Furthermore, the difference in cost between the two treatments is considerable, as maintenance, even on one tablet a day, is nearly nine times as expensive as one injection of 100 μg. of vitamin B_{12} once a fortnight. So long as the nature and mode of action of intrinsic factor remain uncertain it is clearly preferable to rely on the parenteral administration of vitamin B_{12} for the routine treatment of pernicious anaemia. Although the majority of patients prefer taking tablets to receiving injections, more detailed investigation is required before oral treatment can be recommended.

Summary

A clinical trial of oral treatment of pernicious anaemia was carried out with a preparation containing a combination of vitamin B_{12} and intrinsic factor. In four out of five previously untreated patients there was a satisfactory response; in the fifth the treatment was ineffective, but the patient subsequently responded to parenteral vitamin B_{12}. In 12 patients previously treated with vitamin B_{12} by injection and transferred to maintenance treatment with the oral preparation the haemoglobin and red-cell count showed over one year a significant fall. In a further patient on oral maintenance therapy, features of subacute combined degeneration developed. It is concluded that at present oral therapy is not as reliable as vitamin B_{12} by injection.

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REFERENCES

Blackburn, E. K., Burke, J., Roseman, C., and Wayne, E. J. (1952). *British Medical Journal*, **2**, 245.
Callender, S. T., Turnbull, A., and Wakisaka, G. (1954). Ibid., **1**, 10.
Castle, W. B. (1953). *New Engl. J. Med.*, **249**, 603.
Chalmers, J. N. M., and Hall, Z. M. (1954). *British Medical Journal*, **1**, 1179.
Glass, G. B. J., and Boyd, L. J. (1953). *Blood*, **8**, 867.
Lowther, C. P., Alexander, W. D., and Hendry, E. B. (1954). *Lancet*, **1**, 495.
Ungley, C. C. (1950). *British Medical Journal*, **2**, 905.
Vilter, R. W. (1955). *Amer. J. clin. Nutr.*, **3**, 72.

DELAYED SPEECH AND DEVELOPMENTAL APHASIA

BY

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We intend in this paper to consider the causes and management of delayed speech. Delay in this important skill, raising as it does disturbing questions of general mental development, is an embarrassment to the child and an anxiety for the parents. Our continuing uncertainty about many of the cerebral processes involved in speech make it also a difficult diagnostic problem for the family doctor and specialist. In 1950 we (Miller and Morley, 1950) suggested the practical need for a simplified classification of speech disorders, and to relate the present observations to our general approach we have with minor modifications reproduced that classification here (Table I). Yet as some of the terms we use are common both to everyday conversation and to the more specialized terminology of speech disorders we must first say what they mean to us in our work and how they are used in this paper.

TABLE I.—*A Classification of Speech Disorders in Childhood*

Disorders arising from deafness
1. Congenital nerve deafness:
   (*a*) Severe for all frequencies.
   (*b*) Partial or high-frequency deafness.
2. Acquired nerve deafness.

Aphasia
1. Developmental aphasia:
   (*a*) Mainly receptive: (1) auditory imperception; (2) alexia.
   (*b*) Mainly expressive.
2. Acquired aphasia.
3. Aphasia associated with general mental deficiency.

Dysarthria
1. Cleft-palate and other anatomical defects of the speech organs.
2. Dysarthria associated with obvious cerebral palsy or other cerebral disease.
3. Dysarthria associated with minimal signs of cerebral palsy.
4. Isolated developmental dysarthria.

Dyslalia
Transient defects of consonant omission and substitution.

Stammering.

Concepts and Definitions

Although mechanistic analysis of such a finely integrated function cannot be pressed too far, it is helpful to think of speech as a triple process: the reception of words by the ear or the eye; their interpretation and synthesis as language within the brain; and, finally, the expression of this language response in further spoken or written words. We regard *speech* as covering the whole of this receptive, formative, and expressive activity.

Words are symbolic sounds which have a consistent range of meaning. In complete or embryonic form they are constructional units of thought and language, and in terms of the movement of speech are heard and understood, arranged and rearranged, and articulated and spoken. *Language* is both the word library of speech and the sum of those meaningful associations of words current in the life and literature of any society.

Aphasia is a breakdown in the comprehension and formulation of words giving rise to a disturbance of thought and a disorder of language. There is therefore both a receptive and an expressive component of aphasia. In some patients either the one or the other predominates, but in many there seems to be an impairment of more than one of the processes concerned and this impairment may be either partial or severe.

Developmental Aphasia

Our present understanding of aphasia is derived from our experience of cerebral injury and disease as they affect adult patients, and in this context the phrase "breakdown in the comprehension and formulation of words" is a proper description of what has taken place. In the children with whom we are mainly concerned in this paper, however, the process is a failure to develop the central processes of speech; a delay in building up a word library and creating a language. In spite of the difference, we propose to use the word aphasia for this disorder of childhood, and, since it becomes apparent during the time that speech is normally developed, to qualify it with the word developmental. For the delayed ability to read, which is often associated with such aphasia but can occur as a specific isolated difficulty, we use the term *alexia*.

Since some aphasic children develop articulatory defects when speech begins we must consider briefly two other conditions. The simpler is *dysarthria*, and implies to us slow and clumsy articulation arising from dysfunction of the muscles used in speech; such dysfunction is evident on physical examination. It does not involve any interference with the internal comprehension and construction of words. A disorder of articulation more commonly in our experience associated with aphasia seems to be an *articulatory apraxia*, and its relationship to aphasia calls for further consideration.

Articulatory Apraxia

In this condition a similar dysarthric difficulty is encountered in the absence of muscular clumsiness evident on examination. Muscular control appears to be adequate for all purposes other than the highly integrated movements of speech. Brain (1952) defines apraxia in general as an inability to carry out a purposive movement the nature of which the patient understands, in the absence of severe paralysis, sensory loss, or ataxia. It is clear that such a disturbance of cerebral function in relation to speech must be closely akin to expressive aphasia, and indeed Brain suggests that motor aphasia may justly be regarded as an apraxia of the purposive movements concerned in speech. In the present context, however, we would prefer to maintain a distinction between articulatory apraxia and aphasia: we have seen a number of children who had an evident articulatory apraxia without that delay in the development of speech which appears to us to be an essential feature of developmental aphasia. There is in such children no lack of fluency, and no evidence of impairment in the development of language. It appears to us that in these cases of articulatory apraxia the lesion must be at some point intermediate between that responsible for the spastic dysarthria which is evident on objective examination and the global failure of language development which is encountered in true developmental aphasia. For the present, therefore, we consider "articulatory apraxia" and "expressive" or "motor" aphasia as separate though closely related disorders.

Delayed Speech

The normal range of time for speech development in our community is given in the Chart and it is clearly possible to draw the line at which normality ends and delay begins at varying distances from the mean. We are concerned with speech delay which is such that the parents seek medical advice or which gives rise to real difficulty when the child enters school.

In the past six years we have seen 278 children with delayed speech in this sense, and the main conditions accounting for the delay are given in Table II. This is, of course, selected material coming to a speech department and gives no idea of the relative importance of these causes in the community as a whole. In the continuing study of 1,000 children of all social classes in Newcastle from which the chart was obtained there were four children at the age of 6 with delayed speech, and three of these were mentally defective. This observation brings out the predominant position of mental defect in the delayed development of speech. This, however, has always been recognized, and one intention of this paper is to emphasize the importance of developmental aphasia and deafness, especially unsuspected partial deafness.

TABLE II.—*Causes of Delay in Speaking in 278 Children*

Deafness	110	Mental deficiency	71
Developmental aphasia	72	Cerebral palsy	22
Transient	49	Mental illness	3
Prolonged	23		

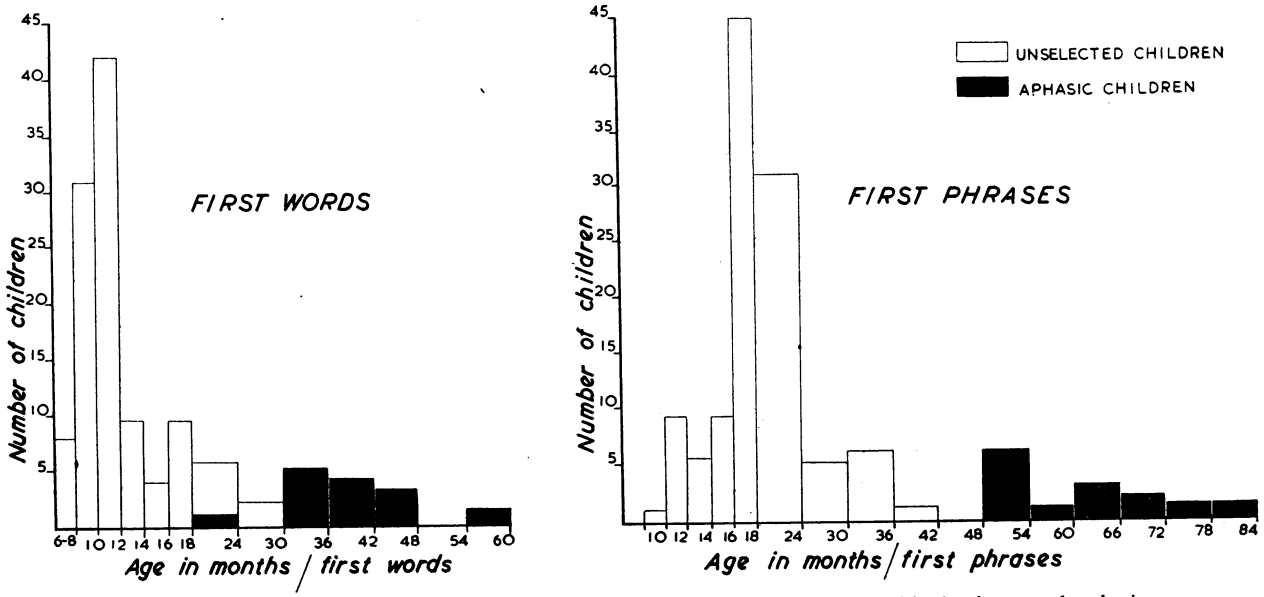
Developmental Aphasia

Before we give descriptive examples of developmental aphasia certain general observations will be made.

The group of children with aphasia with whom we are mainly concerned here did not develop any recognizable words until 2 years or later, or phrases until 4 years or later. What is more, when words or phrases did begin they often developed slowly, and there was an obvious poverty of language. A few children had one or two words early in the third year, but the development of language was arrested at that point for some time, with a further halting advance at 4 or 5 or later. Our definition of developmental aphasia of considerable degree is of course an arbitrary one, but the delay in speech development in this group in relation to a representative group of normal children is plainly shown in the Chart.

The 72 children can be divided into three main groups. There were 49 in whom speech developed fairly quickly and successfully after the age of 4, and who could be regarded as the terminal gradient on the slope of normal development. We have called these transient developmental aphasia. The second and third groups consist of 15 children with a more prolonged delay and inadequacy who are the main concern of this paper, and eight with intelligence quotients between 80 and 90 in which the speech delay was out of all proportion to their mental retardation. We have called these prolonged developmental aphasia. None of the children in the first two groups were below normal intelligence. They appeared emotionally stable, had normal hearing, and were free from other neurological abnormalities. The main features of the 15 children in the second group are given in Table III.

Social Class, Position in Family, and Birth Weight.—The social class of these families was essentially similar to the normal range for our community. None of these children were first children, and, apart from two twins born at term but weighing only 3 and 5 lb. (1.4 and 2.3 kg.) respectively, none were premature.



Age of development of speech in 114 unselected children and in 14 children with developmental aphasia.

TABLE III.—*Main Features of 15 Children with Developmental Aphasia*

Case	Age (Years and Months) First Visit	Age (Years and Months) Last Visit	Social Class of Father	Family Speech Disorders	Place in Family	Birth Weight lb. oz.	Birth Weight kg.	History of Birth Injury or Anoxia	Illness in First 3 Years	I.Q.	First (Years and Months) Words	First (Years and Months) Phrases	Alexia	Defective Articulation
M.B.	8–3	16–2	V	–	5	7 0	3·18	–	–	106	?	?	+	Apraxia
G.D.	7–8	12–4	III	–	3	11 8	5·2	+	–	100	4–9	6	+	,, Dysphonia
D.F.	6–2	10–11	III	–	3	6 0	2·7	–	+	100	3–0	6–6	+	Apraxia
A.H.	4–1	10–0	II	–	2	7 4	3·3	–	–	120	1–8	4–0	–	—
E.M.	3–6	9–4	III	–	2	9 3	4·17	–	–	130	2–6	5–3	+	—
D.P.	2–11	9–3	III	–	3	6 8	2·94	+	–	136	3–6	5–6	–	—
C.S.	4–4	9–10	III	–	2	6 4	2·8	–	–	96	3	4+	+	—
K.S.						3 0	1·36	+	–	93	3	4+	+	—
D.H.	4–5	8–2	V	–	4	6 0	2·7	–	–	100	3–9	5–0	+	Apraxia
D.H.						6 8	2·94	–	–	100	3–9	5–0	+	,,
K.C.	4–3	8–4	III	+	3	10 4	4·65	+	+	100	2+	5–6	+	,,
D.W.	4–4	8–2	III	+	2	7 0	3·18	–	+	106	3+	4+	+	Dyslalia
N.S.	6–2	6–5	III	–	3	5 1	2·3	–	–	100	2+	4+	Not assessed	—
A.W.	4–7	5–10	III	+	4	8 0	3·6	–	–	120	2+	4–6	,, ,,	Apraxia
D.R.	2–11	5–3	V	–	3	Not recorded		–	–	99	2–6	4–1	,, ,,	Dyslalia

TABLE IV.—*Intelligence of 15 Children with Developmental Aphasia*

Name:	M.B.	G.D.	D.F.	A.H.	E.M.	D.P.	D.H.	D.H.	K.C.	D.W.	A.W.	N.S.	C.S.	K.S.	D.R.
First test	A	DC	DC	DC	DC	MP	RM	RM	DC	DC	DC	WCP	WCP	WCP	WCP
I.Q.	106	100	100	114	129	120	100	100	91	106	120	100	96	93	99
Second test		WP	WP	A	WP	A			DC	RM					
I.Q.		91	98	120	130	136			100	105					
Months between testings		42	39	31	46	41	—	—	30	—	—	—	—	—	—

A = Alexander performance scale. DC = Drever Collins performance scale. WP = Performance tests of the Wechsler–Bellevue scale. MP = Performance tests of the Merrill–Palmer scale. RM = Raven's progressive matrices sets A, Ab, B. WCP = Performance tests of the Wechsler intelligence scale for children.

Birth Injury.—A mother's memory on this point is notably fallible and often ill-informed, so that reliable comment is difficult. In four children the possibility of perinatal anoxia or cerebral injury existed; G. D. was blue for several hours after birth; in D. P. normal breathing was not established for half an hour; K. S., the small second twin, was very ill for several days and was not expected to live; and K. C. had the cord tightly round his neck and was blue for the first two days.

Illness in the First Three Years of Life.—A similar factual difficulty is encountered when we try to assess the significance of illness arising during the normal period in which speech develops. Although this was specially asked for, illness was remembered in only three children. D. F. had measles at 9 months; K. C. had severe measles on his first birthday, jaundice at 18 months, and chicken-pox at 2; and D. W. had a doubtful rubella at 1 and measles at 2. None of these illnesses were associated with unconsciousness, prolonged drowsiness, or convulsions, and there is nothing in the histories of these 15 children to incriminate early cerebral inflammation as a cause of aphasia. Infective and, especially, virus illness in the mother during pregnancy may be important, but we have no information on this point.

Intelligence.—The results of the assessment of the intelligence of the 15 aphasic children are given in Table IV. All the I.Q.s are above 90, and therefore all the children may be regarded as not below normal so far as their intelligence is concerned. Non-verbal tests were, of course, used to measure the intelligence of these aphasic children.

Delayed Reading.—This was assessed on the Burt reading scale, and alexia of varying degree was found in 10 of the 12 children who were of sufficient age for testing. This may well continue much longer than the aphasia, and without special treatment it may prove a serious handicap throughout school life. The reading skill shown by these 10 children is given in Table V.

TABLE V.—*The Reading Skills of 10 Children with Developmental Aphasia and Alexia (Years and Months)*

Name:	M.B	G.D.	D.F.	E.M.	D.H.	D.H.	K.C.	D.W.	C.S.	K.S.
Chronological age	10–10	12–4	10–11	9–4	8–2	8–2	8–4	8–2	9–10	9–10
Reading age	Nil	9–3	Nil	5+	6–8	6–4	Nil	6–3	6–5	8–1

Assessment was made on the Burst scale. In addition, the Vernon and Durrell tests were also used in several children.

Defective Articulation was present in 9 of the 15 aphasic children. In two it was a simple dyslalia which responded readily to treatment. The remaining seven, including the fraternal twins, had an articulatory apraxia. One boy had an associated dysphonia, one still stammers under stress, and the identical twins have had a slight but persistent stammer from the age of 6 years.

Laterality.—Eight of the 15 are right-sided, one left-sided, and six show crossed laterality. This is not dissimilar to the pattern found by one of us when testing a large number of schoolchildren in this community.

Delayed and Defective Speech in the Family.—Here again our information is almost certainly incomplete. The mother of D. W. stammered from 4 until 9. A. W.'s brother, who is now 10, was also slow in developing speech, and a paternal cousin of K. C. did not begin to speak until the age of 5. This is a very different family incidence from the children with developmental dysarthria (Morley *et al.*, 1954), where speech disorders in other members of the family were present in more than half.

We have therefore found no evidence that social class, position in family, birth injury, illness during the normal period of speech development, laterality, or family predisposition has any clear relationship to developmental aphasia, the aetiology of which remains unknown.

In view of the considerable delay in starting to speak and the continuing difficulties with language which these 15 children experienced we feel that they probably express a true cerebral disorder and not merely the extreme end of the normal range of speech development.

Receptive Aphasia

(1) N. S., aged 6 years 5 months, the first twin of a second pregnancy, was born at the 35th week and weighed 5 lb. 1 oz. (2.3 kg.). His father is a driver-salesman, and both parents are sensible folk with no history of speech disorder. The boy's birth was normal and he has been healthy since. Until he went to school his parents regarded him as more intelligent than his fraternal twin. They had noticed that he used gesture a good deal, but failed to recognize that they were also using it readily in return. One or two words came late in the second year, but his vocabulary did not increase until he was 4. Phrases developed in the fifth year, but remained few and far

between. He was not silent in his earlier days and often "made a lot of noise which no one could understand." He never seemed to understand spoken requests and made no attempt to lip-read. He responded quickly to gesture, and was so observant that he would often do things without a specific request being made. He could not understand the other children, and was the "odd man out" in the street games, but he was affectionate and helped a great deal in the house and garden. If he could not make himself understood he would often cry, but rarely had a violent tantrum. After eighteen months at school his teachers were quite unable to teach him.

He is a friendly boy who is physically normal, hears the common sounds of the house, and plays sensibly with toys. He does not understand speech beyond a small number of nouns, but responds quickly and accurately to gesture. His speech consists of a small number of words, and phrases which are often noun equivalents—for example, "sit on"=chair, "cut it off"=scissors, "goes up in air"=aeroplane—and he tends to repeat the word or phrase several times. His articulation is good. His I.Q. on two separate occasions was 100. Audiometry has proved difficult, and, though there may be some deafness, it is quite insufficient to account for his delay and difficulty with speech. He would appear to be an example of congenital auditory imperception.

Expressive Aphasia

(2) A. H., aged 10, is the second child of intelligent professional parents. His birth was normal and general development satisfactory. Words began at 20 months, but phrases did not follow until after 4. At 6 he still avoided the use of verbs, and, though willing to tell a story with gusto, got it thoroughly muddled through omitting, transposing, and mispronouncing his words. He has normal hearing and comprehension, and at 8 years and 10 months his I.Q. was 120.

He is, however, a tense, restless boy, and recovery may have been delayed by his personality and the parents' desire for rapid improvement and academic success. Though his speech continues to improve, his mother still considers him "a proper Mrs. Malaprop." His articulation is excellent and his mispronunciations are similar to those met with in acquired aphasia. Presumably this boy recalls sound sequences imperfectly. When he knows exactly what he wants to say his speech is sensible and clear; when he cannot get the word or cannot get the right one, distortions and substitutions occur and speech is jumbled and confused. In writing he expresses himself much more clearly. We consider this boy has an expressive aphasia, perhaps arising in part from a poor auditory memory for speech.

(3) E. M., aged 9 years 4 months, is the second child of three. His father is a skilled worker and there are no speech disorders in the family. His birth was normal and general development satisfactory. Three or four words came by the age of 2½ years, but there was little increase until after 4. Phrases began at 5 years and 3 months, and following this he developed a dyslalia which cleared within the next year. He seemed to understand speech from an early age and obeyed verbal requests very readily. He has always been a very good-tempered boy.

He is happy and healthy, with normal hearing. His I.Q. at 8 years and 3 months was 130. From 8 onwards his speech has improved, though he is still a boy of few words. When excited or anxious his speech may become halting and confused. We have regarded this not as a true stammer but as the disturbance under stress of a newly acquired and precariously balanced function with an underlying impairment of structural origin. At 9 he still has a severe alexia. We regard this boy as essentially an example of expressive or motor aphasia with associated alexia.

Mixed Aphasia

(4) D. F., aged 10 years 11 months, is the third child in a family of five. Both parents are sensible folk and there is no history of speech disorder. He was born normally, but had severe whooping-cough at 3 months and measles at 9 months. After his first year he developed satisfactorily apart from speech. Words began at 3 years, but phrases did not follow until 6 years 6 months. Although he understood some speech before he spoke, his comprehension of quite a number of common words was still uncertain when first seen in his seventh year.

He is lively and active, playing well but always talking much less than his brothers and sisters. On occasion his parents noticed that he whispered to himself as if to rehearse the word or phrase before using it. At nearly 11 he is a shy well-developed boy with normal hearing, and no abnormality in his nervous system. His I.Q. is 100. Even when you know him he is taciturn, and has a marked economy of phrase. Articulation is still hampered by an associated slight apraxia, and he is still unable to read at all. This boy has a severe developmental aphasia, alexia, and an associated articulatory apraxia.

Differential Diagnosis of Delayed Speech

Faced with delayed speech, we have to decide whether this is due to mental deficiency, deafness, developmental aphasia, cerebral palsy, maternal deprivation, or in rare instances to psychotic illness. Children in whom the delay is associated with evident cerebral palsy or cerebral disease will have other and more obvious features pointing to those conditions. In our attempts to master the natural history of speech development and its deviations we have observed a variety of children. The study of the 1,000 children in Newcastle which has already been mentioned is now in its eighth year, and this has provided a representative community context for our hospital experience. In addition to the 15 children with severe aphasia and the eight with aphasia and slight mental retardation, we have observed concurrently 13 selected children with varying degrees of deafness, five children with alexia as their only or dominating disability, and four children with acquired aphasia after head injury or cerebral disease. This experience has made us keenly aware of the diagnostic difficulties and pitfalls which face the family doctor when he first sees a child with delayed speech.

Mental deficiency is without doubt the commonest cause, but information about the child's general development is often hindered by the parents' natural reluctance to consider this explanation and the conscious or unconscious inaccuracy of their answers. On the other hand, the failure of the deaf or aphasic child to understand speech may lead all too easily to the false assumption that he is mentally defective. To complicate the situation further, in all three the absence or limitation of speech rules out verbal methods of testing intelligence.

We have found the following simple questions and observations useful in the initial assessment. Does the child respond intelligently to everyday sounds? Does he respond to speech, especially when his back is turned, or from another room? Does he watch the speaker's lips? Does he grasp simple and more complicated requests easily or with difficulty? How does he make his wants known? How do the family respond to him—by speech or by gesture? What kinds of play interest him most, and can he play for reasonable periods on his own? Does he take care of his toys or is he disturbingly destructive? What is the child's temperament—sensible, good-humoured, and interested, or restless and lacking in attention and concentration? If tantrums occur, are they capricious or only related to failure on the part of others to understand what he wants?

While this history is being taken from the parents the doctor can usefully watch the child's behaviour with the toys in the consulting-room. He can then begin the direct examination by showing the child a number of familiar pictures and asking him to point to the ones he names. After this he can be asked to carry out simple requests, leading up gradually to a careful examination of the ears and the nervous system, with special reference to the range

of tongue movements and the presence of spasticity or athetosis. If speech is forthcoming its extent and quality can be assessed. This initial clinical screening will generally need several interviews, and the first one at least is better carried out in the child's home. This simple clinical approach can give useful if limited guidance in many children. Suspicion of mental defect will be aroused by excessive restlessness, lack of concentration, inability to design and sustain individual play for any length of time, destructiveness, and capricious emotional behaviour. However, the exact diagnosis of moderate mental retardation demands expert assessment before suspicion is allowed to harden into certainty.

With severe deafness for all frequencies, the child fails to respond to some or all of the everyday noises as well as to speech. What is more, he may be so frustrated by his failure to understand and be understood that he has violent tantrums and may be first brought to the doctor as a behaviour problem. On the other hand, in the child with high-frequency deafness, the beginnings of speech are not greatly delayed, and so the condition is often missed, sometimes well into school life. Such a child may be treated for defective articulation unless inquiry has recorded that he responds well to sounds and music, but hardly at all to speech and stories unless he can watch the speaker's face. The most difficult children to distinguish from these with developmental aphasia, however, are children with varying degrees of partial deafness, particularly with loss in the middle frequency range. These children are not silent, even though speech is delayed, and sensible parents have often failed to recognize the true state of affairs. In any child with delayed speech, especially where comprehension is defective, hearing must be thoroughly investigated by an otologist expert in screening tests and audiometric methods suitable for children of different ages. Only in this way will these children with partial deafness be discovered and proper training instituted, with the use of hearing-aids where necessary. The distinction between partial deafness and receptive aphasia can be particularly difficult and may require further observation or even a period of residence in a special school. Moor House School is the only school in the country at present which caters for the more complex disorders of speech.

Where the delayed speech is due to a mainly expressive aphasia there is good understanding for speech, and if the child's intelligence is normal there should be little difficulty in its recognition. The psychotic child with delayed speech, though uncommon, may go unrecognized for a long time under the label of mental defect or behaviour difficulties. Gradually, however, a more bizarre pattern of behaviour becomes evident and psychiatric help is sought.

Treatment

As we believe the delayed development of the use of language is the manifestation of delayed neurological development, attempts to teach a child to speak will produce little result. It is necessary, however, that nothing should be done which would hinder the child's spontaneous development. Overanxiety on the part of the parents, repeated attempts to make the child say words, undue attention paid to any words he may say, producing self-consciousness, will tend to delay rather than assist the development of speech. It is, however, necessary to ensure that there is sufficient environmental stimulus. The natural urge to speak is usually so strong that the child will speak if he can; but contact with children of his own age may be helpful, and, without producing any undue sense of frustration in the child, the mother should not always respond immediately to gestures, but should so manage the situation that the child realizes the usefulness of words rather than signs.

An interest in naming objects and pictures may be stimulated if the child is asked to point to an object named rather than asked, until he is ready, to say the word himself. A collection of pictures in a scrapbook of his own may also help. If the child is more than usually silent and has done little or no babbling, it may be possible to encourage imitation of oral sounds such as consonants or vowels, or animal noises, and in one boy of 6½ years the ability to pick out and make the appropriate sound for a single letter is developing before he is able to use words. If the child of 5 years understands speech he can attend the normal school and can learn even if he cannot speak. If, however, use of expressive speech is delayed beyond the age of 6 years his educational progress will be seriously handicapped.

Summary

We have presented our experience of the delayed development of speech. The main causes are mental deficiency, severe and partial deafness, developmental aphasia, cerebral palsy, and, rarely, psychotic illness.

High-frequency deafness is not associated with any great delay in speaking. In spite of traditional objections we favour the term developmental aphasia for children who are mentally normal, have good hearing and no evidence of cerebral palsy or disease, but show a considerable delay in the development of speech. We have no idea of the true incidence of this condition, but the fact that in the past six years we have seen 15 striking examples and 49 children with a similar though less prolonged delay makes developmental aphasia a more important cause of delayed speech than we expected.

The help which can reasonably be expected from speech therapy has been described. It is important to remember that in many of these children there is a complicating alexia, and that in some there are difficulties with articulation as well.

BIBLIOGRAPHY

Brain, W. Russell (1952). *Diseases of the Nervous System.* Oxford Medical Publications, London.
Drought, C. Worster, and Allen, I. M. (1928). *J. Neurol. Psychopath.*, **9**, 193, 289.
Miller, H., and Morley, M. (1950). *Proc. roy. Soc. Med.*, **43**, 579.
Morley, M., Court, D., and Miller, H. (1954). *British Medical Journal*, **1**, 8.
Spence, J. C., Walton, W. S., Miller, F. J. W., and Court, S. D. M. (1954). *A Thousand Families in Newcastle upon Tyne.* Oxford, London.

SIGNS OF OBSTRUCTION OF THE SUPERIOR LONGITUDINAL SINUS FOLLOWING CLOSED HEAD INJURIES (TRAUMATIC HYDROCEPHALUS)

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The *British Medical Journal* has published some of the most important contributions to the knowledge of the clinical effects of non-infective thrombosis of the intracranial venous structures and the conditions in which such thrombosis is likely to occur. Among the more comprehensive papers we may recall those of Collier (1891), Holmes and Sargent (1915), Symonds (1940), Martin and Sheehan (1941), Martin (1941). Full notice is taken of these contributions in the recent monograph of Garcin and Pestel (1949), in which the whole subject is admirably reviewed.

There is another small group of cases which I believe to be of a similar nature, and which has been slow to receive general recognition. It is a group of cases of head injury in which signs of raised intracranial pressure