Complex psychodynamic and bio-psychological approaches to autism and autistic disorders (a study)

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Abstract:

I do not mention the contemporary pharmacology and our new opportunities since this will be topic of the rest of this tender material. Up to the present day the most accepted and most effective therapy is the cooperation of child psychiatry, clinical child psychology and the specialized special education teaching. There is not even a fully compensating therapy in existence as there is one with adulthood psychosis or psychopathia.
KEYWORDS: special autisms, Kanner, Asperger, Rett, clinical studies, clinical model

It is common to address autistic disorder as "pervasive" or "comprehensive" ontogenetic disorders because they effect all areas of adolescent psychological development negatively. But as the expression itself suggests we are not aware of the specific disorders directly. As it will turn out we cannot deal with autism as on complex disorder, we should rather use the term in plural i.e. autisms and autistic disorders.[1] [2]

E.g. the Asperger-syndrome (AS) has been in use as diagnostic criterion since 1944; it was named as autistic psychopathia then. It only got the name Asperger-syndrome referring to the first diagnostician from Lorna Wing in 1981. After all it was only involved in the official system of DSM-IV [3] [4]as a unique syndrome in 1994 and it is still much debated etiologically and from other aspects.

[1] See: NELSON: Nelson textbook of pediatrics / editor, Richard E. Behrman; Robert M. Kliegman, Valdo E. Nelson, Victor C. Vaughan, Harcourt Brace & Company, Philadelphia, 2002. (First Hungarian Edition, 1995. Budapest, Melánia Kiadó; pp.72-73.)

[2] See: The Merck Manual – Diagnosis and Therapy (MSD ORVOSI KÉZIKÖNYV Diagnózis és terápia) ; Editor in Chief: Robert Berkow, M.D (Melánia Kiadó, Budapest 1994.) ; pp. 2267-2268.

[3] See: DSM-IV. TEXTREVISION (Animula Kiadó, Budapest, 1999.) ; pp. 41-69.

[4] See: DSM idem

It is interesting and its importance will be emphasized later that boy: girl occurrence rate is 6 : 1[5], and it does not show symptoms in early childhood while other autisms do so. Its real occurrence is frequently met after the age of six. It is considered as child psychopathia by clinical child psychologists even today and no etiologic relation is seen between psychotic and neurodegenerative autisms. The fact that the IQ of children with AS is usually outstandingly high – often exceeds the value 140 – further supports this idea. We are going to point out that the classification spread in the '90s was totally wrong by saying that this is a mild form of autisms[6] [7] as opposed to the seriously retarded Kanner-syndrome which was considered as a severe form of autism in this old classification. After 1994 we cannot even talk about such a division.

It is much debated whether it exclusively hits boys. There are many arguments which say that we tend to pay more attention to boy gender samples[8], especially when they show extreme intellectual capacity. Though the above mentioned prevalence rate is generally accepted: it is unquestionable as for the contemporary statistical data. Its frequency is 2/10000 (though as we know the prevalence of every autistic clinical aspect has dramatically grown in the last 20 years, these are also official data from 1995 further on)[9].

If we take a look at the latest psychological function theory of AS (Baron-Cohen EQ – SQ theory)[10] the male –female rate proves even more to be right, functionally too. This is because besides the extremely low EQ and extremely high IQ and SQ AS strengthens the convergent comprehensive function so it results in a high organizational intelligence and in a lower emotional intelligence adversely. So the convergent male-hemisphere function is extremely dominant and the divergent feminine functions are not on only incapable of communication with the convergent functions but they are behind[11].

If we look at the above described model form the aspect of psychodynamics including emotionality it seems to be easy to establish a psychoactive model as for which there is a psychopathic scission between the systematic and unsystematic cognitive, emotional and personality-structure-functions. This gives a good example for the more and more accepted

[5] Wing, L. (1981) Sex ratios in early childhood autism and related conditions. Psychiatry Research, 5, pp. 129-137.
[6] See: DSM idem
[7] See: NELSON idem
[8] Source: http://www.aspergerfoundation.org.uk/infosheets/ta_girls.pdf
The Pattern of Abilities and Development of Girls with Asperger's Syndrome, Sept. 1999
[9] See: NELSON idem
[10] SEE : This paper appered in Tager Flusberg, H, (ed) Neurodevelopmental Disorders MIT Press, 1999. The extreme-male-brain theory of autism ; Simon Baron-Cohen ; Depertments of Experimental Psychology and Psychiatry, University of Cambridge
[11] SEE idem.

model which say that children with AS are likely to become adolescents or adults with borderline personality disorder.[12] [13]

We do not yet hold credible statistical data about this, but more and more psychodynamic system states that there is successive relation between childhood psychopathia with AS and adulthood psychopathic pathographies especially borderline syndrome.[14] It would be a mistake though to apply the so called "minified adult model"; AS is a unique pathography of psychopathia.[15] Its drug therapy has not been worked out even in theory, it will also be discussed later here.

The zero-LORETA 3D EEG examination method[16] could serve as a brand new evolutionist examination, which is much better in localization than any other common EEG-asymmetria examination. fMRI examination that is even better in localizing this function and the corpus callosum MRI on big population can gain great significance; we would like to integrate it by the FDG-PET-MRI examination.

The old and outdated misbelief which says that the childhood autism is a special relapse (shub) of adulthood schizophrenia is absurd and has never even been documented. It is quite obvious that the childhood psychotic autisms (low IQ – often under 75, perinatal neopathia possible) often show a different pathography compared to childhood schizophrenia (relatively high IQ, no perinatal neopathia)[17].This can be well distinguished Psycho-pathologically and dynamically from autistic-psychopathia or from borderline syndromes evolving in late childhood[18] which are basically psychopathic with occasional psychotic relapses (shub) as described by modern etiologic models.

In all the DSM classifications traditionally Kanner Syndrome, which we have known since 1943, counts as the childhood psychotic autism[19]. It is such a standard autism that its discoverer's name is not even noted by the DSM-IV[20] but we will see that it is only a type of

[12] SEE: Infant pain management: a developmental neurobiological approach.Fitzgerald M, Walker SM.Nat Clin Pract Neurol. 2009 Jan;5(1):35-50.PMID: 19129789 [PubMed - in process]
[13] SEE: Otto F. Kernberg: Borderline szindróma és patológiás nárcizmus (Az Autizmus Alapítvány KAPOCS Kiadója, Budapest, 1990.) (Otto F. Kernberg M.D. Borderline Conditions and Pathological Narcissism, Jason Aronson Inc.New York,1957.) ; pp.7-47.
[14] SEE idem.
[15] SEE: DSM idem.
[16] SEE: appliedneuroscience.com
[17] SEE: NELSON idem.
[18] SEE: idem.
[19] SEE: idem.
[20] SEE: DSM idem.

severe autistic disorder. Mental defectiveness is 100% in this case, IQ is under 75 and the pervasive disorder can be recognized before the age of three in communication, social behavior and in flexible thinking. Patients often become echolalic, are incapable of communication even on motor level and the stereotypic behavior in psychomotorics. They cannot recognize the personal pronouns and possessive pronouns or invariably repeat them; meaningless, ritual behavior patterns are often. The syndrome's incidence 3-4/10000 and it is increasing, but the most interesting fact is that the girl : boy rate is 3-4:1[21]. Unambiguous fallback can be recognized in the above mentioned areas compared to healthy coevals before the age of three while they produce the abovementioned pathologic symptoms. This fact will be of greater importance later in our methodic.

Unambiguous neurodegenerative reasons for the Kanner Syndrome are unknown[22].
On the other hand the Rett Syindrome which is recently commonly classified as a type of autistic disorder has an unknown etiology[23] but it is unambiguously a neurodegenerative disease, it only appears with girls and its incidence is 1:15000[24]. Growth seems to be normal until the age of one then the speech and motor skills show a fallback and microcephalia appears. This can cause brain-stem ataxia and minor hand tremor in its early state. Most patients suffering the syndrome produce sigh like breathing with intermitting apneic periods which are accompanied by cyanosis. The stereotypical hand fumbling is very common the spontaneous and aimed hand movement disappears. Interestingly this symptom does not evolve before the age of 2 or 3. Strong epileptiform generalized tonal-clonal seizures emerge at most of the patients in the early period. These can be well treated by antiepileptic drugs though dystrophy and defective weight gain can emerge. The autistic behavior is general which proves to be curious because the initial severe neurologic pathography, which decays, is accompanied by a permanent psychiatric pathography[25].

Today we know that those children who suffer Kanner – or Rett syndromes become mentally retarded people with defective social skills in their adulthood. We also know that development therapies in childhood or adolescence may evoke improvement; thus the lack of such therapies or their application make a big difference in patients lives. Today the

[21] SEE: NELSON idem.
[22] SEE: idem.
[23] SEE: NELSON pp.1528-1529.
[24] SEE: idem.
[25] SEE: NELSON idem.

progression cannot be valuably changed by drug application (this will be detailed later on) but the patients do not develop schizoid personality structures their autism become standardized[26].

It is quite curious to decide what to consider the Rett Syndrome on the basis of classification. The Nelson Textbook of Pediatrix classifies it as "Neurodegenerative disease of various reason"[27]while DSM-IV classifies it as autism or autistic pathography[28]. Clinical child psychologists have only started to discover this syndrome in their field.
If we typically look at the common or at least psycho-dynamically analogous symptoms, which make it possible at all to consider autisms here, then we can create different dynamic categories:

A: Emotional autism

With the Asperger Syindrome this is shown in the psychopathic scission between systematicness and unsystematicness i.e. a typically convergence dominant emotional function phenomenon.
All this is severely diverged with the Kanner Syndrome i.e. it rejects every convergent function i.e. echolalia, stereotypic psycho motor, defective use of personal pronouns.
This pathological divergence appears as neurodegeneration with the Rett Syndrome patients; this is interesting also because, we do not know what it was before.

B: Cognitive autism

It is obvious here that the cause is convergence predominance with Asperger patients while Kanner and Rett patients functionally incapable of basic convergences like complex speech, aimed communication motivated movement etc.

C: Interactive Deficit autism

This is obviously A&B

[26] SEE: idem.
[27] SEE: idem.
[28] SEE: DSM idem.

D: Excited autism

It is implied that with Asperger patients the psychopathic emotional range of systematicness - unsystematicness scission can be observed which can later manifest in various acting-out behavior. It must not be confused with adulthood psychopathia not even in methodical sense. It is more common here that the psychopathic excitation is covered by the emotional bizarreness of the extreme intellect.

With the Kanner Syndrome the excitation can be considered as a negative stereotypic psychotic aggression seizure, though we know very little about its etiology because the psychotic status of retardation as opposed to the numerous life story accounts recorded with aspergeroid patients[29].

Excitation with the Rett Syndrome is neurologically initiated which becomes a chronic psychotic autistic standard status; see above. The emotional and intellectual excitation of both the Kanner and the Rett syndromes show a very divergent feature, in certain cases the convergent intellectual functions cannot even be recognized.

E: Stereotypic autism

See: C&D

Our researchers' discovery

We have said that this is a convergence-divergence balance failure, dominantly "boy-brain breakage" with the Asperger Syndrome, and we have proved psycho-dynamically that the Kanner and even more the Rett syndromes are divergent "girl-brain breakage" or at least they show such pathological brain hemisphere dominance. This is why we suggest applying the above mentioned diagnostic imaging and electrophysiological examinations here as well; results are predicted to be analogous but adverse in meaning. If possible it is worth to expand EQ and SQ tests along with the IQ test and to apply complex convergence-divergence tests, though there seems to be not much chance for this except for with AS patients; not counting the standardized, approved and generally applied IQ tests.

From genetic aspect we know that autisms and autistic syndromes often overlap with the so called "X-Fragile syndrome" (Martin-Bell Syndrome)[30] [31] the genetic etiology of which we do

[29] http://livewithit.blog.hu/2008/08/30/az_aspi_olyan_mint_a_hagyma
[30] SEE: NELSON idem.

not know. This scientific case is even more absurd because we cannot undoubtedly identify it with autistic syndromes. We only know that there is such a parallelism, but this is a blurry fiel both in genetics and child psychiatry, more to that we do not know the exact dynamics of these syndromes in psychology. Neither do we know the genetic origin of premutational X-fragile cases[32] or their etiology. We have worked out a specific examination method in our human research plan which can unambiguously falsify the above mentioned phenomena, problems.

We basically know that X chromosome disorders can cause hypothalamus laesio and since the time of modern diagnostic imaging we also aware that severe hypothalamus laesio[33] and corpus callosum laesio[34] can be identified with many childhood autism patients. We deal with the relation of these in another chapter.
We can talk about a comprehensive autism research along these complex analogy systems from genetics to psychology. Thought these are scientific analogies within which we can build up a complex research by the above mentioned etiologic, progressive diversification of disorder differences within pervasiveness; with the unique and independent discussion of each specificity.

I do not mention the contemporary pharmacology and our new opportunities since this will be topic of the rest of this tender material. Up to the present day the most accepted and most effective therapy is the cooperation of child psychiatry, clinical child psychology and the specialized special education teaching. There is not even a fully compensating therapy in existence as there is one with adulthood psychosis or psychopathia.

The aim of this article and research is to develop such a biologic psychiatric therapy.[35]
It is clear that complex developing therapy cannot be neglected later either.

[31] SEE: http://www.medterms.com/script/main/art.asp?articlekey=5650
[32] We have 18 family members for research reasons
[33] SEE: www.gyogyinfok.hu/magyar/fekvo/hbcs50/torzsek/BNOTORZS_20060701.xls -
[34] SEE: Bibby, Celia M. Autism: A Conditioned Response to Biochemical Toxicity?
[35] SEE: Lehel Simon: Tender fo the Development of a New Autism Drug

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