Characteristics of children with developmental verbal apraxia.

Diane Guthrie

The University of Montana

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CHARACTERISTICS OF CHILDREN WITH DEVELOPMENTAL VERBAL APRAXIA

By
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B.A., University of Victoria, 1979

Presented in partial fulfillment of the requirements for the degree of

Master of Arts
Communication Sciences and Disorders

UNIVERSITY OF MONTANA

Approved by:

[Signatures]

Chairman, Board of Examiners
Dean, Graduate School

Date 8-11-82

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Chapter 1

LITERATURE REVIEW

The greatest proportion of speech defects among all ages are articulation errors. More than 60% of 25 million children and young adults in the United States have been classified as having functional articulation disorders (National Institute of Neurological Disease and Stroke, 1970). According to Silverstein (1971), certain articulatory disorders previously classified as functional may be actually neurologically based. These children have numerous labels such as developmental apraxia, developmental dyspraxia, dilapidated speech, apraxia, verbal apraxia, and even oral motor idiocy (Chappell, 1973; Prichard, Tekieli, & Kozup, 1979). The term used in this paper will be developmental verbal apraxia or DVA.

Definitions

According to Ayres (1975), praxis is a learned ability which allows an individual to plan and direct temporal series of movements in order to produce a skilled and nonhabitual act. This lack of ability to plan and direct can include muscle movements of the face, tongue, hands, legs, or trunk (Gubbay, 1975).

Morley (1967, p. 237) defined DVA as

a defect of articulation which occurs when the movements of the muscles used for speech, that is of tongue, lips or palate, appear normal for involuntary or spontaneous movements such as smiling or licking the lips, or even voluntary imitation of movements carried

1
out on request, but are inadequate for the complex and rapid movements used for articulation and reproduction of the sequence of sounds used in speech.

The disorder is thought to be caused by brain damage or cortical dysfunction present in infancy and to occur in the absence of weakness, paralysis, or muscle incoordination (Morley, Court, & Nutler, 1954; Palmer, Wurth, & Kineheloe, 1964).

Descriptions of DVA have included numerous and variable symptoms. According to Blakeley (1980, pp. 5-6), "The isolation of developmental apraxia of speech as a clinical entity, in light of current knowledge . . . requires clustering of the symptoms most often described." Blakeley described such children as having atypical and inconsistent errors, an expressive language delay, and inordinate resistance to remediation. These symptoms include not only numerous speech and language characteristics but associated symptoms as well.

Gubbay (1975) discussed some of these associated symptoms. She suggested that DVA can be part of a developmental disorder subsumed under the Minimal Brain Dysfunction Syndrome. Included in this syndrome are not only language dysfunctions incorporating DVA but also hyperkinesias, disorders of perception, inefficient body awareness, and control (resulting in clumsiness), language dysfunction including developmental apraxia, emotional lability, specific learning disabilities, and neurological abnormalities. The basis for these developmental disorders is thought to be an alteration in the maturation of brain areas controlling motor coordination and language. Speech and language characteristics, however, are central to the diagnosis of DVA and it can occur without other kinds of disorders (Yoss & Darley, 1974).
**Speech and Language Characteristics**

One of the most consistently reported symptoms of DVA is the inordinate superiority of receptive versus expressive language (Blakeley, 1972; Ferry, Hall, & Hicks, 1975; Rosenbeck & Wertz, 1972; Yoss & Darley, 1974). This characteristic is not thought to be as typical of functional articulation disorders and should, therefore, be useful in differentiating them (Rosenbeck & Wertz, 1972).

The speech of children with DVA is associated with a number of symptoms. Rosenbeck and Wertz (1972) reported that connected speech is more unintelligible than would be expected on the basis of a single word articulation test. Also, while imitation of single words may be reasonable, spontaneous stretches of creative speech may be unintelligible. These children often develop elaborate gestural systems as a result (Ferry, et al., 1975).

Metathetic errors such as transpositions and redundancies of sounds and syllables are said to be common in this disorder. Edwards (1973) and Rosenbeck and Wertz (1972) found transpositions not only within words but in words in sentences as well. This is consistent with the poor verbal sequencing frequently found. For example, oral diadochokinesis is often poorly sequenced and slow (Blakeley, 1980; Yoss & Darley, 1974).

More complex phoneme combinations have been found to be more difficult for children with DVA than children with other articulation problems (Blakeley, 1972; Rosenbeck & Wertz, 1972). Longer polysyllabic words have been found to be more difficult and to contain errors such as omissions, revisions, or additions of syllables (Macaluso-Haynes &
Johns, 1978; Yoss & Darley, 1974). The errors on these motorically complex words were found to occur most frequently on sounds such as fricatives, affricates, and consonant clusters which require the most articulatory adjustment.

Developmental verbal apraxia has been associated with specific types of articulatory errors. Compared to other children who misarticulate, those with DVA make more frequent feature errors per misarticulated sound. Ferry, et al. (1975), Rosenbeck and Wertz (1972), and Yoss and Darley (1974) described inconsistent substitutions, omissions, and distortions. Rosenbeck and Wertz found that, although errors are usually inconsistent, some consistent patterns may be present. These findings are in contrast to a study by Williams, Ingham, and Rosenthal (1981) who found that none of the clusters of error features discussed by Yoss and Darley were predictive determinants of DVA.

Trial and error behaviors in the form of sound prolongations, sound repetitions, and silent posturing have been reported. The patients were described as appearing to "struggle to position their articulators for correct speech production" (Rosenbeck & Wertz, 1972, p. 31). Yoss and Darley (1974) found this behavior was evident only in a few children, most of them older.

The production of vowels and diphthongs occurs early in childhood. Compared to consonants, these sounds are motorically easier to produce (Blakeley, 1980). Errors on vowels and diphthongs are more frequent in children with DVA compared to children with other articulatory disorders (Blakeley, 1972; Macaluso-Haynes & Johns, 1978; Rosenbeck & Wertz, 1972).
Developmental verbal apraxia has been associated with disturbances in prosody. Prosody includes the rhythmic parameters of articulation rate, loudness regulations, phonemic spacing, inflection, and stress (Blakeley, 1980). According to Darley (1969), this prosodic disorder although probably more common in those with acquired apraxia of speech and in high school children with DVA, is believed to be one of the occasional distinguishing differences between so-called functional articulation disorders and DVA. Prosodic disturbances include slowed rate and even spacing. It is suggested that these may be attempts at compensation and self-monitoring (Blakeley, 1980; Rosenbeck & Wertz, 1972). When prosody is not normal, it has been suggested that speech therapy may be valuable (Edwards, 1973).

Slow and limited progress in therapy is another characteristic used to describe DVA (Ferry, et al., 1975). In one study by Pritchard, et al. (1979), six subjects with DVA had had extensive speech therapy and none had made significant progress. Ferry, et al. (1975, p. 751) described "painfully slow progress or minimal improvement" in patients with DVA who had spent one to 11 years in therapy.

Inconsistent hypernasality also has been reported in children with DVA by Blakeley (1972). Their soft palates were described as seeming to "function out of phase with talking but . . . were generally satisfactory in function for non-talking activities such as chewing, swallowing and blowing" (p. 147).

**Associated Symptoms**

Besides these speech and language characteristics, there are other associated symptoms; a number are part of the Minimal Brain
Dysfunction Syndrome mentioned earlier. One such symptom is difficulty with voluntary movements of the oral musculature disassociated with speech production (Blakeley, 1980). This difficulty is called oral apraxia when it occurs in the absence of muscle paralysis or weakness. It may or may not occur in the presence of DVA (DeRenzi, Pieczuro, & Vignolo, 1966; Rosenbeck & Wertz, 1972). Successful performance of volitional oral movements by children with DVA compared to children with other articulation problems requires more demonstration and thus the correct performance of the movements may be impossible.

Another associated symptom that has been discussed is auditory perception. Yoss and Darley (1974) found that, on sound discrimination items on the Denver Auditory Phoneme Sequencing Test, children with DVA performed significantly poorer than normal matched subjects. From this information, it was concluded that children with DVA had poorer auditory perception than normal matched subjects. In contrast, in a study by Williams, et al. (1981), auditory perception was not found to differ significantly, using the same measures as Yoss and Darley. The significance of auditory perception in the diagnosis of DVA is therefore inconclusive.

Emotional and behavioral problems have been associated with DVA. Ferry, et al. (1975) found that a majority of their 60 subjects had significant emotional or behavioral problems. Temper tantrums and outbursts of rage were found in 32, depression or withdrawal in 14, and autism in four. The outbursts of temper and aggressive rage were directly proportional to the inability of these children to communicate with those around them. Their behavior was compared to
that of deaf children who also may be unable to make themselves understood and exhibit similar behaviors.

A high incidence of soft neurological signs involving fine motor coordination, gait, and alternate movements of the extremities has been observed. Yoss and Darley (1972), for instance, found that 94% of the children with DVA in their study had these positive neurological findings. This was contradicted by Williams, et al. (1981) who found virtually no evidence of soft neurological signs.

Ferry, et al. (1975) found all of their 60 patients to be free of obvious cerebral palsy, seizures, an abnormal gag, pharyngeal or jaw jerk reflexes, lingual atrophy, and other major neurological deficits. They did, however, find six associated neurological problems: oral facial dyspraxia, mild spastic diplegia, mild motor retardation, hand tremor, hemiplegia, choreiform movements. Furthermore, abnormal electroencephalograms were found in all 20 of the mentally retarded institutionalized patients included in the study. These patients had mild nonspecific patterns without focal abnormalities.

Rosenbeck and Wertz (1972) found that 22 of 36 subjects had essentially normal neurological findings except for the presence of generalized apraxia. From these studies, the most that can be concluded is that verbal apraxia may be the only evidence of neurological dysfunction or it may occur in the presence of other associated neurological problems.

In a number of studies, children selected for inclusion were required to have intellectual functioning in the normal range (Kornse, Manni, Rubenstein, & Graziani, 1981; Williams, et al., 1981; Yoss &
Darley, 1974). In other studies, intellectual functioning is not indicated. In a study by Ferry, et al. (1975), only 29 of 60 subjects had intelligence quotients of greater than 90, suggesting that normal intellectual functioning is not always a predictive determinant of DVA. Developmental verbal apraxia also has been characterized by a marked discrepancy between verbal and performance IQs on the Wechsler Intelligence Scale for Children. The performance scale was invariably higher in 18 of the 21 cases studied (Walton, Ellis, & Court, 1962).

As the preceding information indicates, not only are the reported symptoms of DVA numerous, but the existence of many of them is questionable or they have been reported to be present only in some of these children. Until further research delineates and describes DVA in a more comprehensive fashion, its diagnosis will continue to be elusive.

**Etiology**

Praxis describes a variety of skilled, learned (noninstinctual) motor acts. The loss of or impairment of these functions results in apraxia which Scheinberg (1981) suggested may be caused by lesions in the parietal lobe or occasionally in the frontal lobes as well. In this instance, Scheinberg referred to all forms of apraxia, not just verbal.

Traditionally, lesions resulting in verbal apraxia have been attributed to the third frontal convolution of the left hemisphere (i.e., Broca's area). In a study by Wertz (1970), however, only 45 of 104 adult patients with acquired verbal apraxia had injury to Broca's area while cortical injury elsewhere was found in 59 cases. Furthermore, of the 21 cases with the best documentation, no consistent
pathology of any specific area of the left hemisphere could be determined.

In addition, even if the praxis centers for the speech musculature were found to be more focal in adults, such findings may not apply to children with DVA. Rosenbeck and Wertz (1972) suggested that in a child's cortex the praxis centers for speech may be diffuse. That is, a larger percentage of the frontal and posterior cortex may be needed to successfully develop and volitionally perform skilled speech movements. This hypothesis is supported by a high incidence of isolated apraxic signs in children.

In adults with acquired apraxia, on the other hand, a right hemiparesis or paralysis usually results, especially when the apraxia of speech is a result of lesions to Broca's area and to the sensorimotor cortex of the left hemisphere. Additional support for the diffuseness of praxis centers are the EEG findings which indicate a lack of focal abnormalities (Ferry, et al., 1975).

Rosenbeck and Wertz (1972) suggested that during development the praxis centers for skilled movement in children lateralize and become more discreetly localized, usually in the left hemisphere. A lesion occurring before this lateralization and localization, if it occurs in any number of left-right anterior-posterior positions, can produce apraxia of speech. They suggested that lesions occurring after this lateralization and localization are likely to be in Broca's area or in the tracts connecting this area with visual, auditory, and tactile-kinesthetic centers.
Kornse, et al. (1981) hypothesized that if DVA was due to damage in or including Broca’s area, motor control of the hand controlled by the left hemisphere should be affected because of proximity to the motor strip. They concluded that DVA was not a result of a defect of motor function in the left hemisphere because neither males nor females performed more poorly with the right and left hands when compared to normal subjects. There is little information, therefore, to support the hypothesis that DVA results from a lesion in a specific area of the cortex.

A sensory based etiology for DVA has been suggested by Edwards (1973). He pointed out that speaking involves ongoing sensory input. That is, the listener’s reaction involves the visual mode and the individual’s utterances involve the auditory and tactile kinaesthetic modes. Processing auditory information involves discrimination, seriation, memory, and proprioceptive feedback. Edwards also suggested that a possible etiology of DVA is an inability to effectively deal with this barrage of multisensory input by integrating and organizing it so that the necessary actions can be produced. This is evidenced in clinical settings by an inability to attend if overstimulated or if exposed to too great a variety of stimuli. These children may have difficulty selecting meaningful input signals and an inability to reject redundancies to which they are exposed in their environment.

Edwards (1973) suggested that proprioceptive input may be especially important to the organization and production of movements necessary to speech. This is supported by the findings that, when proprioceptive feedback of the oral structures is interrupted by
anesthesia, articulatory competence is disrupted in a manner similar to the speech of those with DVA. The longer the interruption, the more severe is the expressive speech disorder. Edwards proposed that if such proprioceptive input is blocked in a child developing language, the consequences for speech may be even more devastating.

Some researchers disagreed with this approach and advanced a motorically based etiology. Perkins (1971), for instance, believed DVA is a motor problem specific to the output transmission channel that occurs in people with intact central language processes. Selection of the proper sequences of phonemes, words, and grammatical structures to express ideas are all intact, but the motor programming of the speech muscles is not. To produce the correct speech sounds in the proper sequence, approximately 100 muscles must be selectively activated at the appropriate time, order, and duration (Darley, Aronson, & Brown, 1975). Developmental verbal apraxia is thought to result when there is an interruption or dysfunction in this programmed sequence.

A genetically based etiology has also been suggested. Ferry, et al. (1975) examined the family histories of children with DVA. They found that 17 of 60 patients with DVA had a positive family history of speech or articulation problems. These problems usually occurred in a male relative such as a father or a brother.

In a study by McLaughlin and Kriegsman (1980), DVA was found in patients with x-linked mental retardation (Renpenning Syndrome). With this form of retardation, affected males and some carrier females share a marker x-chromosome with a constriction of the long arm. The authors propounded that some of these children may have been inappropriately
labelled and that the term x-linked developmental apraxia would be more appropriate.

As the preceding information indicates, the etiology of DVA continues to be a mystery. To begin with, understanding the anatomy and physiology of speech production is an immensely complex task. It may be necessary to first understand speech production before understanding DVA. Also, an understanding of DVA may have to wait until the ultimate speech control center is found, if it exists.

It may be discovered that speech production requires an interaction between a number of cortical areas and, therefore, cannot be localized. Also, the involvement of genetics and the underlying processes of DVA have yet to be fully understood. Research so far has failed to reveal any definite and consistent etiological factors in DVA; however, many etiologies such as the three just discussed have been suggested and research should be directed toward them.

Purpose

The purpose of this study is to describe the characteristics of some children diagnosed as having DVA in a multidisciplinary setting. The diagnosis of DVA has been difficult and, as a result, many children with the disorder continue to be placed in the category of functional articulation disorders. Children with DVA require a more complex remedial approach than those with other articulatory disorders (Blakeley, 1972, 1980). Misdiagnosis can be detrimental to a child's progress because the disorder requires a protracted commitment to long-term individualized speech instruction.
Furthermore, learning disabilities, childhood aphasia, and a number of other problems have been associated with DVA (Ferry, et al., 1975; Gubbay, 1975; Rosenbeck & Wertz, 1972). For these reasons, early identification of these children is urgent. Until these children are identified with the appropriate criteria, success with remedial programs will continue to be disappointing (Blakeley, 1980).

There may not be enough information to conclusively prove that a separate disorder called DVA exists, but there is enough to suggest it. Perhaps the approach to take until knowledge of the problem increases is to base the diagnosis of DVA upon clusters of symptoms as Blakeley (1980) suggested, realizing that none of them alone indicates a definite diagnosis.

Developmental verbal apraxia has been characterized in a variety of ways in the literature, resulting in confusion and controversy about what actually comprises this disorder. The studies in the literature often reported testing not possible in a clinical setting. They also usually concentrated on one specific area (for example, language or fine motor coordination). In a multidisciplinary setting, routine evaluations in these and other areas are possible.

The Language Disorders Clinic at the Crippled Children's Division of The Oregon Health Sciences University is one such setting. This clinic involves one and one-half day evaluations of language disordered children. On the first day, the children are evaluated in audiology, psychology, social work, physical therapy, occupational therapy, special education, and pediatrics. On the second day, a speech and language evaluation, a staff conference, and a parent conference are conducted.
A similar setting is found in the Primary Evaluation Clinic which takes one half day rather than a day and one half.

Such evaluations provide information on many of the symptoms of DVA that are described in the literature.

1. The speech and language evaluation provides information on expressive and receptive language levels and whether or not certain characteristics typical of verbally apraxic speech are present.

2. The neurological examination by the pediatrician and the occupational therapy and physical therapy evaluations provide information on neurological integrity.

3. Psychological testing provides information on intellectual functioning and the presence of emotional or behavioral problems. Additional information in this area may be given by the social worker.

4. The special education evaluation offers information about the existence or nature of any specific learning disabilities.

5. Information on family history, including the presence of any speech and language problems or other related problems, is provided from various sources.

The information in these areas was examined and described relative to the literature on characteristics of DVA. It is hoped that this study will provide more information on characteristics of verbally apraxic children in a clinical setting.
Subjects

The subjects were 11 patients between the ages of 3 years, 4 months and 12 years, 4 months. The patients had been referred to The Oregon Health Sciences University for speech and language evaluations. Nine had been evaluated by a multidisciplinary team through the Language Disorders Clinic and two through the Primary Evaluation Clinic. Ten were males and one was a female. The subjects were from various locations throughout the state of Oregon. The subjects had all received diagnoses of developmental verbal apraxia. Patients with significant hearing losses or cleft lip and/or palate were excluded from the study.

Procedure

The files of the patients diagnosed as verbally apraxic were examined. Information in seven areas was collected if it was available: (1) speech and language, (2) psychology, (3) fine motor, (4) gross motor, (5) neurological, (6) educational, and (7) family history. Some of the tests used for these evaluations are listed in the Appendix.

Because the descriptions are of evaluations done previously, detailed information on exact testing procedures and test results was not always available. Neither, of course, were controls of testing procedures possible except those typically available in a clinical
setting. Also, because of the small number of subjects, statistical analyses of the data were not performed. The purpose of this study, using these data, is to describe rather than define developmental verbal apraxia.
Chapter 3

RESULTS

Speech and Language

The symptom most frequently found in the speech and language evaluations was a discrepancy between expressive and receptive language, with the latter being superior in all cases. Furthermore, of the 11 children, eight had receptive language delay as well (see Table 1).

Table 1

Expressive and Receptive Language Levels

<table>
<thead>
<tr>
<th>Subject</th>
<th>Chronological age (months)</th>
<th>Receptive language (months)</th>
<th>Expressive language (months)</th>
</tr>
</thead>
<tbody>
<tr>
<td>W.H.</td>
<td>80</td>
<td>86</td>
<td>Mild delay</td>
</tr>
<tr>
<td>R.P.</td>
<td>106</td>
<td>57</td>
<td>36</td>
</tr>
<tr>
<td>S.E.</td>
<td>70</td>
<td>60</td>
<td>48</td>
</tr>
<tr>
<td>K.S.</td>
<td>40</td>
<td>36</td>
<td>18</td>
</tr>
<tr>
<td>R.S.</td>
<td>148</td>
<td>148</td>
<td>73</td>
</tr>
<tr>
<td>D.M.</td>
<td>110</td>
<td>41</td>
<td>36</td>
</tr>
<tr>
<td>T.C.</td>
<td>106</td>
<td>96</td>
<td>60</td>
</tr>
<tr>
<td>P.R.</td>
<td>96</td>
<td>78</td>
<td>48</td>
</tr>
<tr>
<td>J.L.</td>
<td>66</td>
<td>54</td>
<td>36</td>
</tr>
<tr>
<td>B.S.</td>
<td>41</td>
<td>36</td>
<td>24</td>
</tr>
<tr>
<td>J.P.</td>
<td>91</td>
<td>48</td>
<td>24</td>
</tr>
</tbody>
</table>

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The children were considered to have a clinically significant discrepancy if the difference was at least six months between receptive and expressive language. Also, if receptive language and expressive language were at least six months less than the chronological age, this was considered a language delay. The decision that a delay existed was based on clinical judgment because most of the evaluations were based on nonstandardized language tests.

Many of the speech symptoms described in the literature on DVA also were used to describe the speech of the children in this study. All the children had at least four of the apraxic speech features described (see Table 2). The excessive use of gestures was found in only two of the youngest children, both three years of age. This determination was based on clinical judgment.

Table 2
Frequency of Apraxic Speech Symptoms

1. Poor diadochokinetic sequencing, 8/11.
2. Difficulty with polysyllabic and/or motorically complex words, 5/10.
4. Transpositions, 6/11.
5. Silent rehearsal, 1/10.
11. Poor verbal imitation, 2/11.
12. Prosody disturbance, 1/11.
13. Hypernasality, 1/11.
The presence of speech problems separate from DVA were found in three subjects. Subject D.M. had dysarthria, T.C. had laryngeal stridor, and P.R. had dysphonia.

Psychology

Of the 10 children who received intelligence tests, one was of normal intelligence, four were of borderline intelligence, four had mild mental retardation, and one had moderate mental retardation (see Table 3). The levels of intelligence were based on criteria by Matarazzi (1972).

Table 3
Intelligence Quotients

<table>
<thead>
<tr>
<th>Subject</th>
<th>Test</th>
<th>IQ</th>
<th>Mental age</th>
</tr>
</thead>
<tbody>
<tr>
<td>W.H.</td>
<td>Stanford Binet</td>
<td>97</td>
<td></td>
</tr>
<tr>
<td>S.E.</td>
<td>Stanford Binet</td>
<td>82-84</td>
<td></td>
</tr>
<tr>
<td>J.L.</td>
<td>Stanford Binet</td>
<td>71</td>
<td></td>
</tr>
<tr>
<td>B.S.</td>
<td>Stanford Binet</td>
<td>-</td>
<td>2 yrs, 4 mos</td>
</tr>
<tr>
<td>J.P.</td>
<td>Stanford Binet</td>
<td>53*</td>
<td></td>
</tr>
<tr>
<td>R.P.</td>
<td>WISC-R</td>
<td>Mild delay</td>
<td></td>
</tr>
<tr>
<td>R.S.</td>
<td>WISC-R</td>
<td>85</td>
<td></td>
</tr>
<tr>
<td>D.M.</td>
<td>WISC-R</td>
<td>42</td>
<td></td>
</tr>
<tr>
<td>T.C.</td>
<td>WISC-R</td>
<td>76</td>
<td></td>
</tr>
<tr>
<td>P.R.</td>
<td>WISC-R</td>
<td>64</td>
<td></td>
</tr>
<tr>
<td>K.S.</td>
<td>Not tested</td>
<td>-</td>
<td></td>
</tr>
</tbody>
</table>

*Prorated to exclude verbal items.
Children given the Stanford Binet Intelligence Scale, who did poorer on the verbal items, had their results prorated to exclude verbal items. The Wechsler Intelligence Scale for Children (WISC) is divided into two sections, verbal and performance. A discrepancy between verbal and nonverbal scores of 15 points frequently is considered clinically significant and was considered so by staff psychologists at the facility where the evaluations took place (Boyd, 1982).

Of the five children given the WISC-R, only one subject, R.S., was found to have a significant discrepancy between verbal and performance scores. Detailed information for subject R.P. was not provided, and subjects D.M., T.C., and P.R. had insignificant discrepancies.

Of the subjects administered the Stanford Binet Intelligence Scale, B.S. was reported to have failed two performance subtests and six verbal subtests. Subject J.P. obtained a performance IQ of 53, but full-scale and verbal IQs were not provided. Subjects J.L., W.H., and S.E. had discrepancies not considered to be significant.

The personality characteristics of four of the children were considered to be of clinical significance. S.E. was described as uncommunicative and obsessive-compulsive, T.C. had a history of temper tantrums, P.R. was described as being a behavior problem, and B.S. was thought to be distractable, active, and noncompliant. These characteristics are based on observation and not formal tests.

**Fine Motor**

Of the 10 children who received a fine motor evaluation, five were delayed, four had normal fine motor functioning, and subject T.C.,
age 8 years, 20 months, performed normally up to the six year ceiling of the test. J.P., who did not receive a fine motor evaluation, was reported to have an "immature grasp" at age 5 years.

**Gross Motor**

Of the nine children who received gross motor assessments, four were delayed. Three had normal gross motor functioning and P.R., age 8 years and T.C., age 8 years, 10 months, performed normally up to the six and one-half year ceilings of the test. J.P. was not evaluated by a physical therapist but was reported by the pediatrician to have an "immature gait."

**Neurological Examination**

The neurological examinations of five of the children were normal. Four patients had clinically significant findings. R.P. had neuromuscular weakness and gait impairment, R.S. had neurofibromatosis (a large number of neuroma with fibromatous elements) and a history of tap seizures, D.M. had seizures and dysarthria, T.C. had seizures, a hypogag reflex, inconsistent velopharyngeal closure, and was observed to drool.

**Educational**

Of the five children who received special education evaluations, two had specific learning disabilities.

**Family History**

Four of the children were reported to have a family history of speech problems. R.S. had a father who stuttered, P.R. had a mother and cousins with speech problems, B.S. had a brother who was reported
to be "slow" to speak and an uncle with speech difficulties, and J.P. had a 12-year-old uncle who "does not speak."

A family history of other neurologically based problems was reported in three of the children. R.C. had a maternal uncle who was mentally retarded and a mother who was described as "slow," R.S. had a mother who required remedial reading when she was in school, and D.M. had an uncle with a possible seizure disorder and a father diagnosed as schizophrenic.

Summary

In summary, the following characteristics were noted:
1. Expressive language delay, 11/11 (100%).
2. Receptive language delay, 8/11 (82%).
3. Apraxic speech features, 11/11 (100%).
4. IQ: normal, 1/10 (10%)
   borderline, 4/10, (40%)
   mild mental retardation, 4/10 (40%)
   moderate mental retardation, 1/10 (10%)
5. Emotional or behavioral problems, 4/11 (36%).
6. Fine motor delay, 5/10 (50%).
7. Gross motor delay, 4/9 (44%).
8. Neurological abnormalities or soft signs, 4/10 (40%).
9. Learning disabilities, 2/7 (29%).
10. Family history of speech problems, 4/11 (36%).
11. Family history of neurologically based problems, 3/11 (27%).
This study described the performance of children with DVA in seven areas: (1) speech and language, (2) psychology, (3) fine motor coordination, (4) gross motor coordination, (5) neurological integrity, (6) educational performance, and (7) family history. The results were fairly consistent with the literature on DVA in that most of the symptoms were usually found in some children but not others.

Speech and Language

The speech and language results indicate that the most frequently found characteristic was a receptive-expressive language discrepancy. This finding is consistent with most of the literature (Blakeley, 1972; Ferry, et al., 1975; Rosenbeck & Wertz, 1972; Yoss & Darley, 1974). Receptive language also was found to be delayed frequently although to a lesser extent than expressive language. This characteristic is less evident in the research on DVA because many of the studies required normal or near normal receptive language as a prerequisite for inclusion in the study. This suggests that, when determining the presence of DVA, the focus should be on the discrepancy between expressive and receptive language and not on whether receptive language is normal or near normal in comparison to expressive language.

With the exception of auditory perception and the presence of more frequent feature errors, many of the apraxic symptoms described
in the literature were found in the speech of the children in this study. Auditory perception and a feature analysis are not often part of a regular clinical evaluation in this setting. This probably accounts for the failure to find either one in the speech and language reports. The testing of auditory perception is a controversial subject and there is little evidence to suggest that it is important to the differential diagnosis of DVA. There also is little evidence to support the presence of more frequent feature errors in children with DVA. Furthermore, auditory perception and a feature analysis of speech would usually be too time consuming in a regular clinical evaluation.

The symptoms such as use of gestures, difficulty with verbal sequencing and complex and multisyllabic words, groping and struggle behaviors, vowel errors, consonant slighting, transpositions, inconsistent hypernasality and poor verbal imitation were all mentioned with varying frequencies. Conclusions based on the relative frequencies will not be discussed, however, because the sample size is too small and the original evaluations from which the information was gathered were not done by the experimenter and, therefore, were not subject to controls for the purpose of this study. For example, tests of oral apraxia or verbal sequencing may not have been done with all the children. The findings, however, suggest that the symptoms found are readily available in a clinical evaluation and, therefore, are of diagnostic use in describing DVA.

Some children were found to have speech problems that were considered to be unrelated to DVA. When examining a child for DVA, this should be kept in mind and efforts made to separate the problems.
For example, if a child has DVA and dysarthria as subject P.M. did, this is important to know because the remedial approaches to the problems are different even if some of the speech characteristics are similar. There is not enough information to determine if voice problems in two of the children are incidental or related to the DVA.

These findings seem to support Blakeley's (1980) suggestion that the diagnosis of DVA be based on a clustering of symptoms. Although an expressive-receptive language discrepancy may be found in all DVA children, this discrepancy may be found in children with other articulation disorders (for example, dysarthria). Therefore, in addition to the receptive-expressive language discrepancy, a number of other speech symptoms appear to be necessary to diagnose DVA. The more speech symptoms a child has, the greater the possibility that the child is verbally apraxic. Before this is possible, however, normative data on the relative frequencies of the speech symptoms in children with DVA and other articulation disorders are necessary. This quantification hopefully would give the diagnosis of DVA a firmer basis than it now has.

Associated Symptoms

In addition to these speech and language problems, a number of associated problems may be found. The psychological findings, for example, suggest that the intelligence of children with DVA is frequently in the borderline or mentally retarded range. This was not always evident in the literature because normal intelligence frequently is a requirement for inclusion in studies. In studies such as the one by Ferry, et al. (1975), however, findings were consistent with this
study because nonverbal IQs were found to be in the retarded range in 22 of the 60 children.

Some of the children in this study were reported to have behavioral and emotional problems. It is difficult, however, to draw any conclusions since the type of problems varied among the children and were based on limited information.

Fine and gross motor abilities are considered part of the assessment of neurological integrity. Fine and gross motor delays were found in approximately one half the children. This is consistent with reports of a high incidence of soft neurological signs such as problems with fine motor coordination, alternate movements of the extremities, and gait (Yoss & Darley, 1974).

Consistent with these findings were the abnormal or soft neurological signs found in four children. The signs varied among the children, with the exception of three children who had a history of seizures. The results of the fine and gross motor evaluations and the neurological examinations suggest that neurological dysfunction is present in at least some of the children diagnosed as DVA.

Educational information was limited, but two of the five children were described as having specific learning disabilities. Further research in this area would be useful to determine if learning disabilities are associated with DVA.

Some of the children were found to have family histories of speech problems or neurologically based disorders such as mental retardation, seizures, and learning disabilities. Information about the type of speech or language disorder and whether the disorder was
on the maternal or paternal side of the family often was not provided. Support for an etiology of x-linked verbal apraxia or mental retardation therefore was not provided. One exception was the finding that only one of the 11 subjects was a female, suggesting a possible genetically based etiology.

A possible explanation for this variability among children with DVA may lie in the etiology suggested by Rosenbeck and Wertz (1972). They posited that the praxis centers for speech and other activities may be diffuse at first and that, if a lesion occurs before lateralization and localization take place, apraxia can be produced by lesions in a number of places. If this is true, perhaps the absence or presence of various problems other than apraxia is dependent on the severity or location of a lesion or dysfunction even if DVA is not.

The suggestion that DVA can be produced by lesions in a number of areas before lateralization and localization takes place may mean that praxis is more dependent on the integration of various parts of the brain than some activities such as fine motor coordination of the extremities. It is possible only to speculate about this until further research provides more information on the brain's functioning in regard to speech and language and other abilities.

The presence of problems other than speech and language in DVA children is consistent with Gubbay's (1975) suggestions that DVA can be part of the Minimal Brain Dysfunction Syndrome. The Minimal Brain Dysfunction Syndrome includes neurological abnormalities, specific learning disabilities, emotional lability, clumsiness, disorders of perception, and hyperkinesias. In this study, some children were found
to have low intelligence quotients, fine and gross motor delays, abnormal or *soft* neurological signs, emotional or behavioral problems, learning disabilities, and positive family histories. It is possible that some of these children may have what Gubbay termed the Minimal Brain Dysfunction Syndrome and suggests that, in many cases, DVA may be part of a generalized deficit.

Furthermore, the findings in this study seem to support the idea that DVA typically occurs with other deficits because all but one of the 11 children had problems in speech and language only. The rest of the children fell somewhere on a continuum between the child with only speech and language problems at one end and a child with problems in all the areas examined at the other end.

It is not clear in this study, however, whether the associated symptoms such as fine and gross motor functioning are unique to children with DVA or whether these difficulties are as frequently found in children with, for example, delayed intelligence or other types of neurologically based speech disorders. Comparative research on associated problems in children with various speech disorders would be necessary to arrive at conclusions regarding the uniqueness of their association with DVA. In other words, DVA as well as other speech disorders may occur with the associated symptoms described as being part of the Minimal Brain Dysfunction Syndrome or DVA may occur in isolation. As a speech therapist, it is useful to know that children diagnosed as having DVA often have many other organically based problems as well.

This study also supported the idea of the existence of a cluster of speech symptoms in children diagnosed as having DVA (Blakeley,
1980; Rosenbeck & Wertz, 1972; Yoss & Darley, 1974). These symptoms are considered by these authors to separate children with DVA from those with other articulatory problems. This study did not, however, compare children with DVA to those with other articulation problems. It confirms only that these symptoms exist in children already labelled with the term DVA.

The terminology used to classify articulation disorders should be defined in order to discuss the use of the label DVA as a means of separating children with this disorder from those with other articulation problems. Articulation disorders can be separated into functional (in that they have no known organic or psychological cause) and nonfunctional (in that they have a known organic cause) (Leonard, 1973; Yoss & Darley, 1974). Functional articulation disorders can be delayed or deviant.

On the basis of the functional articulation definition, it might be argued that the children diagnosed as DVA belong in the functionally deviant articulation group in that conclusive evidence of an organic etiology for children labelled DVA has not been found. This study and others, however, suggest that these children frequently have soft neurological signs related to the speech mechanism such as oral-motor problems, difficulty with diadochokinesis, inconsistent hypernasality, and those other soft neurological signs not related to speech such as fine and gross motor coordination and seizures. Frequently, intelligence is reduced; this also indicates neurological dysfunction. These indications of neurological dysfunction can suggest an organic cause of DVA and exclude those so identified from the category of functional articulation disorders.
Not all children diagnosed as having DVA have these indications of neurological dysfunction. This may mean that these children have been mislabelled or that the tools to determine neurological dysfunction are imperfect. The difficulty of accurate diagnosis lies in part in the controversy over what soft neurological signs are and when they are present.

Children with known organic causes of articulation disorders such as dysarthria are another major grouping. Although DVA has a suspected organic etiology, it is not a proven one. DVA, therefore, does not appear to belong completely to the category of functional or organic articulatory disorders. The only statement that can be made at this time with certainty is that DVA is an articulation deviancy, not a delay.

Another approach in dealing with the problem of labelling and diagnosis is to ignore the etiological basis and attempt to assign a label on the basis of speech behaviors that characterize DVA. If the errors made by children diagnosed as DVA are similar to those with functionally deviant articulation, this label might be more appropriate. There appears, however, to be little similarity between the descriptions of children's speech with functionally deviant articulation (Leonard 1973; McReynolds & Huston, 1971) and those with DVA (Blakeley, 1980; Yoss & Darley, 1974). With respect to its relationship to organically caused articulation disorders, Darley, Aronson, and Brown (1975) described organic articulation problems as differing from DVA in a number of ways.
It is not possible, therefore, to determine conclusively if DVA is a valid label. Neither does it seem to belong under the current classification system of functionally deviant or organically deviant articulation disorders, both of which are still ill defined and vaguely categorized.

In conclusion, although not experimentally validated at this time, this writer feels that the DVA label appears to have clinical applicability. These children do appear to have speech characteristics not identifiable as functional or that are recognized as organically based articulation deviances. Ultimately, however, the decision in regard to using the label or not depends upon the clinical reliability with which it is used in a particular setting and the agreement among the professionals as to its meaning. It well may be, however, that the best approach at this time is to describe the articulation disorder whenever possible rather than use labels that are ill defined and too broad to be helpful unless specific circumstances so demand.
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APPENDIX
TESTS USED IN EVALUATIONS

Language

1. Peabody Picture Vocabulary Test.
2. Test of Auditory Comprehension of Language.
3. Sequenced Inventory of Communication.
4. Articulation tests (not specified).
5. Screening Test for Developmental Apraxia of Speech.

Psychology

2. Wechsler Intelligence Scale for Children.

Fine Motor

1. CCD-motor Test for Upper Extremities.

Gross Motor

1. CCD-scale of Motor Progression for Control of the Head, Trunk, Lower Extremities and Locomotion.

Education

1. Developmental Assessment for Young Children.
2. Peabody Individual Achievement Test.
3. Woodcock-Johnson Psychoeducational Battery.
Neurological (typical examination as described by Scheinberg, 1981).

1. Mental status.
2. Gait and station.
3. Head and spine.
4. Cranial nerves.
5. Motor function
7. Reflexes.
8. Autonomic and sphincter function.