Henry Ford Hospital Medical Journal

Volume 4 | Number 3 Article 8

9-1956

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THE WORTH OF SERIAL ELECTROENCEPHALOGRAMS

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Often it is hard to decide with certainty whether a patient with signs of an intracranial lesion has a neoplasm or a disorder of cerebral circulation. Observation of the course of events — the progress or subsidence of symptoms and signs — must be relied upon in practice to clarify this issue. The problem could be resolved sooner in most instances by angiography or pneumoencephalography. Unfortunately, there is some risk of doing harm to the patient attending the performance of these procedures; so they should be used judiciously upon those patients in whom tumor is strongly suspected and when a delay favors the disease. Electroencephalography‡ is a harmless procedure which provides a mode of information concerning cerebral function that is different than those utilized in physical examination or x-ray study, and the test may be performed as often as needed. Although an EEG provides useful information about patients who have a tumor or a CVA, a single one does not supply definitive points enabling one to distinguish tumor from CVA.

Tumors are electrically inert[1], of themselves unable to influence the brain's electrical patterns. But when they interfere with the function of neurones around them, distortions in the electrical activity of the affected neurones[2,3,4,5], cause disturbed electrical fields which are detectable at the scalp surface by EEG. Thus indirectly is the presence and approximate location of a tumor discovered. Similarly, a part of the brain infarcted by occlusion of its blood supply is electrically inert, since the neurones in the affected part are either destoryed or made incapable of electrical activity. But around the infarcted region there may be a zone of injured neurones from which emanate distorted electrical waves[6,7,8,9]. Whether injured by tumor or by interruption of blood supply the neurones react electrically much the same, yielding in either case few or uncertain clues to the identity of the lesion. Time then is the crucial factor which is required to solve this problem. Tumors enlarge and should cause increasingly intense and wide-spread EEG disturbances (Fig. 1), while the disturbances caused by cerebral vascular lesions should subside when repair occurs and collateral blood supply is established (Fig. 2). It is logical to assume that the data obtained from a series of EEGs taken at proper time intervals would help solve the tumor or CVA question. This survey was undertaken to learn how reliably tumors could be distinguished from CVA with EEG data collected on two or more occasions.

METHODS AND MATERIALS

The records of all patients upon whom more than one EEG had been performed in the EEG laboratory of the Henry Ford Hospital since January 1953 were reviewed. The records of those patients proven to have brain tumors by pathologic means

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‡The terms electroencephalogram and electroencephalography are both designated by EEG; and the different forms of disturbances in cerebral function due to localized or generalized insufficiency of cerebral circulation are designated CVA.

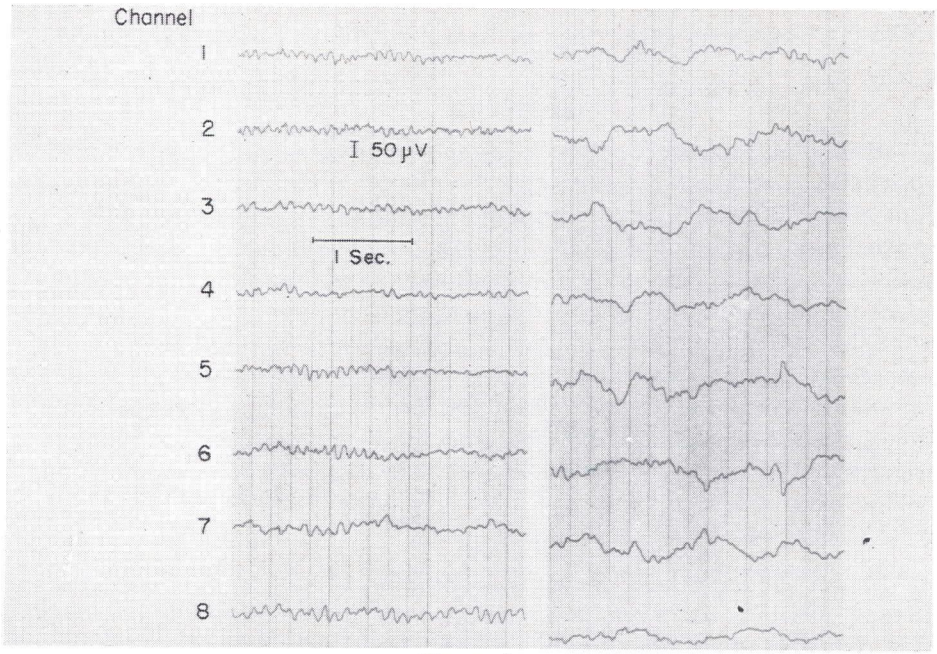

Figure 1

EEG of patient with metastatic tumors. Note the marked increase in slow waves in progress EEG one month after the first. Electrode arrangement is: channel 1 — left frontal to left central, 2 — left central to left temporal, 3 — left temporal to left parietal, 4 — left parietal to left occipital, 5, 6, 7, 8 are right-sided couples in same order as left.

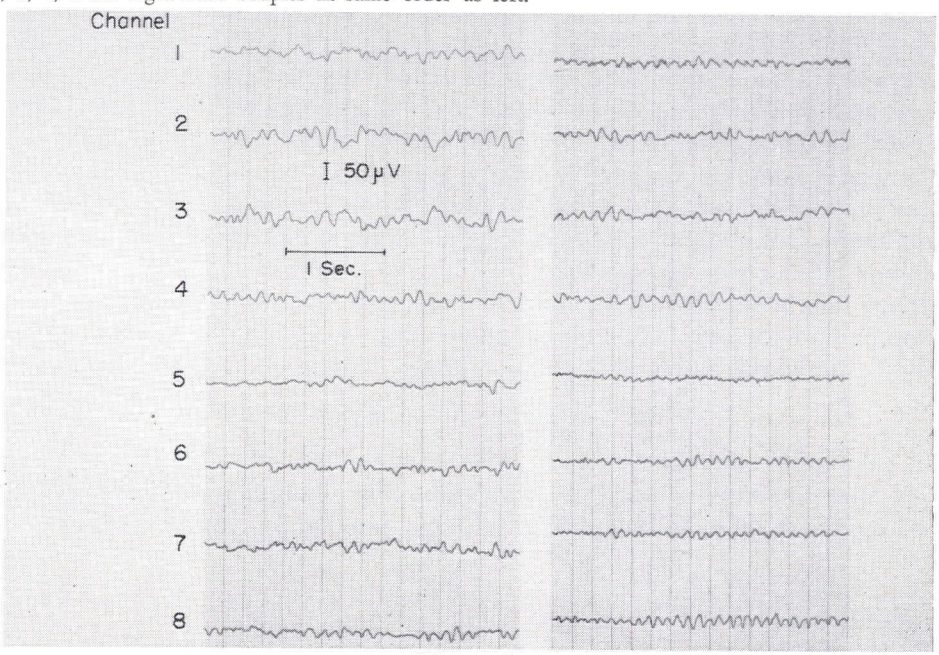

Figure 2

EEG of patient one week after abrupt right hemiplegia. Note loss of rhythmic activity especially on left side which reappears in EEG taken six months later. Note slow waves in left temporal lead sudsiding in later EEG. Electrode arrangement same as in Fig. 1.

(Table I) and records of patients who had CVA proven by pathologic means or confirmed by contrast-media x-ray studies (Table II) were selected for study. Also reviewed were records of patients with CVA that were diagnostically well documented by neurologic evaluation and adequate follow-up (Table III). Case records were not admitted to this study if the nature of the intracranial lesion remained in doubt. Initial EEG tracings were rated normal, borderline, and abnormal; the criteria of normalcy being those generally accepted among electroencephalographers[10,11,12]. EEG patterns are quite variable even in normal persons; so that judgments of EEG records are to some extent matters of opinion.

In this laboratory progress EEGs were considered to be worse than the original or conversely improved by these criteria: If the number per minute of slow waves longer than 2 seconds in duration had increased 10% or more, if the duration of the waves had increased by one-half cycle or more, if the amplitude of the slow waves

Tab. 1 Brain Tumors Verified by Pathology

Case	Tumor Type	First EEG	Progress EEG — Time in Months							
			.5	1	2	4	6	8	12	over 12
1	Meningioma	±		W						
2	Astrocytoma	±		U		B				
3	Astrocytoma	+								W
4	Meiningioma	O	W							
5	Glioblastoma	+	W							
6	Carcinoma	O			W					
7	Carcinoma	+	U							
8	Carcinoma	+	U							
9	Glioblastoma	+		W			W			
10	Carcinoma	±		W	W	W				
11	Astrocytoma	±	W							
12	Astrocytoma	±		W						
13	Glioblastoma	+					U U			
14	Glioblastoma	O					W W			
15	Meningioma	+		W						
16	Glioblastoma	+				W				
17	Glioblastoma	+		W						
18	Carcinoma	+	W							
19	Oligodendroglioma	O							W	
20	Astrocytoma	+								W
21	Meningioma	±								U U W

+ = Positive
± = Borderline
O = Negative

B = Improved
U = Unchanged
W = Worse

Tab. 2
CVA Verified by Contrast X-ray Studies or Pathology

Case	First EEG	Progress EEG — Time in Months							
		.5	1	2	4	6	8	12	Over 12
1	+		U	U		B		B	
2	+		B			B			
3	+				B				
4	+		U						
5	+		B						
6	+	W W		B					B
7	O			W				W	
8	+	B							
9	+			B					
10	+		U						
11	+		U				B		
12	+						B		
13	+	B B							
14	O	W							
15	+	U							
16	+	W							
17	+		NEW	CVA	W				
18	+							W	
19	+		B						
20	+		U	U	B B				
21	+			B					
22	+		B						

had increased 20% or more, or if the source of slow waves at the surface of the scalp had increased. Other points taken into consideration were an increase in the content of 5-7 per second wave activity and an increase of depression and irregularity of the alpha and rapid frequency activities.

RESULTS

The data under consideration have been tabulated in Tables I, II, III. The original EEGs in the group of twenty-one patients with brain tumor were abnormal in eleven patients, borderline in six and negative in four. Eight of the eleven patients with abnormal EEGs developed greater abnormalities in their progress EEGs. The progress EEGs of the other three patients remained unchanged; however, the time between the first and final EEG was only two weeks in two of these patients; hardly enough time to expect a change. Progress EEGs of the patients whose original EEGs were negative or borderline became abnormal except in one borderline case which showed improvement. The tracings in this case being borderline and of dubious

Tab. 3

Case	First EEG	CVA Diagnosed by Clinical Means — Progress EEG — Time in Months							
		.5	1	2	4	6	8	12	Over 12
1	+		U	B		B			
2	+		B			B			
3	+				B				
4	+	W W	B B						
5	+	U			B				
6	+						U		
7	+		B						
8	+					B			
9	+	W							
10	+	B		B				B	
11	+	U							
12	±		B						
13	+	B		B					
14	+					B			
15	+				B				
16	+		B	B					
17	+	B							
18	+							W B	
19	+			U					
20	O		U						
21	+		B						
22	O				U				
23	±			B					
24	+	U U							
25	+		B					B	
26	+			U					
27	+		B						
28	+		W	B		B			
29	+			B					
30	+				U				
31	+	B							
32	+		B						
33	+	W	B						
34	+		U		U				
35	+	B							
36	+		U						
37	+		U			W		B	
38	+			B					
39	+			B					
40	+				B			B	

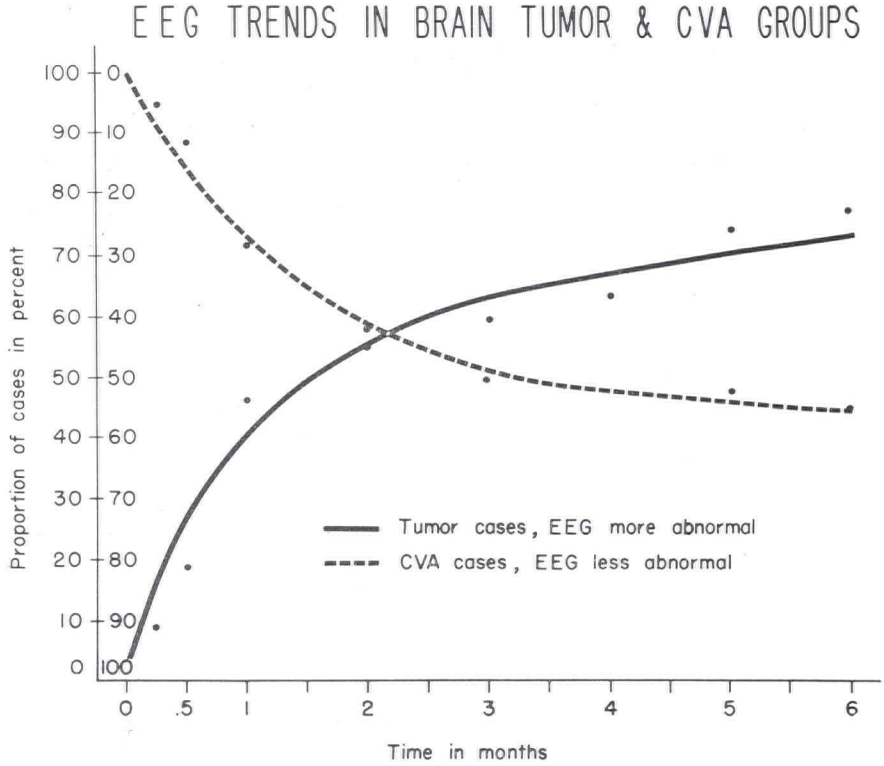

Figure 3

Curves showing when progress EEG's in the tumor group were found worsened and in the CVA group improved. Cases in per cent of total group becoming more or less abnormal for tumor and CVA groups respectively plotted on ordinate. Time after initial EEG that progress EEG was found changed on abscissa. In both tumor and CVA groups more cases changed within the first two months than in equal periods later.

localizing value were difficult to judge; the improvement noted in the final one taken at the fourth month was meagre. By the end of the second month 52% had already established a trend of increasing abnormality in their progress EEGs (Fig. 3). Although an additional 24% of the group worsened, their progress EEGs were not obtained within this period. No EEG was obtained in 9.5% after the one taken in two weeks. Another 9.5% had unchanged progress EEG after six and eleven months respectively. One to three years elapsed in three cases before the EEG was obtained and found to be more abnormal than the original.

All first EEGs of the CVA cases were abnormal except four which were negative and two which were borderline. Of the 62 cases with CVA it was found that 44 (71%) showed improvement, 50% having shown this improvement in their progress EEGs within the first two months. Twenty-one percent remained unchanged but in only four had the progress EEG been obtained after the second month. The EEG became worse in twelve cases (19%), but in six of these after a period of deterioration the EEG improved, thus leaving 10% with final EEGs remaining worse than the first. The increase of abnormality occurred within the first month in seven cases.

DISCUSSION

Most of the patients with brain tumor composing the group under consideration were those in whom the diagnosis was made only after difficult and prolonged study. The original diagnosis of tumor had been tendered in only four of these patients. This group was diagnostically difficult because those patients in whom the diagnosis of tumor was made easily were treated promptly, there being no need and no opportunity for repeated EEG testing. In this group of patients the progress EEGs with one exception became more abnormal. Progress EEGs in the majority (58%) of cases revealed increasingly abnormal trend prior to the end of the second month following the day on which the first EEG had been done. It can be assumed quite safely that had progress EEGs been obtained earlier in all cases, the proportion of the group revealing early EEG trends would have been considerably greater.

One case of improvement of the progress EEG required explanation. It was thought a small hemorrhage had occurred within this tumor just before the first EEG was done, progress EEGs showing apparent improvement while the hemorrhage was being absorbed. Improvement in progress EEGs also might be anticipated in patients with brain tumors when the first EEG had been obtained soon after the occurrence of a convulsive seizure. In addition, transient EEG disturbances caused by certain drugs, metabolic changes, and head trauma, if present when the first EEG was obtained but absent later, could be misinterpreted as an EEG improvement in a patient harboring a brain tumor. However, from the evidence at hand it would appear that such complicating conditions occur infrequently.

Most cases (71%) in the CVA group revealed a trend of decreasing abnormality in their progress EEGs (Fig. 3), but this direction of change was less definite than was the opposite trend in the tumor group. The number improved undoubtedly would have been greater if more progress EEGs had been obtained at least until a time was reached when new CVAs surpassed lesions improving. The proportion of the group (19%) that worsened was fairly large; however, all but 10% finally improved. It was noted that increased abnormality frequently occurred within the first few weeks, later followed by improvement.

The cases that did worsen posed a special problem for they gave the EEG appearances of tumor. Explanations for this transient increased abnormal state were: extension of a thrombus in a vessel, venous congestion, cerebral edema, or circulatory incompetence in a patient first tested in the early promotory period preceding a complete ictus. The few instances when after several months EEGs were more abnormal than the original were best explained by the occurrence of fresh occlusions of other diseased vessels. It is suspected that patients in whom the EEG remained unchanged had lesions which had healed as much as they could before the first test was done.

The patient whose progress EEG is more abnormal than the first should be suspected of having a brain tumor, particularly if the abnormality continues to increase after the first few weeks. On the other hand if the progress EEGs improve, the probability is great that the patient does not have a tumor; a CVA being by far the most likely possibility. The value of progress EEG is enhanced when information derived from them is integrated with clinical data, EEG changes often being explained

clearly by clinical facts and findings. When there is the problem of the patient having a tumor or a CVA it is recommended that a progress EEG be obtained in one month and again in two months. When the situation remains unclarified after two months, progress EEGs should be taken at two to four month intervals.

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