Reprint (1-1)8

Positive Outcome With Neurofeedback Treatment In a Case of Mild Autism

Arthur G. Sichel, Lester G. Fehmi, and David M. Goldstein

This article looks at the experience of Frankie, an autistic 8 and 1/2 year old boy. He was diagnosed mildly autistic by several specialists. One specialist claimed he was brain damaged and "autistic-like " and that there was no hope for improvement. At Frankie's mother's request, neurotherapy diagnosis and treatment was begun. After 31 sessions, Frankie showed Positive changes in all the diagnostic dimensions defining autism in DSM-111-P, This has profound implications for treatment in a field with few low-risk alternatives.

Introduction

The DSM-III-R (American Psychiatric Association, 1987) defines autism as "characterized by qualitative impairment in the development of reciprocal social interaction, in the development of verbal and nonverbal communication skills, and in imaginative activity. Often there is a markedly restricted repertoire of activities and interests. . ."

The mother of an 8 1/2 year old autistic boy contacted one of the authors seeking treatment for her son, Frankie. She was at first referred elsewhere but returned and we agreed to evaluate him. The first referral was to Jonathan Cowan who, in verbal communication to one of the authors, reported symptom amelioration in EEG treatment of an autistic child.

Pretreatment Behavior

Frankie exhibited a seeming lack of awareness of the existence of others. He once forgot his glasses after a training session. When he came for his next session, one of the authors held his glasses up for him to take. He did not appear to visually focus on or attend to the author. He focused on the glasses. He reached out and took them, looked at them, looked up, as though focusing on the wall through and behind the author, put on the glasses and walked away. The author had the distinct impression of being looked through, never looked at or attended to.

Frankie did not seek comfort when distressed. He showed no imitation of his siblings and did not engage in ' social play. His mother reported that he did not vocalize until the age of three, when he began to babble. She said she has worked a great deal on his verbal communication. Verbalizations appeared quite minimal at the beginning of treatment. He did not make eye contact, did not look at the person or smile in social approach. He had a fixed stare in social situations.

Frankie did not change facial expression or respond verbally when addressed. However, he usually did as his mother directed. Directions were simple and responses were slow. When questions were asked of him, his mother would repeat the question until he made some minimal response and she would interpret that response to the neurofeedback provider. He showed very

little imaginative play. He read with a monotonous tone of voice. His brief sentences often had odd inflection, almost a sing song quality. He referred to himself as "Frankie" and rarely spoke unless spoken to.

He displayed stereotyped body movements in the form of hand flapping. He was attached to a number of objects which he insisted on carrying around with him. In summary, pretreatment behavior included symptoms which meet the diagnostic criteria for Autistic Disorder listed in the DSM-III-R, sections A, B, C and D.

Patient Assessment

Two separate psychologists, each in private practice, one also a school psychologist, diagnosed him autistic. A neurologist specializing in autism, who is on the faculty of a medical school, diagnosed him autistic. A special education professor at a state college said he was autistic-like but brain damaged and said there was no hope for improvement. His mother said the psychologists and physician described him as high level or mildly autistic.

A test of variables of attention (T.O.V.A.) was administered. It is used as an aid in diagnosing attention deficits in children and adults. He did not respond to the tester, but appeared to be participating in the test during practice and during the test. His T.O.V.A. performance showed a variability score which was statistically interpreted by comparison with age related norms to be consistent with an attention deficit disorder.

Electrical activity of the brain was recorded at 19 sites (jasper, 1958) using a Lexicor Neurosearch-24 while Frankie was engaged in six different activities (sitting still with eyes closed, with eyes opened, reading, listening, doing a mental arithmetic task and drawing). Calculation of the percent power ratios of theta to beta brain wave activity showed the kind of deviations from normal which are seen in attention deficit disorders (i.e., percent power ratios above 3). Mean ratios, averaged across the five conditions in which his eyes were open, were highest in the parietal and central regions, as shown in Table 1. For the three parietal sites, averaged across the five eyes opened conditions, the pre-therapy theta (4-8 Hz) to beta (13-21 Hz) ratios were 4.07 (P3), 3.98 (PZ) and 3.63 (P4).

Neurotherapy

Because of high theta/beta ratios and with his mother's urging, it was decided to give Frankie the kind of theta/beta/EMG treatment which is being used successfully for attention deficit disorders (cf. 4, 5, 6, 7, 8).

As of this writing, Frankie has received 31sessions of training in which he has been rewarded for raising his SMR (12-15 Hz) and decreasing theta (4-8 Hz) activity at various brain loci on the sensory-motor strip and parietal lobe. EEG training during early sessions was provided by an EEG Spectrum and for later sessions on the Lexicor using the Biolex program, based on Frankie's choice of computer game. Both utilized a monopolar electrode placement with the ear

patient assessment, with the sites showing the highest ratios receiving the most neurotherapy.

Post Treatment Behavior

The following description of changes since initiation of neurofeedback training comes from his mother, from a female caretaker who has seen him daily for 3-4 hours per day, and from our own observations. His mother reported significant changes after three training sessions. She said he was talking more and had been affectionate with his siblings. For the first time in his life he played with his sister, and even kissed her, and he put his arm around his older brother.

Over the course of training Frankie's behavior continued to change. He began attending to and reacting to others. He started making eye contact. He presented his biofeedback trainers with valentine cards he had made; he appeared shy while presenting them and seemed thrilled when the cards were praised.

After 31 neurofeedback sessions he notices his sister's distress and tries to interfere when she resists taking a bath or going to bed. He seeks comfort when he reads something upsetting. He imitates his older brother and plays with his brother, his sister and a friend. He no longer tires easily and no longer has trouble falling or staying asleep. His headaches are significantly reduced, as is his tendency to appear anxious and worried. He is much less shy and withdrawn.

At this point in treatment, Frankie's verbalizations are still limited and responses continue to appear slow. He now sometimes makes eye contact and no longer has a fixed, vacant stare in social situations. He engages in a lot of imaginative play with his sister. He now reads with some expression. He does not speak much and speaks monotonously, but a singsong quality was not present during later sessions. He now refers to himself as 'T" He initiates conversations at home and asks for what he wants. Before, he frequently engaged in a repetitive jumping activity. Now, he rarely does this. Before, he showed great attachment to a number of unusual objects, insisting on carrying them around. He now carries markedly fewer things around with him.

He is evaluated annually by a speech therapist. His most recent evaluation was just prior to this writing. The speech therapist reported that he has improved one whole diagnostic category since his last evaluation. Last year he was found to have profound language deficits (over 40 months delay in development). This year he showed severe language deficits (30 months delay). The speech therapist specified that no hand flapping or self-stimulating behavior was observed. He did confuse pronouns and omit articles. He could not follow two and three step commands and echolalia was present. However, he had improved so much that, for the first time, the speech therapist was able to use age appropriate tests. In summary, Frankie has demonstrated positive changes on all the diagnostic dimensions defining autism in DSM-III-R.

Brain Wave Changes

QEEG mapping of Frankie's brain activity was repeated after completion of 31 sessions of neurotherapy. The pre- and post-neuro therapy theta to beta percent power ratio for each of the 19 sites recorded, averaged across the five eyes opened conditions, are shown in Table 1. Prior to neurotherapy, seven sites had percent power ratios above 3.00 (see values denoted by

asterisk), and the highest ratio (4.07) was at P3.

As shown in Table 1, two sites (P3 and CZ) remain slightly above 3.00 after neurotherapy. Fifteen of the 19 sites showed reduction in their power ratios after neurotherapy. Ranked among the largest reductions in percent power ratios were the changes that occurred at P3 and PZ. These represent the sites which received the predominant proportion of training time.

Discussion

The behavioral changes and the brain wave changes in this 8-year-old autistic boy are viewed as a positive outcome of neurotherapy. These results are suggestive that neurotherapy can be an effective treatment for some of the symptoms of mild autism. It would be interesting to follow possible further gains with additional neurotherapy sessions.

Table 1

Mean percent Power Ratios for 19 brain sites across five eyes open conditions (sitting still, reading, listening, arithmetic and drawing) before and after Neurotherapy

	Neurotherapy	
	Pre	**Post**
F7	1.72	1.57
T3	0.25	1.90
T5	1.41	1.84
FP1	1.80	2.08
F3	2.00	2.54
C3	3.40a	2.99
P3	4.07a	3.04a
01	2.92	2.02
FZ	3.02a	2.73
Cz	3.59a	3.15a
PZ	3.98a	2.98
FP2	1.97	2.08
F4	2.50	2.26
C4	3.03a	2.94

P4	3.63a	2.90
02	2.76	1.83
F8	1.54	1.39
T4	0.95	1.10
T6	2.08	1.00
a Denotes percent Power Ratios above 3.00.		

The core deficit in autism as discussed by Pennington (1991) is the inability to imagine what is going on inside another person in terms of thoughts, feelings and images. It seems reasonable that one has to discriminate and be able to represent these internal states to oneself before one can imagine what internal states another person might be experiencing. Neurotherapy has led to the reduction of the power ratios in the parietal region, where Frankie's ratios were highest prior to neurotherapy, and where the experience of his body is mediated. The findings reported here support the hypothesis that neurotherapy training has led Frankie to pay attention to the experience of his body, or to attend to it or experience it differently, we suggest both more objectively and more intimately. We believe this newly learned and qualitatively different way of attending to and experiencing his body has had profound consequences (Fehmi, In press).

The same type of neurotherapy which is used to treat attention deficit disorders has initiated a process which reduced autistic symptoms and supported the development of normal patterns of social interaction and communication. This has profound implications for treatment in a field with few low risk alternatives. These results are consistent with the view that a basic defining characteristic of autism is the failure to pay attention appropriately to the experience of one's body. That is, mild autism may be profitably considered a form of attentional limitation or rigidity to which other attention treatments may also be useful (cf. 10). The authors look forward to further clinical research with mild autistic patients to support or refute the above findings and interpretations.

References

American Psychiatric Association (1987). *Diagnostic and Statistical Manual of Mental Disorders*, (Third Edition, Revised). Washington, DC: American Psychiatric Association, 1987.

Fehmi, L. G. (In press). Attention to Attention. In J. Kamiya (Ed.), *Applied Neurophysiology & EEG Biofeedback.* Future Health.

Jasper, H. H. (1958). The Ten Twenty Electrode System of the International Federation. *Electroenceph. Clin. Nuerophysiol*, 10, 371-375.

Lubar, J. F., & Shouse, M. N. (1976). EEG and behavioral changes in a hyperkinetic child concurrent with training of the sensorimotor rhythm (SMR): A preliminary report. *Bio-feedback and Self-Regulation*, 3, 293-306.

Lubar, J. F., & Shouse, M. N. (1976). Use of biofeedback in the treatment of seizure disorders and hyperactivity. *Advances in Clinical Child Psychology, 1*, 203-265.

Lubar, J. F., & Deering, W. M. (1981). *Behavioral Approaches to Neurology.* New York: Academic Press.

Lubar, J. F. (1991). Discourse on the development of EEG diagnostics and biofeedback for attention-deficit/hyperactivity disorders. *Biofeedback and Se~(-Regulation,* 16, 201225.

Pennington, B. F. (1991). *Diagnosing Learning Disorders.* New York: The Guilford Press.

Tansey, M. A., & Bruner, R. L. (1983). EMG and EEG biofeedback training in the treatment of a 10-year old hyperactive boy with a developmental reading disorder. *Biofeedback and Self-Regulation,* 8, 25-37.