Acquired Aphasia in Childhood with Seizure Disorder : A Heterogeneous Syndrome

Deonna, Th., Beaumanoir, A., Gaillard, F. & Assal, G.

Neuropädiatrie, 1977, 8, 3, 263-273

Summary

The authors report six children with acquired aphasia of unknown etiology. The clinical picture was clearly different from that seen in the usual childhood aphasias and resemble other cases initially reported as “syndrome of acquired aphasia with convulsive disorder”. All had associated paroxysmal EEG abnormalitics and 5 have had clinical seizures. The language disorder has improved or remained stationary and no other neurological signs have developed. Our review of the literature and the study of our personal cases show no uniform clinical picture in these children. Three different clinical patterns seem to emerge. The first group show rapid onset and recovery of aphasia, frequent fluctuations in the severity of the language deficit typical of so-called epileptic aphasia. Thèee children appear to have a better prognosis. The second group show worsening of the aphasic deficit after repeated seizures or episodes of aphasia. In the third group progressive deficit in language comprehension (auditory agnosia) with a variable degree of recovery and rare or no clinical seizures.

The possible significance of the EEG abnormalities has been discussed and the importance of the aphasia on general behavior and the problems of differential diagnosis have been stressed.

Key words

Aphasia, seizure disorder, paroxysmal electroencephalographic abnormalitics

Résumé

Les auteurs rapportent six cas d’aphasie acquise d’origine indéterminée dont les caractéristiques cliniques sont très différentes des aphasies de l’enfant habituelles. Nos cas ressemblent à d’autres décrits initialement par Landau et Kleffner sous le titre de « syndrome d’aphasie acquise avec convulsions ». Notre revue de la littérature et l’étude de nos cas personnels montrent de grandes différences cliniques entres les cas. Trois groupes principaux semblent se dégager. Le premier groupe comprend des enfants dont l’aphasie a un début et une récupération rapide, avec des fluctuations fréquentes du déficit du langage, typique de l’aphasie épileptique. Ces enfants semblent avoir le meilleur pronostic. Dans le second groupe, il y a une aggravation et une persistance de l’aphasie après des crises épileptiques ou des épisodes répétés d’aphasie. Dans le dernier groupe, on observe un déficit progressif de la compréhension du langage (agnosie auditive) avec un degré variable de récupération et de rares ou même une absence de crises épileptiques.

L’importance des anomalies électroencéphalographiques et les conséquences de l’aphasie sur le comportement de l’enfant ainsi que les problèmes de diagnostic différentiel sont discutés.

Mots clés

Aphasie, comitialité, épilepsie, électroencéphalographie

Acquired Aphasia in Childhood with Seizure Disorder : A Heterogeneous Syndrome

Introduction

Unusual forms of acquired aphasia in children associated with a seizure disorder and paroxysmal electro-encephalographic abnormalities, quite different in their clinical characteristics from the usual childhood aphasias have been increasingly reported since et Landau and Kleffners original publication (1957).

These children usually have a normal psychomotor and speech development with no history or signs of previous encephalopathy. They develop an elective loss of acquired speech with a total or relative preservation of nonverbal intellectual functions and usually show no evident cause of acquired focal brain lesion. However, all constantly display paroxysmal electroencephalographic abnormalities and most children have clinical seizures at sometime or other during the course of their illness. The course of the illness, although sometimes protracted is usually self-limited and the children either recuperate or remain permanently aphasic.

The purpose of the present paper is to report a personal series of 6 such cases with longitudinal clinical, electroencephalographic and neuropsychological evaluation and to review the literature on this "syndrome of acquired aphasta with convulsive disorder" with regard to the following main questions: Is the syndrome uniform due to a specific etiology, and what is the relation of the aphasia to the seizure disorder, i.e. its relation to epileptic aphasia?

Our review of the literature and the study of our personal cases suggest that at least 3 different varieties of the syndrome can be distinguished on the basis of their clinical characteristics.

Material and methods

6 children, 5 boys and 1 girl with the syndrome have been observed in the department of Pediatrics of the University Children's Hospital of Lausanne and Geneva. All children have been hospitalized sometime in the course of their illness though none was known to the authors before the onset of the aphasia. Case histories are thus mainly retrospective and in many cases incomplete. The histories have been taken from former clinical records and from the parents. All children have undergone detailed neurological examination at various times in the course of their illness. All have had a normal psychomotor development prior to the onset of aphasia, all have shown paroxysmal EEG abnormalities in association with the aphasia, 5 have had clinical seizures, none have manifested any fixed neurological deficit besides the aphasia and some have presented associated neuropsychological disturbances. In all cases the clinical course was either stationary or regressive.

Detailed neuropsychological examinations have been performed at the time of the fixed deficit or after recovery by the same psychologist (F. G.). Psychological assessments at the onset of aphasia have not been collected systematically and only sparse notes could be obtained from the records. The neuropsychological evaluation comprised of the following items

a) General intelligence rating: all children but one (case 1) were assessed with the Wechsler intelligence scale (only the performance part could be completed with cases 2, 3 and 5). Case 1 bas been tested with the Borel-Maisonny scale.

b) Language: oral expression and automatic speech (recorded), object naming (visual and tactile), imitation of sounds, words, sentences and digit series, singing with and without words, rhythmical tapping, comprehension of language, object pointing, dichotic listening (case 6 only), production of sentences with given words, verbal fluency, verbal memory (learning, short and long term recall), reading, writing ability, mental and written arithmetics. Sound recognition was tested with familiar sounds and with a task of matching 3 new sounds (learning).

c) Gnosias and praxias: body part identification, right-left discrimination and Head's "hand-eye-ear" test; tactile finger identification, stereognosia. Bucco-linguo-facial imitation, ideomotor gestures, imitation of movements without meaning, object utilization, dressing and undressing.

Fine motor development and lateralization: hand strength, Purdue pegboard, cutting with scissors, finger elevation, feet and eye dominance.

e) Visuo-spatial abilities: drawing of familiar shapes, copying and reproducing from memory (short and long term), visual gnosias for simple, schematic, overlapped and incompleted figures, colour perception and colouring.

Electroencephalograms have been obtained in all cases at some time during the illness, rarely in the initial phase but then checked repeatedly over the course of many years. All were systematically reviewed (A. B.).

Results

Table I summarizes the main clinical data on these 6 children. We have chosen a diagrammatic representation to allow easier comparison of the children. We believe that a detailed case history for each child would not reveal any further pertinent information. A more detailed discussion of all relevant data (aphasia, seizure disorder, EEG abnormalities, neuropsychological evaluation) is given for the whole group and references to any particular case is made when necessary.

Table 1. Summary of clinical findings

Patients:	AT □ 6 years	MR □ 9 ½ y.	RG □ 10 y.	PA □ 10½ y.	CS □ 11½ y.	MZ □ 20 y.
Age at onset of aphasia (y.)	4	5½	3½	9	4	6½, 7½
Language prior to aphasia	Normal	Normal	Moderately retarded	Normal	Normal	Poor prononciation
Mode of onset of aphasia	Abrupt	Progressive (months) first thought	Progressive (months) suspicion of hearing	Progressive (weeks) comprehension	Progressive (months) lack of comprehension	First episode: gradual (weeks), second episode:

		deaf	difficulties	diff., stuttering	and expression	rapid (5 days)
Fluctuation of aphasia	No	No	No	Yes	No	Yes, 2 separate episodes

Table 1. Summary of clinical findings (continuing)

Clin.seizures Prior aphasia At time of aph. Later	1½ y. - +	- - +	- - -	17 months + +	- - +	- + (2d.episode) +
Seizures types Frequency	½clonic +	Myoclonic(few) 1 x GM 1x psychomot	-	Febrile seiz. Psychomot. s.	1 x nocturnal type	1xFocal motorR 1 x GM
Evolution of seizures	Still heaving seizures	No seizure for the past 2½ y.	Never any	Occasional "absences", controlled with medication	No seizure for past 9 years	No seizure since age 7 (normal EEG)
Number of EEG	11	6	4	15	5	4
Time between onset of aphasia and first EEG	-	6 months	1½ y.	-	1 y.	1 month

Table 1. Summary of clinical findings (continuing)

Patients:	AT □ 6 years	MR □ 9 ½ y.	RG □ 10 y.	PA □ 10½ y.	CS □ 11½ y.	MZ □ 20 y.
EEG follow-up from onset of	1 y.	2½ y.	4 y.	1 ½ y.	5 y.	6 y.

aphasia						
EEG Normalizat.	-	-	+	-	+	+
Neuropsych. Manual dom.	R (?)	Ambidextrous	R + +	Ambidextrous	Ambidextrous	L + + Dichot: L-ear superiority
Intelligence rating	Borel-Maison. 3-4 y.	WISC V=0 P=64	WISC V=0 P=87	WISC V=62 P=107	WISC V=0 P=87	WAIS V=109 P=119
Sound recognition	Intes-table	Normal	Complete failure	Normal	Possible with training	Normal
Visuo-spatial abilities	Intes-table	5 y.-level	Normal	Normal	7½ y.-level	Normal

Table 1. Summary of clinical findings (continuing)

Outcome of aphasia duration of follow-up:years	No recovery, no compre-hension, no expression Durin 2 y.2 y.	Progressive continuing recovery, compre-hension: 5 y.-level, expression: 2 y.-level 4 y.	No recovery after 7 years, auditory agnosia, no verbal expression 6½ y.	Persistant moderate dysphasia, comprehension & repetition deficits, dyslexia, dysgraphia 1½ y.	No recovery after 8 years, pure word deafness 7½ y.	1) total recovery in 3 months 2) total recovery over years 13 y.

1. Diagnosis and evolution of the aphasia

4 children have had normal speech (case 1, 2, 4, 5) prior to the onset of aphasia. One has had “poor prononciation” but has been able to understand everything (case 6), one girl (case 3) has spoken “poorly for her age” but has understood everything and bas been able to make herself be entirely understood. It is very difficult to learn in retrospect how speech loss has occurred with only the interview with the parents and the review of clinical records as a guide. In 3 cases, it has occurred gradually over the course of a few months (cases 2, 3, 5), one case (case 5) within weeks with fluctuations. In one case (case 6), aphasia has occurred in two separate episodes the first being gradual and the second rapid and total within 5 days. One child has lost speech over night (case 1). In all but one case (case 4) speech production has been totally lost at some point and comprehension has been absent in 3. Two of these children have been initially regarded as becoming deaf. It is impossible to be sure what has been lost first, but in two cases lack of speech comrehension appears

predominant while in two other cases both expression and comprehension seem to have disappeared simultaneously.

In 5 of 6 cases, a primary psychiatric reaction has been thought to be the cause of speech loss and has in each case been connected with a traumatic psychological event. In one child, speech deterioration has been interpreted as linked with the seizure disorder (case 4). Four children were initially treated with psychotherapy (cases 2, 3, 4, 5).

One boy, now 20 years of age, has recovered completely (case 6) one child (case 2) now 9½ years old, is gradually recovering from total speech loss and verbal comprehension deficit (case 5); one child (case 4) has a moderate mixed persistent dysphasia with fluctuations. Three children (cases 1, 3, 5) have remained totally aphasic with no language comprehension (auditory agnosia) and have a limited or absent verbal output; two of them are being treated in a school for deaf children (cases 3 and 5).

2. Seizure disorder and EEG findings

The family history is negative for epilepsy in all cases. The time relationship between onset of seizures and aphasia, the type and frequency of seizures and the outcome of the disorder have been studied. There is no constant relationship in time between the occurrence of clinicallv recognized seizures and the onset of aphasia. Two have had seizures a long time ago (2½ years and 17 months), only 2 have had seizures on the day preceeding the onset and two have had seizures long after aphasia had developed. Seizures have been of various types. Two have had right focal motor seizures, two psychomotor seizures, one a long history of convulsions with fever (case 4). In all cases, the seizures have been infrequent and no episode of status epilepticus has been recorded.

44 EEGs recorded over a period ranging from 1 to 6 years have been reviewed. In 4 cases, a significant delay has occurred between the onset of aphasia and the first EEG, owing to the initial lack of recognition of the organic nature of the problem. The duration of follow-up EEG recordings has been variable, but 3 children whose tracings have been registered over the longest interval have since returned to normal.

The major abnormality consists of slow spikes sometimes followed by a slow wave which is either isolated or grouped in long bursts or even continuous in the same tracing. The spikes are predominantly located over the posterior territories of both hemispheres, in the posterior temporal and parietal regions. No paroxysmal abnormalities can be seen in the anterior temporal or fronto-central regions. These anomalies are always bilateral and asynchronous over the hemisphere with a left sided predominance except in one case where it is consistently right sided (case 1). A striking feature in all cases is the variability in space, time, and intensity of these focal discharges, which are best recorded in the children who have had frequent tracings. In addition to focal anomalies, multifocal slow spikes and bursts of spikes which are never synchronous can be noted at one time or other in all tracings, in 2 cases in the first tracing. There are also bilateral slow wave anomalies the theta range (in 12 tracings regarding 4 children) and these are located in the same region as the spikes and are predominant on the side where the spikes are most frequent. The basic rhythm is normal in all 11 tracings; in 2, multifocal spikes obscure any basic rhythm; in one tracing abnormal background activity is due to drug intoxication.

Neuropsychological evaluation and outcome of the aphasia

Because of the wide variation in age and in the degree of preservation of language and of intellectual ability at the time of testing, no significant comparison can be made. Two children have persistent auditory agnosia (cases 3 and 5), 6½ and 7½ years after the onset of their aphasia. One of them (case 5) can identify non-speech sounds after training, but does not recognize any speech sounds (verbal auditory agnosia or pure word deafness). He is being treated as if he were deaf but despite good non-verbal performances (WISC performance 87) has difficulties with deaf sign language and in lip reading. The other child (case 3), 7 years after the onset of his aphasia, has no understanding of the meaning of sounds despite normal hearing and has no means of verbal expression. One child (case 2) who initially showed global mental deterioration in addition to total speech loss has also retarded non-verbal functions (5 years level) and an even more severe verbal deficit (2 or 3 years level) four years after the onset of the disease.

Our youngest child (case 1) shows no speech comprehension and expression at the age of 5, 2 years after the onset. It is impossible to get him to collaborate adequately for psychological testing. A 2 years discrepancy between his verbal and non-verbal performances is evident. One child (case 4) with fluctuating dysphasia has a striking discrepancy between his verbal and performance IQ (62/107) with particular difficulties with speech comprehension and repetition and marked dysgraphia, dyslexia and dyscalculia.

Our oldest case, now 20 years of age, is entirely normal.

Review of the literature

Reports published in psychiatrie, neurological and specialized speech literature have appeared under various titles. This reflects the many different aspects of this syndrome and the varied interpretations of the behavior of a these children (Landau and Kleffner, 1957; Rose 1969; Worster-Drought, 1971; Stein and Cury, 1968; Huskisson, 1973; Gascon, 1973; Shoumaker, 1974; McKinney and McGreal, 1974; Brissaud and Richardet, 1974; Koupernik, 1969). Because of the heterogeneity of the syndrome, a complete review of all available literature is not to be expected.

39 cases in 10 publications over the past 20 years have been reported in sufficient detail to cover some of the following important points: developmental history, particularly the quality of speech prior to aphasia, the mode of onset of aphasia and its major clinical characteristics, the fluctuations in the severity of the aphasia, the mode of recovery and the temporal relationship of the aphasia to the seizure disorder.

The majority of the children are between 3 and 7 years of age (35/39) at the onset of the aphasia and only one has its onset after 10 years (youngest 2 years, oldest 13 years). Data on sex is too incomplete to be significant. 35 have normal speech before the onset of aphasia and in only 5 cases is speech reported as having been “retarded or abnormal”. The mode of onset of aphasia is described as having occurred rapidly within one to two days in 4 children, over a few days in another 4, progressively over weeks or months in 6 children; in the rest of the cases the description is not sufficiently detailed.

Fluctuations in the severity of aphasia suggestive of a functional deficit related to the seizure mechanism are described in 8 cases, 6 of which finally make a good recovery and 2 of which show little or no recovery. In 17 cases, no fluctuation of the aphasia is reported, 7 of these make a good recovery and 10 show little or no recovery.

The temporal relationship of the seizures to the aphasia is clearly stated 21 cases. In 7 children, seizures occur immediately preceeding and in 1 case immediately following the onset of aphasia. 9 children have seizures temporally unrelated to the aphasia, 4 in the preceeding weeks or months and 5 after the onset of aphasia. In several cases, focal motor seizures and post-ictal focal neurological disturbances are noted.

No attempt has been made in the above review to give detailed reports of EEG abnormalities. By definition (see introduction) all children had paroxysmal EEG abnormalities (spike-waves usually diffuse but more often with temporal predominance or more rarely an isolated temporal spike dysrhythmia).

It is remarkable to note that even though many children had spinal fluid examinations, contrast studies (pneumo- encephalograms, arteriograms) in no case is any specific diagnosis made, either at the time of onset or later. Except for some dilatation of lateral ventricles and asymmetry of temporal horns mentioned in 3 cases, no significant structural abnormalities are described in the air encephalograms.

Discussion

The clinical course and the characteristics of the aphasia in our children and in those quoted in the literature viewed are different from the usually described childhood aphasias with brain lesions. This has been noted by most authors. However, it is apparent that these children do not constitute a homogeneous clinical group. On the basis of clinical features of the aphasia (mode of onset, type of aphasia, evolution of the language disorder) 3 different clinical varieties the syndrome seem to emerge.

The first would include those children in which aphasia develops abruptly, regresses rapidly or shows marked fluctuations in severity usually in association with seizures, clinically suggesting a cortical onsetin areas closely related to the speech regions (i. e. right focal motor seizures, psychomotor seizures). These are examples of epileptic aphasia, similar to those described in adult patients (Hécaen and Angelergues,1960; Alajouanine and Sahouraud 1960). Although the language deficit persists sometimes for a longer period then would be expected in post-ictal aphasias, the recovery seems nonetheless too rapid as to be explained by a shift in speech dominance. The aphasia probably represents a functional disconnection of language mechanisms without any newly acquired lesion. The nature of the underlying focus is unknown but could be congenital in some cases, especially those with retarded speech development.

The second group concerns those children where no recovery takes place after a seizure or after repeated episodes of aphasia. The mechanism here may be analogous to the progressive hemiparesis which sometimes follow recurrent focal motor seizures during the course of chronic focal motor epilepsy. When recovery gradually takes place in the course of months or years, however, it may be assumed that the other hemisphere has “taken over speech”. This is well illustrated by our case 6, who probably had left hemispheric dominance for speech at the age of 6 (aphasia with a left sided EEG focus and right focal seizures) and who now at the age of 20 is completely left handed and has a left ear predominance in the dichotic listening test suggesting a right hemispheric dominance for speech.

The third group consists of children who gradually develop a marked deficit in auditory comprehension, very unlike what is observed in the more usual childhood aphasias. They may have few or no seizures at all, and are often initially diagnosed as becoming deaf. These children show variable degree of recovery, often minimal even after many years. Worster-

Drought (1971) has suggested that the pathological process in these children may be some sort of "slow grade non progressive encephalitis of the autoimmune variety" because of the subacute and non-progressive course of the aphasia. So far, no laboratory evidence either in the cerebrospinal fluid or in the blood has proved this hypothesis, but it should be remembered that the initial examinations are usually carried out rather late in the course of the aphasia, due to its insidious onset. Considering the capacity of an intact hemisphere to "take over" speech in a child with unilateral hemispheric lesion, bilateral lesions may be assumed in those children who show no recovery after many years. 2 of our children with prolonged follow-ups seem to belong to this group. One has verbal auditory agnosia (pure word deafness), the other has a total absence of auditory comprehension. These cases resemble adult cases with proven bilateral temporal lesions involving Heschl's gyrus (Jerger 1972). In some of these children, the visuo-verbal functions can be remarkably preserved and they can be taught to write quite well (Worster-Drought 1971).

In addition to the underlying cerebral pathology and influence of the seizure disorder in these children, many other factors can probably influence the course of the aphasia. Hécaen and Piercy (1956) and Brown and Hécaen (1976) from studies of aphasia in left handers and children, suggest that lateralization of language functions to one hemisphere and localization within this hemisphere are variable and progressive phenomena occurring at different rates with maturation.It is possible that the children under discussion have an unusual genetic or acquired pattern of cerebral organization which renders them particularly sensitive to brain damage or seizure activity as far as language is concerned. The problem of handedness has not yet been studied in enough detail in any of the children with the syndrome of acquired aphasia with seizure disorder. It is however interesting to note that one of our patients is completely left-handed, 3 have no definite hand preference and only one is fully right handed.

We have not found any definite correlation between the evolution of the paroxysmal EEG abnormalities and the clinical outcome nor any significant electroencephalographic differences between the three groups described. The spontaneous fluctuations of the abnormalities, the tendency to improve with time, the usually normal fundamental rhythm and the focalisation of the discharges argue strongly against a diffuse progressive encephalopathy even at the onset of the illness. These anomalies might correspond to discharging foci with frequent diffusion, although focal seizures and localised spike discharges in no way always imply a focal cortical lesion in the child, as exemplified by the so-called benign focal epilepsy of childhood with rolandic spikes (Beaussart 1972). The possibility that these paroxysmal discharges are the expression of an auditory functional disconnection has been suggested by Gascon et al. (1973) because of analogous observations in the EEG of visually defective children where occipital spikes can be recorded. In any event, no evident correlation between these discharges and clinical seizure activity or the severity of the aphasia has been noted in most of the children. This is quite unlike what is observed in the absence-status or Lennox syndrome (Gastaut 1966) where a marked effect on consciousness and mental activity are seen together with the spike-wave discharges.

Finally, we would like to emphasize the importance of the aphasia on the childs' general behavior and the diagnostic problems which may arise. A bizzare, sudden or insidious onset of speech loss in a previously normal child, with no other evidence of organic illness, can easily be mistaken for a psychiatric reaction as in fact happened in most of our cases and in some quoted in the literature. Unexpected sudden improvement or worsening of a language disorder (Koupernik 1969) may well be interpreted as being psychological, particularly if a normal EEG is recorded.

A prolonged loss of speech comprehension and of power of verbal communication may be accompanied by severe behavior disturbances, thus creating a complex psychiatric picture. It is probable that recognition of the syndrome will contribute to a better understanding of some children presently regarded as psychotics.

In conclusion, the syndrome of acquired aphasia with paroxysmal EEG abnormalities does not appear to be a uniform syndrome due to any specific etiology but differences in the course of the disease, individual characteristics of the aphasia, and long term prognosis suggest different pathological precesses and mechanisms as the underling cause of the aphasia. The "lesions" or defective "mechanisms", however, involve the speech areas selectively and are associated with paroxysmal discharges in the EEG and frequently with clinical seizures as well.

* * * * *

Références

1. Alajouanine, T., Sabouraud, O.: Les perturbations paroxystiques du langage dans l'épilepsie. Encéphale 49 : 95 (1960).

2. Beaussart, M. : Benign epilepsy of children with rolandic (centrotemporal) paroxysmal foci. A clinical entity, study of 221 cases. Epilepsia 13: 795 (1972).

3. Brissaud, H. E. et Richardet, J. M.: Syndrome de désintégration du langage et comitialité. Journées parisiennes de Pédiatrie, 1974.

4. Brown, J. W., Hécaen, H. : Lateralization and language representation. Observations on aphasia in children, left-handers, and anomalous dextrals. Neurology 26: 183 (1976).

5. Gascon, G., Victor, D., Lombroso, C. T.: Language disorder, convulsive disorder and electroencephalographic abnormalities. Archives of Neurology 28: 156 (1973).

6. Gastaut, H., Roger, J., Soulayrol, R., Tassinari, C. A., Régis, H., Dravet, E.: Childhood epileptic encephalopathy with diffuse slow spike-waves (otherwise known as petit mal variant) or Lennox syndrome. Epilepsia, Amst. 7: 139 (1966).

7. Hécaen, H., Angelergues, R.: Epilepsie et troubles du langage. Encéphale 49: 138 (1960).

8. Hécaen, H., Piercy, M.: Paroxysmal dysphasia and the problem of cerebral dominance. Journal of Neurology, Neurosurgery and Psychiatry 19: 194 (1956).

9. Huskisson, J. A.: Acquircd receptive language difficulties in childhood. British Journal of Disorders of Communication 8: 54 (1973).

10. Jerger, J.: Auditory disorder following bilateral temporal lobe insult. Journal of Speech and Hearing disorders 37: 523 (1972).

11. Koupernik, C., Masciangelo, P. M., Balestra-Beretta, S. : Conséquences d'un attentat sexuel commis sur une fillette de 4 ans. La psychiatrie de l'enfant. Vol. XII : 267 (1969).

12. Landau, W. M., Kleffner, F. R. : Syndrome of acquired aphasia with convulsive disorder children. Neurology 7 : 523 (1957).

13. McKinney.W., McGreal, D. A. : An aphasic syndrome in children. Canadian Medical Association Journal 110 : 637 (1974).

14. Rose, F. C. : Receptive aphasia in childhood (abst.) Proceedings of the Society of British Neurological Surgeons. Journal Neurology, Neurosurgery and Psychiatry 32: 65 (1969).

15. Shoumaker, J. R., Bennett, D. R., Bray, P. F., Curless, R. G.: Clinical and EEG manifestations of an unusual aphasic syndrome in children. Neurology 1: 10 (1974).

16. Stein, L. K., Cury, F. K.: Childhood auditory agnosia. Journal of Hearing Disorders 33: 361 (1968).

17. Worster-Drought, C.: An unusual form of acquired aphasia in children. Developmental Medicine and Child Neurology 13: 563 (1971).

* * * * *