

5-1-1969

Convulsive disorders in the mentally retarded

David E. Magaret

University of Nebraska Medical Center

Follow this and additional works at: <http://digitalcommons.unmc.edu/mdtheses>

Recommended Citation

Magaret, David E., "Convulsive disorders in the mentally retarded" (1969). *MD Theses*. Paper 103.

This Thesis is brought to you for free and open access by the College of Medicine at DigitalCommons@UNMC. It has been accepted for inclusion in MD Theses by an authorized administrator of DigitalCommons@UNMC. For more information, please contact digitalcommons@unmc.edu.

Convulsive Disorders in the Mentally Retarded

by

David Ernest Magaret

The College of Medicine in the University of Nebraska

In Partial Fulfillment of Requirements

for the Degree of Doctor of Medicine

Under the Supervision of Frank Menolascino, M. D.

Associate Professor of Psychiatry and Pediatrics

Omaha, Nebraska

February 3, 1969

Table of Contents

I. Introduction	1
II. Method	1
III. Results	2
Sex and age	2
Intelligence3
Types of Seizures	4
Maternal age at confinement	4
Family history	5
EEG	5
Etiology	5
Physical examination	6
Psychiatric diagnosis6
Control of seizures	7
IV. Discussion	11
V. Summary and Conclusion	20
VI. Bibliography	22

Convulsive Disorders in the Mentally Retarded

Introduction

The purpose of this thesis is to study convulsive disorders in the mentally retarded and examine the data concerning the relationships of these two symptoms.

Clinical investigation is made into the histories, physicals and management of 126 institutionalized mentally retarded patients with documented convulsive disorders. Special attention is given to the degree of mental retardation and its correlation with the type of convulsions, family histories, maternal age at confinement, electroencephalographic (EEG) aberrations, neurological abnormalities, psychiatric complications, etiologies of the mental retardation and the effectiveness of control of seizures.

It is clear that convulsive disorders and mental retardation have a recognized relationship, but the exact nature of this relationship is still a matter of controversy. There is no evidence to support a firm conclusion of intellectual deterioration on convulsive attacks. It is held that the occurrence of convulsive disorders and mental retardation is not coincidental, but that both result from the same underlying central nervous system disorder and that the extent of malfunction can be estimated by EEG, neurological and intellectual dimensions.

Method

All clinical investigation was done at Glenwood State Hospital and School near Glenwood, Iowa. This institution is state supported and provides care and habilitation for the mentally retarded patient of all ages. The enrollment is approximately 1,000 with variation hinging

primarily upon the boarding of patients in nursing homes and authorized visits to the homes of parents and relatives.

Before enrollment the patient is given a complete evaluation. A medical and social history is taken and a physical examination including chest and skull x-rays, complete blood count, urinalysis and EEG is performed by the hospital staff. When a psychiatric problem is suspected, the patient is seen by a psychiatrist. In addition, the patient is given an appraisal by a psychologist, physical therapist and occupational therapist. This information is then compiled in a permanent record. When the patient enters Glenwood State Hospital and School, a record of his medications, activities and difficulties is kept daily and remains on permanent file.

I began the research for this thesis during the summer of 1967. Using a computer list, I obtained the names of the 126 registered patients with histories of convulsive disorders. I studied the records of these patients and investigated the following: sex, age, I.Q., type of seizure, maternal age at confinement, family history, etiology, EEG reports, physical examination, psychiatric diagnosis and the frequency of seizures. The information was found to be very complete with the exception of the family histories.

Results

Sex and Age: I found sex distribution in the 126 patients to be of little significance.

Table #1

Age (years)	Number	Percent
1-10	25	20.6%
11-20	58	46.0
21-30	23	18.2
30+	20	15.2
Total	126	100.0%

There is a slightly larger number of males than females with 65 males and 61 females. The ages ranged from 2 years to 52 years with the greatest number of patients being between 11 and 20 years of age.

Intelligence: All of the patients had undergone psychological testing and an estimate of the intelligence of each was rendered.

Table #2

I. Q.	Number	Percent
less than 25	68	54.0%
26-50	40	31.8
51-70	15	11.8
71+	3	2.4
Total	126	100.0%

An attempt was not made to give each patient an exact I. Q. rating, but only to place him in an I. Q. range. I found the majority of the patients to have I. Q.'s less than 25 with the least number being borderline mentally retarded.

Types of seizures: I found that all patients were classified according to the type of seizures. This was determined by historical description and actual accounts by staff.

Table #3

Type of seizure	Number	Percent
major motor	82	65.0%
petit mal	12	9.6
psychomotor	8	6.4
focal	5	4.0
mixed	19	15.0
Total	126	100.0%

The largest group suffered from major motor or grand mal seizures.

Maternal Age at confinement: I obtained an accurate maternal history on all the patients.

Table #4

Maternal age (years)	Number	Percent
15-20	12	9.6%
21-25	31	24.6
26-30	32	25.4
31-35	35	27.8
36+	16	12.6
Total	126	100.0%

I found 40.4% of the mothers were over 30 years of age at confinement of the patients.

Family history: Because of illegitimate births, divorces, etc., I ran into difficulty finding accurate family histories in 25 of the patients. Only near relatives, which consists of siblings, parents, grandparents, aunts and uncles were considered. I found 11 of the 101 patients with adequate histories had a positive family history of convulsive disorders of some type and that 19 of the patients had a positive family history of mental retardation to some degree.

EEG: I found that one or more EEG's had been performed on all the patients and the tracings were sent to the University of Nebraska for interpretation. The four basic abnormalities noted were: (1) activity slower than 8 per second and of moderate or high voltage while the patient is awake; (2) activity faster than 12-13 per second and greater than 15 microvolts in amplitude either during wakefulness or sleep; (3) spikes; (4) amplitude asymmetry. I found that 102 or 84.2% had one or more of the abnormalities listed above.

Etiology: I found that all patients were classified according to The American Association on Mental Retardation coding method for the etiologies of mental retardation. It is interesting that 72 or 56.6% were distributed among one of the three unknown etiology categories. The largest single group was that due to trauma or physical agents.

Table #5

Etiology	Number	Percent
I. Infection	18	14.3%
II. Intoxication	4	3.7
III. Trauma or physical agents	29	23.2
IV. Disorders of metabolism, growth or nutrition	0	--
V. New growths	3	2.3
VI. Unknown prenatal influence	28	22.1
VII. Unknown causes with structural reactions manifest	21	17.1
VIII. Unknown cause with functional reaction manifest	23	18.3
Total	126	100.0%

Physical exam: I found that all patients had received complete physical examinations with accurate recording of neurological abnormalities including paraplegia, hemiplegia, quadriplegia, spasticity, abnormal coordination, hyperreflexia, and pathological reflexes. I determined that 80 or 63.5% of the patients had abnormal neurological findings consistent with upper motor neuron malfunction.

Psychiatric diagnosis: All of the patients had had psychological evaluation and the diagnostic problems had been seen by a psychiatrist. I discovered that 14 or 11.2% of the patients had suffered from

psychotic reactions and 34 or 27.0% had difficulty with behavioral reactions.

Control of seizures: I examined the ward charts and discovered that the nurses kept monthly records of the number and type of seizures which occurred in each patient.

Table #6

Number of seizures the previous 12 months	Number	Percent
0	72	57.2%
1-5	22	17.4
6-10	11	8.7
11-20	13	10.3
21+	8	6.4
Total	126	100.0%

I found the greatest number of seizures in a single patient for the previous 12 months to be 133, but that the majority of the patients had had no convulsions in that time period.

I then attempted to examine the degree of mental retardation in more detail. I was able to find a correlation between the degree of mental retardation and the types of seizures.

Table #7

Seizure type	Less than 25	26-50	51-70	Total
Major Motor	52 (76.5%)	20 (50.0%)	8 (54.2%)	80
Petit mal	6 8.8%	4 10.0%	2 13.0%	12
Psychomotor	1 1.5%	5 12.5%	2 13.0%	8
Focal	2 2.9%	1 2.5%	2 13.0%	5
Mixed	7 10.3%	10 25.0%	1 6.8%	18
Total	68 100.0%	40 100.0%	15 100.0%	123

Major motor seizures were found to be the most common type of seizure in all three I. Q. groups, but when comparing these groups, a greater percentage of the patients with an I. Q. less than 25 had major motor seizures.

When I studied maternal age at confinement and degree of mental retardation, I could not find any significant positive correlation.

Table #8

Maternal age	Less than 25	26-50	51-70	Total
15-20	9 (13.2%)	2 (5.0%)	1 (6.8%)	12
21-25	17 25.0%	13 32.5%	1 6.8%	31
26-30	16 23.8%	8 20.0%	8 54.2%	32
31-35	18 26.5%	12 30.0%	3 20.0%	33
36+	8 11.5%	5 12.5%	2 13.0%	15
Total	68 100.0%	40 100.0%	15 100.0%	123

I also found no correlation between a positive family history of mental retardation and/or epilepsy and the degree of mental retardation.

(See Table #9)

Table #9

Pos. histories	Less than 25	26-50	51-70	Total
Pos. epilepsy	5 (7.4%)	4 (10.0%)	2 (13.0%)	11
Pos. M. R.	7 (10.3%)	7 (17.5%)	5 (33.3%)	19

I found a positive correlation between abnormal EEG tracings and the degree of mental retardation. I noted that only 6 of the 68 patients with I. Q.'s less than 25 were reported to have normal EEGs.

Table #10

	Less than 25	26-50	51-70	Total
Abnormal	62 (91.0%)	31 (78.0%)	9 (60.0%)	102
Normal	6 (9.0%)	9 (22.0%)	6 (40.0%)	21
Total	68 (100.0%)	40 (100.0%)	15 (100.0%)	123

It was my finding that abnormal neurologic findings consistent with upper motor neuron lesions were positively linked with the degree of mental retardation. I noted 57 or 84.0% of the patients with I. Q.'s less than 25 had abnormal neurological findings compared to 4 or 26.6% of the mildly retarded.

Table #11

	Less than 25	26-50	51-70	Total
Abnormal neurological findings	57 (84.0%)	19 (47.5%)	4 (26.6%)	80

I was able to show an inverse relationship between behavioral reactions and the degree of mental retardation. Behavioral reactions were most common in mildly retarded and least common in the severely

retarded.

Table #12

	Less than 25	26-50	51-70	Total
Behavioral reaction	6 (8.8%)	17 (45.0%)	11 (73.0%)	34
Psychotic reaction	6 (8.8%)	3 (7.5%)	0	9

I was surprised that I wasn't able to find any significant correlation between the control of seizures, etiology and the degree of mental retardation.

Table #13

Control of seizure	Less than 25	26-50	51-70	Total
0	35 (51.5%)	24 (60.0%)	10 (66.6%)	69
1-5	13 19.1%	6 15.0%	3 20.0%	22
6-10	7 10.3%	3 7.5%	1 6.7%	11
11-20	9 13.2%	3 7.5%	1 6.7%	13
21+	4 5.9%	4 10.0%	0	8
Total	68 100.0%	40 100.0%	15 100.0%	123

Table #14

Etiology*	Less than 25	26-50	51-70	Total
I	9 (13.2%)	7 (17.5%)	2 (13.0%)	18
II	2 3.3%	2 5.0%	0	4
III	12 17.5%	13 32.5%	4 26.8%	29
V	1 1.5%	1 2.5%	1 6.8%	3
VI	23 33.6%	4 10.0%	1 6.8%	28
VII	12 17.5%	5 12.5%	4 26.8%	21
VIII	9 13.4%	8 20.0%	3 19.8%	20
Total	68 100.0%	40 100.0%	15 100.0%	123

Discussion

In early life mental retardedees are difficult to ascertain except in extremely severe cases and up to the age of 4 years the mildly or moderately retarded children are not called upon to perform any duties of significance. During the school period however, intellectual deficiencies are brought into the foreground. Also between the ages of 11 and 14 more accurately standardized tests can be applied to determine the degree of intellectual capacity. After the end of the school period, the proportion of retardedees suddenly drops because the standards of school environment no longer apply and there are more employment opportunities for the mildly retarded.

Penrose²² and Tredgold²⁴ found the peak incidence age group of the non-epileptic institutionalized mental retardedees of all degrees to be between the ages of 11 and 14 with the second highest incidence between the ages of 5-9 and 15-20 years.

* American Association on Mental Retardation nomenclature.

Since the epileptic subject with mental retardation has double handicaps, seizure frequency can make adjustment outside of institutions impossible and therefore necessitate earlier institutionalization. I suspected the group of patients with mental retardation and epilepsy to have a larger proportion of younger patients when compared to the non-epileptic retardee. I found this not to be the case. The largest age group was the 11-20 year olds, similar to that for the non-epileptic retarded.

Investigations have been made to determine the sex frequency of the non-epileptic retarded patient. It was found that while there was no significant difference in sex, there was usually a slightly greater number of male patients. Tredgold²⁴ studied 41,439 retardees in institutions in the USA and found 26,489 were males and 20,950 were females. I similarly found no sex differences, but a slightly greater number of males. Therefore, I was not able to establish any positive correlation between sex and the mental retarded with epilepsy.

In an institution for the mentally retarded those who need care and supervision have the most urgent claims on admission. Therefore a considerable proportion of all cases of severe mental retardation in the community need to be cared for in hospitals. The severely retarded need nursing care because of their helplessness, but in addition many of the moderately and mildly retarded often need supervision on account of their propensities for maladjustment in the community. The intelligence of the non-epileptic mental retardee therefore is spread over a range from nearly untestable to borderline mental retardation. Pen-

rose²⁰ studied 5,238 residents living in the Massachusetts State Schools for Mental Retardation, and found the following I. Q. distribution:

Table #15
(after Penrose ²⁰)

I. Q.	Number	Percent
0-25	1478	28.2%
26-49	1676	32.2%
50-69	1771	33.8%
70+	313	5.8%
Total	5238	100.0%

Another study by Penrose²² of 1280 similar patients gave nearly identical results.

I found that the patients I studied had lower intelligence than the general non-epileptic mentally retarded patient. Keith¹¹ found that 56% of his patients with convulsive disorders had I. Q.s less than 25 and 10% were in the borderline range. The relationship of convulsive disorders and intellectual levels is not clearly understood. In the past it was believed that intellectual deterioration was a consequence of seizure attacks, but now there is no evidence to support this. Convulsive disorders should always be regarded as a symptom of some type of underlying brain damage and/or disorder although in practice it is often not possible to establish the exact nature of the disorder. It is axiomatic that the occurrence of epilepsy and mental retardation is not coincidental, but that both result from the same underlying central nervous system disorder.

There are probably two reasons for the increased prevalence of epilepsy among the more severely retarded patients. The first is the rarity of epilepsy among mongols, who are usually higher grade retardees. The second is that patients with I. Q.s less than 25 nearly always have severe brain damage and therefore increases their chances for the occurrence of epilepsy.

Although the means by which the fetus is influenced is obscure, there is little doubt that the age of the mother is very significant in the etiology of mental retardation. A disease dependent upon maternal age is difficult to separate from one dependent upon the number of previous pregnancies, because the two effects are so closely correlated. There is evidence that order of birth can have independent effects and that the first born, as well as those children born at the end of a long series of pregnancies, are less viable than those born in between. Lilienfeld¹⁶ believes maternal age is a possible factor in production of intracranial injury because of increased danger to the fetus in the very young and the elderly mother. Lilienfeld¹⁷ also found that the incidence of mental retardation with epilepsy was lowest in mothers of the 20-29 year old group and was greatest in those less than 20 and over 30 years. Penrose²¹ found in 121 cases of mental retardation without seizure disorders that the majority of the mothers were over 30 years old at confinement and only 1% were less than 20 years old. I discovered that 40% of the mothers of my patients were over 30 years old and 9.6% were less than 20 years at confinement. This indicates while there is a dependence of mental retardation upon

maternal age, it is not different from that in the epileptic mental retardee. In addition, the patients with severe retardation did not tend to have more elderly mothers, when compared with the mildly retarded patient.

For years the convulsive disorders have been neatly separated into several clinical types of seizures. But in spite of much research, we are still ignorant of the reasons why various forms of central nervous system disorders are more liable to result in one of the types of seizures. Collins³ believes that gray matter lesions are more epileptogenic than white matter lesions and in his series of mental retardees with convulsive disorders he found the motor cortex to be especially epileptogenic. In his series of 400 patients he found that 80% suffered from major motor seizures. He also found that the patient with petit mal seizures usually had higher I.Q's than the patient with grand mal seizures. The patient with psychomotor seizures fell someplace in the middle. This coincides well with my results. I found 65% of my series to have major motor seizures and that 76.5% of the patients with I.Q's less than 25 suffered this type of seizure activity. This evidence would tend to link the degree of mental retardation reflected in severe retardation and I. Q. less than 25 with major motor seizures. Therefore, it would seem that major motor seizures often indicate a more extensive brain disorder than does petit mal or psychomotor seizures.

Many investigators have been interested in the role heredity plays in epilepsy and mental retardation. For many years idiopathic

epilepsy has been suspected of having a familial basis. Epilepsy secondary to trauma, infection or tumors would not be hereditary. Grinker¹⁰ found that 3.2% of 20,000 near relatives of 4,231 non-retarded epileptic patients had positive history of seizure disorders of some type. Penrose²² found that of 210 retarded patients with epilepsy, 6.9% had positive family histories of epilepsy. He also found that 2.7% of the severely retarded patients and 12.9% of borderline cases had positive family histories of mental retardation. I discovered a higher incidence of positive family histories for epilepsy and mental retardation than the previous studies. In addition, I was not able to demonstrate any correlation between positive family histories and degree of retardation. Since 20% of the patients studied in my series had inadequate histories, this data is probably not significant.

The EEG is a record of electrical activity of the brain. Only gross changes in electrical activity can be recorded in this way, but in principle the data obtained does not differ from that recordable from electrodes placed directly on the brain. Since most mental retarded have some type of central nervous system damage or disorder, the general effects of brain damage in the EEG explains most of the changes seen. There is no EEG characteristic of mental retardation and there are no correlations between any EEG changes and the level of mental retardation. In most cases of mental retardation, the EEG abnormality is not specific and only indicates that some cerebral dysfunction is present without offering a clue to its nature. Ellingson⁵ found the EEG's of 90% of non-retarded epileptics examined by standard methods were

abnormal. Walter²⁶ found after examining 200 retardees without epilepsy that the undifferentiated group of retarded patients generally have high incidence of abnormal EEG tracings. One exception to this rule is the patient with Down's Syndrome who often has near normal tracings.

I noted that 84.2% of the patients in my series had abnormal EEG tracings and there was a positive correlation with the degree of mental retardation. This suggests that the severe brain disorders resulting in profound retardation also have an increased chance of producing abnormal EEG recordings.

When taken together as a group the mentally retarded patients are physically inferior to the rest of the general population. In a study by Penrose²⁰ of 400 mentally retarded patients, he showed that neurologic abnormalities were to be found in many of the borderline cases and most all of the low grade cases. In a similar study by Keith¹³ 263 spastic children were examined and a positive correlation between severity of the neurologic abnormalities and the degree of mental retardation was found. He discovered that quadriplegics had a mean I. Q. of 40 and hemiplegics a mean I. Q. of 74. He concluded that patients with abnormal neurological findings had organic lesions and showed a higher incidence of mental retardation. I also found a high incidence of abnormal neurological findings and that the patient with severe retardation had the highest incidence of these neurologic findings. This suggests that the central nervous system disorder producing abnormal neurological function is related to the level of

intelligence and the degree of brain impairment can be determined often times by a neurologic exam as well as intelligence testing.

Mental retardation is a symptom associated with a large number of disease entities which affect the organism in its earliest stages of growth and development. Noyes¹⁹ states that 50-65 percent of mental retardation arises from poorly understood causes existing prior to birth. It is generally agreed that hereditary transmission of mental defects plays a much less important role than previously believed. Waggoner²⁵ studied 254 mental retarded and found a higher incidence of epilepsy among secondary acquired etiologies including: infections, trauma, tumors and intoxicants, than in the primary or endogenous group of etiologies. He found that patients with acquired lesions showed a greater degree of mental retardation and a higher incidence of epilepsy. Collins³ found in his study of epileptic intelligence there was a superiority of the idiopathic group over the brain damaged group and that therefore brain damaged epilepsy was most deleterious. In my study, I was not able to demonstrate any increased incidence of epilepsy among the secondarily acquired etiologies. On the contrary, the majority of the patients had primary endogenous etiologies and there was no correlation between the etiology and the level of retardation. It appears that primary and secondary etiologies had similar distributions of intelligence.

The relationship of psychiatric disturbances and forms of mental

retardation, which rapidly leads to social incompetence is not simple. Cases where theft, violence or larceny are involved, children whose intellects are below normal are more likely to be found out than children with greater ability. Penrose²² found the number of neurotics among higher grade cases to be increased and in these patients, neurosis was a principle reason for admission. I discovered that the majority of the mildly retarded patients were having behavioral reactions. This was present in only a small minority of the severely retarded patients.

The aim in the treatment of seizures is to give medication which will counteract the explosive tendency of the involved neurons. Anticonvulsant medication has no deleterious effects on intellectual activity and controlling seizures in mentally retarded rarely results in improvement in intellectual development. Nevertheless, attempts of adequate control in retarded patients must be done, since seizures can cause physical injuries and complicate management. Keith¹¹ found after studying 296 mentally retarded epileptic patients that there was an association between severity of mental retardation and the control of seizures with adequate therapy. Penrose²² states that the majority of the severely retarded with seizures had attacks at the frequency of more than 10 per month and only 9% had seizures less than one per month. In my series I discovered that the majority of the patients were well controlled having no seizures in the previous 12 months. In addition, there didn't seem to be any correlation between the degree of retardation and the control of seizure. There is often a problem deciding what constitutes adequate

therapy and therefore, the treatment should always be performed by a physician familiar with convulsive disorders and anticonvulsant medications. The medical care of the institutionalized mentally retarded has greatly improved in recent years and this could likewise bring better control of seizures in these patients with convulsive disorders.

Summary and Conclusion

- (1) There is no significant variation in age or sex between the epileptic and non-epileptic institutionalized mentally retarded patient.
- (2) There is an increased prevalence of severe mental retardation among the retardee with convulsive disorders.
- (3) Major motor seizures are most common among mental retardees with epilepsy and there is a positive correlation between major motor seizures and the degree of retardation. The major motor seizure as with low intelligence is often an indication of the degree of central nervous system damage.
- (4) There is no significant difference in maternal age at confinement between the retardees with convulsive disorders and those without.
- (5) There is a large number of patients with retardation and epilepsy with near relatives suffering from epilepsy and/or mental retardation.
- (6) The mentally retarded with convulsive disorders have a high incidence of abnormal EEG and the incidence is highest among the severely retarded.
- (7) Neurological abnormalities are common in the mentally retarded

with seizures and is most common in the severely retarded patient.

(8) In the majority of the patients poorly understood causes lead to their mental retardation and there was no association of specific etiologies and the degree of mental retardation. Brain damage was not found to be more deleterious.

(9) Behavioral reactions are most prevalent in the mildly retarded.

(10) The majority of retarded patients with seizures can be well controlled and there is no association between severity of retardation and control of seizures.

(11) The occurrence of mental retardation and convulsive disorders results from the same underlying central nervous system disorder the severity of which is reflected in major motor seizures, low I. Q., abnormal EEG tracings and neurological abnormalities.

Bibliography

1. Alford, A. F.: Mental Health Despite Mental Retardation, Lancet 1:1233-1235 (June 18, 1955).
2. Churchill, J. A.: Relationship of Epilepsy to Breech Delivery, Electroencephalography, 11:1-4 (February, 1959).
3. Collins, J. C.: Epileptic Intelligence, J. Consult. Psych. 15:392-399 (October, 1951).
4. Crome, L.: The Brain and Mental Retardation, Brit. Med. J. 1:897 (March 26, 1960).
5. Ellingson, R. J.: Clinical Electroencephalography in Convulsive Disorders, Nebr. State Med. J. 36-37:396-412 (December, 1951).
6. Ellingson, R. J.: Clinical EEG in Non-Convulsive Disorders of the Brain, Nebr. State Med. J. 37:180-86 (June, 1954).
7. Ellingson, R. J.: On the Reliability of Clinical EEG Interpretation, J. of Nervous and Mental Disease 128:425-437 (May, 1959).
8. Gibbs, F. A., Gibbs, E. L.: Atlas of Electroencephalography, 2nd Edition Addison-Wesley Press (1951).
9. Gordon, R. G., Norman, R. M., Berry, R. J.: Neurological Abnormalities--Their Occurrence and Significance as Illustrated by Examination of 500 Mental Defects, J. of Neurol. and Psychopath. 14:97-107 (October, 1933).
10. Grinker, R. R., Adolph, L. S.: Neurology- 6th Edition, Lippincott, (1965).
11. Keith, D. C., Ewert, B. J.: Mental Status of Children with Convulsive Disorders, Neurology 6:419-425 (1955).
12. Keith, H. M., Gage, R. P.: Neurologic Lesions in Relation to Asphyxia of the Newborn and Factors of Pregnancy, Pediatrics 26:616 (1958).
13. Keith, H. M., Norval, M. A.: Neurological Lesions in Relation to the Sequelae of CNS Injury, Neurology 3:139, (1953).
14. Lennox, W. G. Epilepsy and Related Disorders 1st Edition, McGraw-Hill (1960).
15. Lennox, W. G., Gibbs, E. L., Gibbs, F. A.: Inheritance of Cerebral Dysrhythmia and Epilepsy, Arch. of Neurol. and Psychiat. 44:1155 (November, 1940).

16. Lilienfeld, A. M., Pasamanick, B.: The Association of Maternal and Fetal Factors with Development of Cerebral Palsy and Epilepsy, Am. J. Obst. and Gyn. 70:93 (July, 1955).
17. Lilienfeld, A. M., Pasamanick, B.: The Association of Maternal and Fetal Factors with Development of Mental Retardation, Am. J. Ment. Def. 60:557 (January, 1956).
18. MacGillivray, R. C.: Epilepsy in Down's Anomaly, J. Ment. Def. Research 11:43-48 (May, 1967).
19. Noyes, A. P., Kolb, L. C. Modern Clinical Psychiatry 6th Edition W. A. Saunders Co. (1966).
20. Penrose, L. S.: Mental Defect, Farrar and Rinehart, New York (1934).
21. Penrose, L. S.: Maternal Age, Order of Birth and Developmental Abnormalities, J. Ment. Sci. 85:73 (1939).
22. Penrose, L. S.: The Biology of Mental Defect 3rd Edition, Grune and Stratton, New York (1963).
23. Phillips, G.: Traumatic Epilepsy After Closed Head Injury, J. Neurol., Neurosurg. and Psychiat., 17:1 (February, 1954).
24. Tredgold, R. F.: Textbook of Mental Deficiency, Williams and Wilkins, Baltimore (1963).
25. Waggoner, E. J., Sheps, F. D.: The Frequency of Convulsive Disorders in the Mentally Retarded - Clinical and Patho-Anatomical Considerations, Am. J. Psychiat. 100:497. (1944).
26. Walter, R. D., Yeager, F. L., Rubin, H. K. Mongolism and Convulsive Seizures, Arch. Neurol. and Psychiat. 74:559-563 (May, 1955.)