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Electroencephalographic Findings in Deaf Psychotic Patients

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ELECTROENCEPHALOGRAPHIC FINDINGS IN DEAF PSYCHOTIC PATIENTS

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INTRODUCTION

The literature on the relationships between clinical EEG abnormalities and deafness is very sparse. Studies of the incidence of clinical EEG abnormalities in the psychotic deaf are nonexistent. A group of deaf subjects selected for suspected psychosis was used to study the incidence of EEG clinical abnormalities and to assess the characteristics of this population for which, so far, there is no clinical definition.

Profound hearing loss may occur as a result of the most varied etiologies involving either central or peripheral lesions and with characteristic differences in the degrees to which other brain structures are impaired. The problems incurred in dealing with this diversity of etiologies were well brought out by Azoy *et al.* (1952) and will not be discussed here.

As Vernon (1968) pointed out, four of the five leading causes of deafness are also major etiological factors in brain damage, a fact which emphasizes the need for improving the methodology for differentiating types of brain damage contributory and non-contributory to deafness. When the dimension of psychosis with its organic or functional possibilities is added, the difficulty in determining the relatedness of given items in the symptomatology increases.

Given the diversity of etiologies it becomes apparent that 1) the degree and location of damage to cerebral structures even within a given

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process show great variance (Villa-Badó & Samsó-Dies, 1953), and 2) since often the brain damage is subcortical, only a small portion is detectable by scalp recordings. In addition, localization of auditory functions in the brain is non-specific, and Derbyshire *et al.* (1956), analyzing the EEG response pathway involved in audition, re-emphasized the nonspecific nature of the cortical responses.

The only EEG clinical study of the deaf in English is by Gibbs and Gibbs (1964, pp. 9, 231). In their run of 396 patients with deaf symptomatology they concluded that there were no clear relationships between the EEG and hearing disorders. They also found that while deafness and athetosis were clinically associated the EEG could not discriminate between them.

The broad area of attempts to utilize the EEG methodology in the study of deafness includes the estimation of hearing threshold by EEG. Perl *et al.* (1953) concluded that the EEG changes described following auditory stimulation were nonspecific in that they could be induced by stimuli other than sounds. Another approach is the experimental analysis of EEG spectra (Giannitrapani, 1970) which is not presently usable for clinical diagnosis. For clinical purposes therefore, the method for determining EEG abnormalities with particular reference to deafness has not improved beyond traditional clinical interpretation.

In summary, the variety of etiology of deafness, the super-imposed psychotic dimension, the lack of EEG abnormalities in the deaf without psychiatric symptomatology, and the availability of only traditional clinical methods for interpreting the EEG constitute the field within which this study was conceived.

METHOD

Twenty-six subjects were selected, primarily from Chicago and the adjoining areas including the state hospitals, to participate in a demonstration research project on the psychotic deaf (Grinker *et al.*, 1969). Criteria for inclusion were on the basis of previous audiologic as well as psychiatric examinations. All of these patients were deaf in the sense that they could not understand speech with or without a hearing aid, and they underwent an extensive psychiatric and psychological evaluation.

The subjects were also administered a routine clinical electroencephalogram with 16 monopolar electrodes. The temporal line of electrodes was identical to that of the 10/20 system (Int. Fed., 1958). For the three coronal planes, however, there were only two additional electrodes rather than three as per the 10/20 system to obtain a total of 16 virtually equidistant electrodes throughout the scalp.

Some problems were encountered when attempting to communicate to the patient the essence of the routine and what was expected of him. Communication was established via an interpreter, but anxiety was often high and some of the patients did not understand or found it difficult to keep their eyes closed during the recording session.

Due to the small size of the sample, expecially when the group was divided into subcategories, statistical tests were not deemed suitable for establishing significance.

RESULTS

Table 1 shows the incidence of psychiatric categories as compared to the etiology of auditory impairment among these patients. The tabulation shows that in this sample the largest occurrence was of schizophrenics with unknown auditory impairment etiology.

TABLE 1

Distribution of Psychiatric Diagnosis and Etiology of Auditory Impairment

	Unknown N=10	Rubella N=4	Hereditary N=5	Scarlet Fever N=3	Meningitis N=2	Comp. Rh Fac. N=1	Prematurity N=1	Totals
Schizophrenics N=16	6	3	3	3	1			16
Other Psychotics N=6	2		2		1		1	6
Neurotics N=4	2	1				1		4
Totals	10	4	5	3	2	1	1	26

Table 2 shows the distribution of EEG characteristics according to etiology of auditory impairment. A patient may appear more than once in Table 2, depending on the number of his scoreable EEG characteristics. Of the 26 subjects tested, 19 had abnormal EEGs as determined by a routine clinical interpretation of the tracings.

Table 2 shows that the most common EEG abnormality found in these records was excessive slow activity, proportionately distributed throughout the deafness etiologies listed, except for scarlet fever. Patients with the latter etiology showed no EEG abnormality. The asymmetries noted occurred primarily in the temporal areas, while sharp wave activity was prevalent in the parietal areas. The temporal-areas depressions were

either left, right or bilateral, while the sharp waves of the parietal areas were only on the right side or bilateral. Very little true spike activity was noted and seizure patterns were absent. Positive spike activity occurred for only two cases, both having a rubella etiology, and seizure activity was not present in the sample.

TABLE 2

EEG Characteristics According to Etiology of Auditory Impairment*

	EEG Characteristics						
	NORMAL	ABNORMAL					
Auditory Impairment Etiology		Excessive Fast	Excessive Slow	Mixed Fast & Slow	Asymmetries	Sharp Waves	Spikes
Unknown N = 10	2		5		3	2	
Rubella N = 4	1	1	2	1	1	2	2
Hereditary N = 5	1	1	2	1	1	2	
Scarlet Fever N = 3	3						
Other N = 4			1	1	2	1	
Totals 26	7	2	10	3	7	7	2

*A patient may appear more than once in the Table depending on the number of EEG abnormalities

TABLE 3

EEG Characteristics According to Psychiatric Diagnostic Categories*

	Excessive EEG characteristics						
	NORMAL	ABNORMAL					
Psychiatric Diagnostic Categories		Excessive Fast	Excessive Slow	Mixed Fast & Slow	Asymmetries	Sharp Waves	Spikes
Schizophrenics N = 16	6		4	2	2	4	2
Other Psychotics N = 6		1	4	1	2	2	
Neurotics N = 4	1	1	2		3	1	
Totals 26	7	2	10	3	7	7	2

*A patient may appear more than once in the Table depending on the number of EEG abnormalities

Table 3 shows the same data reorganized according to psychiatric diagnostic categories. The six normal EEGs which were observed among the patients with a diagnosis of psychosis occurred in every instance in patients who were diagnosed as schizophrenics. The excessive slow activity

is primarily of a "diffused" nature and is proportionately distributed throughout the psychiatric categories with perhaps a stronger incidence in the "Other Psychotics" group. The two patients with positive spike discharges (parietal) are schizophrenics.

An analysis was also attempted to establish the relationship between the degree of EEG abnormality and signs of organicity in the psychological test battery. The agreement between these two measures was not significant.

DISCUSSION

It is difficult, with the small sample of patients in the subcategories of this study, to apply statistical tests to the data. However, the paucity of EEG studies of deaf patients, the total absence of EEG studies of the psychotic deaf and the difficulty in acquiring a sample with these characteristics emphasizes the significance of these findings. It is to be noted that Gibbs and Gibbs (1952) had found incidence of deafness in less than 1/1000 of their patients. When studying only the psychotic deaf, the available population becomes even smaller, and the absence of studies in this area can be appreciated.

The greater percentage of the present sample was diagnosed as schizophrenic, a diagnosis which might have been overdetermined by the isolating nature of the hearing handicap. An individual, therefore, may show what can be referred to as withdrawal symptoms either because of a schizophrenic process or because the hearing handicap prevents him from interacting in a normally accepted fashion.

Of the total sample of the study, four patients, or over 15%, showed no psychotic process at closer scrutiny. These were patients who had previously received, perhaps in a state hospital setting, a diagnosis which assumed a psychotic process. These diagnostic errors resulted probably from a misinterpretation of the autistic features of the adjustment to problems of communication to which the deaf will resort.

There is a great diversity in the EEG abnormalities noted and in the variety of the loci in which they occurred. This diversity is in accord with the etiological diversity of hearing defects. The abnormalities noted are likely to be related to the process involved in the etiology of the deafness but may be only indirectly related to the deafness per se.

The question arises as to whether the deaf psychotic population presents organic problems different from those presented by a psychiatric population without deaf symptomatology. Hanretta (1965), in reviewing over 100 EEG studies of patients with psychiatric diagnoses, concluded that abnormal EEGs occurred on the average in 30% of schizophrenics, 20% of manic-depressives and even less in neurotics.

The EEG abnormalities of the present sample are 62% for the schizophrenics, 100% for the "other psychotics" category and 75% for the neurotics. By mixing categories the EEG abnormalities in the present sample occur in 73% of the patients. This study then demonstrates that a greater degree of EEG abnormalities occurred among deaf with presumed psychosis than was observed among psychiatric patients without deaf symptomatology.

These abnormalities were characterized by a dominance of slow activity, a relatively high degree of sharp wave and spike activity in the parietal regions and the absence of seizure activity. The lack of association between grand mal epilepsy and deafness as well as the low incidence of focal activity had already been noted (Gibbs & Gibbs, 1952, pp. 114, 216).

The fact that of the seven normal EEGs six belonged to patients with the psychiatric diagnosis of schizophrenia might indicate one of two things: 1) the nature of the brain damage responsible for the hearing loss in the schizophrenic deaf might have been different from the damage in the other patients or 2) the schizophrenic group might have included patients incorrectly diagnosed as deaf but who did not respond to verbal stimulation because of the schizophrenic process.

There is no evidence for greater incidence of EEG abnormalities among the deaf without psychiatric symptomatology than among individuals with normal hearing. The only two extant studies are by Gibbs & Gibbs (1964, pp. 9, 231), and by Zislina & Novikova (1960), both negative.

The present sample of deaf psychotics, with an overall 73% of EEG abnormalities, is definitely different from a deaf sample without psychiatric symptomatology. As demonstrated earlier, it also shows a greater percentage of organic involvement than a psychiatric sample without deaf symptomatology. One would be tempted to draw generalities from the type of EEG features noted in the patients of this study. It is important to emphasize the diversity of the abnormalities and warn against the temptation of accepting cause-effect relationships from evidence presented from any one patient.

One important question relates to the consequence of misdiagnosis or incomplete diagnosis for the rehabilitation of these patients. Rehabilitation procedures are dependent upon correct differential diagnoses. In cases where deafness is found in conjunction with presumed psychosis, the rehabilitation counselor will be aware of the possibility that the normal presenting mechanism of the deaf person might be interpreted as a psychotic process where such process might not exist.

Given a presumed double diagnosis, the psychotic problem might be regarded to be the most urgent, and the tendency might be to shunt the patient to a psychiatric ward where his behavior would be misinterpreted even further because of general unfamiliarity with deaf problems. The

caution to be exercised is to explore all avenues for a better differential.

In the case where psychosis is definitely ascertained, the possibility for a coordinated interdisciplinary approach would be to treat both handicaps simultaneously. In these instances the goal would be to determine the assets and support the strengths that remain available for full utilization. For the deafness, treatment would rely on techniques used with patients with similar residual hearing, while for the psychiatric disorder treatment techniques will have to be developed in concert with the rehabilitation counselor.

Close interaction between the two disciplines might well result in restoring the patient to maximum level of functioning and prevent him from being led into a hopelessly misdiagnosed life with an unproductive future.

The relevance of this study is that it demonstrates the high percentage of organic involvement in patients with deaf psychotic symptomatomatology, whether actual or presumed, and the need for comprehensive evaluations for the correct determination of the assets and the liabilities of each individual patient. In many cases the findings of the EEG examination will only confirm the existence of known or suspected brain damage, but occasionally the EEG abnormalities will point to the damage in a manner that will stimulate a full neurological workup and clearer delineation of the intact functions of the patients.

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