Phonological Awareness in Specific Learning Disability: A Journey into Search for Genetic Inheritance

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Abstract

Specific learning disability (SLD) is a condition debated for long particularly for the causation. The genetic nature of specific learning disability in particular, has been widely discussed. The recent literature increasingly highlights the associated phonological skill deficits as the underlying core cause for the genetic inheritance with SLD. The present study aimed at comparing the phonological skills of families of children with SLD with that of children and adults without SLD. Three families of children with first cousins, the siblings and only one child in family who were diagnosed as having SLD were selected for the study along with the normal sibling and both the parents. All were tested on phonological tasks. Results were compared with normal children and normal adults. The results indicate that both the qualitative and quantitative performance of children with learning disability matched with that of their fathers suggesting a possibility of tracing genetic inheritance with behavioral measures such as phonological awareness skills. The study opens-up a possibility of devising low cost and non-invasive protocol to explore genetics as a causative factor by clinician that would facilitate better counseling and prognosis. An investigation of performance of children with SLD and their family on phonological tasks revealed a possibility of underlying phonological skill deficits as the cause for SLD, than the inheritance of specific learning disability per se.

Key words: phonological awareness, Learning disability, genetic inheritance

The terms learning disability, learning disabilities and learning disorders (LD) refer to a group of disorders that affect a broad range of academic and functional skills including the ability to speak, listen, read, write, spell, reason and organize information. A learning disability is not indicative of low intelligence. Indeed, research indicates that some people with learning disabilities may have average or above-average intelligence. Causes of learning disabilities include a deficit in the brain that affects the processing of information.

Learning disabilities can be categorized either by the type of information processing that is affected or by the specific difficulties caused by a processing deficit. Historically the reading disorders that are acquired as a result of brain injury were investigated the most. Gradually the reading disorders of congenital origin caught the attention of medical specialists, psychologists, special educators and speech language pathologists. The term SLD (Specific Learning Disability) has been widely accepted for those disorders of congenital origin. A specific learning disability is a disorder in one or more of the central nervous system processes involved in perceiving, understanding and/or using concepts through verbal (spoken) or written language or nonverbal means. This disorder manifests itself with a deficit in one or more of the following areas: attention, reasoning, processing, memory, communication, reading, writing, spelling, calculation, coordination, social competence and emotional maturity.

The causative factors for SLD have been debated over the decades without consensus from the researchers. The investigative research, however, made a clear distinction between acquired and congenital disorders of reading, especially with respect to their causation. The notion of SLD as being genetic in origin has been emphasized in a majority of studies (Franck's & Monaco, 2002; Schulte-Korne, 2001; Smith & Pennington, 1990; Grigorenko, 2001; Remschmidt, 1996).

Dyslexia, one of the SLDs, is a disorder of reading and spelling is a heterogeneous neurological syndrome with a complex genetic and environmental etiology. People with dyslexia differ in their individual profiles across a range of cognitive, physiological and behavioural measures

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related to reading disability. Some or all of the subtypes of dyslexia might have partly or wholly distinct genetic causes. An understanding of the role of genetics in dyslexia could help in the diagnosis and treatment of children more effectively than is currently possible and also in ways that account for their individual disabilities. This knowledge will also give new insights into the neurobiology of reading and language cognition. Genetic linkage analysis has identified regions of the genome that might harbour inherited variants that cause reading disability. In particular, loci on chromosomes 6 and 18 have shown strong and replicable effects on reading abilities. These genomic regions contain tens or hundreds of candidate genes, and studies aimed at the identification of the specific causal genetic variants are emerging in more number in the recent years.

Evidences for Genetic Inheritance of SLD

Recent advances in the understanding of the genetics of SLD based on family studies show a moderate to high familiality and heritability which is evident in studies done by Silver (1971); Omenn and Weber (1978); Singer, Stewart and Pulaski (1981). They studied the pedigree of family history and reported that SLD phenotype is genetically influential. Apart from family studies, twin studies offer crucial support for an understanding of the hereditability of reading, spelling and correlated cognitive phenotypes (example: phonological and orthographic processing). Early twin studies found concordance rates of around 100% in monozygotic twins and about 50% in dizygotic twins, indicating a substantial heritability for reading disability (Zerbin-Rubin, 1967; Bakwin, 1973). Stevenson, Graham, Fredman and McLaughlin 1987), ascertained twins from the general population and found heritability of spelling disability of 0.53, which increased to 0.75 with the control on intelligence. High and significant heritability was for phonological awareness in Colorado twin study (Defries, Fulker & La Buda, 1987; Stevenson, Graham, Fredman & McLaughlin, 1987; Olson, Gillis, Rack & Fulker, 1989; Castles, Datta, Gayan & Olson, 1999).

Patrick, Pearson, Halpin and Jackson (1973) did genetic investigation in a community based population of children with mild-moderate learning disability in 'Human Genetic Unit' Edinburgh University. Age range considered was 5-12years and 13-18year. Sex ratio was 1:62 (Male: Female). 50.6% of them had family history. They performed clinical genetic studies. 4% of them had affected parents as was siblings. Highly significant genetic contribution to etiology of SLD was reported in this genetic investigation.

Different genetic models of reading and spelling disorders have been postulated and there is evidence for both polygenic and monogenic inheritance. For example, the finding that the rate of affectedness in siblings is mainly influenced by whether one or both parents are affected (Hallgren, 1950; Wolf & Melngalis, 1994; Gillis et.al 1996) can be best explained by a polygenic model. Further evidence for this model comes from the findings that siblings in family with two affected parents were more severely impaired than siblings in families with one affected parent (Wolf & Melngalis, 1994) and also provides evidences for an autosomal-dominance transmission with sex-dependant penetrance (Fisher, 1905; Stenphenson, 1907; Hishelwood, 1907; Hallgren, 1950).

A Dominant Gene for Developmental Dyslexia on Chromosome 15 and 3

Smith (1983) found a linkage of reading and spelling disorder to chromosome 15. Grigorenko (1997) reported locus on the long arm of chromosome 15. Morris, Robinson and Goldmuntz (2000)used family based associated mapping with two independent samples and suggested one or more genes contributing to reading disorder.

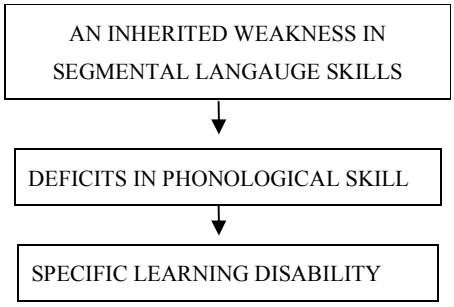
Hemmia, Myllyluomaa, Haltiad, Taipaleb et al (2002) stated that developmental dyslexia is a neurofunctional disorder characterized by an unexpected difficulty in learning to read and write despite adequate intelligence, motivation and education. They studied a large pedigree, ascertained from 140 families considered, segregating pronounced dyslexia in an autosomal dominant fashion. Affected status and the subtype of dyslexia were determined by neuropsychological tests. A genome scan with 320 markers showed a novel dominant locus linked to dyslexia in the pericentromeric region of chromosome 3. Nineteen out of 21 affected pedigree members shared this region identical by descent (corrected $p<0.001$). The new locus on chromosome 3 is associated with deficits in all three essential components involved in the reading process, namely phonological awareness, rapid naming and verbal short term memory.

Evidences for Deficiencies in Phonological Skills in SLD

Vellutino (1977), Liberman and Shankweiler (1979) and Mattingly (1980) postulated that learning disability is a language disorder and that poor readers are not aware that spoken and printed words can be segmented into individual phonemes. This inability in turn prevents them from coding information phonetically. Various

investigators have demonstrated that the ability to segment words into phonemes develops at about the time children learn to read and is highly correlated with reading achievement in beginning readers but is deficient in children with reading disability (Liberman, & Shankweiler ,1979; Bradely & Bryant, 1983;). Mattingly (1980) hypothesized that children who have little or more difficulty in understanding spoken language may be unable to abstract phonemes from spoken words, a step that is necessary for learning to read and that this inability for phonemes segmentation is because of severe language impairment (Vellutino, 1979; Tallal, 1980)

In the literature of the recent past, arguments are put forth by researchers that SLD is a specific developmental language disorder involving deficits in the underlying processes that contribute to the language disorder. And that, the manifestation of the SLD is due to the deficiencies in these underlying processes such as deficits in phonological awareness, sequencing, segmentation and naming. A synthesis of the studies that support the genetic cause for SLD and those that suggest the deficiency in the phonological skills in the SLD leads one to speculate that the SLD is invariably genetic. However, what is inherited is not the SLD itself but, the phonological processing skills, which when deficient can produce the symptoms of SLD. Doyle (1996) has shown that deficits in phonological skills are themselves the result of an inherited weakness in segmental language skills, which can be summarized as in Figure 1.



(Source: Doyle, 1996; Dyslexia an introductory guide)

Figure 1: SLD as specific developmental language disorder.

Do Behavioral Measures Tap Genetic Inheritance?

As we learn from the model (Doyle, 1996), though the causation for SLD is genetic, there are very few measures which can tap its origin. There are many tests used to screen for learning Disability in children. Phonological awareness task is the most commonly used in all the tests.

Kumar and Prema (1999) compared 2 subjects (6years/F & 21years/M) the first cousins with diagnosis of SLD, on their performance on phonological tasks. They reported that despite the age, educational and language differences, both the subjects manifested similar qualitative errors/deficits on phonological awareness tasks, suggesting possibility of inheritance of phonological skill deficits rather than SLD per se.

A retrospective study conducted by Prema and Jayaram (2001-02) to evaluate the relationship between demographic factors and reading skills in Indian children learning to read and write English reported that despite variations in demographic data the children did manifest a strong correlation between language and reading variables and not between reading and demographic variables suggesting that the underlying core issue behind SLD could be the language phenomenon which is the crucial factor. They used Early Reading Scales (ERS) in English. The test consisted of the many tasks related to phonological awareness, such as Reading (words and non-words), Rhyme recognition, Syllable stripping, Syllable oddity, Phoneme stripping and Phoneme oddity.

The review of literature suggests that a considerable percentage of children with SLD evidence genetic inheritance and that this could be tapped through family studies. Further the review suggests that SLD could be the manifestation of underlying language deficiency, the phonological skill deficits, in particular.

Phonological Awareness Training

The review of the literature suggests that improvement in the phonological awareness skills would improve reading and writing skills in individuals with learning disability.

Phonological skills involve manipulations of the phonological constituents of spoken words in tasks such as blending, segmenting and rhyming. Students who learn to read well can rhyme at approximately age 4 (Maclean, Bryant & Bradley, 1988) and blend and segment orally presented words and sounds by the end of the 1st grade (Perfetti, Beck, Bell & Hughes, 1987). But most poor readers, by the end of the 2nd grade, still cannot blend or segment words as well as normally reading younger children (Vellutino & Scanlon, 1987).

Rollanda, Jenkins, Leicester, and Slocum (1993), evaluated the effects of training in phonemic segmentation and instruction in letter names and letter sounds on kindergarten children's reading and spelling skills. Ninety students from three urban public schools in the U. S. were randomly assigned to one of three groups. The first group received training in segmenting

words into phonemes, as well as training in correspondences between letter names and letter sounds (phoneme awareness group). The second group received only the training in letter names and letter sounds (language activities group). The third group received no intervention (control group). Results indicated that phoneme awareness instruction, combined with instruction connecting the phonemic segments to alphabet letters, significantly improved the early reading and spelling skills of the children in the phoneme awareness group.

Alexander, Andersen, Heilman, Voeller and Torgesen (2007), studied the effect of Phonological awareness training and remediation of analytic decoding deficits in a group of severe dyslexics. A group of ten students with severely dyslexia ranging in age from 93 to 154 months were treated in a clinic setting for 38 to 124 hours (average of 65 hours). Pre- and post-treatment testing was done with the Woodcock Reading Mastery Test and the Lindamood Auditory Conceptualization to assess changes in phonological awareness and analytic decoding skills. Results revealed statistically significant gains in phonological awareness and analytic decoding skills.

Hence there is strong correlation between phonological awareness and learning disability. Any measure consisting of phonological awareness tasks can trace out the existence of learning disability.

Need For Phonological Awareness Measures

Although genetic investigation methods can give a significant correlation between genetic inheritance and SLD there are many disadvantages such as:

- Gene mapping is invasive as it requires, blood, hair or nail samples.
- Gene testing methods are not cost effective.
- These methods are very time consuming.
- Genetic analysis is a complex process.
- As a routine, clients and their families cannot be recommended for genetic investigations. Consequently, a genetic analysis serves only the interest of researchers and not practicing clinicians.

However, if behavioral measures with phonological awareness tasks are found suitable for tracing inheritance, these measures are,

- Cost effective.
- Practical and clinician friendly and
- Time required for testing is comparatively very less.

Aim of the Study: The present study aimed at comparing the phonological skills of families of children with SLD with that of children and adults without SLD. Study also aimed at looking for genetic inheritance in SLD through behavioral measure.

Method

Subjects: Three families of children diagnosed as having SLD were considered for the study. 20 normal children between age range of 9-14years and 10 normal adults in the age range of 28-50years were used for comparison. The families consisted of,

- Two children (Ma and Ki) who are first cousins having SLD
- Siblings (Ab and Ra) who were diagnosed as SLD
- Single child (Ka) in a family diagnosed as SLD

Criteria for subject selection

- All the subjects were selected from AIISH (All India Institute of Speech and Hearing) clinical population with provisional diagnosis as SLD and those who underwent therapeutic program for the same for minimum of one year.
- Both clinical as well as normal group of population did not exhibit any peripheral sensory or motor problems.
- All the subjects should have English as their second language.

The details of three groups of children, their siblings and parents are as shown in Table 1.

Materials: Metaphonological test developed by Prakash et.al., (2002) was used to test phonological awareness in subjects for English language. Test was modified and the reading subtest, Syllable oddity and Phoneme oddity was prepared using the textbooks for standard V- IX. This test consist of 6 subtests including Reading, Rhyme recognition, Syllable stripping, Syllable oddity, Phoneme stripping and Phoneme oddity. In Reading subtest, subjects were required to read words and non words in rows. It consists of 20 words and 20 non-words starting from simple to complex.

Except for the Reading task, all other subtests are presented via auditory only mode. In Rhyme recognition task, subjects were presented pairs of words and they were required to answer whether the words are rhyming or not. For the syllable stripping task, subjects were presented with a

word and syllable which has to be deleted out of the word and pronounce the remaining sounds in the word. Eg: /Corona-ro-cona/. In the word "Corona" if we remove "ro", remaining is "Cona".

Subjects with SLD	Group I (first cousins)		Group II (siblings)		Group III (single)	Normal adults (N = 10) (5 M & 5 F)	Normal children (N= 20) (10 M & 10 F)
	Ma	Ki	Ra	Ab	Ka		
Age (in years)/sex Home language Other language	13/M E K	16/M E K	13/M K E	10/M K E	11/M K E	28-50 K E	9-14 K E
Education Medium of instruction	VI Std E	Diploma E	VII Std E	V Std E	VI Std E	UG-G K/E	V-IX E
Siblings	Brother	Brother	Brother	Brother	Brother	NA	NA

(UG= Undergraduate, G= graduate, E= English, K=Kannada, M=Male, F=Female, NA= Not applicable)

Table 1: Details of the subjects

For the subtest of Syllable oddity, subjects were presented a set of four words. They are required to print the word with odd syllable out of four words. Eg: Mesh Melt Meant Milk. Here "milk" is the odd word, as the word "milk" starts with syllable /mi/ and all other words start with "/me/".The odd syllable can occur either in the initial or final position of the word and was uniform throughout the set of words. The procedure is same for the tasks of Phoneme stripping and Phoneme Oddity except that here phonemes are tests and not the syllables.

Eg: Phoneme stripping : /Drink-d-rink/
Phoneme oddity: /Pip Pin Hill Pig/

Parts	Tasks	No.of items	Total score	Examples
A	Reading words Reading nonwords	20 20	20+20 = 40	Treatment Nastami
B	Rhyme Recognition	12	12	Call & Wall
C	Syllable stripping	12	12	Corona-ro-cona
D	Syllable Oddity	12	12	Mesh, Melt, Meant, Milk
E	Phoneme Stripping	12	12	Drink-d-rink
F	Phoneme Oddity	12	12	Pip, Pin Hill, Pig

Table 2: Phonological awareness in English (Prakash et.al., 2002).

Every correct response was scored as "1" and incorrect response as "0". Total score for the complete test is 100. Approximate duration taken to complete the test is 45minutes. The test was administered in a single sitting for all the subjects. Table 2 gives the example of items in the test. Complete test is provided in Appendix along with the score sheet. This test was administered to all clinical population including their parents.

Results and Discussion

The data obtained for the test for both children and adults were noted in the score sheet. Maximum score for the complete test was 100. Mean was calculated for each group. The performance of the three groups of subjects and their siblings was compared with the mean scores of age matched normal children and that of parents with mean scores of adults as given in Table 3.

Table 3 shows almost equivalent mean scores for normal children and adults (90.45 & 93.1 respectively) indicate that their performance paralleled with each other. On the other hand, when the performance of three groups of children was compared with normal children and that of the first cousins (Group I), the score was lower relative to parents with normal children.

Compared to the performance of normal children, siblings (Group II) were much lower; single child (Group III) was in between the two. The higher mean scores of group I could be because of the intensive remedial training given to them for over a period of 3-4years.

The performance of parents on phonological tasks reveals interesting results too. In Group I and Group II families, performance of fathers was relatively low in comparison to mothers and that scores were almost equivalent to that of their sons despite the educational, experiential and age differences. In Group III, however, both the parents performed well in comparison to normal adults. Further analysis of performance in subtests of phonological tasks indicated that in Part A, subjects (Group I, II and III) and the fathers had more problems in reading complex non-words. While Part B (Rhyme recognition) was performed well by all of them, in Part C (syllable stripping) vowel substitutions were seen (Eg: 'Potato-to-pota' the subjects said 'poto'. In Part D (syllable oddity),

the subjects looked for spellings rather than concentrating on auditorily similar words. In Part E (phoneme stripping), the subjects deleted the wrong phoneme. (Eg: 'Drink-d-rink' they said 'Dink').

Groups	Subjects	Normal children Mean score = 90.45	Siblings	Normal adults Mean score = 93.1
I	Ma	82.0	95.0	Father = **69.0** Mother = 97.0
	Ki	90.0	87.0	Father = **49.0** Mother = 92.0
II	Ra Ab	**48.0** **40.0**	NA	Father = **67.0** Mother = 93.0
III	Ka	**60.0**	86.0	Father = 87.0 Mother = 98.0

Table 3: Mean scores on Phonological Tasks

The performance of Group II was analyzed separately on the sub-tests in comparison to that of their father and the details are as shown in Table 4. The table 4 indicates that the scores of father match with his children's scores.

When comparing the results of three groups of children (group I, II and III) with normal children and that of the first cousins, the scores were lower relative to parents of normal children. It is the clear indicator of inherited phonological deficit. This is in agreement with Doyle (1996), who stated that the manifestation of the SLD is due to the deficiencies in these underlying processes such as deficits in phonological awareness, sequencing, segmentation and naming. Also, deficits in phonological skills are themselves the result of an inherited weakness in segmental language skills.

Analysis of results of three groups of children with SLD indicated that performance on phonological awareness tasks could be graded from high to low in first cousins, single child and siblings in that order. This could be due to the remedial training given to the first cousins at very early stage for around 3-4years. This result is in agreement with Alexander, Andersen, Heilman, Voeller and Torgesen (2007) who studied the effect of phonological awareness training and remediation of analytic decoding deficits and found statistically significant gains in phonological awareness and analytic decoding skills in a group of severe dyslexics. However the performance of children with SLD did not match the normal children. The results indicate that with remedial help, the phonological awareness of these children can be facilitated yet they fail to match the normal children.

Subjects	Part B (Max 12)	Part C (Max 12)	Part D (Max 12)	Part F (Max 12)
Ra	10.0	5.0	6.0	3.0
Ab	12.0	8.0	0.0	0.0
Father	11.0	5.0	6.0	2.0

Table 4: Comparison of siblings vs. father on sub-tests.

The above findings are also supported by Rollanda, Jenkins, Leicester and Slocum (1993), who found that severity in siblings with history of SLD in family, is much higher than the other groups.

While comparing the scores of adults on phonological tasks, the performance on phonological awareness tasks of fathers was relatively lower in comparison to that of mothers. Also it was equivalent to the scores of their sons. The results strongly agree with Kumar and Prema (1999) and Prema and Jayaram (2002) who suggested that the underlying core deficits in children with SLD could be in the domain of language, the phonological deficits per se and that it could manifest similarly in families with strong genetic inheritance. Not only the overall scores of fathers matched with their sons but also the scores in individual subtests of the test did match between father and sons. This was observed in the group with both siblings exhibiting SLD. This suggests a strong possibility of inheritance. The results are also in support of Omen and Weber (1978), Silver (1971) who studied pedigree of family history and reported that SLD is genetically influenced.

Owing to the limited sample size (normal N= 10 or 20, subjects N = one or two in each group), one sample t-test (two tailed) was employed for analysis. The data of subjects and their parents was compared with the data obtained from normal children and normal adults respectively. The t values were very highly significant (children: $t = 7.43$, $p<0.05$; adults: $t = 15.59$, $p<0.05$). The results suggest that children in Group I, II and III children showed significant difference from those of normal children. Also the performance of fathers showed significant difference in comparison to the normal adult group. The findings of the present study on performance of first cousins, siblings and single child on phonological awareness tasks, support our premise that there is likelihood to trace genetic inheritance of SLD through behavioral measures.

Conclusions

The purpose of the present study was to look for genetic inheritance of SLD through behavioral measures, i.e., phonological awareness tasks. Three families of children with learning disability, their siblings and their parents were assessed for

phonological skills in English. The performance on phonological awareness tasks of the subjects along with their parents were compared with another group of normal children and normal adults respectively. The results indicate that both the qualitative and quantitative performance on phonological awareness tasks of children with learning disability matched with that of their fathers. The findings of the present study on performance of first cousins, siblings and single child on phonological awareness tasks, support our premise that there is likelihood to trace genetic inheritance of SLD through behavioral measures. The study opens-up a possibility of devising low cost and non-invasive protocol to explore genetics as a causative factor by practicing Speech-Language pathologists that would facilitate better counseling and prognosis. The performance of Group I (first cousins) subjects highlights that the severity of genetic influence could be reduced, if not entirely alleviated, by undertaking intensive remedial measures. Further studies in this direction would be important to investigate the interaction of inheritance versus environment.

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APPENDIX

Metaphonological Test

A. READING:

SL	WORDS	NONWORDS	WORDS	NONWORDS
1	English	Dymicna	Treatment	Posicomption
2	Mystery	Rymemo	Cruelity	Acquisition
3	Explore	Lisheng	Crucified	Mofiableodi
4	Furnace	Nastami	Fathomable	Caltechni
5	Canvas	Noroco	Prospectus	Dentpinindo
6	Chairman	Myryst	Modifiable	tusticontion
7	Courage	Apdixpen	Democratic	Tusprospect
8	Decades	Mafeo	Categorically	Pralityacti
9	Operation	Plexore	Publication	Adtraminist
10	Philateraly	Spirein	Eternity	cephalotry

B. RHYME RECOGNITION:

ILLUSTRATION:

Fame game	Mind fame
Call wall	Mind kind
Wall head	Kind game

TEST ITEMS:

1	Kill mill	7	Deep heep
2	Gate heep	8	Gate hate
3	Lift gift	9	Kill lift
4	Cable table	10	Silly hilly
5	Hate beep	11	Mill lift
6	Cable silly	12	Lable silly

C. SYLLABLE STRIPPING:

ILLUSTRATION:

Tomato-ma-toto
India-a-indi
River-ri-ver

TEST ITEMS:

1	Carona-ro-cana	7	Potato-to-pota
2	Manila-lla-mani	8	Holiday-day-holi
3	Dynamic-dy-namic	9	Jupiter-ju-piter
4	Gallery-lle-gary	10	Memory-mo-mery
5	Jocular-cu-jolar	11	Stamina-na-stami
6	Banana-ba-nana	12	Indigo-in-digo

D. SYLLABLE ODDITY:

ILLUSTRATION:

Class	Clamp	Splass	Cleft
Shadow	Shallow	Sharon	Shop
Muffle	Chappell	Cripple	Stample
Lock	Hawk	Clock	dock

TEST ITEMS:

1	Mesh	Melt	Meant	Milk
2	Went	Bent	Bond	Sent
3	Fable	Cable	Table	Stable
4	Master	Poster	Tastier	faster
5	Beat	Wheat	Site	Seat
6	Pole	Goal	Mel	Role
7	Neck	Knock	Nest	Knelt
8	Muller	Ruler	Puller	Killer
9	Bench	Belt	Boost	Best
10	Biology	Zoology	Sociology	Trinity
11	Lot	Cot	Hat	Pot
12	Lamb	Bomb	Comb	tomb

E. PHONEME STRIPPING:

ILLUSTRATION:

Alive-a-live
Ideal-I-idea
Open-o-pen

TEST ITEMS:

1	Drink-d-rink	7	English-l-engish
2	Egard-e-gard	8	Erupt-t-erup
3	Facula-f-cula	9	Forgo-g-foro
4	Abduct-b=aduct	10	Crawl-l-craw
5	Droop-r-doop	11	Drown-n-drow
6	Freak-r-feak	12	Epode-e-pode

F. PHONEME ODDITY:

ILLUSTRATION:

Jug	Mug	Rug	Fun
Doll	Hop	Pop	Top
Held	Gild	Bold	Mat
Just	Jump	Dung	junk

TEST ITEMS:

1	Pip	Pin	Fig	Pig
2	Bud	Bun	Bus	rug
3	Gave	Give	Hive	live
4	Lot	Cot	Had	pot
5	Pin	Sit	Sin	sip
6	Ket	Ken	Kemp	Pet
7	Disk	Damp	Dusk	husk
8	Cock	Cork	Coke	Hawk
9	Pack	Back	Lack	Punk
10	Fun	Pit	Bun	Gun
11	Freak	Break	Friend	frank
12	Dark	Mark	York	park