COMPLEX PARTIAL EPILEPSY: A THERAPEUTIC MODEL OF BEHAVIORAL MANAGEMENT AND EEG BIOFEEDBACK

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INTRODUCTION

Individuals with complex partial epilepsy" are managed most often by medical neurologists whose primary focus is the treatment of seizures with anticonvulsant medication. Paradoxically, often these medications produce interference with thinking and memory, and drowsiness. Yet complex-partial seizures are often poorly controlled by these medications. Glaser et al. (1) described a high incidence of interictal psychological abnormalities in patients with complex-partial seizures, characterized by fluidity of thought process, loss of trains of association, word finding difficulties, and fluctuations in the accuracy of perceptions. It is no wonder that these individuals experience difficulties in psychosocial functioning.

The intent of this clinical pilot project was to develop a behavioral therapy approach to the seizural and non-seizural problems of a group of individuals with complex-partial epilepsy. A counseling and EEG-biofeedback model was developed to reach skills that could be used to reduce seizure frequency and improve neuropsycho-logical and emotional functioning. Careful attention was paid to not only the frequency of seizures but also the degree to which interictal phenomena such as memory disturbance, dissociations and difficulty in organizing thoughts interfered with the individual's daily life.

Previous attempts to influence the incidence of seizures with EEG biofeedback techniques are well summarized in reviews by Kuhlman & Kaplan (2). Yates (3), and Olten & Noonberg (4). EFG biofeedback is based on the theory that the feedback can modify any individual's EEG and thereby change the clinical condition. Sterman et al. (5). Finley et al. (6), Lubar & Bahler (7), Kuhlman (8), Wyler et al. (9), Quy (10) and Sterman & MacDonald (I 1) all report reduction in seizure frequency with EEG biofeedback training. Sterman et al. (5) and Lubar (7) reported increase in seizures with training breaks. These studies focused on controlling seizures inside the laboratory. The present research extends these feelings by following the patients' progress for 12 months after the completion of the therapy program.

SUBJECTS

Five complex-partial epilepsy patients were referred from a private practice in neurology (Joel M. Reiter, M.D.). They were representative of the general population of the disorder insofar as their seizures were characterized by preseizure anxiety and visceral symptoms, alterations in consciousness, involuntary motor behavior, and at least partial amnesia. In addition, seizures were poorly controlled by medication, and extensive medical histories were available (see pp. 29-31). Individuals were sufficiently stable within the community to make possible an extensive follow-up.

Three subjects were female and two male, all right-handed. Ages ranged from 17 to 36 at the start of the project.

MEASURES

1.Clinical Laboratory Measures. Baseline serum levels of anticonvulsant medications and 12 channel clinical EEGs were obtained from the subjects during the four-week baseline period and again after 12 months of therapy.

2.Seizure Frequency Records. Subjects kept a daily record of seizure activity during the baseline period and continued through the first 12 months of data collection. During the next six months, subjects mailed in their charts, monthly. No contact was made during the final six-month follow-up period. At the end of two years, seizure frequency was determined by personal interviews. Seizure data were confirmed periodically by interviewing family members.

3. Neuropsychological Testing. Tests were administered to assess the effects of the therapy program on cognitive, perceptual, and emotional impairment during periods between seizures. An extensive battery of measures was administered because of the wide variation in neuropsychological deficits in this subject population. The tests employed assessed a broad range of abilities dependent on cerebral functioning (Reitan [12]). Tests were administered in the following order: Bender-Gestalt Test. Wechsler Memory Scale. Wechsler Adult Intelligence Scale, Halstead-Reitan Neuropsychological Test Battery. Rorschach Inkblot Test, and the Minnesota Multiphasic Personality Inventory (MMPI). Tests were administered during the baseline month, and again after one year. One subject entered the project late and was not tested.

BIOFEEDBACK INSTRUMENTATION

A late model Autogenic Systems 120 EEG analyzer was used for the study. The in is a single channel operational amplifier which signal conditions with period to period analysis to compute real-time dominant frequency and amplitude into analog output signals ranging between 0 and 10 volts DC for conversion to audio feedback signals and further data recording. The frequency output and "raw" EEG were recorded on Channels I and 2, respectively, of a Grass Instrument Model 79D polygraph. The analyzer and polygraph were interfaced using two Grass 7PO3 adaptor panels. One channel of EEG was used to independently record data on the polygraph's third channel using a Grass Model 7PB3 wide-band AC amplifier.

Beckman standard-size Ag/Ag C1 electrodes with adhesive discs and Parker Signa electrode gel were applied after cleaning the scalp with alcohol and abrading with Hewlett Packard Redux electrode paste. Electrode attachment was tested using a J&J Limited electromyograph (EMG) electrode test circuit accepting a resistance of < 5.000 ohms. Ground electrode was placed on the contralateral mastoid process. Electrode placements were anterior temporal (F7 or F8) and posterior temporal (T3 or T6). A medical neurologist selected the more abnormal hemisphere; if the clinical EEG's were not focal, the right hemisphere was recorded (F8 and T6). Each recording electrode yoked into respective grids of the Grass and A.S.I. amplifiers for simultaneous comparison recording of each instrument.

Bilateral anterior temporalis surface EMG was recorded with the electrodes and procedures described above. Placement was determined by palpation of the muscle under contraction. 'Me muscle action potentials were amplified and signal conditioned with a J&J Limited Model 55 EMG using a bandpass of 100 to 200 Hz and displaying RMS amplitude

values. The full wave rectified surface EMG output was fed into a Grass adaptor panel and recorded on Channel 4 of the polygraph with a driver-amplifier setting 1/2 amplitude high frequency at 0.5 Hz.

') Includes temporal lobe epilepsy. psychomotor epilepsy

TREATMENT PROGRAM

1.EEG Biofeedback. EEG normalization rather than selective enhancement of specific frequency ranges is most clearly associated with a reduction of seizures (Lubar (151). Accordingly, individuals were reinforced for normalizing their awake, relaxed EEG pattern. To reduce possible muscle artifact from the EEG recordings, subjects were first trained to maintain relaxed scalp muscle tonus via anterior temporalis EMG feedback. Audio output and threshold were set to produce a loud tone burst (65 db) if 2 uV/sec was exceeded for 0.5 seconds. The tone also interrupted subsequent EEG normalization training as a time-out when these values were exceeded. Each subject was able to maintain normal resting muscle scalp tonus.

EEG biofeedback began with the therapist observing five minutes of baseline recording. Minimum amplitude of 10-uV/sec and dominant frequency were selected on an individualized basis. The goal was to achieve an amplitude greater than 10-uB/soc and selectively to shape the frequency toward the 8 to 12 Hz range. Then the individual was trained to increase the amplitude within this range.

2.Behavioral Counseling. Behavioral counseling was conducted in 15-minute sessions preceding and following EEG biofeedback sessions. The sessions took place at weekly intervals for six months and monthly intervals for six months.

Counseling involved three objectives: Identifying the pre-seizure warning and/or aura; identifying emotional, behavioral, physiological and/or environmental mechanisms which trigger seizure activity; and learning a suitable relaxation technique which is effective in aborting seizure activity.

a. *Pre-seizure Warning and Aura*. Patients were taught the importance of identifying the symptoms that precede seizure activity. These symptoms are consistent in individuals and are subtle and varied, ranging from rushes of thought to focal disturbances. In most patients, the pre-seizure symptoms are consistent and therefore a reliable indicator of impending seizure activity. At the start of the project, two subjects were aware of pre-seizure symptoms; by the end of the month, two others were, and the fifth one discovered these symptoms in the fourteenth month.

b. The second objective was to help the individuals become aware of stressful factors in their lives which tend to trigger their seizures. They were made aware of common life stresses that tend to aggravate their seizure condition such as sleep loss, poor eating habits, personal conflicts, job or school pressure. They were encouraged to allow self-awareness to develop through the use of a daily log of seizures. This log focuses attention on the relationship between daily activity and seizure frequency.

c. *Relaxation*. The third objective involved instruction in relaxation techniques. Our patients were taught deep breathing techniques to initiate relaxation. They were instructed then to use

progressive muscle relaxation exercises. A written instructional hand-out was given for home use. During weekly EEG biofeedback sessions, individuals were given reinforcement for producing awake relaxed EEG patterns (alpha range 8-12 Hz and 50 uV or greater). These weekly sessions were intended to enhance relaxation gained by doing daily breathing and relaxation exercises. The individuals were encouraged to use these techniques when they felt tense or in a pre-seizural state.

Andrews Fights Epilepsy and Wins, Middletown Time Star, Feb 13, 1986

RESULTS

1.Clinical Laboratory Measures. Clinical anticonvulsant levels are shown in Table 1. There is no apparent relationship between anticonvulsant medication levels and changes in seizure frequency or neuropsychological test results. Clinical EEG's remain fundamentally unchanged (Table 2).

2.Seizure Frequency. Figure I depicts monthly seizure frequency of four subjects over a period of 24 months. A consistent decreasing trend can be seen in all individuals. Continued decrease in seizure frequency in the one-year period after cessation of therapy can be seen with the three individuals who persisted in having seizures during the actual clinical treatment time. Individual differences in reduction of seizures are outlined in case histories (see pp. 29-31).

3. Four out of five of our subjects carried out significant positive *life changes.* These included enrollment in college, an increased social life, directing a training project for epilepsy and running a major political effort, job employment for the first time in eight years, and teaching nursery school. In every instance, personal and family life showed significant improvement (cf. pp. 29-3 1).

4.Neuropsychological Testing. Subjects showed improvement as assessed by the Wilcoxon Signed-Ranks Test, on the following measures (** a = .062. * a = .125):

Wechsler Adult Intelligence Scale

- ** Full Scale I.Q.
- ** Performance I.Q
- * Verbal I.Q.
- * Block Design
- * Picture Arrangement

Wechsler Memory Scale

- * Memory Quotient
- * Mental Control
- * Visual Memory
- * Paired Associations

Halstead-Reitan Neuropsychological Test Battery

- * Category test

- * Tactual Performance test
- * Dominant Hand
- * Non-Dominant Hand
- * Total Time
- * Memory
- * Localization
- * Trails A
- * Rhythm test
- * #Writing Dominant Hand
- * Impairment Index

Consistent patterns of improvement are apparent (see Table 3). The Impairment Index provides an overall measure of the degree of cerebral dysfunctioning. On this measure, three of the four subjects showed improvement. Specific gains were made on measures of abstract reasoning, intelligence, memory, attention, and concentration. In addition to these higher functions, improvements were also made on certain sensory (Fingertip Number Writing) and motor (Tactual Performance Test) tasks. Pre-therapy MMPI profiles are consistent with substantial personality disturbance in two of the four subjects (C.A. & K.W.). On re-testing, two (C.A. & K.P.) showed improvement, one (D.P.) remained essentially the same, and one (K.W.) showed greater degree of disturbance. (A subsequent third administration of the MMPI for this subject nine months later did show significant improvement over the first two MMPI profiles.) On the Rorschach test, all but one of the subjects (D.P.) manifested an excessive degree of fabulized imagery and marked looseness of association prior to therapy. On re-testing, all three of these subjects' protocols showed a reduction in these features.

DISCUSSION

Analysis of the results indicates that the clinical therapy model developed in this pilot study was successful in producing favorable changes in seizure frequency, psychological tests and emotional functioning. Furthermore, these improvements were maintained in the five subjects during the 12-month period following the therapy program. It is beyond the scope of this study to isolate variables in the therapy model responsible for the positive changes. Further study using control groups might isolate the more important components. Our work with partial complex epilepsy leads us to propose that to some extent the seizures may be within the realm of voluntary control. Our study indicates that individuals with partial complex epilepsy are sometimes able to diminish the frequency of daytime partial-complex seizures.

The standard medical approach has been to use pathological phenomena described by patients in a diagnostic way. The usual approach to treatment of complex-partial epilepsy is to take careful history, outline aberrancies such as frequent deja vu, dissociation, floating sensations, disturbed memory, and then pronounce the diagnosis of complex-partial epilepsy and the treatment as anticonvulsant medication.

Our counseling method is designed to allow the aberrant phenomena to tell us something of the individual's capacity to control seizures. Often, the perceptual aberrancies described by individuals with complex-partial epilepsy present more of a problem in their daily activities than their actual seizures. When individuals gain control over their thought processes, they can effect positive changes in interpersonal relationships, jobs and school performance.

Our counseling includes: 1) self awareness; 2) awareness of parapsychological phenomena as normal and not to be feared; 3) identification of subtle internal changes that precede seizures; 4) identification of factors in their daily lives that trigger seizures; 5) recognition of their ability to change the "feeling state" of the brain by using relaxation techniques and reinforcing these techniques with weekly EEG biofeedback training.

Because the results we obtained in this pilot project were uniformly good, some investigators might raise the question as to whether we were working with people who had pseudo-seizures as opposed to those with organic seizures. We think the summaries and case histories outline clinical histories compatible with actual seizures. In addition, focal temporal slowing in the 24 Hz and the 5-6 Hz range had been documented in past EEGs in all of our five subjects. Two of our subjects had exhibited focal spikes over temporal region in the EEGs at some time in their clinical seizure history. One of our patients had bilateral temporal spikes at the beginning of our project, with improvements of her EEG during the course of therapy.

At the beginning of our study, the five subjects were on from one to four anticonvulsant medications. These medications may contribute, to increased problems with memory function, flow of thought and body awareness (Matthews & Harley [13] and Hutt et al. [14]). It is possible that anticonvulsants "re-set' the central nervous system and make it difficult for patients to perceive subtle internal changes that indicate the onset of a seizure. Our study leads us to speculate that prescription of anticonvulsants should be directed at minimally influencing a patient's level of alertness and ability to perceive subtle internal mechanisms that can herald increased seizure frequency. Within this context, the full potential of a behaviorally oriented approach in rehabilitation of individuals with complex partial epilepsy will be understood only when it is instituted early after the initial diagnosis and, when possible, before the institution of anticonvulsant medication.

CASE HISTORIES

C.A. (17, female) was the product of a normal pregnancy, labor and delivery. Her early development and milestones were normal. At age 5, she began to have seizures characterized by brief absences with movements of her hands, chewing, dilatation of pupils and a "spacey" look. On occasion, she would lose her balance and fall. She also had auras of dizziness, which allowed her to sit down. These episodes lasted 30 seconds and occurred with variable frequency. Both C.A. and her mother related increase of seizures during stressful times like the beginning of the school year or with lack of sleep. She had been on Dilantin, Mebaral, a trial of Depakene and Diamox. Despite adjustment of medications, she continued to have psychomotor seizures averaging 12 per week. C.A. had great trouble with interpersonal relationships. She had virtually no interactions with classmates in school. At 16, she carried a small stuffed animal held with an elastic band on her arm, which she called "Teddy". Clinical EEGs in 1979 and prior to the initiation of our project demonstrated general bursts of high amplitude slow and spike and sharp activity, more abnormal on the right, but generalized and superimposed on an irregular 7-8 Hz background. During the 12 months of therapy and the 12 months following therapy, C.A. experienced a continuing and marked decreasing trend in seizure frequency. She became involved in social activities at school. During the summer after graduation from high school, she toured the United States with a high school choir group. She has entered college where she is performing acceptably. Neuropsychological testing demonstrated an increase of memory quotient 12 months after entertaining into the project. Her medications were unchanged.

D.C. (24, female) had onset of grand mal seizures in Kindergarten. The earlier seizures had no associated aura. Subsequent seizures had "an impulse of knowing the seizure was going to happen and tunnel vision". The seizures have consisted of tonic-clonic movement with tongue biting. She was placed initially on Dilantin with some intermittent seizures. Between age 17 and 19 the seizures remissed. At age 19, she had seizures occurring at 2-3 month intervals while maintaining therapeutic levels of Dilantin. She also experienced frequent deja vu, one episode of absence seizure, and difficulty with organization of thoughts. She started taking Phenobarbital at age 20. She experienced recurrence of seizures at age 21 during menstrual periods. She continued to have from one to three seizures per month at the time of her menstrual period despite therapeutic levels of Dilantin, Phenobarbital and the use of Diamox. She was reluctant to use other types of medication and entered our project for that reason.

A clinical EEG in 1975 demonstrated generalized mild slowing with right temporal slowing intermixed. At the beginning of our project, she continued to have generalized mild slowing. D.C. experienced no psychomotor or grand mal seizures after initiation of the project. Diamox was withdrawn and she remained on Phenobarbital after Dilantin was stopped at the end of the first year. During the course of the project, she developed a more active social life. She became an effective and well-regarded nursery school teacher.

D.P. (36, male) experienced a nocturnal grand mal seizure lasting four minutes at age 32. A clinical EEG at that time showed right temporal delta slowing. He then began to have three to four episodes where his face would turn red and he would lose track of his activities. These occurred from three to six times per day in clusters and then spontaneously abated for several weeks. He was initially placed on Dilantin and then Dilantin and Phenobarbital. The "absence" seizures continued with the same frequency and intensity. He had no further grand trial seizures. Because of continuation of seizures after initial evaluation, he was seen at University of California, San Francisco and Stanford neurology departments. Clinical EEGs demonstrated mixed slowing over the right temporal region and then generalized 5-6 c/s slowing, more-marked in the temporal leads. Repeat CT scans were negative. He took various medications including Dilantin 400 mg per day, Tegretol 1200 mg per day, Mysoline 500 mg per day, and Depakene 1000 mg per day in divided doses. Because of continuation of seizures, he was seen at the neurology department at U.C.L.A. where he was suggested his Tegretol be increased. In addition to clusters of absence seizures, D.P. complained of memory problems and difficulty thinking. Despite these problems, he was able to continue working long hours as a concrete finishing foreman in a family business.

D.C. experienced a significant reduction in psychomotor seizures during the first eight months of the project. At that time, he underwent major urinary tract surgery. He then had an increase in seizure frequency. Because he complained of difficulty with memory and thinking, his medication was reduced to include Dilantin and Tegretol alone in therapeutic doses. His seizure frequency showed a progressive decline after his release from clinical sessions. He has been seizure free for the last six months. He underwent no major changes in his job or family situation. Neuropsychological testing demonstrated improvement of memory quotient from 90 to 112 with concomitant increase in performance I.Q.

K.P. (35, female) started having seizures at age 4. These consisted of involuntary laughing. She then started to have grand mal seizures. She was placed on various medications and a ketogenic diet She continued to take phenobarbitol, Celontin, and Mysoline from age 11 to age 29. At that time, she began to have focal seizures with flailing of the right arm for 15

seconds. This was associated with lapse of memory. Anticonvulsants were increased to Mysoline 500 mg and Celontin 600 mg daily. From age 29 to 34, she remained under the care of a neurologist and medication was reduced with an attempt at elimination of medication. In each instance when medication was withdrawn, she experienced focal seizures with twitching of the right face and eye. At the time she entered our project, she was taking Dilantin 300 mg, Mysoline 250 mg and Celontin 300 mg daily, and she was having difficulty continuing with school. She had been unable to maintain a regular job. During the early years of her seizure disorder, K.P. had been evaluated at Mayo Clinic with normal pneumoencephalography and arteriography. Clinical EEGs before her teenage years at Mayo Clinic had shown diffuse slowing with high voltage spikes in the right frontal area. An EEG prior to entry into this project demonstrated irregular 7-8 Hz background with intermittent theta and sharp activity greatest over the frontotemporal regions. During the course of our project, K.P. stopped having seizures. One year later, she had a single grand mal during a period of sleep deprivation and was started back on Dilantin 200 mg per day. She has had no other seizures. K.P. made impressive changes in her life activities. She entered a Master's Program in psychology. She obtained full-time employment. She developed and implemented a comprehensive program for instructing people and their families with epilepsy in the nature of the disorder. Over 50 people attended each of eight sessions. She assumed directorship of a major political campaign.

K.W. (28, male) was the product of a normal pregnancy, labor and delivery with normal milestones and development. He had no neurologic problems until age 19. During his first year at college, he developed headaches characterized by visual aura, and sensations of movements in his lower peripheral visual field. Four months later, he had a grand mal seizure. He was started on Dilantin and phenobarbitol. After initiation of medication, he experienced seizures preceded by heavy pressure 'in his chest and the sensation of burning rubber with loss of consciousness following. The seizures would last from 30 seconds to several minutes. These seizures would occur in series and then he would be seizure free for several weeks. Prior to the onset of his first seizure, he had an increase of deja vu phenomena occurring four to five times per week. In addition, he had a sensation of floating out of his chair, which he described as being somewhat pleasant. K.W. underwent several medical work-ups with multiple normal CT scans. Clinical EEG's demonstrated 5-6 Hz activity on repeated occasions with no well-defined alpha activity. K.W. had taken a variety of medications, including Tegretol, Dilantin, Mysoline and Depakene. Following a consultation at Stanford University, Celontin and Mesantoin were used. Despite this, he continued to have seizures at three to five week intervals. At the time he entered our project, he was taking Tegretol 1200 mg per day, Dilantin 500 mg per day, Depakene 1250 mg per day and Mysoline 625 mg per day. He complained of drowsiness as a side effect of the medication. He was unable to pursue school studies or obtain a job. K.W. experienced a gradually declining trend in seizures with some exacerbation in the first year of therapy. After one year, there was a consistently declining trend in seizure frequency and he has been seizure free for eight months at this time. Neuropsychological testing demonstrated an increase in performance I.Q He now manages two bowling alleys, his first job in eight years.

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Note:This paper has not been published. I am very much pleased that it is made available to the group now: Not least, it provides some impression about the historical and scientific background of the working book, which was introduced in our first circular issue. (B.M.)

Patient	Medication	Dose (24Hrs)	Levels (Pre)	(Post)	Therapeutic Range (mcg/ml)
C.A.	Dilantin	250mg	24.0	28.0	(6<17)
	Mebaral	300mg	4.0	34.0	(10<40)
	Diamox	750mg			
D.C.	Dilantin	300mg	9.0	19.0	(6<17)
	Phenobarb	60mg	18.0	21.5	(10<40)
	Diamox	500mg			
D.P.	Dilantin	500mg	3.0	2.0	(6<17)
	Tegretol	1200mg	5.0	4.2	(3<10)
	Depakene	1500mg	42.0	40.0	(50<100)
	Mysoline	500mg	5.0	5.0	(1<15)
K.P.	Dilantin	200mg	8.0	0.0	(6<17)
	Mysoline	250mg	1.0	0.0	(5<15)
	Celontin	30mg			
K.W.	Dilantin	500mg	19.0	16.0	(6<17)
	Tegretol	1000mg	2.6	2.2	(3<10)
	Depakene	1250mg	40.0	42.0	(50<100)
	Mysoline	500mg	4.6	4.0	(5<15)

Neuropsychological Test DataSummary

Table 3

C.A.		**D.P.**		**K.P.**		**K.W.**		#	#
Pre	**Post**	**Pre**	**Post**	**Pre**	**Post**	**Pre**	**Post**	**impr**	**low**

WECHSLER ADULT INTELLIGENCE SCALE

Information	9	9	7	7	11	9*	13	14	1	1
Comprehension	9	9	13	10*	12	10*	13	12*	1	3
Arithmetic	7	7	6	6	8	10	11	11	1	0
Similarities	10	12	11	10*	12	12	12	14	2	1
Digit Span	9	9	11	14	11	10*	7	10	2	1
Vocabulary	10	11	9	10	13	13	14	13*	2	1
Digit Symbol	7	7	8	8	10	12	7	7	1	0

Picture Completion	7	7	12	14	7	9	12	13	3	0
Block Design	4	2*	7	11	9	12	9	10	3	1
Picture Arrangement	6	12	11	13	12	12	6	9	3	0
Object Assembly	10	8*	6	10	10	12	9	9	2	1
Verbal I.Q.	99	102	95	97	106	104*	109	113	3	1
Performance I.Q.	81	83	101	112	98	114	9	98	4	0
Full Scale I.Q.	91	94	97	103	103	109	102	107	4	0

WECHSLER MEMORY SCALE

Information	5	5	4	5	5	6	6	6	2	0
Orientation	5	5	4	5	5	5	5	5	1	0
Mental Control	4	7	5	7	6	7	9	9	3	0
Logical Memory	9	7*	6	7	9	12	11	10*	2	2
Digit Span	10	9*	12	14	12	12	9	11	2	1
Visual Memory	4	5	6	10	9	11	12	11*	3	1
Paired Associates	9	13	16	19	19	17*	8	14	3	1
Memory Quotient	77	84	90	112	105	118	94	103	4	0

HALSTEAD-REITAIV NEUROPSY-CHOLICAL TESTS

Category Test Errors	98	96	58	64*	34	23	26	10	3	1
TPT Dominant Hand	10'(6)	10'(9)	10'(5)	5'54"	10'(9)	6'57"	4'47"	7'52"	3	1
TPT Nondom Hand	10'(5)		10'(5)	8'03"	3'18"	2'34"	5'41"	4'40"	3	0
TPT Both Hands	8'49"	6'15"	3'55"	7'34"	8'00"	2'22"	3'48"	3'24"	3	1
TPT Total Time	28'49"	24'45"	23'55"	22'31"	21'18"	11'53"	14'16"	15'56"	3	1
TPT Memory	7	9	6	8	7	8	9	40		
TPT Location	1	1	6	8	0	5	5	6	3	0
Trails A	44"	33"	22"	30"*	35"	23"	43"	31"	3	1
Trails B	u	u	84"	104*	121"	52"	73"	63"	2	1
Speech Sounds Errors	8	4	4	4	5	4	2	7*	2	1
Rhythm Test Correct	16	15*	11	28	26	27	24	25	3	1

Fingertap Dom	31	37	46	42*	49	52	45	37*	2	2
Fingertap Nondom	32	u	40	36*	41	47	32	32	1	1
Tactile Forms Right	0	0	0	0	0	0	0	0	0	0
Tactile Forms Left	0	u	0	0	0	0	0	0	0	0
# Writing Right	13	r	5	0	9	6	2	0	3	0
# Writing Left	13	r	2	1	5	4	0	0	2	0
Finger Agnosia Right	1	1	1	1	0	1*	0	0	0	1
Finger Agnosia Left	1	1	2	1	2	3*	0	0	1	1
Impairment Index	.9	.7	.7	.6	.4	.0	.3	.3	3	0
No. of Tests Improved	19		27		28		21		95	
No. of Tests Impaired	5		8		7		8			28