STUDY OF EMOTIONAL-VOLITIONAL DISRUPTIONS DYNAMICS AND INTELLECT PREREQUISITES IN CHILDREN WITH ATYPICAL FRONTAL ABSENCES.

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Aim: Studying of principal psychological functions habilitation in children with atypical frontal absences upon compensation of epileptic process in order to comprehend the importance of organic locus in triggering intellectual disruptions and personality alterations.

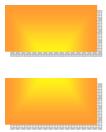
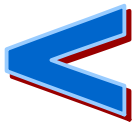
Materials and methods: study included children with absences without concomitant brain organic lesion proved by MRI (I group), and with an organic brain lesion proved by MRI (II group), all of them had been in a remission during three-five years. The following methods were employed: Wechsler subtests adapted to children (“picture to cut”, “remove the excessive”, “subject images”); “Ray-Osterrieth test”, overall assessment scales of psychological state (OASPS): emotions, sense of humor, mimics and gesture recognition and interpretation. The first psychological assessment was carried out three or four weeks after the last absence detected by EEG. The second assessment was carried out, on average, a year later the remission, the third one – in four years, the last assessment – in five years of successful anticonvulsive therapy, i.e. before planned discontinuation of anticonvulsants.

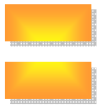
I group (no CNS organic lesion detected by MRT, with atypical frontal absences) N=69, ♀=34, ♂=35	II group (patients with a CNS organic lesion detected by MRT, with atypical frontal absences) N=143, ♀=70, ♂=73
Four neuropsychological assessments were carried out on all children: in 3 months, 1 year, in 3 and 5 years since the last seizure	
Irregularity of intellect structure for the reason of attention function deterioration. (baseline) 50 children on 69 (72.5%)	Irregularity of intellect structure for the reason of attention function deterioration. (baseline) 140 children (97.9%) on 143.
In 1 year – 46 children (66.7%).	In 1 year – 139 (97.2%)
In 3 years 44 children (63.7%)	In 3 years 138 children (96.5%)
In 5 years 44 children (63.8%)	In 5 years 138 patients (96.5%)

*validity $p \leq 0.05$

Deficiency of visual-spatial analysis and synthesis at first assessment in 40 children on 69 (57.9%)		Deficiency of visual-spatial analysis and synthesis at first assessment in 143 children (100%)
In 1 year – 38 children (55%)		In 1 year – 120 children (83.9%)
		
In 3 years – 36 children (52.1%)		In 3 years – 110 children (76.9%)
		
In 5 years – 34 children (49.2%)		In 5 years – 104 children (72.7%)

*validity $p \leq 0.05$

Immaturity of autoregulation mechanisms and cognition activity in 59 children (85.5%) on 69. (baseline)		Immaturity of autoregulation mechanisms for cognition activity in 143 children (100%) on 143. (baseline)
In 1 year –50 children (72.4%)		In 1 year – 130 children (90.9%)
		
In 3 years – 50 children (72.4%)		In 3 years – 120 children (83.9%)
		
In 5 years – 42 children (60.8%)		In 5 years - 119 (83.2%)

Comprehension of logical temporal relations, level of accessible generalization in 55 children (79.7%) (baseline).		Comprehension of logical temporal relations, level of accessible generalization in 120 children (89.3%). (baseline).
In 1 year – 51 children (73.9%)		In 1 year – 110 children (76.9%)
		
In 3 years – 40 children (57.9%)		In 3 years – 98 children (68.5%)
		
In 5 years – 40 children (57.9%)		In 5 years – 96 children (67.1%)

*validity $p \leq 0.05$

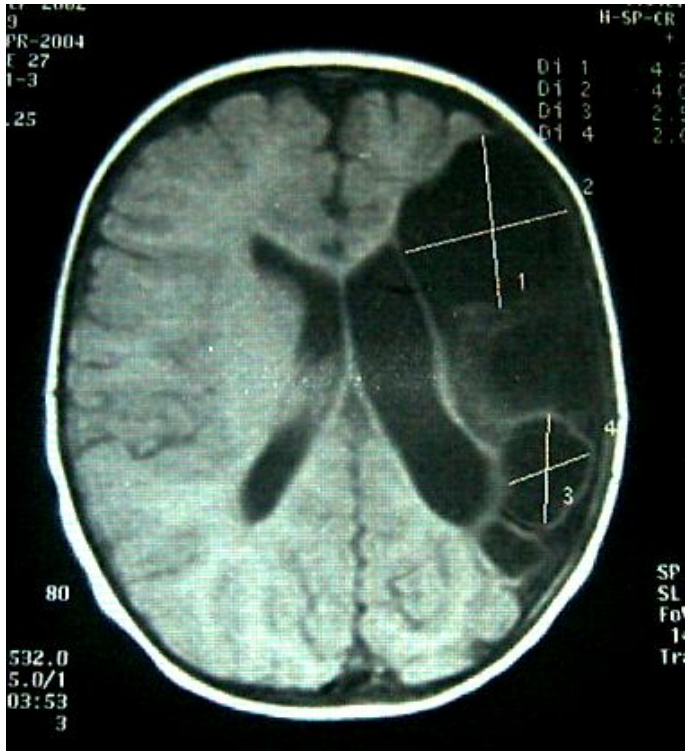
Patient M., 6 years 2 months old.

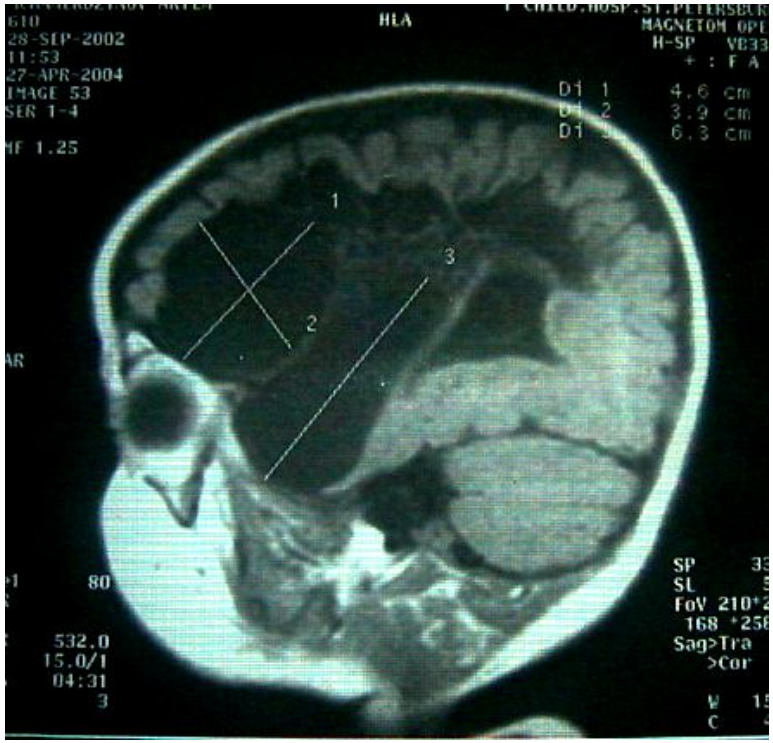
Anamnesis extract: IV pregnancy (I-childbirth, II-miscarriage, III-abortion), acute bronchitis at 15-16 weeks. Childbirth in 40 weeks, quick (2.5 hours), body mass - 2930 g., height - 51 cm, immediate cry, Apgar test assessment – 9 points, put to breast immediately, did not suck, was sluggish, manifestation of toxic erythema. By day 2 eyelid and body oedema aggravated; transferred to the Pediatric Academy. According to neurosonography by day 9 was diagnosed a stroke in the arteriae mesencephalicae basin (to the left), symptomatic therapy administered. At the age of 2 months received therapy for the right limbs paresis (nootropy therapy). Repetitive massage up to the age of 1 year.

Psycho-motor development – held head since the age of 3 months, turned over and sat since 10 months, actively cooing and babbling, walking at 2.5 years old. Simple speech since the age of 3 years.

Diagnosis: Cerebral spastic infantile paralysis, right-sided hemiparesis at the age of 3 months.

Repetitive brain MRT. Detection of evident cyst-atrophic alterations in the left hemisphere.





See explanations below.

Anamnesis: epileptic seizures onset at the age of 3 years, 2 months.

Seizure types at the moment of the age 5 y.o.

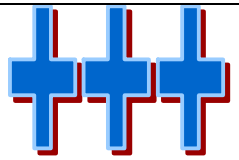
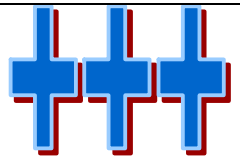
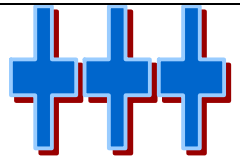
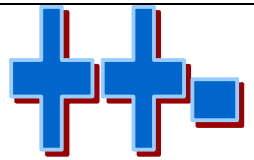
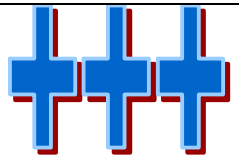
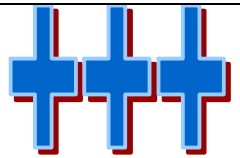
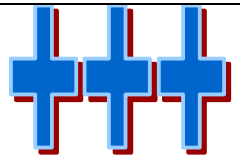
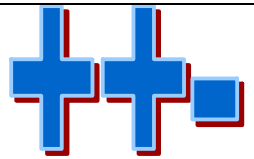
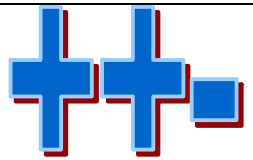
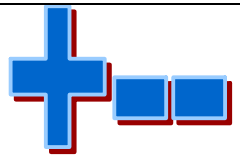
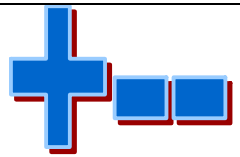
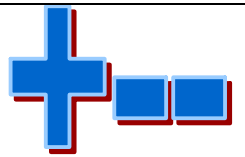
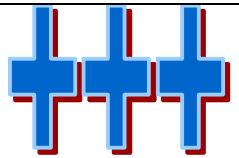
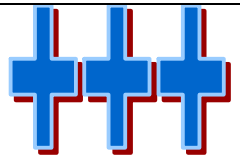
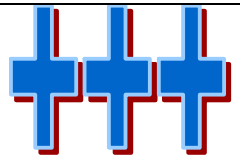
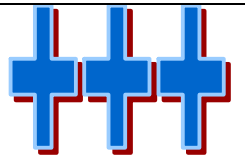
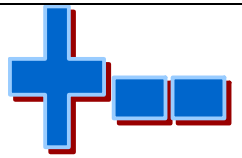
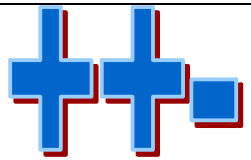
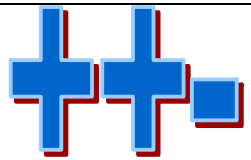
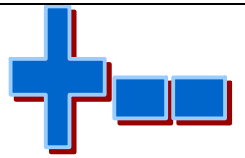
1. simple focal motor seizures with eye adversion to the right, clonic jerks of the right hand; at times clonic movements of the right leg without loss of consciousness for 1-2 min, concomitant salivation and vomiting (at times);
2. serial course of simple focal motor seizures (5-7 seizures per series) with consciousness depression;
3. focal simple motor seizures secondarily generalized as generalized convulsive seizures up to several times per month.
4. "Freezing" and drops.

Status course of focal motor seizures registered thrice over 2005, treatment administered in resuscitation department.

Main diagnosis – symptomatic epilepsy with right-sided focal motor seizures with generalization and atypical frontal absences. Psycho-motor development retardation.

Concomitant conditions – right-sided hemiparesis, bilateral partial atrophy of visual nerves discs.

Treatment administered: sodium valproate (up to 30 mg/kg) and carbamazepin (up to 15 mg/kg) as well as topiramate dosed 3 mg/kg over 6 months. Thereupon topiramate was replaced with lamotrigine and carbamazepin with oxcarbazepin. No effect noted. Since the age of 5 received calcium valproate with lamotrigine and clonazepam according to the age dosage norm. The number of generalized seizures was up to 5 per month. As Keppra was titrated, at the very beginning the number of seizures decreased to one simple focal seizure per month which allowed gradual discontinuation of lamotrigine and calcium valproate. Keppra dosage at this moment is 750 mg/day (41.6 mg/kg) and rivotril 0.0005 mg/day, acetazolamid 30 mg/day.

Index	1 month	1 year	3 years	5 years
Intellect structure irregularity (WISK, norm about 10)	 2-3	 2-3	 3-4	 5-10
Random otic attention concentration				
Deficiency of visual-spatial analysis and synthesis				
Immaturity of autoregulation mechanisms and cognition activity				
Comprehension of logical temporal connections, level of accessible generalization				

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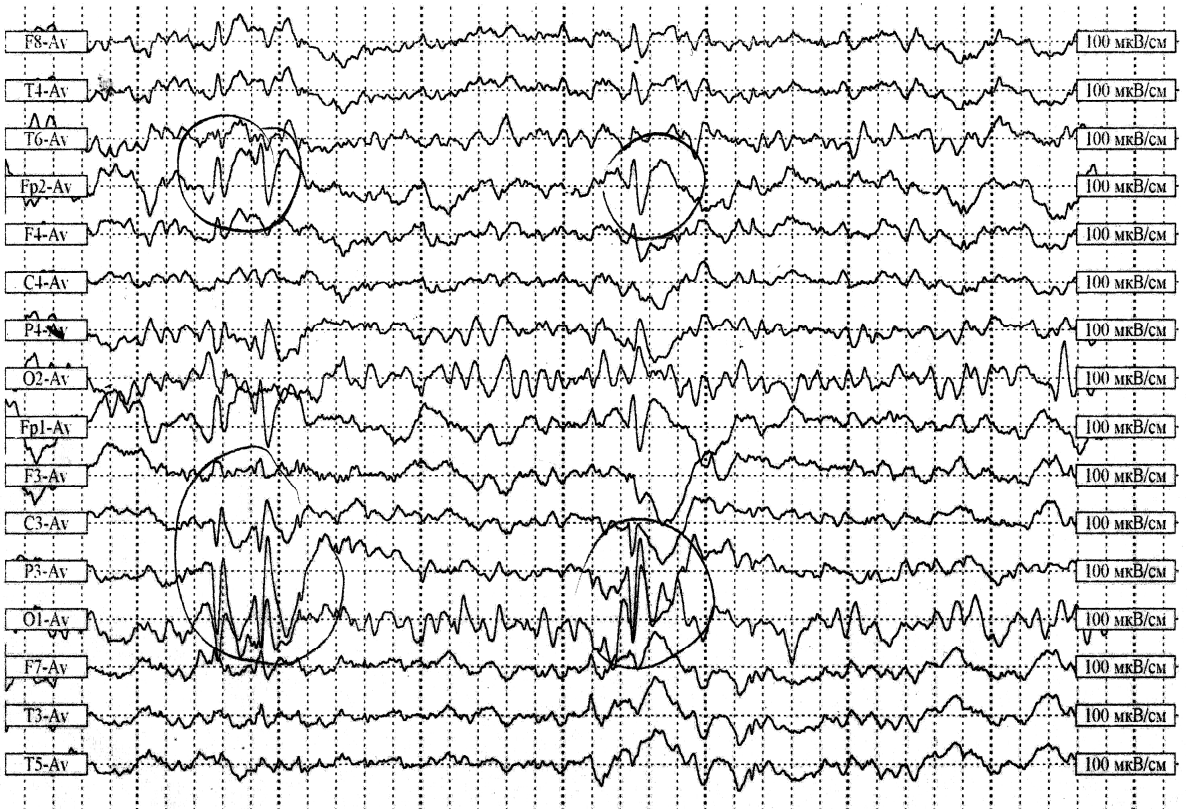
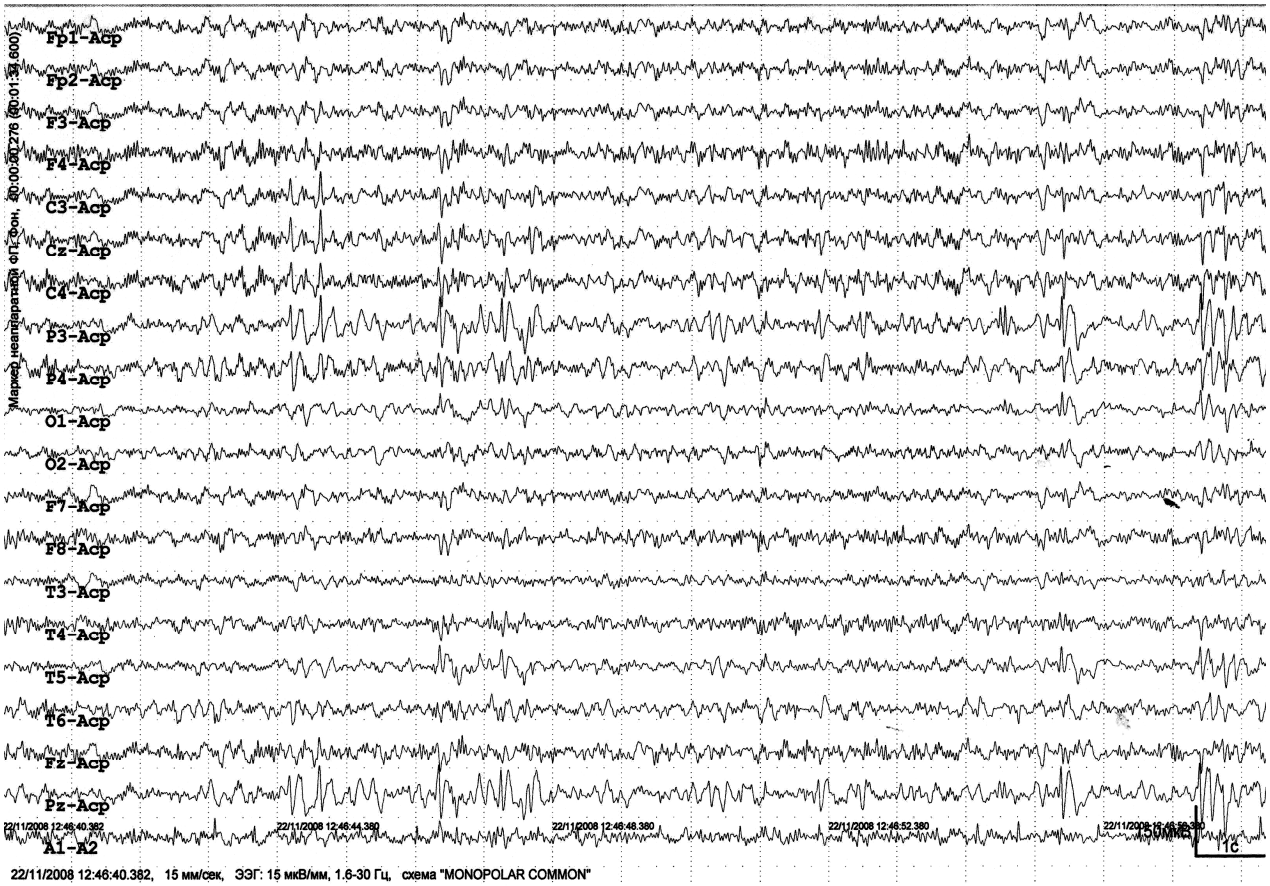
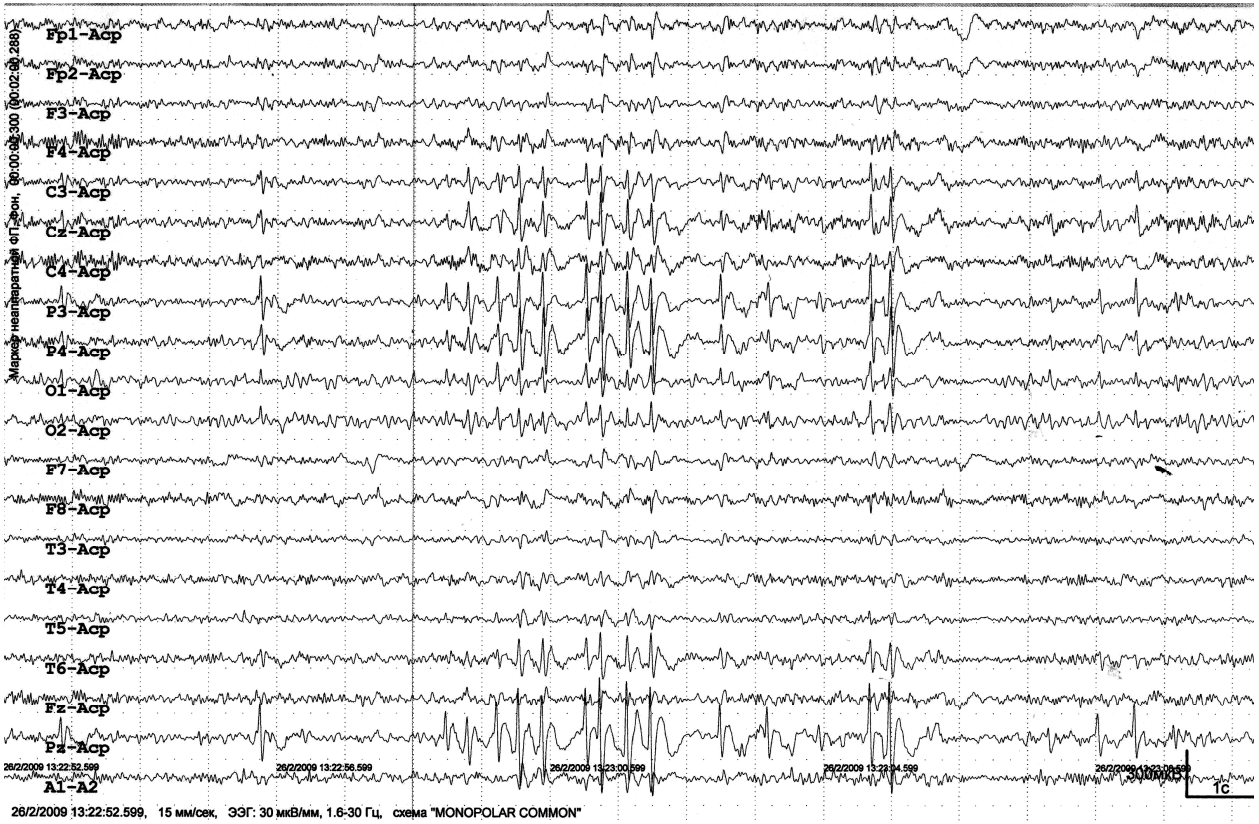


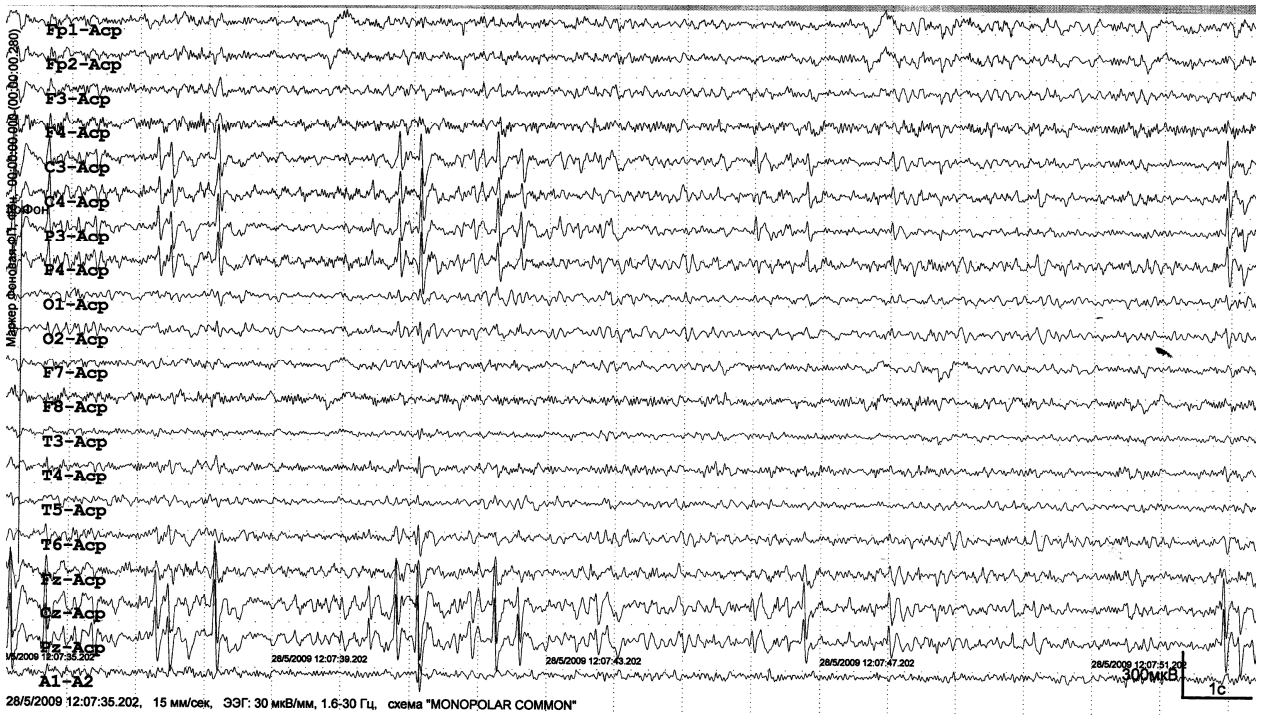
Epileptiform activity in frontal zones as high amplitude slow waves and in parietal-central and temporal leads (spike-wave complex) with an accent being in the left hemisphere. 4 years old.



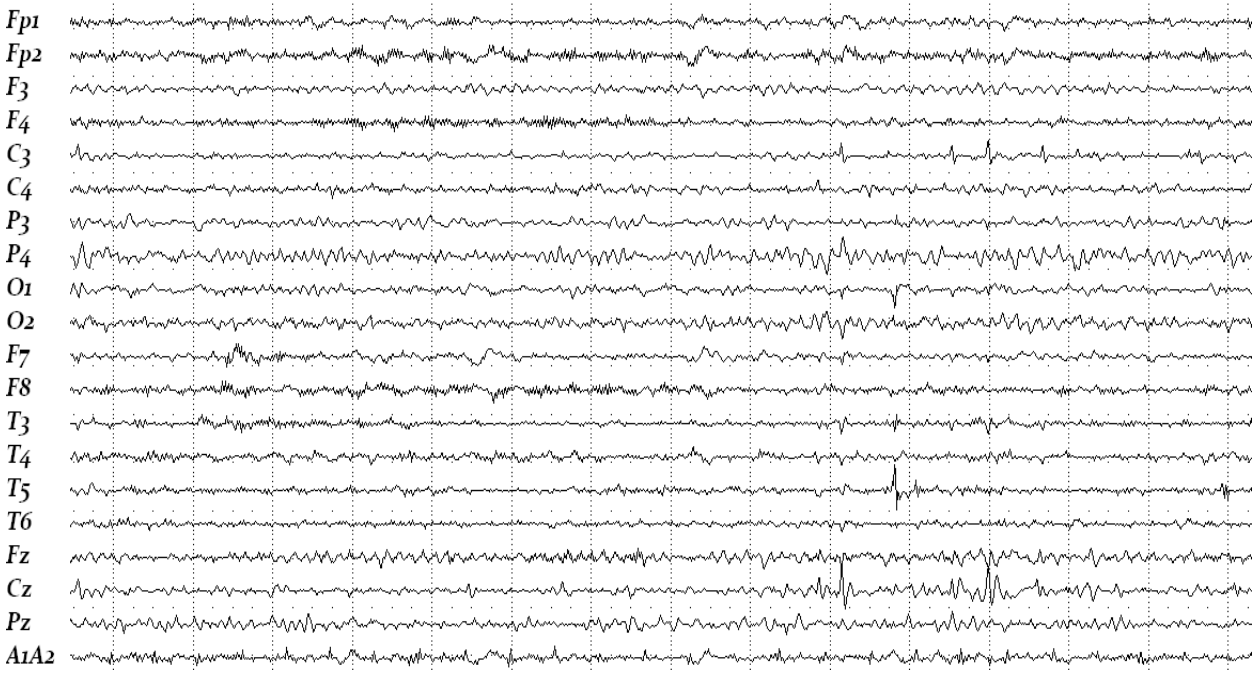
EEG-diffuse alterations of brain bioelectrical activity having regular traits and manifestations of a beta2-rhythme which is more evident in the frontal lobe; stable manifestations of localized paroxysmal activity in parietal-central lobes with an unstable left-sided accent, no lateralization. 5 years old.



EEG-expressed diffuse disregulatory alterations of brain bioelectrical activity, with manifestations as well as intense residual paroxysmal damages in occipital-parietal-back-temporal lobe, with a right-side tendency; at hyperventilation moderate increase of paroxysmal intensity. 6.5 years old



EEG-diffuse disregulatory alterations of brain bioelectrical activity with manifestations as well as less intense residual paroxysmal disruptions in central-parietal zones. No lateralization. Decrease of discharges amplitude. No seizures over more than 8 last months. 7 years old.



Diffuse disregulatory alterations of brain bioelectrical activity with manifestations of significantly reduced residual paroxysmal disruptions pertaining primarily to central zones with left-side lateralization. 8 years old.

Patient M, DOB 16 SEP 1999

Patient's anamnesis – first pregnancy with toxicosis of 1 and 2 half, acute respiratory virus diseases, flu at 2-3 weeks, threat of miscarriage at week 20, mother suffered German measles at weeks 24-25. Childbirth at week 40, duration 10 hours. Immediate cry, Apgar scale assessment 8-9 points, put to breast immediately. Due to milk absence artificial feeding was used. BCG at a maternity hospital. Right-sided pyramid syndrome noted since birth, supervised by a neurologist. Psychomotor development – stood up since 5 months, overstepped with a support since 6 months, at 9.5 months walked by herself, first words at the age of 1 year and 1 month. At the age of 7 months, after DPT and poliomyelitis revaccination, right leg paresis occurred, outpatient treatment administered. Paresis had been regressing over 3 weeks. At the age of 8 months, after electro treatment, short "freezings" occurred that disappeared gradually by 10 months with no treatment administered. Received nootropic therapy. EEG registered generalized outbursts of paroxysmal activity in the parietal zone.

Antecedent anamnesis: at the age of 1 year and 5 months the right-sided paresis aggravated and generalized seizure with a right-side tendency developed: concomitant salivation, mental confusion for up to 15 minutes. Hospitalized to a Child Infection Clinic, received phenobarbital. At the same tine simple motor focal right-sided seizures occurred; no loss of consciousness. Outpatient treatment, phenobarbital and lamictal prescribed. On the background of seizure frequency increase the right-sided paresis manifestations aggravated, ataxia occurred. Natrium valproate (syrup) was added, but because of the excessive dose and nausea and vomiting apparition it was discontinued. At the age of 1 year and 7 months right leg jerks appeared: series course and occurred during sleep and upon wake-up. Calcium valproate and clonazepam were prescribed. Over 2 weeks these seizures reduced, but absences appeared. At the age of 1 year and 9 months two atonic seizures occurred, right leg paresis aggravated, ataxia developed. Intoxication reoccurred at administration of natrium valproate up to 1200 mg/day and clonazepam 3 mg/day.

By the age of 2 intoxication manifestations persisted (nausea, fatigue, etc.) absence status developed. Ataxia occurring had been growing on. Repetitive hospitalization to Child Infection Clinic, depakine dose reduced to 600 mg/day, clonazepam discontinued. Girl's somatic state improved, absence status persisted ("froze constantly").

By the age of 2 years and 3 months absence seizures were fortified by frequent simple focal motor right-sided seizures, right-sided paresis and ataxia occurring had been growing on. Along with neurological conditions new psychological disruptions developed. Fatigue, lethargy, sleepiness as symptoms of a psychological retardation became evident. Received depakine 150-150-300 mg up to 900 mg/day, succinimid 250 mg/day. Absence course was of a status type. Natrium valproate dosed 600 mg/day + carbamazepin/retarded release = seizures persisted. Motor and psychological development retardation was in progress.

By the age of 3 topamax 200 mg/day + natrium valproate 600 mg a day produced no effect.

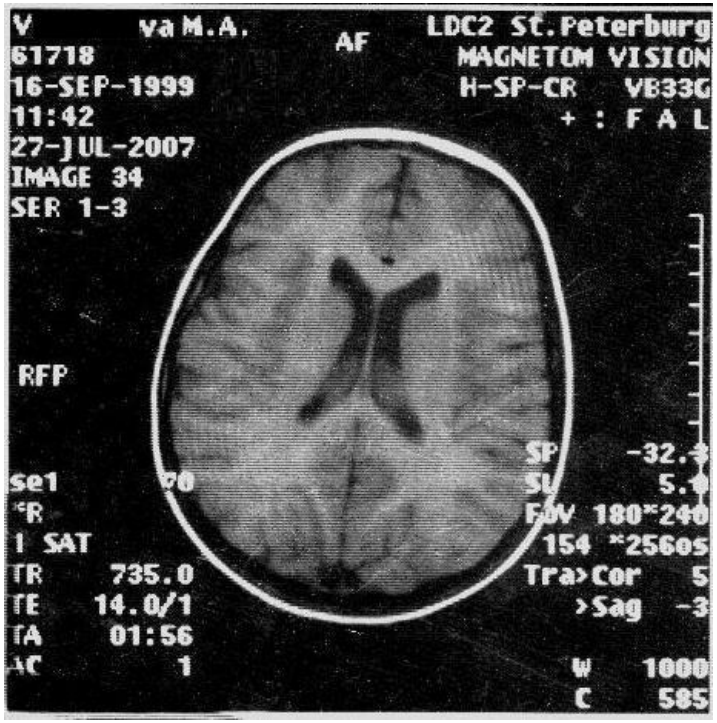
In Feb 2003 (3 years, 8 months) the authors started curation. Topamax was replaced with finlepsin 250 mg/day + hydantoin 125 mg/day, the seizures reduced up to complete halt over a week. Over the next month ataxia manifestations stopped (better walking, speech development).

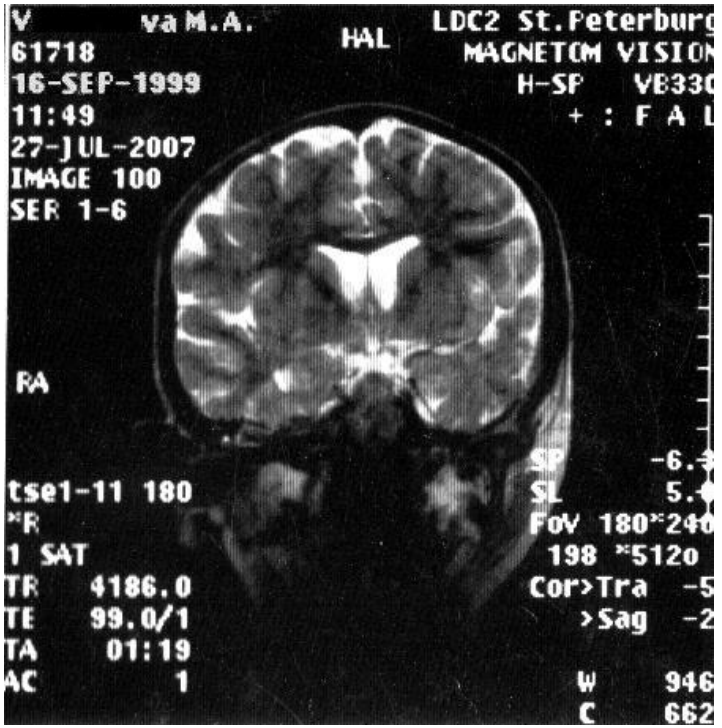
Since 2006 (6 years) switched to natrium valproate 300 mg/day, lamictal 100 mg/day.

At present: depakine chrono 450 mg/day, lamictal 100 mg/day.

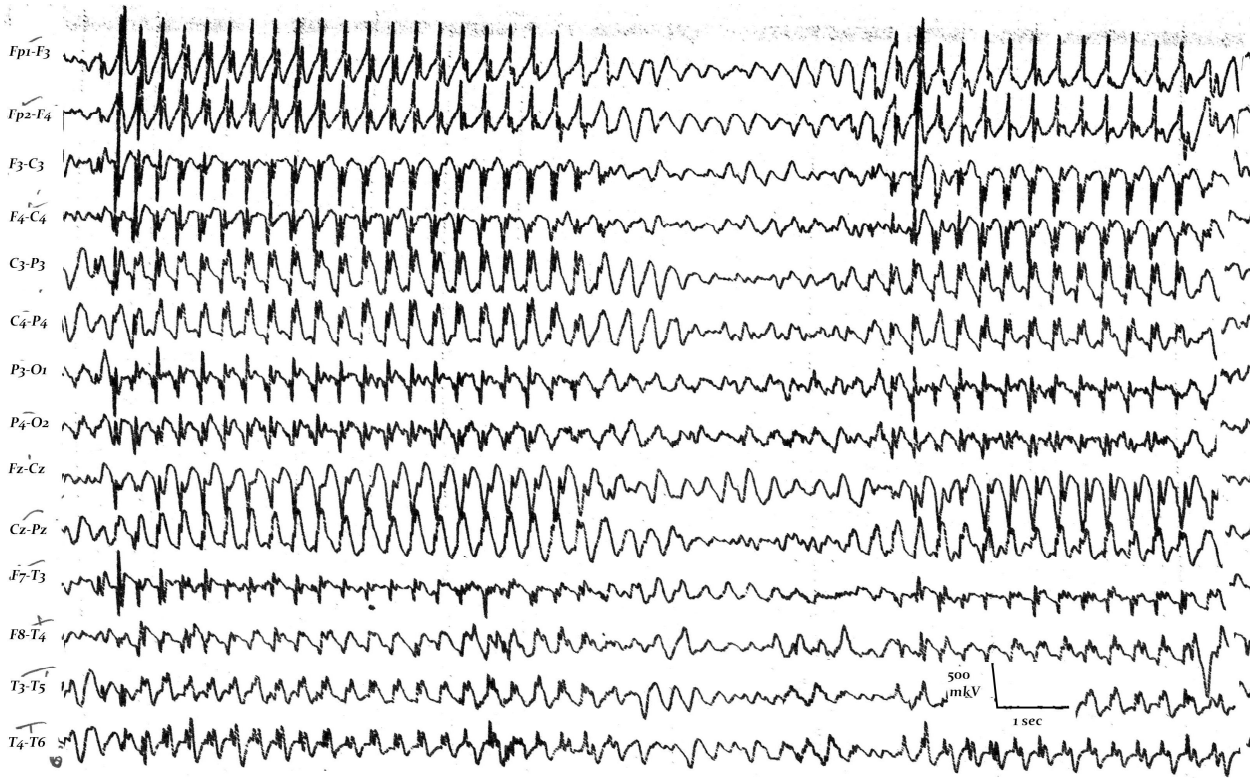
Brain MRT (at the age of 3) – MR image of encephalopathy, mixed hydrocephaly with preponderance of the inner.

Diagnosis: Epilepsy - focal cortical, frontal-temporal, generally idiopathic, status course of atypical frontal absences and simple focal motor seizures.

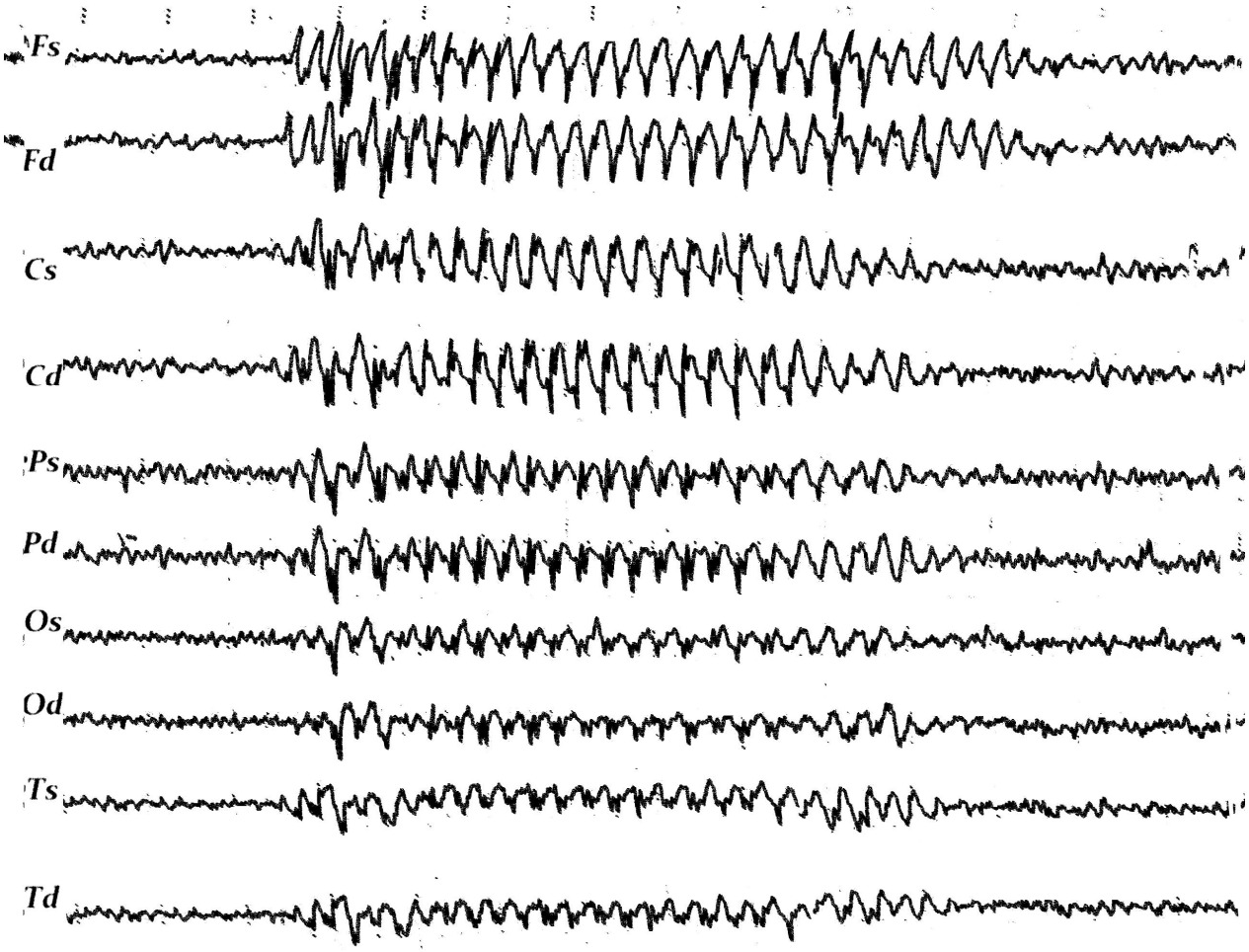




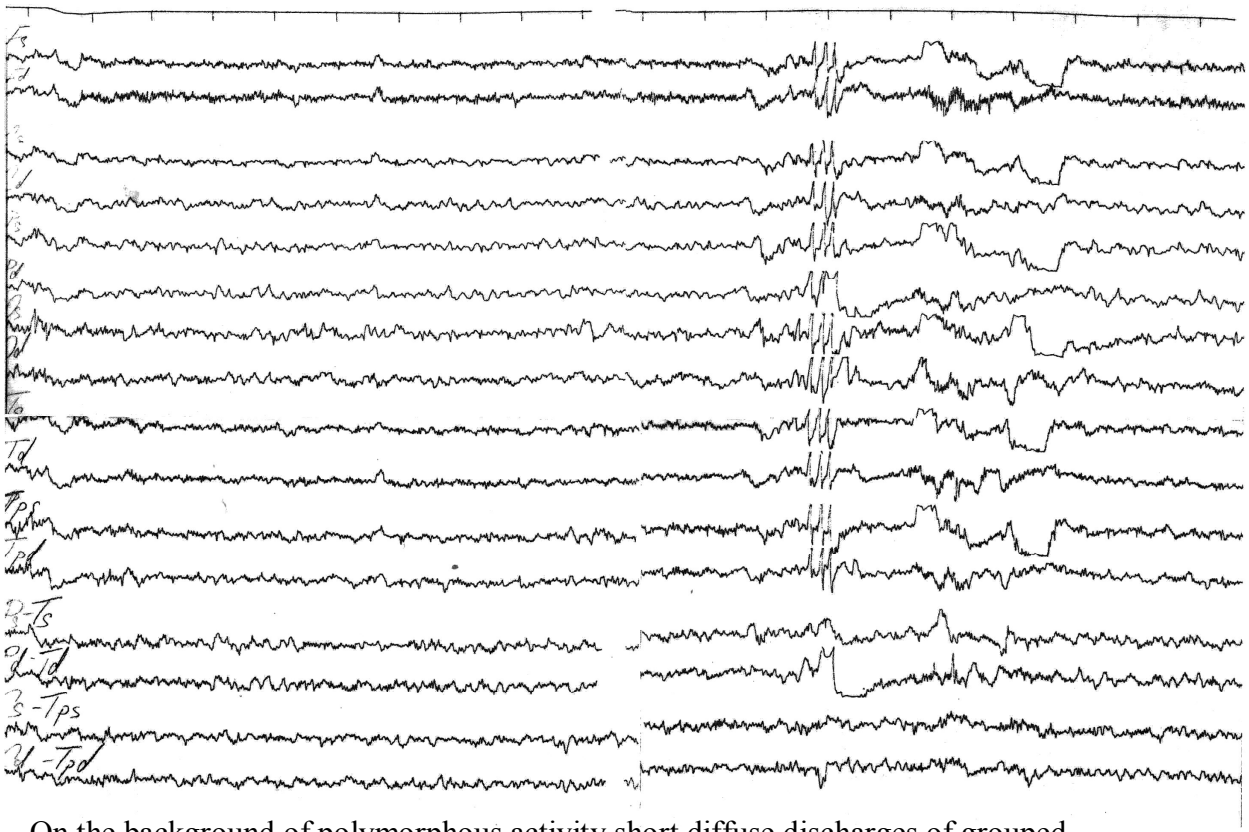
See explanations below



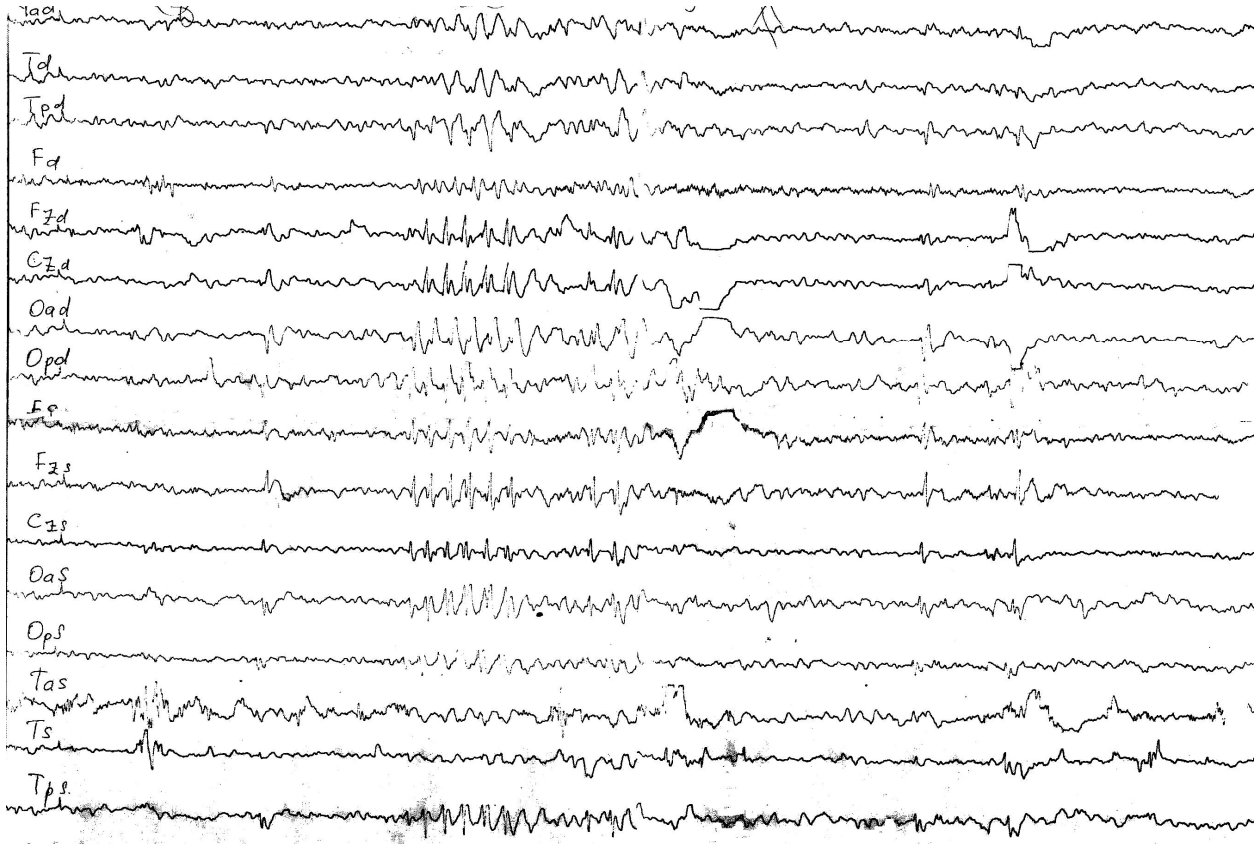
Grouped diffuse discharges of slow wave/spike complexes with left-sided lateralization in lobe-lobetemporal leads, frequency 2.5-3/sec, duration 7 -10-14 sec. Age 1 year, 5 months.



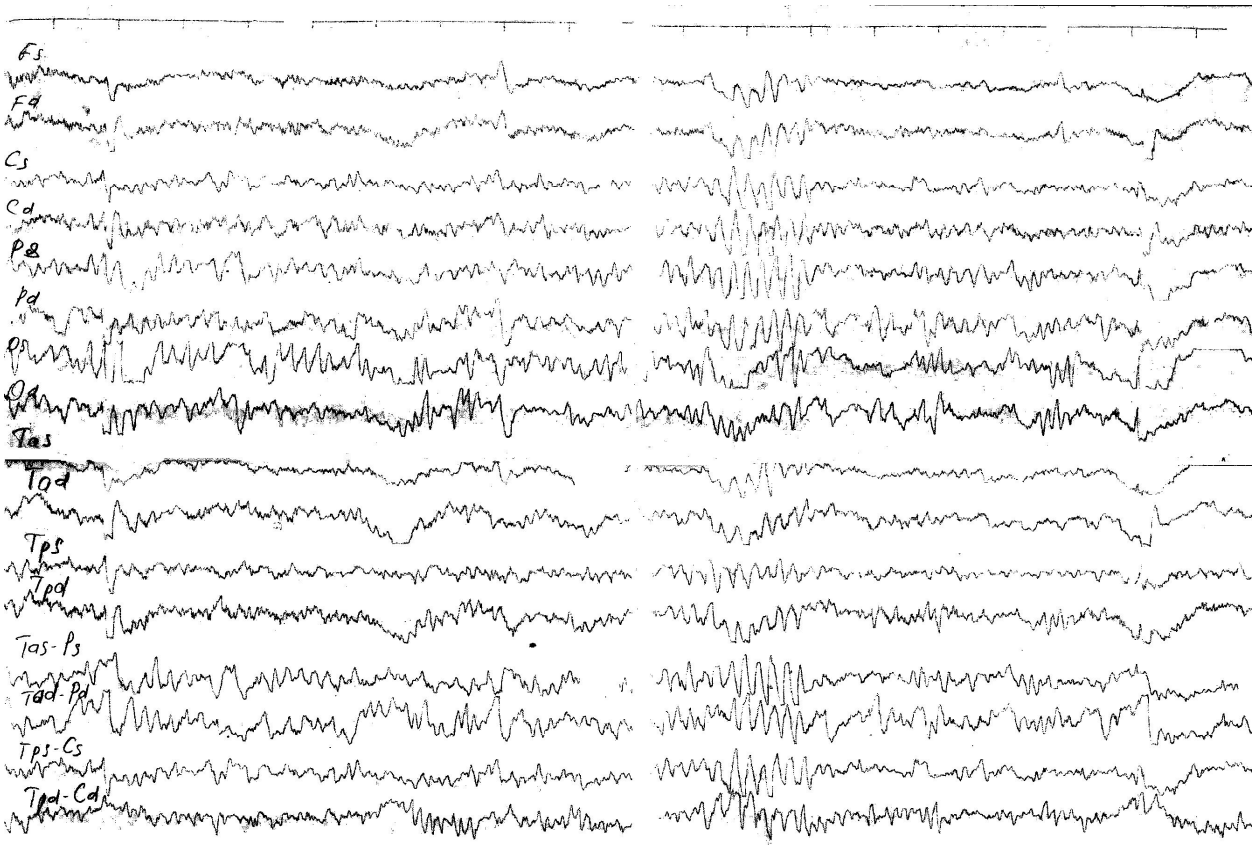
Grouped diffuse discharges of slow wave/spike complexes with left-side lateralization in frontal-temporal zones; frequency 2.5-3 /sec, duration up to 7 sec.
2 years old.



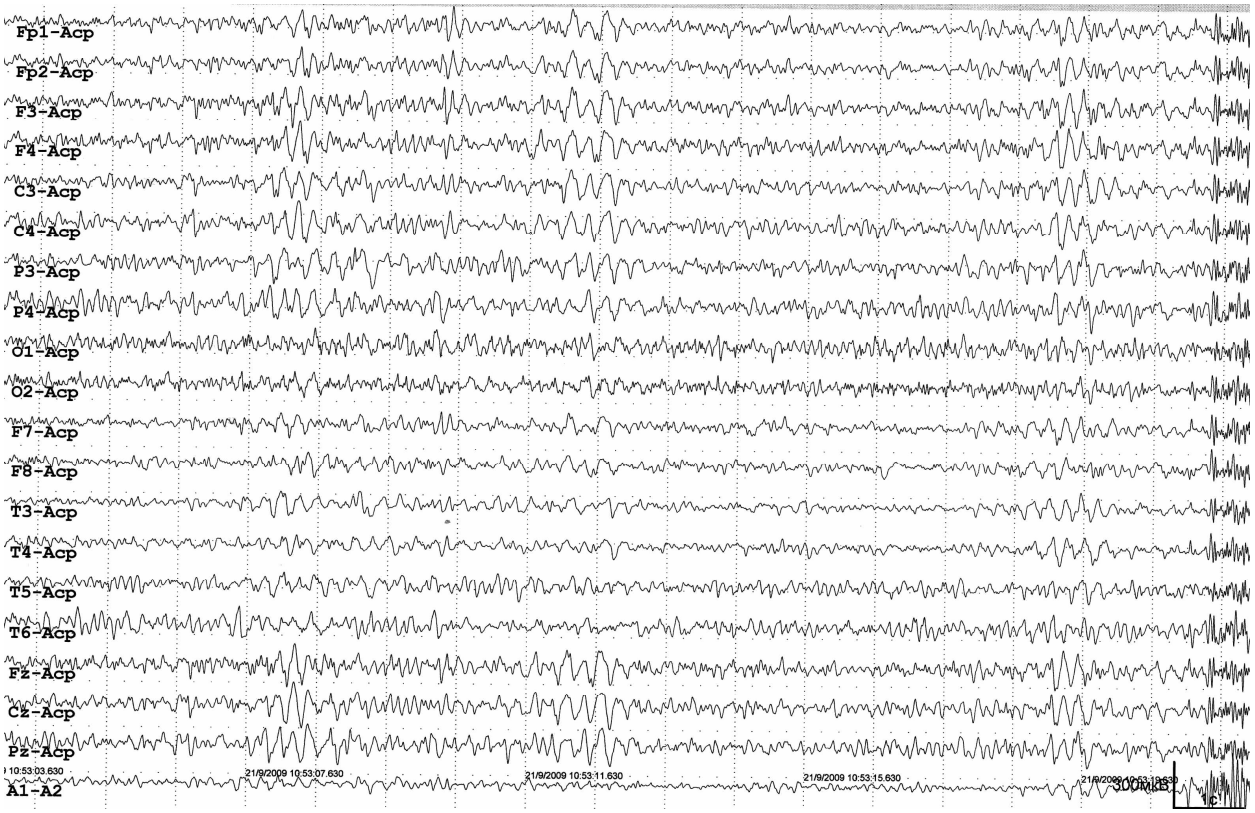
On the background of polymorphous activity short diffuse discharges of grouped reduced slow wave/spike complexes of a left-sided lateralization in the frontal-temporal zone. 4 years old.



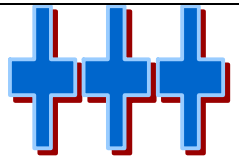
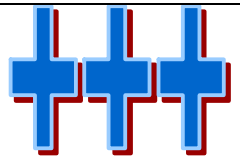
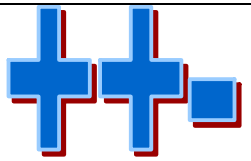
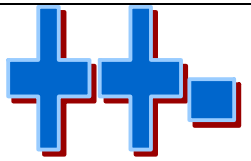
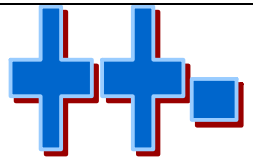
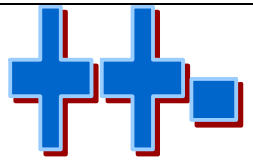
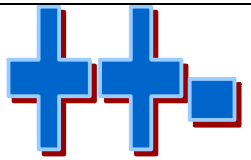
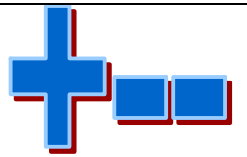
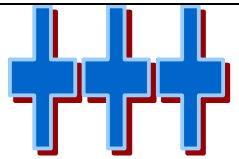
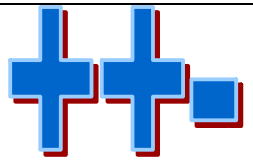
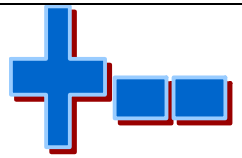
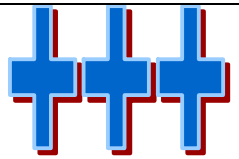
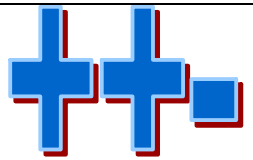
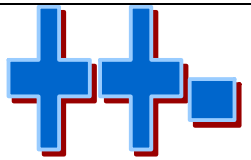
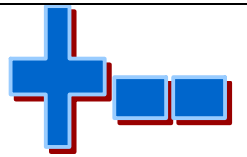
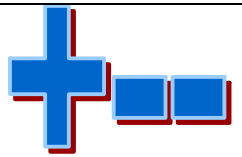
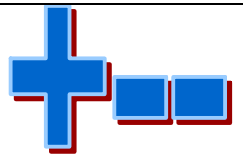
On the background of polymorphous activity diffuse discharges of grouped reduced slow wave/spike complexes of a left-sided lateralization in the frontal-temporal zone.
(6 y.o.)



On the background of polymorphous activity diffuse outbursts of grouped high-amplitude wave of teta range (5 Hz) without a definite lateralization.
(8 y.o.)



On the background of polymorphous activity diffuse outbursts of grouped high-amplitude wave of a teta range (4-5 Hz) without a definite lateralization.
(11 y.o.)

Index	1 month	1 year	3 years	5 years
Intellect structure irregularity (WISK, norm about 10)	 4-6	 4-6	 7-8	 5-10
Random otic attention concentration				
Deficiency of visual-spatial analysis and synthesis				
Immaturity of autoregulation mechanisms and cognition activity				
Comprehension of logical temporal connections, level of accessible generalization				

+ + + - max intensity; + + - - average intensity; + - - - min intensity

- - - - no symptom

Conclusion: concomitant brain organic lesion in case of atypical frontal absences affects severity of psychological disruption at the beginning of the rehabilitation process but does not affect the rehabilitation quality which may imply that there is no correlation between ontogenesis recovery and a child's brain organic lesion upon compensation of the atypical frontal absences.