condition. It is necessarily a generic concept which refers to a number of types of reading disability which may or may not be related in aetiology and in the underlying disturbance of function. He added:

> Clearly, a careful delineation of the various dyslexia syndromes is of the greatest importance for treatment. At present there is little solid knowledge of any of these syndromes and only hints as to the ways in which different types of reading disability are linked to salient features of development. (Shankweiler, 1964b, p.61).

A few attempts have been made to delineate the subgroups among dyslexic children. Ingram (1960) distinguished three different clinical syndromes of developmental dyslexia from reading and spelling errors. Some children had predominantly "visuo-spatial" difficulties, others had mainly "correlating and synthesising" problems" and others had "dysphasic" difficulties.
Ingram et al. (1970) divided their sample of eighty-two highly pre-selected dyslexic children into two categories: the "specifics" (N=62) whose disability was limited to reading and spelling, and the "generals" (N=20) who also performed below expectation in arithmetic. While both groups made audio-phonic and visuo-spatial errors in oral reading, the "specifics" made significantly more audio-phonic errors.

Silver and Hagin (1960) identified two major subgroups of dyslexia amongst 150 disabled readers. Ninety-two per cent of the total cases were found to have perceptual deficits, mainly of a visual nature. The remaining eight per cent of cases were free from perceptual deficits but had emotional disorders. The authors also noted that twenty-two per cent of the children with perceptual problems showed positive signs of specific organic defects.

Benton (1962) proposed that there are at least two varieties of developmental dyslexia, one type being associated with perceptual disorders (for example, difficulties in the apprehension of shapes and shape relations) and another type linked with conceptual or language anomalies.

Kinsbourne and Warrington (1963) studied a group of thirteen backward readers who showed a discrepancy of more than twenty points between their verbal and performance IQs on the WISC or the Wechsler Adult Intelligence Scale (WAIS) and delineated two distinct subgroups. Group 1, a language-retarded group, with a low verbal IQ showed other clinical evidence of language disorder that was almost completely absent in Group 2, the Gerstmann group. The latter group, with a lower performance IQ, exhibited specific difficulty with tests of finger differentiation and order, right-left discrimination, mechanical arithmetic and constructional tasks. The two groups are viewed by Kinsbourne and Warrington as syndromes of developmental cerebral deficit based on difficulties in language skills and in sequential ordering respectively. They suggested that each syndrome may give rise to a characteristic type of delay in learning to read and write.

On the basis of assessment of perceptual and motor abilities Shankweiler (1964a) divided his series of twelve cases of reading disability into three groups: (a) those cases in which dyslexia occurred in relatively pure form; (b) those cases in which dyslexia was accompanied by marked spatial and constructional disabilities; (c) an expression of a general disturbance of language development. He concluded that developmental dyslexia should not be viewed as a unitary clinical entity.

Bannatyne (1968) identified two main subgroups among dyslexic children: genetic dyslexia and minimal neurological dysfunction dyslexia. The first group comprise pure dyslexics whose disability is essentially an inherited lack of ability to use language fluently. The condition is characterised by difficulty with fine auditory discrimination, auditory sequencing and the association of sound sequences with visual symbols. The second dyslexic group was differentiated from the genetic group in that they generally have difficulties in a variety of functions: visuo-spatial, auditory, motor-kinesthetic, tactile-integrative and conceptualisation.

Wolf (1967) delineated three syndrome patterns of specific dyslexia as a result of scores on the WISC: Group 1 was low on the Verbal Scale, Group 2 was low on the Performance Scale, and Group 3 was approximately equal on both scales. Johnson and Myklebust (1967) described two forms of dyslexia based on differing symptomatology. The two syndromes manifested deficits in the auditory and visual processes pre-requisite to reading. The visual dyslexic tends to be characterised by slow and faulty visual discrimination, poor retention of form and sequence, analysis and synthesis defects, letter reversals and inversions and poor whole-word recognition. Auditory dyslexics often find difficulty in auditory discrimination, retention, analysis and synthesis. It should be noted that Johnson and Myklebust (1967) did not provide empirical evidence for their claims.

Other authors who also postulated that the two major syndromes of dyslexia are either visual or auditory in nature include De Quiros (1964) and Holroyd and Riess (1968).

By studying the diagnostic patterns in the total reading and spelling performance of dyslexic children, Boder (1971) identified three clinical syndromes: dysphonetic (a sound-symbol deficit), dyseidetic (poor memory for visual gestalts) and a mixed group who possessed both defects.

McGlannan (1968) postulated the existence of three types of dyslexia: (a) genetic dyslexia; (b) specific dyslexia; (c) complex dyslexia. Klasen (1972) considered that dyslexia constitutes a varied syndrome with multiple causation and proposed the following classification: (a) somatogenetic dyslexia; (b) psychogenetic dyslexia; (c) sociogenetic dyslexia.

Naidoo (1972) studied ninety-eight boys retarded in reading and spelling. A cluster analysis was carried out to discover whether subgroups of dyslexia could be identified. No definite clusters or groups emerged to support the existence of clearly distinguishable subgroups. Instead there appeared to be a continuum, at one end of which there was a predominance of boys with a family history of reading or spelling difficulty while at the other was a small number of boys in whom there was evidence suggestive of neurological dysfunction, but with no family history.

Because of these findings, Naidoo believed that only a multiple aetiology would account for the observations in many boys. She added that varying aetiological factors seem to be associated with different patterns of disability, but concluded that these patterns are insufficiently distinct to allow a clear-cut identification of subtypes.

Mattis et al. (1975) studied eighty-two children and young adults aged between eight and eighteen years who were classified as either: (a) brain damaged dyslexics (N=53), or (b) non-brain damaged dyslexics (N=29). As a result of neuro-psychological testing, three main syndromes of dyslexia were identified: (a) language disorder; (b) articulatory and graphomotor dysco-ordination; (c) visual perceptual disorder.

Whether these findings will decrease the need for research into the delineation of types of specific reading disability that many investigators have stressed is necessary (Critchley, 1970; Ingram, 1969; Rutter et al., 1970; Spreen, 1970; Vernon, 1965) remains to be seen. The issues involved are not only of theoretical import but crucial to the planning of appropriate and effective remedial programmes. Such analyses will provide a basis for the classification of dyslexic children into subgroups and the specification and remediation of their particular disabilities.

SUMMARY

The task of adequately evaluating the research related to specific reading disability is extremely difficult because investigators have usually employed different diagnostic criteria. As Ravenette (1971) reported, diagnosis is a private and personal matter. He added:

> (because) the presentation of 'specific developmental dyslexia' is like a theme with variations where the theme is never formally stated, it has to be deduced from the context in which it appears. (Ravenette, 1971, p.106).

Indeed, very few authors have selected children in accordance with any generally accepted criteria

The early writers generally assumed, because of teachers' or parents' reports, or clinical observations, that their subjects had at least average intelligence. Other researchers included in their sample children with IQ ratings below 90. Moreover, many authors included in their studies children with concurrent pathologies, for example, neurological dysfunctions, speech disorders, sensory defects or behavioural problems.

By not restricting their attention solely to genuine cases of specific reading disability, these authors are not likely to have contributed very much to its understanding. Any unique or distinctive features of specific reading disability would most probably be masked or distorted by factors commonly associated with other forms of reading problems.

Although specific reading disability can obviously co-exist and be aggravated by other academic or environmental circumstances, a positive diagnosis in any particular instance can only be made with confidence when factors known to inhibit the learning of reading are shown not to have played a causal role. As Benton (1975) observed, the literature is marked by the failure of many researchers to distinguish specific reading disability from reading difficulties that are due to extrinsic causes.

Hence, after more than seven decades of clinical study and research, often involving interdisciplinary collaboration in the fields of medicine, psychology and education, the nature of specific reading disability still remains a matter of considerable controversy.

The review also shows that there is great variation in the estimated frequency of the disability. This is not unexpected because of the different criteria used to delimit the disability and the different ages at which children were studied. Apart from the Isle of Wight study (Rutter, et a1.,1970) there have been no large-scale surveys reported in the literature reviewed.

The most common estimate made by investigators from a number of different countries seems to be between two per cent and five per cent of the total school population. It should be reiterated that children with specific reading disability form only a small proportion of the total number of retarded readers. There is general agreement that a much greater incidence of the disability occurs in boys than in girls.

Researchers have to date been unable to specify any pathognomonic sign or cluster of signs invariably co-existing with dyslexia. This finding lends support to the notion that there are different varieties of dyslexia, recognisable by different patterns of disability which may or may not be related in aetiology and in the underlying disturbance of function.

That there are different manifestations of specific reading disability or different accompanying patterns of disorders is not surprising. Obviously, all disabled readers are not failing to read because of the same faulty skill or ability (for example, visual discrimination, auditory memory or visual-auditory associations). Reading is a complex process requiring for its elaboration the normal functioning of a host of factors including visual and auditory processes, any one of which may be disturbed, faulty or underdeveloped for a number of biological or socio-cultural reasons. Consequently, the behavioural profiles of disabled readers are likely to show considerable variability.

Specific reading disability is currently viewed as a learning disability of variable aetiology. Several writers consider it to be a manifestation of inherited traits. Some attribute it to subtle or minimal neurological defects or injury of brain structure or delay in development of brain function. Others contend that the disability results from a number of different factors which are usually found in combination. Some of these are constitutional or intrinsic; others depend upon the social and educational milieu of the child.

It is not adequately stressed, however, that causal factors rarely operate in isolation. It is widely accepted that behaviour is dependent upon inherent genetic endowment, upon modifications of brain growth by illness or injury and the manner in which environmental influences interact with the individual.

As the review illustrates, because most authors have been motivated primarily to search for an intrinsic or endogenous cause of specific reading disability, they have tended either to ignore or to minimize the effects of exogenous factors. For instance, factors such as adverse conditions within the school and the home may operate either singly or conjointly to prevent or disrupt learning. Although it is seemingly implied by investigators that environmental factors have not contributed to specific reading disability, very few authors have provided evidence to show that such is the case.

Although concerned about this lack of evidence, Vernon (1971) contended that the defects associated with specific reading disability are "not in the main of the type which could be caused by environmental handicaps; thus their occurrence provides additional evidence for the constitutional nature of the disability." (p. 130).

Though it is still the object of active research and considerable topical interest, specific reading disability, not surprisingly, remains surrounded by a number of obscurities regarding its precise nature, its diagnosis, prevalence, aetiology, prognosis and appropriate educational management.

PHASE 2 (1977 to Present)

I am not attempting to replicate the very detailed literature review presented in Phase 1, but will highlight some major developments that have occurred over the past 37 or so years.

The literature reviewed in Phase 1 helps to provide a useful historical background and identifies some important issues and controversies in the field of dyslexia.

While dyslexia continues to be a very popular field for research from many different perspectives, important developments are most apparent in the following areas-

- Definitions
- Cognitive Deficits
- Neurological and Genetic Bases
- Treatment

Definitions

Basically, most researchers and writers continue to follow the accepted tradition of defining dyslexia as an unexpected underachievement in reading and other literacy skills, despite many integrities including average or above intellectual ability. Most definitions now specifically state that dyslexia is a neurological dysfunction with genetic origins.

Coltheart (2010) chairman of the Dyslexia Working Party, (See below for details, pages 54-55) defined dyslexia as a language-based learning disability of neurological origin. It primarily affects the skills involved in accurate and fluent word reading and spelling. It is frequently associated with difficulties in phonological processing. It occurs across the range of intellectual abilities with no distinct cut-off points. It is viewed as a lifelong disability that often does not respond as expected to best-practice evidence-based classroom methods for teaching reading.

As mentioned above, the DSM-5 redefines Specific Learning Disorders and includes comments on the two most common types of SLD- Dyslexia and Dyscalculia (problems with maths). Dyslexia is an alternative term used to refer to a pattern of learning difficulties characterised by problems with accurate or fluent word recognition, poor decoding, and poor spelling abilities. If dyslexia is used to specify this particular pattern of difficulties, it is important to specify any additional difficulties that are present, such as difficulties with reading comprehension or math reasoning.
The severity of Dyslexia needs to be classified as Mild, Moderate or Severe.

(For extensive comments on the DSM-5 reclassification and new definition of Specific Learning Disorders, see Section-**Learning Disability**
pp. 9-13).

Despite the general agreement about the definition of dyslexia, a careful study of much research reveals that critically important selection criteria have been severely compromised. This has resulted in research samples including students with general reading problems, not exclusively those with a specific reading disability or dyslexia. Hence, some researchers are working with different groups of children and therefore, selection biases may be contriving to distort results.

To ensure that the population under investigation includes only dyslexic students is a very difficult, demanding and time consuming exercise. I am also very perturbed by other selection trends; perhaps the most common of which is a failure to control for chronological age. Too frequently, the age range of students is large, often exceeding three or more years. Such an age difference has very significant consequences, especially in young students. Including, say Grade 3 students with Grade 6 students in the same sample of dyslexic students, results in at least two distinctly different groups, who are highly likely to be at different stages on the learning to reading continuum. Having such a heterogeneous group in terms of age and nature of reading problem makes interpretations about cognitive, linguistic and academic characteristics spurious.

The keen reader is encouraged to investigate the sample characteristics of dyslexics in reported scientific studies. Such characteristics including, age, gender, IQ and reading ability, can usually be readily located in the methodology section of Journal articles. Be prepared for some surprises!

Cognitive Deficits

Researchers have refined their search for the cognitive deficits in dyslexia. Three of the more highly regarded hypotheses follow.

The Verbal Deficit Hypothesis

Vellutino and associates (1979, 1987) conducted a series of elegant visual perceptual studies on poor and good readers. They concluded that the dyslexic readers were inferior in the cross modal transfer between visual and verbal codes. In other words, they were deficient in the verbal labeling and language encoding of visual stimuli. It is argued that acquiring visual-verbal relationships is a basic skill prerequisite to learning to read.

The Phonological Deficit Hypothesis

Through the 1980s and 1990s, there has been a growing shift from the verbal deficit hypothesis to a more specific theory; that dyslexia is characterised by phonological processing difficulties. Key researchers proposing this theory include the following-Liberman and her colleagues (1974, 1989), Torgesen (1993, 1999), Snowling (1987, 2000, 2004), Stanovich and Siegal (1994).

Converging current evidence continues to supports the specific theory that dyslexic readers have phonological (speech) processing deficits. This hypothesis focusses directly on basic reading skills.

To be more specific, it is postulated that dyslexia stems from a deficit in phonological processing or difficulty in recognising that spoken words are formed by discrete phonemes (for example, the word "cat" is composed of the sounds c-a-t). As a result, affected individuals have difficulty associating these sounds (phonemes) with visual letters (graphemes) that make up written words and performing other phonological skills such as sound blending and sound analysis. Hence, there is a complex hierarchy of functions related to processing speech sounds or phonemes.

It is interesting to note that Stanovich (1993) questioned whether all the phonologically related deficits are reflective of a single underlying processing problem. I would content that the processing and retention of **sequential information** is such a critically important underlying single factor. (For a very comprehensive account of Directionality and its two components- sequencing and orientation, see the following **Sections** -**Early Learning Essentials 1 : Directionality, Learning Disability).**

Phonological skills are causally related to the normal acquisition of beginning reading skill. (Stanovich, 1993). Their role is critical and central at the learning to read stage. At later stages of reading, more sophisticated language abilities including syntactic and semantic skills and higher-order verbal thinking skills play more important roles.

Other Deficit Hypotheses

O' Brien et al., (2012) investigated types of developmental dyslexia. Their study provided support for separate subtypes of dyslexia, with and without phonological deficits.

Perez et al., (2012) uncovered a severe impairment of short-term memory for serial order information in dyslexia that cannot be reduced to a phonological processing impairment.

Elsewhere I have acknowledged the central role played by phonological processing skills in the acquisition and development of reading and spelling skills. However, I do not accept the proposition that dyslexia is caused by phonological deficits. I see a more basic and pervasive problem underpinning dyslexia-a directional confusion/uncertainty. Directionality has two components-sequence and orientation. The processing of both sequential and orientational information, visual (visual sequences and visual/spatial orientation) and auditory (auditory sequences) is involved in early reading, spelling and handwriting.

The critical role sequencing plays in the development of phonological skills can be seen in the tasks commonly used to measure such skills-

Rhyming-e.g., what word rhymes with "top"?

Sound Awareness-identifying initial, final and medial sounds in spoken words.

Sound Blending-making words out of a sequence of letter sounds (e.g., hearing "s-t-o-p-s" and saying "stops"-not spots, spot, tops, pots, post, posts!)

Sound Manipulation-removing sounds from a spoken word and identifying the new word-(e.g., take the "r" sound out of "cramp" and give the new word)

I contend that fundamental sequencing problems are at the core of most phonological deficits. Other sequencing problems are also commonly seen in children with dyslexia-serial learning difficulties, reading and spelling errors, fine-motor and fluency problems in handwriting (e.g., motor patterns or "motor melodies"), confusions with algorithms, slow acquisition of "tables" in mathematics.

As mentioned in the **Reading Section**, reading is composed of two main processes: decoding and comprehension. Decoding words has two distinct elements- auditory strategies- the constructive, methodical, step-by-step "sounding-out" of words (using phonological and phonic skills) and visual strategies -the instantaneous, visual recognition of words. In dyslexia, the original deficit occurs at the early decoding stage.

The increasing evidence of the linkage from phonological processing ability to reading skills provides the rationale for the explicit, systematic teaching of phonological skill to young dyslexic students. (For a very comprehensive account of these phonological skills and learning to read, see **Reading Section**).

Neurological and Genetic Bases of Dyslexia

There has been quite spectacular development in research into brain functioning and genetics in recent years.

Dyslexia is now firmly considered to be neurobiological in nature. (Galaburda, 1993; Grigorenko, 2000; Shaywitz and Shaywitz, 2003). It is interesting to recall that pioneer researchers including Hinshelwood and Orton hypothesised that dyslexia was a brain-based disorder with a hereditary component.

The advent of neuroimaging and neuromagnetic techniques to study brain structure and function enhanced dyslexia research in the 1980s and 1990s. Since then, interest in the neurologically based causes has persisted. Current models of the relation between the brain and dyslexia generally focus on some form of defective or delayed brain maturation. More recently, research has provided increasing evidence supporting a genetic origin of dyslexia. Interestingly, phonological processing emerges as a central causal feature under significant genetic control. (Snowling, 2005).

Galaburda et al., (2006) continued their outstanding work on genetic studies and dyslexia. Their perspective on dyslexia is that some of the brain changes cause phonological processing abnormalities as well as auditory processing abnormalities; that the latter, resolve in a proportion of individuals during development, but contribute early on to the phonological disorder in dyslexia.

Goswami (2008) contended that neuroimaging provides unique opportunities for understanding the acquisition of reading by children and for unravelling the mystery of developmental dyslexia. In this article, she offers a selective overview of recent neuroimaging studies, drawing out implications for education and the teaching of reading.

The different neuroimaging technologies available offer complementary techniques for revealing the biological basis of reading and dyslexia. Functional magnetic resonance imaging (fMRI) is most suited to localisation of function and hence to investigating the neural networks that underpin efficient (or inefficient) reading. Electroencephalography (EEG) is sensitive to millisecond differences in timing, hence it is suited to studying the time course of processing; for example, it can reveal when networks relevant to phonology verses semantics are activated. Magnetic source imaging (MSI) gives information about both localisation in the brain and the time course of activation.

The author illustrates how each technology is most suited to answering particular questions about the core neural systems for reading and how these systems interact and what might go wrong in the dyslexic brain.

Pennington (2009) provided a detail study of the neuropsychological bases of learning disabilities.

Scerri and Schulte-Korne (2010) reported that developmental dyslexia is a highly heritable disorder with a prevalence of at least 5% in school-aged children. The authors stated that linkage studies have identified numerous loci throughout the genome that are likely to harbour candidate susceptibility genes. Association studies and the refinement of chromosomal translocation break points in individuals with dyslexia have resulted in the discovery of candidate genes at some of these loci. A key function of many of these genes is their involvement in neuronal migration.

The DSM-5 identifies a Specific Learning Disorder (including Dyslexia) as a neurodevelopmental disorder with a biological origin that is the basis for abnormalities at a cognitive level that are associated with the behavioural signs of the disorder. The biological origin includes an interaction of genetic, epigenetic, and environmental factors which affect the brain's ability to perceive or process verbal or nonverbal information efficiently and accurately.

Treatment

Research continues to support the argument that a modified educational program with a strong emphasis on the explicit and systematic teaching of phonological skills is the most appropriate course of treatment for young dyslexic students. (Muter, 2004; Rack, 2004; Snowling, 2004).

Shaywitz (2005) included a section, titled "Helping Your Child Become a Reader" in her excellent book on dyslexia. It covers strategies to assist a child with dyslexia to learning to read using effective reading strategies. This section of the book gives an extensive list of references and resources for parents and teachers and many illustrations and lists of examples of concepts and strategies are used. Shaywitz provided a detailed plan for parents and teachers to teach a child to break the coding required to learn to read, improve reading skills to a proficient level and finally become a skilled reader with a high level of reading fluency and accuracy.

Shapiro and Solity (2008) investigated the efficacy of intensive phonological awareness and phonics training that was integrated into whole-class, mixed ability reading lessons. They found that such training was highly effective for children with poor phonological awareness, even when incorporated into whole-class teaching.

Lawrence (2009) has written a very interesting and informative book on dyslexia and gives practical advice for parents and teachers.

Torgesen et al (2010) evaluated two computer-assisted programs (Read, Write and Type (RWT; Heron, 1995) and The Lindamood Phoneme Sequencing Program for Reading, Spelling and Speech (LIPS; Lindamood and Lindamood, 1998). The students receiving the computer-assisted programs performed better than the control group who were exposed to their school's normal reading program. The authors found that significant differences were obtained for phonemic awareness, phonemic decoding, reading accuracy, rapid automatic naming and reading comprehension. A follow-up at the end of second grade showed a similar pattern of differences although only differences in phonemic awareness, phonemic decoding and rapid naming remained statistically reliable.

Snowling and Hulme (2010) reviewed evidence concerning the nature, cause of and treatments for children' reading difficulties. They argued that any well-founded educational treatments must be based on sound theory of the causes of a particular form of learning difficulty, which in turn must be based on an understanding of how a given skill (e.g., phonological skills) is learned by typically developing children.

Berninger et al., (2011) gave details of a computer-based program for teaching spelling skills to children with dyslexia.

However, not all students will improve with a focus on phonological skills. If the student does not respond or if their progress has plateaued, other strategies should be employed. For instance, the explicit and systematic teaching of phonic skills (the relationship between letters and sounds) through the developmental stages, beginning with single letters, then consonantal clusters through to the vowel blends. In addition, the use of visual activities, such as flashcard drill to establish and develop instant word recognition, should be considered. At all times, the teacher must be mindful that reading is a language based skill, hence a student's general language development including vocabulary development and enrichment and higher-order thinking and reasoning skills, will need attention.

For the older student with dyslexia, it is highly likely that they have acquired important phonological and phonic skills, albeit very slowly. The focus should be on the rapid and automatic development of decoding skills through flashcard drill, paired-reading strategies and general reading fluency activities. Following the acquisition of these lower-level reading skills, the higher-level reading skills of vocabulary knowledge, thinking and understanding became central to reading success.

More specific information on the teaching of reading can be found in the **Reading Section**.

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Another Important Reference

Helping People with Dyslexia: A National Action Agenda. A Report to the Hon. Bill Shorten, Parliamentary Secretary for Disabilities and Children's Services, from the Dyslexia Working Party. Chairman-M. Coltheart, January 2010.

This report made some 19 recommendations.

Recommendation1-Definition of Dyslexia

Dyslexia is a language-based learning disability of neurological origin. It primarily affects the skills involved in accurate and fluent word reading and spelling. It is frequently associated with difficulties in phonological processing. It occurs across the range of intellectual abilities with no distinct cut-off points. It is viewed as a lifelong disability that often does not respond as expected to best-practice evidence-based classroom methods for teaching reading.

Recommendation 2-Legislative Recognition of Dyslexia as a Disability

Recommendation 3-A National Dyslexia Advisory Council

Recommendation 4-Compliance with the Act

A further 15 Recommendations are made touching on areas such as teaching, schools, assessment, instructional materials, information for families, technology, community awareness, employment and training and research funding.

These are very important and most welcomed recommendations and hopefully they will see the light of day.

I participated in the Australian Government's House of Representative's Select Committee on Specific Learning Difficulties, (including Dyslexia) in 1976 and sadly witnessed little positive, practical outcomes.

I have some fundamental concerns with this definition which is obviously a derivative of the International Dyslexia Association's definition of 2002. These concerns stem from many decades of experience in private practice, in a University Clinic and as a member of a review panel assessing applications for Special Examination Arrangements in VCE examinations.

Certainly, acquiring and developing basic reading and spelling skills including word recognition and phonological processing skills, are critical in the early stages of learning to read and spell. However, as is frequently seen in the reading performance of many older students, they develop satisfactory higher-order reading skills including vocabulary knowledge and reading comprehension, even though they had a history of basic reading problems, or in some cases, continue to struggle with these basic skills (e.g., reading pseudo-words, phonological skills). Surely, the purpose of reading is to gain meaning from print. Hence, if a person can read with adequate fluency and understanding, even though they struggle with some of the basic skills of reading and spelling, should they still be considered to have dyslexia?

I personally have seen numerous instances where senior secondary school students continue to have problems with these basic reading skills (as measured by standardised reading tests), yet perform at, or above, age/grade expectations on advanced reading vocabulary and reading comprehension tests.

Reading involves more than basic word recognition and word-attack skills. Important language skills including syntactic skills, the higher-order skills of semantics and perhaps certain non-verbal organisational skills, play critical roles in accurate word recognition, reading fluency and reading for understanding.

Reaching a satisfactory standard in spelling and written expression is different. Often the student who had problems attaining the basic skills continues to experience spelling and essay writing difficulties throughout their secondary school years and beyond.

Another concern relates to the operational use of the definition by practitioners, such as teachers and psychologists. There is no mention of what constitutes defining symptoms of dyslexia or the level of underachievement or impairment that determines a significant problem. Most established definitions refer to a significant or substantial gap between learning expectations (e.g., age, IQ or Grade level) and current reading performance. Often this gap is quantified, for instance, a discrepancy between one and two standard deviations.

I have one further concern. Traditionally, most researchers and authorities defined dyslexia by exclusion. Established reasons for academic underachievement such as intellectual disability, social and emotional problems, vision and hearing impairments, low motivation, socio-cultural disadvantage, inappropriate instruction were taken into consideration in making a differential diagnosis. Of course, this approach has always presented some concerns as dyslexia can co-exist with other problems. However, there was a determined effort in the past to eliminate comorbidity, i.e., dyslexia co-existing with some known cause of reading problems, such as an intellectual disability. Hence, the preference was to define dyslexia by exclusion; all known causal factors were eliminated before a positive diagnosis was made.

I have found that many boys with reading problems have had a long history of Otitis Media (middle-ear infection), usually with attendant intermittent hearing loss. It is not surprising that such students have difficulty acquiring and developing early reading skills, especially phonological skills. In my opinion, these students should not be labelled dyslexic, but I suspect that many are falsely identified as such. There is a known reason for their reading problems and the causation is not neurological but sensory and/or perceptual.

By stating that dyslexia occurs across the range of intellectual abilities with no distinct cut-off points, the definition breaks with long established practice of excluding individuals with below average intellectual abilities. This clearly has important theoretical and practical implications including the following- research, identification, incidence statistics (an increase from 5%-10% to perhaps 20% and above), a huge increase in requests for services and funding and changes in special examination regulations.

Without the usual exclusions, any reading problem, whatever its cause (except for those with neurological differences/problems-how will this be determined-neuroimaging?) will be called dyslexia. This moves against long established tradition, where dyslexia was seen as a small subtype within the field of reading problems.

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