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Contribution

Psychopathology and psychological semiology of autism. Hypotheses about psychological rhythms and autistic development.

Summary:

As a psychiatrist, I have carried out parallel clinical evaluations of people presenting with a pervasive developmental disorder (PDD) over the last 12 years. This has involved around 100 people, of whom half presented with an intellectual disability or mental impairment (mental retardation ranging from mild, severe to profound), often without verbal language abilities. These were clinically severe situations.

The symptoms of autism are now well known (communication difficulties, limited interests, difficulty in socializing) (1). However, I realized how hard it is to connect the various symptoms described. This aspect can affect the clinical course and development, particularly when it comes to evaluating the efficacy of patients' treatment.

The current wide recognition of a neurodevelopmental cause of autism has enabled me to establish the hypotheses that I outline below. Clinically, these hypotheses aim to touch on the psychopathology of autism that, in my view, still seems unknown. My approach seeks to encourage interest in the general treatment of adults with autism and help train a new generation of therapists.

Introduction:

Scientific literature describes the main symptoms of autism without going into detail on the psychopathology (2). I have performed a lot of research, and continue to do so. Why is this so important? It can have a concrete impact, for example on evaluating the efficacy of a treatment. This evaluation may be carried out indirectly using tools such as the ABC (Aberrant Behavior Checklist (3) that is widely used with autism, giving quantitative evaluation of irritability, hyperactivity or rather psychomotor agitation, social withdrawal, inappropriate speech, stereotypic behavior). However, this scale can be too descriptive. It is then a question of explaining the implications of the results for aspects of each autistic person's daily life to relatives. I propose a quick schedule assessment (Table 1) on the base of general principle of assessment of the severity of the symptoms.

	Table 1	Rapid Autism Severity Schedule (RASS)(*)	
		Severity Score Features	Score
Level 1	Common autism symptoms without troubled adaptative functioning		1
Level 2	like level 1, but adaptative functioning moderate troubled		2
Level 3	Risk severely of disruptive behavior (Self-Injury, Aggression)		3
Exemple 1	Physical ma	nnifestations :	
Level 1	Searching social worker to show a choice, food, objects		
Level 2	Keep on disruptive behavior with moderate troubled adaptative functioning		
Level 3	Grabs at clothes and to pinch injury others		3
Exemple 2	Verbal beha	aviors :	
Level 1	Vocalize without troubled adaptative functioning		1
Level 2	Making lou	d noises	2
Lebel 3	Screams rep	petead	3
	(*) Mario Navarro Chafloque, psychiatrist, Lausanne, Switzerland-2018		

Understanding the psychopathology would not just enable the medical profession to assess the patient's development since birth (history), it would also help to answer specific questions in the clinical development. It would facilitate comparison with the family's or colleagues' knowledge of the autistic person. This permanent dialog has enabled me to develop hypotheses and find a clinical equivalent in practice.

My approach is now part of the psychopathology of autistic manifestations. This constitutes an effort towards clinical understanding of manifestations in people with an autistic disorder or a pervasive developmental disorder. More specifically, the manifestations involve a sub-section of the population that cannot give clear indications of their malaise because of significant (mild to profound) intellectual impairment and a complete lack of verbal language. These types of situations make psychological clinics (4) very difficult for healthcare professionals, which explains the interest in facilitating clinical understanding and the work of autism specialists.

Scientific research could give a more exact response on the relevance or otherwise of these clinical hypotheses.

Hypotheses:

I therefore put forward the hypothesis that there are neurobiological rhythms in the clinical development of the symptoms of autism. The neurobiological rhythm would consist of changes to or a deterioration in the symptoms of autism when there is no clear a priori causal factor (Stress) at the time.

We therefore see elation, autoagressive or heteroagressive behavioral issues, more verbal stereotypies, more psychomotor agitation crises or conversely psychomotor inhibition or "catatonia", a deterioration in social withdrawal.

I have also observed a rise in repetitive behavioral problems, and the list is a long one. For example, (loud, shrill) screams, tears, grunting, rocking, striding back and forth across a house, the autistic person running away, refusal to take part in day-to-day activities, deterioration in rituals (drinking too much coffee, opening water taps, knocking over objects, breaking objects, throwing objects, tearing clothes, etc.), less concentration, lower attention span, etc. Clinical manifestations can disrupt the relative autonomy achieved: the person cannot retain and understand simple information from people around them, the ability to use toilets may be limited or basic personal hygiene may deteriorate.

Hypotheses on the connections between the main symptoms of autism:

I think that there is a destructuration of the autistic person's thoughts. This situation would lead to a loss of basic psychological reference points, such as orientation in time (if the person has this ability), space (for example, the person starts pacing up and down or running away) or context (the person no longer

knows whether it's time for a meal or to go to a workshop or to chase all passing vehicles on surrounding roads). All this disorientation would cause heightened repetitive autistic behaviors, resulting in a significant decline in the autistic person's participation in everyday life. Other issues can arise, such as anxiety, depression, obsessive-compulsive disorder (OCD), etc.

My clinical hypotheses form part of the reflection and results of scientific research into the neurodevelopmental origin of autism. I therefore believe that faulty connections in the development of cerebral neuronal migration (5) lead to retardation in neural communication, translating into a lack of language development or inability to inhibit repetitive behaviors (stereotypies) in the prefrontal cortex (6) and excessive functioning of some regions of the brain. This hyperfunctioning can lead to highly developed skills (people who are gifted mathematicians, for example) because there is no compensation or investment of other cerebral functions, or these will be very defective because of an imbalance of neural or axonal connections in the brain.

Another consequence might be sensory hyperexcitability due to a lack of balanced sensory regulation, for example in the skin or an absence of pain or rather a significant increase in the pain threshold that seems to be an absence of pain. Or retardation in expressing emotions about a life-changing event (Stress) that gives the false impression in the moment that the autistic person does not show emotion or have emotional capacity, which is completely wrong. Emotions are expressed with a time lag, with the impression that their appearance is delayed. The time lag of this emotional expression can range from some days to some weeks. This phenomenon can be understood as an emotional "backlash".

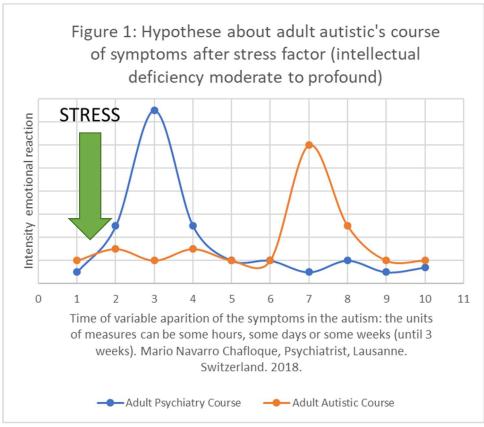


Figure 1. Diagram of a psychopathological model of the apparition of the emotional reaction in the adult autism with an important intellectual deficiency. Exposed to the stress, the curve of the course in adult general psychiatry show symptoms rather immediately. However, in the adult autism with an important intellectual deficiency, stress exposition provoked an emotional reaction curve to occur too late", backlash ", having the false impression that the emotion doesn't exist.

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