

The Structural Dynamics of Autism

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Abstract - Autism is considered here as a set of regulations necessary for the structural stability of the self — a psychic organization that emerges from neuronal complexity. The cohesion of the self is undermined by an alteration in the constitution of mental objects unified through intentionality (objective synthesis). The failure of structural stability in the autistic self in turn produces a negative semiology (regulatory disturbance, dissociation, functional disorganization). A new typology of autistic signs, built on their regulatory functions, is proposed (in seven classes), and the outlines of a phenomenological psychotherapy, centered on understanding the autistic relation to the world, are sketched.

Translated from the French original, "Dynamique structurelle de l'autisme" (www.benoitvirole.fr)

Introduction

Research on autism requires scientific approaches grounded in standardized protocols and statistical evidence. It is enriched by clinical monographs and first-person accounts of experience. But it also requires the construction of models. This essay belongs to the latter category. It proposes a theory of autism built on the following axiom : autism spectrum disorders (ASD) are forms of regulation necessary for the structural stability of the self — a psychic agency emergent from neuronal inter-connectivity — whose cohesion is undermined by an alteration in the constitution of mental objects unified through intentionality (objective synthesis). Given its aim and format, this essay is not concerned with refutations, demonstrations, examples, or the establishment of proof. Its purpose is to construct a theory that renders autism intelligible while meeting the criteria of concision, coherence, and adequacy to the facts.

Our theory of autism is built upon four classes of facts, corresponding to the main sections of this essay.

The first concerns the difficulties psychoanalysis has faced in constructing a complete theory of autism. Its presuppositions regarding a primitive mother-infant symbiosis, and its conflation of the construction of object relations (affective bonds to persons) with that of objectivity (relation to the physical world), account for this relative failure. The second class concerns the findings of neurobiology, which have led to autism being considered a neurodevelopmental disorder ; we shall present these fundamental — though not decisive — findings synthetically, insofar as neurobiology has not succeeded in proposing a unified interpretation of autism. The third class of facts derives from complexity science. The development of neuromimetic models, connectionism, and models of self-organization has opened new ways of approaching the complexity of neurobiological organization. Neuronal interconnection and the multiple loops of neuroregulation (dopamine, serotonin, noradrenaline) evoke a complex dynamical system endowed with properties such as emergence, self-organization, and an attractor structure. The self, a mental agency emergent from this complexity, will be treated as the appropriate level for understanding autism. The fourth class of facts concerns the possibility, opened up by work in cognitive science, of understanding the constitution

of mental objects from perceptual data. The dualist notion of mental representation is an obstacle to understanding the link between the physical world and the mental world. Thanks to cognitive science, and to its reinterpretation of intentionality, it has become possible to follow, step by step, the process of reception, coding, and integration of physical objects up to the synthesis of mental objects. Objective synthesis through intentionality allows for the constitution of mental objects, generates semiotic prototypes, and constitutes the cognitive basis of intersubjectivity. We will ultimately define autism as a modality of structural stability of the self, arising from a specific dynamic of objective synthesis modified by neurodevelopmental singularities[1]. The final section sketches the foundations of a phenomenological psychotherapy of autism.

Defining Autism

Historically, descriptions of autism begin with the work of Leo Kanner and, subsequently, Hans Asperger[2]. Current international terminology considers autism a neurodevelopmental disorder belonging to a broad spectrum (Autism Spectrum Disorder, ASD). The most recent nosographic system enabling scientific communication among researchers, the DSM-5, characterizes ASD by a semiological triad comprising (DSM-5, 2013) : (1) persistent deficits in communication; (2) an inability to develop and maintain appropriate social relationships; (3) restricted, repetitive behaviors, accompanied by stereotypies and unusual sensory behaviors (see Table 1). All autistic individuals present these similar manifestations, allowing for consensual identification among observers. Yet their expression borders on the idiosyncratic — one subject, one unique semiology. We regard this unity/diversity paradox as a fundamental fact. Relative to DSM-IV, the DSM-5 broadened the inclusion criteria for ASD by incorporating Asperger's syndrome, excluding Rett syndrome, and widening the criteria for membership in the category. This extension increases the number of children falling within ASD to a prevalence rate approaching 1% of the general population (Fombonne, 2007), or even 1 in 88 (Constantino et al., 2010). This extension is problematic : it tends to include, within a single category,

clinical profiles as different as a self-injuring autistic child, locked into stereotypies and refusing all human contact, and an adult who has founded a high-technology company, capable of mentally manipulating complex data but whose affective contacts are limited or nonexistent. The inclusion of such divergent profiles within a single entity raises the question of the unity of the underlying determining process.

Contributions of Psychoanalysis

Until the end of the 1980s, psychoanalysis dominated the theoretical interpretation of autism. Beyond lexical differences among its various currents (Freudian, Kleinian, Lacanian, Kohutian. . .), psychoanalysis approached autism through the postulate of a primary disorder of psychic individuation (Mahler, 1968, 1970),[3] unfolding along two axes : that of object relations and that of the construction of the unconscious body image (see Figure 1). For Meltzer (1984), the child in utero is animated by the desire to be born in order to escape the uterine claustrum. Birth occurs within an intense aesthetic experience. The infant experiences primary anxiety arising from the gap between the surface qualities of the object and the unknown of its inner psychic qualities. Autistic children lack the possibility of projecting parts of the self into a containing object (the mother's "breast"), since neither their own self nor the object possesses interiority. The processes of projective and introjective identification cannot develop, hindering psychic development and confining the child to adhesive identification (a behavior in which the autistic child takes hold of another's hand in order to carry out an action). An attempt is made to maintain primary non-differentiation (a psychic symbiosis between mother and infant), comparable to a negative hallucinatory conduct. Because of this lack of psychic separation, the autistic child experiences actual separation from the mother as an anguishing discontinuity, felt as the tearing away of a part of the child's own body, leaving behind a persecutory black hole (Tustin, 1972). To fight against the risk of annihilation, the autistic child constructs a "psychic shell," built from autistic objects and intracorporeal identifications (Haag, 1985). The autistic object (a physical object, a toy, a fragment of a toy) is neither a symbol of lack nor

A. Persistent deficits in social communication and social interaction (all symptoms) 1. Marked deficit in the verbal and nonverbal communication used 2. Deficit in social interactions 3. Lack of social reciprocity 4. Inability to develop and maintain relationships appropriate to the developmental level with others B. Restricted, repetitive patterns of behavior, interests, or activities 1. Motor or verbal stereotypies, or unusual sensory behaviors (echolalia, repetitive use of objects, idiosyncratic phrases) 2. Excessive attachment to routines and ritualized patterns of behavior, resistance to change, repetitive questioning, distress in the face of change 3. Restricted, fixed fields of interest 4. Hyper- or hypo-reactivity to sensory input, or unusual interest in certain aspects of the environment (indifference to pain, heat, or cold, excessive sniffing or touching of objects, fascination with lights or spinning objects) C. Symptoms must be present in early childhood (though they may not become fully manifest until social demands exceed limited capacities). D. Symptoms cause clinically significant limitations in social functioning, occupational functioning, or other areas of daily life. E. These difficulties cannot be explained by intellectual disability or severe developmental delay.
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Tableau 1 – DSM-5 Diagnostic Criteria

a transitional object enabling the tolerance of absence, but rather an object that palliates the anxiety of the void generated by an unmentalized separation (Houzel, 1988). Through these intracorporeal identifications, the autistic child seeks to construct an individuated psychic envelope out of a symbiotic envelope shared with the mother (Bick, 1968; Haag, 1985). The attraction autistic children show for certain forms (folds, corners, cones, angles) evokes the folds of this symbiotic psychic envelope. This envelope is double-layered and becomes invaginated at the moment when the child, emerging from symbiosis with the mother, moves out of adhesive identification in an attempt to construct its own psychic space from a fragmented body image. The attraction to circular dynamics (whirlpools) reflects attempts to cathect a body image that has been dismantled along the lines of cleavage between sensory modalities (Meltzer, 1975).

The psychoanalytic theory of autism has been the object of several criticisms. Those concerning psychogenetic causality are the least interesting : most contemporary psychoanalysts no longer share the

psychogenetic convictions once expressed by Bruno Bettelheim. Other criticisms, not always objective, have targeted the perceived inefficacy of psychoanalysis with autistic children. Let us recall the technique : an autistic child is accompanied, once or several times a week, into a room equipped with play materials, figurines, water, modeling clay, and so on. The analyst observes, comments, and suggests activities or interpretations according to the meaning inferred from this material. The principle is one of observing a phenomenon, inducing meaning through the therapist's own mental association, analyzing the therapist's own emotional reaction, and attempting to communicate this to the subject. These inductions offer an increased intelligibility of the manifestations of autism, fostering better adaptation and, ultimately, sometimes a reduction in anxiety. An expansion of the possibilities for contact can be observed. However, these interpretive inferences in no way modify the structure of autism itself. One way of understanding the limits of psychoanalysis is to consider that the material from which it infers its theory of autism consists only of secondary products — products of

distortion — arising from a deeper dynamic to which it has no direct access, situated as it is in a space distinct from psychic reality. If the uncontested domain of psychoanalysis is that of psychic reality, the domain of autism is that of developmental reality. If psychoanalysis wishes to describe autism in its full complexity, it must at some point be able to connect with developmental reality. On this precise point, however, psychoanalysis is hampered by its own presuppositions.

The psychoanalytic conception of autism is inseparable from the notion of primary narcissism (or of an anobjectal, autoerotic, undifferentiated, fusional, symbiotic stage), whose reality has been called into question in light of the knowledge acquired concerning newborn cognition. New findings in infant developmental psychology attest to the existence of an innate, differentiated recognition of the mother[4]. The notion of primary narcissism makes it difficult to account for autistic children's behaviors, which are bound up with the construction of an objective world (the perceptual and representational integration of real elements of the physical world) and are not simply the projection of object relations (affective relations to persons as psychic beings) onto this world. Even if it is possible that the autistic child perceives the physical singularities of the world as elements of their own corporeality (Ferenczi), or projects onto them emotional or even sexual content in the Freudian sense, the physical world must nonetheless first be perceived and integrated "cognitively." Yet on this central point — the construction of objectivity, i.e., the mental construction of the objects of the world — psychoanalysis is silent. The link between the perceived world and mental representation raises the problem of the first mnemonic inscription. As Jean Laplanche writes : how, strictly speaking, could pure perception already supply signs ? If it is merely perception, this at best supplies only indices ; and if these were merely indices — purely factual traces, residues without semiological intentionality — how could they be offered up for a first translation by the subject ?[5]

This problem is a consequence of preconceptions regarding the separate representation of the "thing," or, in its structural (Lacanian) version, the opposition

between signifier and signified. It treats as equivalent the construction of object relations (the psychic representation of the other, or of the drive-cathected partial object) and the construction of objectivity (the construction of mental objects from perceptual data). Yet in order to cathect an object with drive-energy, that object must first be constructed. Psychoanalysis's difficulties in describing the fundamentals of autism stem from this conflation between the construction of objectivity and that of object relations, and from its incapacity to conceptualize the intermediate levels — the dynamic interfaces — between psychic reality and neurophysiological reality.

Contributions and Limits

In conclusion, psychoanalysis has contributed to a better understanding of the affective life of autistic children who, like all children, experience suffering and pleasure, have desires and fears, an infantile sexuality, and a need for love and security. It has achieved a description of the emotional states experienced by the autistic child and of the pathognomonic qualities of their anxiety (dismantling, precipitation). It has been extended through interpretations of intracorporeal identification processes, rendering intelligible part of the behavior of autistic children (retreating into a corner, leaning against a wall, looking sideways, and so on). It remains at the forefront in describing the drive-based movements of infantile sexuality observable — often in exacerbated form — in autistic children. It inspires practices that provide real help to the autistic child and their family, when not perverted into an implicit accusation of the parents[6]. It convincingly accounts for the function of autistic objects, from which many children cannot bear to be separated.

By contrast, other elements receive no specific explanation : the intellectual disability present in some autistic children ; the so-called "restricted" but highly specific interests, such as fascination with subway maps or trains ; the peculiarities of autistic language and its temporal developmental dynamics ; the adaptive capacities of autistic adults and their particular cognitive competencies. The limits of psychoanalysis regarding autism reside above all in its metapsycho-

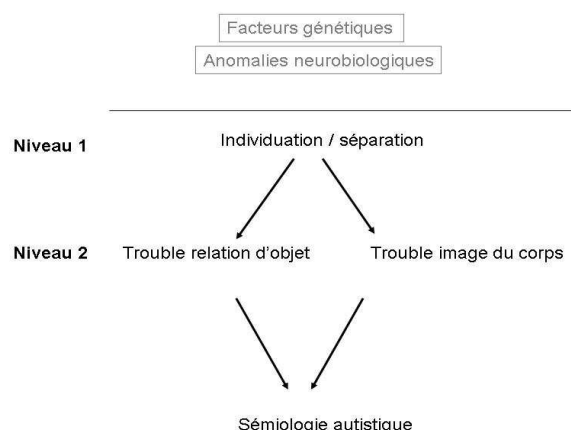


Figure 1 – Structure of the psychoanalytic approach to autism. Neurobiological and genetic factors remain in the background.

logical presuppositions. Its failure as an explanatory paradigm is sometimes — though not always — accompanied by a rejection of the scientific approach to autism in the name of subjectivity, the superior status of the psyche, the singularity of every child, and so on — an ideological degradation that we should not confuse with psychoanalysis in its essence, a discipline that explores the human soul (Freud's *Die Seele*), aware of both its areas of operative validity and the limits of its application.

Contributions of Neurobiology

The First Level : Genetics

Neurobiology has upended the theoretical landscape of autism by establishing its neurodevelopmental nature. Ideally, the neurobiology of autism aims to establish a deterministic sequence across three levels : (1) the genetic level, where a particular gene (or gene sequence), defective or mutant, codes for the synthesis of a protein involved in a process of neuronal maturation, synaptogenesis, impulse conduction, or the architectonics of a structure ; (2) the level of neurobiological structures and functions ; (3) the level of cognitive functions (see Figure 2).

At the genetic level, ASD manifestations are phenotypic expressions of gene sequences still being

progressively identified, generally following non-Mendelian transmission. Mendelian transmission of a single gene responsible for autism is estimated to account for 3% of cases ; the presence of chromosomal anomalies, 5% ; de novo mutations of a single gene, between 5 and 10% ; leaving 80 to 85% of cases in which gene identification remains unknown (Guo, 2011). The risk of recurrence within families of autistic individuals is 45 times higher than in the general population. When one monozygotic twin is affected by autism, the second has a 60% probability of also being autistic, whereas this risk is virtually nil among dizygotic twins. The sex ratio shows four autistic boys for every one girl. The generally greater severity of autism in girls (with frequent accompanying intellectual disability) is linked to several abnormal gene sequences on the X chromosome. More than twenty other regions of susceptibility have been identified across numerous chromosomes, particularly chromosomes 2q, 7q, 16p, and 15q. Anomalies have been observed in genes coding for proteins involved in stages of neurogenesis (selection of neuronal networks, apoptosis, transport of neurotransmitters such as serotonin and glutamate). Autism is frequently, though not systematically, associated with genetic conditions such as Fragile X syndrome, tuberous sclerosis, Rett syndrome, neurofibromatosis, and phenylketonuria. Table 2 presents, for illustration, the main genes implicated in autism.

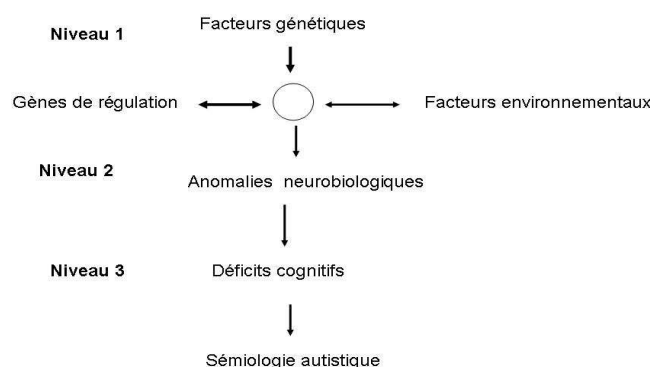


Figure 2 – Structure in three levels in Neurobiology.

Most genes implicated in the occurrence of autistic states are not part of a linear causal series. The genetic program offers a possible choice among various developmental bifurcations, upon which environmental factors exert an influence. Alongside the immutable phenotypic expression of encapsulated genes, there exists a form of environmental selection of the individual characteristics best adapted to the regulation of the organism. Certain genes exert a regulatory function over dynamic processes of neurogenetic development. These processes are not controlled by encapsulated genes indifferent to the environment, but interact with environmental factors.

Environmental Factors

The environmental factors implicated in the occurrence of autistic states may be toxic (valproic acid, thalidomide...), viral (cytomegalovirus, rubella...), anoxic, or consequent upon acquired brain lesions, neonatal jaundice, nutritional deficiencies, and so on. Any factor that disrupts neuronal migration during embryogenesis can set off developmental trajectories toward autism, provided it is combined with a genetic susceptibility. Other environmental factors are linked to relational interactions. The human infant, like the young of many mammal species, requires care and feeding, but also relational imprinting and an attachment relationship. Traumatic conditions (long early hospitalizations, ruptures of attach-

ment at critical moments, prolonged affective deprivation) are associated with signs such as gaze avoidance, rocking, and avoidance of contact. If addressed promptly, these forms — sometimes termed "autistic phenocopies" or "autistic-like" presentations — are reversible, though not without leaving psychic aftereffects (Brown, 1997; Rutter, 2001). The existence of these partially reversible autistic disorders, secondary to deprivation or traumatic factors, is not in contradiction with the fundamentally neurodevelopmental nature of autism. In traumatic situations, a child may reorganize around an earlier developmental level. Children suffering from hospitalism develop repetitive behaviors as a way of relying on an orderly structure to reassure themselves in the face of a reality perceived as chaotic. Variability in resilience produces variability in these secondary reactions. Those in whom autism persists present genetic vulnerability factors. Psychoaffective disturbances are not, in themselves, causal factors of ASD; they are insufficient to initiate an autistic trajectory unless combined with a genetic predisposition. The interplay between predisposing factors, determining factors, and aggravating factors — some genetic, some environmental — produces variability in autistic presentations, along a gradient from severe autism to isolated autistic traits. Genetic sequences statistically implicated in the occurrence of autism may fail to be expressed in an autistic phenotype; conversely, other genes implicated in autism are also found implicated

Gene	Implication
AVPR1A	Social behavior
CAMK2B	Calcium-dependent synaptic protein kinase
DISC1	Binding protein, Asperger syndrome
FMRP	Chromatin regulation and remodeling
FMR1	Fragile X syndrome
GRIK2	Glutamate receptor (intellectual disability)
GRIN2A	Glutamate receptor (intellectual disability)
MECP2	DNA transcription, methylation, Fragile X-type autism (type 3)
MECP2	Chromatin regulation and remodeling
NF1	Neuronal cytoskeleton formation
NLGN3 (X-linked)	Neuroligins and neurexins, synaptogenesis, neuronal conduction
NLGN4 (X-linked)	Neuroligins and neurexins, synaptogenesis, neuronal conduction
NRCAM	Neuronal adhesion, growth and guidance of the axonal growth cone, migration
NRG1	Neurologin ligand, cell membranes, neuronal conduction
OXTR	Transsynaptic receptors and transporters, oxytocin receptor
RELN	Neuronal migration, serine/threonine/tyrosine kinase activity
SHANK3	Cell adhesion, synaptogenesis, dendritic induction
SYNGAP1	GTPase activation, phospholipid binding

Tableau 2 – Selected genes implicated in autism and their developmental implications

Sources : *Neurobiology of Mental Illness*, Charney D.E et al. 2013 et *Neurobiology, Diagnosis and Treatment in Autism*, Riva et al. 2013 Advances in autism genetics : on the threshold of a new neurobiology, *Nat Rev Genet.* 2008 Mays, 9(5) : 341-355.

in other conditions (particularly intellectual disability).

Genetic determinism is thus neither univocal, nor linear, nor specific. The causes of autism are complex in the scientific sense of the term. The plurality of factors involved does not interact as a simple sum or complementary series, but rather as external parameters (control factors) acting upon a dynamical system. Recognizing this complexity is not a failure of understanding the causes of autism — it is an epistemic advance. It allows autism to be conceived as a developmental trajectory in which initial conditions and environmental factors modulate genetic sequences and orient them (or not) toward autistic states. Thus, clinical experience with autism attests that very early therapeutic interventions have a measurable efficacy[7].

The Second Level : Structures

Neurobiological anomalies in autism may be distributed across several structures, or selectively marked in a single one (the amygdala, for example), or affect neuronal conductivity, or the overall quantity of neurons (in excess or deficit, due to abnormal apopto-

sis), or their morphology. Sometimes these anomalies are observable through investigative techniques (imaging, etc.). Electrophysiology has established that one-third of autistic children present with epilepsy. Yet in some cases, nothing distinguishes the brain of an autistic subject from that of a non-autistic subject. This indistinguishability does not preclude the presence of anomalies, since these may escape available investigative means. Several hormonal regulation anomalies have been identified, and a theory of early testosterone exposure has been proposed (Auyeung & Baron-Cohen, 2009). Elevated microbiota levels have been noted in animal models of autism, giving rise to hypotheses of a bacterial determinant (Reardon, 2013). Numerous biochemical investigations likewise reveal anomalies. The neurobiology of autism is in full expansion, but the findings remain heterogeneous. Table 3 presents the main neurobiological anomalies and their functional implications. They are not linked within a biologically coherent structural system and may be entirely absent in a given autistic individual. Despite this inter-individual variation in anomalies, specific manifestations common to autism do exist (the triad of cardinal symptoms). The neurobiology of autism thus finds itself in the uncomfortable position of demonstrating an un-

derlying substrate of anomalies that are not systematic — as though several distinct causes could produce the same effect.

The Third Level : Cognitive Functions

Cognitive approaches have produced convergent findings (for a review, see Mottron, 2006). The method employed is as follows : a group of autistic subjects is selected on the basis of diagnostic criteria (DSM, supplemented by questionnaires and tests). Autistic subjects with intellectual disability are most often excluded, as being unsuited to informing on the specificities of autism proper. A control group is formed of non-autistic subjects, free of other disorders, matched for age, sex, and intellectual level. Subjects in both groups are exposed to a single test, chosen according to the study's prior hypothesis, under identical administration conditions. Results are compared using statistical analysis (ANOVA, t-test, etc.), selected according to group size and the number of variables tested. The statistical procedure concludes (or fails to conclude) that a significant difference exists between the two groups. Published results from such studies are subsequently the subject of meta-analyses.

We list below the principal convergent findings of these studies, bearing in mind that these cognitive traits are distributed variably and with differing intensity among autistic individuals — some may be entirely absent in a given subject, others extremely pronounced.

1. **Sensory and perceptual peculiarities.**

Hyper- and hypo-sensitivities may coexist across different sensory domains (hearing, vision, touch, pain sensitivity) (Ornitz, 1968; Bruneau, 1999).

2. **Distortions in the referential and pragmatic aspects of language,**

following a developmental progression through seven successive phases : (1) mutism; (2) immediate echolalia; (3) immediate, delayed echolalia with pronominal reversal; (4) sporadic immediate echolalia with delayed echolalia and stereotyped language; (5) delayed echolalia with stereotyped language and productive language in context;

(6) stereotyped language with productive out-of-context language; and finally (7) hypergrammatical productive language with thematic repetitiveness (Prizant, 1983). Some children remain fixed at an initial phase, while others progress through all phases, though with variable and unpredictable timing.

3. **Difficulties in the expression, regulation, and recognition of emotions,**

giving rise to motor manifestations (hand-flapping, abnormal movements, jumping) (Hobson, 1989).

4. **Difficulties in decoding emotions and in acquiring a theory of mind**

(understanding the mental states of others) (Baron-Cohen et al., 1985; Frith et al., 1991; Gepner, 2010).

5. **Singularities in executive functions**

(planning, flexibility, working memory), marked by difficulty maintaining provisional goals in working memory and an imperative need to close out an already-initiated task (Pennington et al., 1996). Deficits exist in the coordination and planning of sequences of simple goal-directed movements. Certain postural control anomalies have been interpreted as dysfunctions of anticipatory systems (Schmitz et al., 2003).

6. **Attentional singularities.**

Autism is associated with a specific form of attentional functioning, comprising both a positive facet (enhanced perceptual discrimination, except for social stimuli) and a negative facet marked by difficulty orienting the attentional beam, while exogenous attention remains preserved (Damasio & Maurer, 1978)[8].

7. **Dyspraxic elements,**

particularly among Asperger children (dressing dyspraxia) (Ioan, 2005). These dyspraxias result from the impossibility of self-referencing motor conduct to one's own body. Difficulties also exist in imitating postures and symbolic or non-symbolic body movements, linked to a deficit in coordinating self- and other-representations (Rogers & Pennington, 1991).

8. **Above-average competencies in certain cognitive activities**

(visuospatial ability, eidetic memory). Serial systems (letters, num-

Neurobiological anomaly	Functional implication
Superior temporal sulcus / secondary auditory cortex (Zilbovicius et al., 2000)	Social cognition ; voice control
Prefrontal cortex / frontal lobe (Damasio & Maurer, 1978)	Delayed metabolic maturation ; deficient functional connectivity ; executive functions ; gaze/attention ; theory of mind
Cerebellum — reduced Purkinje cell number ; hypoplasia of lobules VI and VII (Courchesne et al., 1988)	Modulatory functions ; posture ; executive functions
Central nuclei / amygdala — increased or decreased size	Emotion recognition
Corpus callosum — total or partial agenesis	Central coherence
Hippocampus — reduced dendritic connections	Immediate memory ; encoding
Neuronal conduction — reduced neuron size ; reduced number of cranial motor nerve nuclei	Central coherence ; intellectual disability
Serotonergic system	Abnormal movements ; ritual stereotypies
Dopaminergic system — frontal lobe, nigrostriatal, mesolimbic, mesocortical projections (Damasio & Maurer, 1978)	Attention ; regulation of executive functions (Courchesne, 1991)

Tableau 3 – Principal neurobiological structures implicated in autism.

bers...), maps, and calendars are privileged objects of interest (Fitzgerald, 2004).

1. **Developmental heterochrony** (Crespi, 2013). Cognitive functions are arranged in an abnormal temporal sequence, with original delays, advances, and developmental plateaus.

Several recent models have proposed that autistic manifestations do not stem from a localized cognitive determinism but from a global adaptation to an abnormally perceived world. According to Bruno Gepner, autism is marked by anomalies (deficits or excesses) of connectivity and spatiotemporal synchronization among multiple cerebral territories, causing the world to appear both too fast and too fragmented to autistic individuals, generating a range of perceptual, imitative, cognitive, and socio-communicative deficits (Gepner et al., 2010). Other authors have attempted to conceive of autism as a developmental adaptation to a world perceived as too intense (Markram et al., 2010), requiring the autistic subject to regulate a state of hyper-perception, hyper-memory, and hyper-emotion.

A Primary Disorder in Autism ?

Despite the accumulation of convergent data on altered cognitive functions, no primary cognitive disorder of autism has been convincingly identified. A primary disorder is defined as the proximal cause of the behavioral disturbances characteristic of a given pathology (Pennington & Ozonoff, 1996). It must therefore satisfy the following criteria :

1. **Universality.** The primary deficit must be observable in all subjects presenting the pathology, without exception — that is, in all subjects identified as ASD.
2. **Specificity.** The deficit must be observable only in this pathology — it must be pathognomonic.
3. **Necessity and sufficiency.** The deficit must account for the full range of the disorder's manifestations.

None of the cognitive theories proposed to date simultaneously satisfies all three criteria. The hypothesis of a primary deficit in affective contact and emotional regulation, affecting early relationships (Trevarthen, 1989), has been refuted. Ornitz and Ritvo's (1968) hypothesis of a primary auditory-perceptual

Domain	Observable	S	T	No.
Behavior	Relational indifference	X	IV	1
	Impulsive flight from groups or places		IV	2
	Imperative repetitive behavior		VI	3
	Constant use of an invariant object		VI	4
	Abnormal reaction to separation from mother (indifference, tantrum)	D*	V	5
Language	Absence of language development	X	I	6
	Inappropriate vocalizations		V	7
	Prosodic disturbance		I	8
	Immediate or delayed echolalia	X	IV	9
	Pronominal reversal — no use of "I"	X	IV	10
Cognition	Hypergrammaticality		VII	11
	Verbal stereotypy	X	VI	12
	Marked elective interest (numbers, letters, trains, maps)	X	VII	13
	Exceptional specific competence	X	VII	14
	Marked intellectual disability		I	15
Attention	Ritual lining-up of objects, vehicles	X	VI	16
	Agitation, hyperactivity, instability		III	17
	Attentional/concentration disturbance		I	18
Motor	Dressing dyspraxia	A*	I	19
	Visuospatial dyspraxia / fine motor skills / laterality		I	20
	Absence of initiation, hand-leading of others	X	I	21
	Complex gestural stereotypies	X	II/VII	22
	Hand-flapping	X	V	23
Perception	(Facial) tics / oral bruxism		V	24
	Abnormal auditory reaction, hyper/hypo, sound phobias	X	IV	25
	Gaze avoidance	X	IV	26
	Finger agitation / objects in peripheral visual field		IV	27
	Object rotation, external morphodynamism, dropping	X	VI	28
Posture	On/off conduct with light	X	VI	29
	Anomalies of hearing, vision, touch, taste, smell		I	30
	Abnormal reaction to pain		I	31
	Preferential lying-down posture		IV	32
	Jumping and/or toe-walking		IV	33
Emotions	Tonus disturbance, listlessness, hypotonia		II	34
	Marked anxiety manifestations		III	35
	Emotional deficit	X	I	36
Feeding	Depressive sadness	D*	V	37
	Feeding disturbance		II	38
Toileting	Difficulty acquiring toilet training		II	39
Aggression	Marked self-injury		III	40
	Marked hetero-aggression		III	41
Sleep	Sleep disturbance		II	42

Tableau 4 – Principal observable manifestations in autism. S : pathognomonic specificity of marked ASD (X) ; A* : specific to Asperger presentations ; D* : specific to anaclitic depression. Types — I : Deficits ; II : Functional disorganization ; III : Dissociation ; IV : Adaptive behaviors ; V : Emotional regulation ; VI : Invariance ; VII : Emergence of schemas.

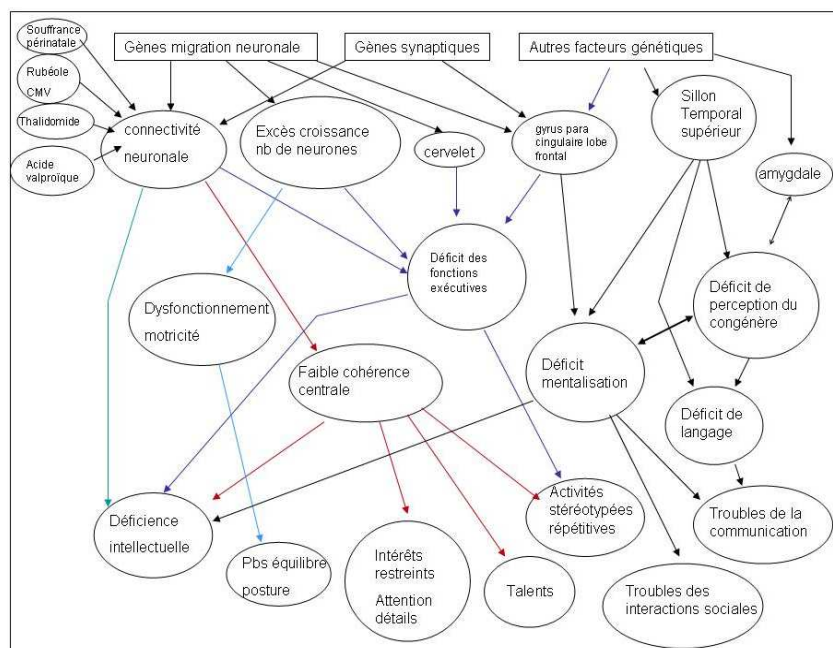


Figure 3 – The complexity of the links between the determinants of autism (partial overview). No simple bijection exists between the set of cognitive functions and the set of observables. For example, stereotypes are linked both to executive-function deficits and to a lack of central coherence. Adapted from Ramus, F., "L'hypothèse neurodéveloppementale de l'autisme," Laboratoire des sciences cognitives et psycholinguistique, ENS/EHESS/CNRS, Paris, 18/10/2012.

disorder, involving central rejection of input from ascending auditory pathways, has likewise been refuted (Klin, 1993), although singularities of auditory perception, objectified through anomalies in late auditory evoked potentials, remain markers of autistic disorder (Bruneau, 1999). Theories that appeared to satisfy the universality criterion, such as executive-function disorder (Baron-Cohen, Leslie, & Frith, 1985) — present, with variable form and intensity, in all autistic individuals — also account for attention-deficit/hyperactivity disorder and frontal-lobe syndromes. Theory-of-mind deficits are likewise found in certain deaf children without language but without autism. Finally, the criterion of necessity and sufficiency renders the very definition of a primary disorder illusory, given how far apart the two cardinal features — social-communication disorder and elective/restricted interests — appear to be from one another. Models seeking a single primary disorder are

thus doomed to a dead end, since no simple bijection exists between the set of cognitive functions and the set of observable manifestations of autism (see Figure 3). A given trait may appear in one case where the associated cognitive function is deficient, and in another case where the same trait appears alongside an intact version of that same function. One consequence is the imprecision of semiological classifications and diagnostic tools (PEP, ADI, ADI-R, CARS, ECA-R), which rely on clinical empiricism[9]. To date, then, cognitive theories have failed to identify a primary disorder capable of explaining the variety of observable singularities. The conclusion is unavoidable : autism is not a conglomerate of neuropsychological deficits.

The Autistic Self

The Complexity Approach

If, despite diverse etiologies affecting distinct neurobiological structures and different cognitive functions, we nonetheless observe an apparent unity in the behaviors of autism (a singular pattern of communication, social anomalies, fixed and restricted interests), we must infer the existence of an entity distinct from the underlying biological and cognitive structures, whose properties account for the constancy of these observables. We are thus led to introduce an entity for which cognitive functions serve as partial components, and which presents a sufficiently constant unity to be identified as an autistic state. This entity logically fulfills functions with a broader scope than those achieved by the mere conjunction of the underlying cognitive structures — a property evoking the phenomenon of emergence found in complex dynamical systems. Research on complex dynamical systems, subject to the necessity of maintaining their own stability, has shown the existence of an emergent phenomenon : the appearance of a form of "cognitive" agency responsible for coordinating stabilizing regulations[10]. This emergent agency is delocalized across the full set of interactions among the system's elements and is irreducible to their physical properties. It possesses novel qualitative properties that cannot be deduced from the properties of the system, and it exerts downward causation upon the underlying, less complex systems.

Defining the Self

We identify this integrative entity — the sole solution to the diversity/unity paradox of autism — with an agency emergent from neurophysiological complexity, tasked with ensuring its own structural stability. We name this entity the **self**, and attribute to it the fundamental mental functions of : cognitive cohesion (allowing for the stability of a perceived world) ; individuation (the unity and singular identity of an individual) ; and virtualization (the anticipatory mentalization of actions, the phantasmatic fulfillment of desires) (see Figure 4). In this broad conception, the self can fully integrate the unconscious and the

agencies discovered by psychoanalysis, while also assimilating the functions described by cognitive science. Adopting this epistemological perspective, both psychoanalysis and cognitive science can be seen as describing partial cross-sections within the complexity of the self (Virole, 2011).

The application of complex systems theory to psychic life is an axiomatic analogy, but its reification is plausible. Neuronal interconnectivity, the dynamic behavior of neuronal networks, the oscillations observable within cortical columns, the dynamics of neuroregulation, and the kinetics of neurotransmitter action are but a few among many elements justifying the application of complex dynamical systems theory to neuronal functioning (Pezard & Nandrino, 2001). Neuromodulators — dopamine, serotonin, noradrenaline — allow "information" to be initiated, inhibited, or enhanced by increasing or decreasing contrasts between activated neuronal zones. The three neuroregulatory systems, each with distinct kinetics, generate dynamic coupling effects (resonances, phase shifts) whose modeling requires recourse to the mathematical models of complexity theory (definitions of attractors, phase space)[11]. This methodological choice allows the properties of a complex system to be exploited without needing to specify its substrate or precisely know its content. Any complex dynamical system endowed with self-organizing capacities, immersed in an environment involving chance, evolves by modifying its internal states and generating new morphologies and functions (Atlan, 1979). The ultimate evolution of a system endowed with self-organizing properties is the generation of an agency capable of anticipation and decision-making, through reflexive consciousness[12]. The system may evolve toward a macro-system of higher complexity : the complexity of a biological cell integrates into a tissue, an organ, an apparatus, an organism. Conversely, a system may degrade : following regulatory failures, a reduction in complexity leads to stabilization at a lower level, while still retaining the holistic properties of the higher level. Complete degradation of complexity carries the system beyond the threshold that defines its existence, resulting in its disappearance (death). By defining the self as a mental entity emergent from complexity, we need not concern our-

selves directly with its content, and can instead focus on its properties.

1. **Holistic character.** The self maintains structural stability through regulations. One such regulation is the invariance of form. Form invariance across different sensory modalities is a characteristic of the newborn's cognitive system. This supra-modal system runs counter to theories postulating that inter-sensory differentiation is acquired only later, through learning, enabling the unified perception of the world (Bullinger, 1989). Perceptual completeness, functional constancy, and sensory intermodality[13] reveal the existence of other regulations. These are thus multiple : some innate, others acquired, some idiosyncratic. The self-regulation of the self is teleonomic — it is coextensive with its very form of existence. It compensates for disturbances, maintains the cohesion of parts and the integrity of the system's form, repairs and delays degradation, favors adaptation, and anticipates variation (Rumelhard, 1995)[14]. It is an aspect of the self's self-production (Morin, 1977).
2. **Sensitivity to initial conditions.** The orientation of the self's developmental trajectories is constrained by initial conditions (genome, environment, early clinical history). The self integrates the history of its regulations as acquired experience (memory). Initially, the unfolding of the self is controlled by regulatory genes and by human interactions. Any alteration of genomic regulation, combined with an alteration of interactive human regulation, produces — if it exceeds certain critical thresholds, variable across subjects — a bifurcation toward an autistic configuration,[15] marked by specific cognitive, perceptual, and emotional functioning.
3. **Singularity.** By virtue of its history, each self is singular and seeks to distinguish itself through the construction of an identity. Autism does not erase the personalities, characters, and identities of each subject.
4. **Regulatory figure.** The structural stability of the self depends on the limits of the fields of regulation defining a plateau of stability (the system's attractor). Beyond these limits, the self un-

dergoes a catastrophic bifurcation toward another plateau. The set of these limits constitutes the system's regulatory figure. It is topological in nature and includes critical singular points. If the regulations fail to maintain structural stability, the autistic self may dissociate.

5. **Emergence.** The structural stability of the self initiates dynamics of emergence — new morphologies and functions not deducible from its prior states. Autism reveals the emergence of unprecedented manifestations and behaviors.

Classification of Autistic Observables

The vicissitudes and successes of the structural stability of the autistic self manifest themselves in the observable behaviors of autism. These are variable, reflecting the diversity of underlying neurobiological determinants, but they are unified insofar as they all aim at establishing and maintaining the structural stability of the self. They can be classified into seven types. Each autistic individual presents a mosaic of these different types. Table 4 (above) presents the list of the most frequent autistic observables, classified by domain and numbered according to their typology.

Type I — Functional deficits. These are basic deficits of motor, executive, linguistic, or social functions, without regulatory compensation by the self. One observes the absence of a cognitive function's establishment and its consequences for the development of other functions. Observable traits of this type are privative (for example, total and definitive absence of language development, deficit in emotion recognition, massive intellectual disability, visuospatial dyspraxia). Their existence is due to the severity of the underlying neurocognitive alterations. In other cognitive domains, normal development may be observed. These deficits are comparable to ordinary neuropsychological deficits; they are not specific to autism, and are the least theoretically interesting.

Type II — Functional disorganization (regression to an earlier developmental plateau). If one mode of regulation fails, the self regresses to a stability plateau attained earlier in development. Disturbances of major physiological functions (feeding, sleep) represent a regression of the self toward ear-

lier states of physiological stability. The appearance of motor automatisms is explained by a failure to maintain attention : the alteration of attention reveals the underlying function that serves as a fall-back plateau against the risk of decohesion. In the autistic child, the appearance of cyclical stereotyped behavioral sequences is explained by this deficit in the cohesion of the self. The line of disorganization runs from an intentionality that is either unacquired or deficient, down to the underlying level of voluntary attention, revealing the automated cyclical behaviors beneath. The cyclical character of stereotypies and repetitive behaviors is linked to the underlying cyclical structure common to every natural dynamical system (Maturana & Varela, 1973; Delattre, 1979). It is often sufficient to draw the autistic child's attention and help them resume an intentional process in order to attenuate their stereotypies. However, in certain autistic individuals — particularly those with alterations of the serotonergic system — the disorganization of the self is such that it precludes any intentionality, rendering the motor stereotypies invasive and sometimes complicated by disturbances of pain and pleasure sensation[16].

Type III — Dissociation of the self. Regulatory mechanisms are insufficient to maintain the structural stability of the self, which dissociates into unstable partial entities, allowing drive-based manifestations to emerge. The so-called "psychotic" behaviors of autistic children result from these dissociations : self-injury, hetero-aggressiveness, motor agitation, fecal play, bulimia, rumination. When the clinical picture of an autistic individual is dominated by such dissociations, differential diagnosis with psychotic — particularly schizophrenic — forms becomes highly difficult. Primary neurobiological deficits sometimes account for these severe neuropsychiatric presentations, calling first for a psychopharmacological response (neuroleptics) before the initiation of psychotherapy. In other cases, these are autistic individuals engaged in a developmental trajectory of the self that has encountered obstacles, whether endogenous or environmental. Nosographically, the existence of these mental-dissociation presentations raises the question of the structural links between autism and schizophrenia. Before the introduction of neurolep-

tics, the natural course of schizophrenia often evolved toward a terminal form once called "autism."

Type IV — Adaptive behaviors in response to partial functional deficits. Finger agitation in the peripheral visual field, used to maintain foveal vision, is a paradigmatic example. Toe-walking, leaning against a wall to stabilize gaze, or seeking a lying-down position to counterbalance postural instability, are further examples. The autistic individual develops behaviors whose purpose is to enable optimal adaptation of their cognitive functions. These behaviors should therefore not be indiscriminately counteracted, and should be approached non-invasively. It should nonetheless first be ascertained that they are not primary functional deficits (Type I) — for instance, toe-walking arising from a pyramidal syndrome, or from visuospatial dyspraxia. Visuospatial dyspraxias are favored by early neurodevelopmental disorders but are not specific to autism. Other praxic disorders specific to autistic children arise from difficulty maintaining the intentionality that prepares the execution of a motor act. An autistic child may correctly orient themselves in familiar space, descend a staircase, and so on, so long as these actions are governed by automatized movement patterns. Everything changes when the child must perform these actions intentionally, anticipating them through mentalization (virtualization) — at which point difficulty arises. This is the case with ideomotor dyspraxias in Asperger individuals, which resemble automatic/voluntary dissociations but chiefly reflect the difficulty of mentalizing a space that would allow for the virtualization of a three-dimensional act.

Type V — Anxious or aggressive emotional reactions to the failure of a regulation, or to a trauma resulting from a breach of regulatory thresholds (perceptual thresholds, pain thresholds), taking the form of an abnormal expression of emotion (motor movements, hand-flapping, unmotivated laughter, inappropriate cries). This is a form of regulation through the dissipation of emotional excitation : the emotion has exceeded the threshold of regulation and triggered a dissipative catastrophe. These reactions are signs that the cohesion of the self has been placed at risk. Phylogenetically, emotions serve as triggers for the major vital reactions

of flight, the seeking of protection (anxiety in the face of danger, pleasure in security), and sexual attraction. In the autistic child, these abnormal emotional reactions are observed during disruptions of "scripts" (ordered sequences of action, Schank, 1975). Moving from a script suited to one context (a space, a time, a situation) to a second script (another space, another time, another situation) requires crossing a discontinuity. This crossing is only possible through a self-consciousness that anticipates the second script (mentalizing the action, virtually seeing oneself perform it before initiating it). If a script is interrupted before its closure, or if the succession of scripts occurs without anticipatory intentionality (self-consciousness), a painful affect arises, ranging along a gradient from unease to anxiety. A typical example is that of autistic children who become engaged in an activity linked to a spatial context (a room) and who experience anxiety when interrupted in what may be judged repetitive or inadequate activities, or when a change of room is imposed on them without prior mentalization (hence the necessity of image-based supports in care settings, to allow for anticipation). These emotional reactions are linked to experiences of individuation. One function of the self is to maintain individuation; if the cohesion of the self is not maintained, experiences of separation are lived as terrifying. On this point, the contribution of the psychoanalysis of autism remains fundamental. But care must be taken to distinguish emotional reactions linked to the interruption of ongoing behaviors from those generated by separation situations that reactivate experiences of attachment rupture.

Type VI — The search for invariance and immutability. This type does not include repetitive stereotypies, which are functional disorganizations of mentalization revealing the underlying cyclical structures (Type II above). It does, however, include the search for fixed behaviors, such as an unchanging way of opening a door, or of taking a particular route. Any modification of these immutable behaviors may trigger anxious reactions — the consequence of the interruption of a fixed behavioral script whose function is to ensure the constancy of a reference point.

Type VII — The emergence of intentional, regulatory-oriented behaviors. In the autis-

tic child, the alteration of cognitive functions generates instability in the structure of the self and entails the necessity of specific regulations, identified as autistic symptoms. These take the form of a search for an external kinetic as a support for intentionality; elective interests in forms, typed schemas, or mechanisms; and particular competencies appearing in the establishment of spatial or temporal reference systems, in the iteration of algorithms, and sometimes in eidetic memory.

We now have a new conceptual framework at our disposal. The notion of regulation for maintaining the structural stability of the self has allowed us to classify the observables of autism and to infer a generative logic from them. But if we wish to avoid a purely top-down construction, moving from a global level (the self) to the local level (the observables), we must also describe the internal dynamics by which the autistic self is constructed, accounting bottom-up for the phenomena observed — moving from basic functions up to the self.

The Synthesis of Mental Objects

The structural stability of the self requires the cohesion of elements of physical reality that are given only partially by perception, through the generation of mental objects that can be manipulated by thought. This structural dynamic is that of **objective synthesis**[17]. Objective synthesis involves the acquisition of the multiple facets of physical objects. We see a cube only through one of its faces exposed to our vision, and we mentally reconstruct a cube endowed with its three spatial dimensions. In vision, the constitutive singularities are the contours and discontinuities of forms. According to David Marr (1982), vision extracts information about objects from the way light, reflected off physical surfaces, generates patterns of luminance. Through transduction by the retina's photoreceptors, these patterns become discretized. Downstream of the visual system, higher-level transformations take place. Vision allows for the formation of a primary two-dimensional (2D) sketch: this level extracts local qualitative discontinuities that can be interpreted as objective if they remain stable across changes of scale. The second, in-

intermediate level ($2\frac{1}{2}$ D), between the 2D and 3D levels, represents the external world as composed of sensitive surfaces moving through three-dimensional space (R^3); at this level, discontinuities are interpreted as the apparent contours of objects. The third and final level (3D) is that of volumes and their geometric properties; at this level, the prototypes of objects are constituted. In addition, the transitions between spectral formants are privileged for the reconstruction of timbre (Botte, 1988). These reconstructions depend on intentionality (through gaze, through listening), which allows for the cohesive fusion of the different facets of real-world objects, always only partially given by perception (see Figure 5).

Derived from medieval thought, the term "intentionality" was reintroduced into contemporary philosophy by Franz Brentano to designate the property of mental states of being directed toward objects or states of the world. Unlike Husserl's conception, which identified intentionality with the property of a conscious mental act, intentionality is here understood as independent of any conscious awareness of a thought-content (Proust, 1998). The ray of intention (gaze, listening), passing through the various sketches and adjusting them to their common points, places them in perspective (in the same way that numerical stitching operators function in computerized image synthesis). Intention performs the stitching-together of the primary sketches of the object (dynamic, spatial, temporal, frequency-based, and energetic cues), transforming them into a mental object available to cognition. Every object — visual, tactile, auditory — is constructed through the synthesis of its various perceptual facets around an optimal facet, what Husserl called the *optimum*, corresponding to the semantic prototype of the object's class. For Husserl, the data of perception are not distributed within pre-existing isotropic and isochronous spaces, contrary to the Kantian thesis, but organize themselves around privileged intentional modes — the *optima* of the domain under consideration and its extremities (*extrema*). In the visual field, the optimum is the point of fixation, where the thing displays and unfolds its internal determinations; the extremum is the edge of the field, where differences blur and lose clarity and distinctness. The optimum is the ideal visual proximity; the extremum, the horizon of perspectival re-

moteness. For the temporal field, the optimum is the living present, the focal point of the actuality of intentional acts, with two extrema corresponding to the respective limits of retention of the immediate past and of protention toward the imminent (the window of action). The optimum of an object entails the presence of an extremum corresponding to its most distant facets; beyond the extremum, the absence of the object begins, its disappearance into the horizon of other objects.

Remarkably, Husserl's theses have since been confirmed by contemporary cognitive science, which has shown that an object is indeed perceived according to its best facet (optimum), the one that facilitates manual grasping[18]. The notion of *affordance*, proposed by Gibson's ecological psychology, accounts for the same phenomenon. Perception is ecologically oriented and processes only those elements possessing a behavioral valence — "the object's offer of meaning" (Wells, 2002). The constitution of physical objects in perception (phenomenal consciousness) is thus the correlate of a directed act[19]. Perception is not a passive reception of information but a transformation of sensory data through an intentional act aiming at the optimum of the object. But since this cannot always correspond to an act that is actually carried out, any intention directed toward an object requires the inhibition of the motor act. From sketches encoded within the attractor states of neuronal networks, icons (mental images) of physical objects are synthesized and can be manipulated within internal virtual micro-worlds constituting thought (implicit hypothetical, imaginary) — designed to simulate action and reflect upon it rather than execute it.

Perceptual Singularities

In the very young autistic child, the neurocognitive resources necessary for the development of intentionality (implementation of neuromotor gaze control, maturation of attentional processes, neurosensory integration, and so on), which enable objective synthesis, are insufficient or delayed (heterochrony), owing to neurobiological factors (conductance disorders, cortical lesions, structural anoma-

lies, etc.). Physical objects are perceived as discontinuous sketches, and are not integrated into unified mental objects with pragmatic value. The facets of objects become morphological singularities that trigger grasping-motor behaviors (non-inhibited affordances). The autistic child rushes toward a salient object — a doorknob, a slat in a blind — perceiving it as an attractive topological singularity, a trigger for manual grasping behavior. In a non-autistic world, such an object would evoke an unconscious affordance whose motor execution would be inhibited. The autistic child seeks to render the external world coherent with their internal world by electively seeking out elements congruent with their capacities for objective synthesis (caustics, plays of light...). These elements are fundamentally apragmatic. The autistic self ignores the horizon of intentional objects and becomes absorbed in the contemplation of their constitutive facets. It enacts a kind of methodological solipsism, constructing the conditions of a controlled experiment : it delimits a closed system that allows a dynamic phenomenon to be reproduced. To ensure cognitive cohesion, young autistic individuals need an attentional fixation on external kinetics, often cyclical, but also linear, such as the movement of a vehicle (a train, a car). The necessity of an external kinetic is explained by the difficulty of maintaining objective synthesis, itself due to alterations of the attentional systems. The intentionality constitutive of the synthesis of mental objects requires an external kinetic to serve as its support. This explains the interest autistic children show in video games, which generate the illusion of an avatar's movement at the center of a pseudo-3D virtual world. The rapid movement of the background images produces the illusion of movement in the central figure (the vehicle), sustains visual attention (by stimulating peripheral vision while favoring foveal vision), and allows for the unfolding of intentionality (advancing, retreating, orienting, acting...).

Intersubjective Otherness

For Husserl, the construction of intersubjectivity requires the perception of intentionality enacted in the body (*Leib*) of the other[20]. It involves kinesthetic resonance with the body of the other, consid-

ered the foundation of empathy (emotional resonance). Becoming aware of the existence of mentalization in the other (a "theory of mind") requires the anticipatory intuition of the other's movements. Intersubjectivity — the foundation of the relation to the social world and of the communication of mental states — depends on the recognition of intention as embodied in the body of the other.

The construction of intersubjective otherness follows a development marked by critical phases, each involving the reflexivity of intentionality. Every intention to move is realized through an afferent signal sent to the motor (effector) center, and a copy of the efferent signal sent to integration centers, allowing the movement to be anticipated and, if necessary, corrected (a "feed-forward" anticipatory regulation). The intention directed at the object feeds back onto the subject. Perception and attention are controlled by feedback and regulatory systems (efferent systems, corollary discharge, mirror neurons). Since there is no intention of the self toward the object without a corresponding return to the emitting self, every intention participates in the reflexive construction of the ego — that is, of the self[21].

At the beginning of life, the self is constituted of nuclei centered on sensory experiences that remain disjointed, owing to the neuronal immaturity of the multimodal integration structures. The lasting integration of these various nuclei of the self occurs through precurrence. The English biologist Sherrington (1857–1952) noted the link between the evolution of the nervous system and sensory organs capable of detection at a distance (distal senses : smell, vision, hearing). Distal senses possess a distinctive functional characteristic : they allow the organism to anticipate a predatory attack and prepare flight or counter-attack behaviors. This anticipation — termed *precurrence* by Sherrington — induces the emergence of an "inner" space, distinct from reflex reactions. It enables anticipatory representation and complex behavioral decisions. Precurrence favors the constitution of primitive intentional transitivity (subject $\Rightarrow$ object), observable clinically through visual pursuit of an object and the search for the spatial source of a sound. The gaze directed at another's gaze holds a particular status : when the infant encounters the

gaze of another, and thus the mobility of another's eyes, the infant must couple their own ocular movements to those of the other in order to sustain visual contact — resulting in an exchange of gazes accompanied by smiling, which corresponds to René Spitz's (1965) first organizer.

Any alteration of sensory functions (sensor deficits, delayed myelination, neuronal lesions...) that limits precurrence deflects developmental trajectories, undermines the cohesion of the self, and induces autistic behaviors (gaze avoidance, indifference to sound). The second critical moment occurs between the 6th and 9th months and involves the constitution of joint attention along with the emergence of proto-imperative, then proto-declarative, manual pointing (gestural deixis). The extended index finger embodies the beam of intention. Remarkably, this critical moment requires the intervention of a partner : the infant's endogenous attention toward the external object is prepared by the mother, through her own attention to the object. By watching the object, then the mother's face, and by perceiving the difference between the object's behavior as directed by the infant's own intention and by the mother's, the infant comes to distinguish between self-awareness and awareness of the other. The third moment in the constitution of intentionality is that of the mirror stage (around the 11th month), described by Wallon and taken up by Lacan (1966)[22]. It allows for the emergence of a fully assumed form of reflexive consciousness, and enables the self to take itself as its own object of intention.

The Generativity of Language

This reflexivity modifies the generativity of language. Generativity is expressed in the competence to produce an infinite variety of linguistic utterances from minimal units, enabling the evocation and communication of ideational content beyond any immediate reference. In autism, generativity is expressed in a singular manner. The successive phases of language development in the autistic child — some autistic individuals remain fixed at initial phases — reveal a trajectory toward linguistic performance from a competence initially modified by neurobiological factors

(particularly anomalies in the superior temporal cortex). Yet this alteration of the neurobiological foundations of language does not, by itself, account for the specificities of autistic language. Phonological structure, phonemic oppositions, syntactic rules, and semantic permutations appear intact in subjects who have progressed beyond the initial phases, but the generativity of language, bearing on minimal units of meaning (morphemes) syntactically arranged, is established in a singular way. An autistic person may use a whole sentence out of context. The intention to communicate is present, but it makes use of units without intelligible meaning, owing to the repercussions of the disturbance of objective synthesis upon the foundations of meaning itself. For the autistic person, the minimal signifying element (the whole sentence) is indeed associated with a signified, but this signified is not commonly shareable (a shared convention of meaning). It can, however, be understood if one takes the trouble to try to grasp the communicative intention behind it. The uttered sentence does not carry ordinary meaning ; it is a whole, undivided unit, a kind of deictic, pointing toward an experience or a desire that the autistic person wishes to signal to us. The specific delay in acquiring the full pronominal system and in using "I" is explained by the singularities in the development of intentionality, which is only belatedly performed in language. More profoundly, the generativity of autistic language engages objective synthesis itself. For the identity of an object to emerge from the continuous flux of its appearances (the succession of retinal images for vision, of cochlear patterns for audition), several presentations of the object are required. Since every perceptual sketch is linked to the horizon of other sketches, it generates an anticipation of them — it becomes the sign of other, virtual sketches. Every perceptual sketch is thus, by nature, semiotic. The semiotic — and, more precisely, the symbolic, if we accept that these sketches form among themselves a system of differences — emerges from perception and is naturally grounded in it. Any disturbance of objective synthesis, for instance through a failure to constitute the horizon of other sketches, induces a disturbance in the genesis of meaning, and consequently in the generativity of language. Language disorders in autism are thus explained by the repercussions of the deficit in objective

synthesis upon semantic construction. If the mental object is not synthesized, and thus not unified across the horizon of its sketches, it cannot be individuated and cannot be associated with a meaning.

Understanding Autism

According to our model, autism is determined by neurobiological factors (genetic factors first and foremost) acting as external control factors that parameterize the internal dynamics of the development of the self (see Figure 6). Autism is thus neither a disease nor a psychopathological structure, but a stable form taken by a self constrained to establish stability from neurocognitive resources distinct from those of non-autistic individuals. The apparent behaviors of autism are not the symptoms of a pathology, but the visible facets of a developmental structure. The difference is radical : in one case we are within the register of the dynamics of an illness, a disorder, a deficit ; in the other, we are describing a subjective entity. For example, the repetitive behavior of an autistic subject can be interpreted as the sign of a defense process against anxiety (the psychoanalytic model), or as the sign of a serotonergic-system disorder (the neurobiological model) — but it is also the form taken by the autistic subject's existential relation to time. This singular relation to time is not, in itself, the sign of a disorder ; it is neither an indicator, nor an index, nor a symptom. It is the autistic form of the apprehension of time and space. One helpful way of representing this singular apprehension is to imagine a geological world in which time has been accelerated : mountains become mineral waves. If markers are placed on these mountains, they become temporal markers, clocks. Conversely, if time is slowed, the grains of an hourglass become motionless — they become spatial markers instead. If time slows sufficiently, fleeting forms take on lasting consistency (Guy, 2011).

Our model invites us to sketch the outlines of a phenomenological psychotherapy of autism, distinct from psychoanalytically inspired psychotherapies. The emotional experiences felt by the "embryonic self" are not linked to symbolic representations that can be analyzed in terms of verbal representations, nor in terms of a signifier/signified relation

— semiotically, they are indices. Interpretations offered in verbal language cannot reach the indexical traces of these experiences. Infra-verbal interpretations — mimicry, posture — are more effective, but remain represented at a level of complexity distinct from these early experiences. Nonetheless, the phenomenological approach we advocate cannot dispense with psychoanalysis : it requires the analysis of the therapist's own defenses against their own anxiety, so as to allow them to approach the autistic world with benevolent curiosity. It thus implies psychoanalytic training, complemented by a phenomenological sensibility of the kind initiated by Binswanger (1947).

The phenomenological approach centers on the singularity of the unfolding of autistic thought ; it does not aim at its normalization, but instead seeks, together with the patient, the conditions for their adaptation to an actual reality. It treats the lived experience of early childhood as already accomplished and unmodifiable — though partly reactualized within the emotional relationship with the therapist — and is primarily concerned with the orientation of the developmental trajectory, that is, with the realization of the autistic self. It requires the therapist to exercise a non-invasive vigilance regarding the way the autistic subject constructs their own regulations to maintain the structural stability of their self. The therapist must understand a stereotypy, or the pursuit of a play of light, a series of objects, or an on/off behavior, not as pathological manifestations but as singularities performing a regulatory function in the service of objective synthesis.

The psychotherapy session is organized around identifying the morphological and dynamic singularities sought out by the autistic person. The session reveals each autistic person's existential position — that is, the full set of regulatory modes established to sustain their intentionality. At appropriate moments, the therapist endeavors to support the intentionality that enables objective synthesis, through the establishment of joint attention, co-action where possible, or co-immersion where digital interfaces are used[23]. A phenomenological psychotherapy of autism consists in fully accepting the existence of autistic subjects endowed with a psychic organization distinct from that of non-autistic individuals — one

in which a complete transmutation occurs of the values we conventionally attribute to the human psyche. The communication of thoughts and emotional states is not strictly necessary for existence. Most autistic subjects have no need for such communicative exchanges. What they do need, however, is a non-invasive co-presence, respectful of their autism, and a response to their search for contact, which follows unprecedented paths. Phenomenological psychotherapy of autism consists in being-with, in understanding, in accepting the radical nature of this difference in existence — rather than seeking, at all costs, an interhuman communication that we should remember is often itself illusory and degraded within the empty speech of social convention. Therapeutic efficacy is not measured by the disappearance of autistic manifestations, but by their most harmonious possible integration into a life.

Phenomenological understanding of autism, always incomplete and tangential, is not, on its own, sufficient. The bringing-together, into a coherent whole handled with tact, of cognitive, behavioral, and psychoanalytic approaches seems to us the right path. But the phenomenological approach whose broad outlines we have tried to describe is the only one that allows for an intimate understanding of the autistic position in the world. If it does not always translate into an extension of the autistic person's contact with others, and sometimes appears merely to accompany their existence, this does not mean it is without value. The authentic accompaniment of an existence is, in itself, the highest task of any psychotherapy.

Conclusions

Our interpretation of autism is far from meeting all the criteria of coherence, unity, concision, and adequacy to observed facts that one is entitled to expect of a model (Delattre, 1979). But with autism, we are not confronted with the demand for a deterministic model, but rather with the necessity of a new way of seeing — a new theoretical space. We hope to have tangentially approached this objective. It is possible that new discoveries in neurobiology — particularly in molecular genetics — and in cognitive science (the neuronal implementation of consciousness) will ren-

der obsolete this or that assertion in this text, perhaps even the whole of it. But we strongly doubt the capacity of reductionist approaches, seeking to isolate a single gene, a single biological structure, or a single cognitive function, to erect a deterministic model of autism without directly confronting the question of complexity. We equally doubt the capacity of psychoanalysis, in the current state of its conceptual apparatus and clinical practices, to fully account for autism, whose deep determinism lies within the intermediate spaces between the psyche and its neurophysiological substrate. Nonetheless, a metapsychological renewal — a "neuropsychanalysis," or, better still, a "topology of complexity" — could open new perspectives.

In any case, whatever the fate of this essay, it will have been an attempt at coherence within a fragmented field. And if we accept the assertions presented here as valid, we can begin to become familiar with a new way of considering autism within the diversity of the human condition. Given the suffering of parents, and of the many autistic individuals left behind by our society, it may seem inconceivable that one could hold a positive view of the existence of autism. And yet we must consider that within the autistic person there exist unprecedented potentialities, ones that overturn our normative certainties about human relationships and our way of seeing the world.

References

- Abraham, F. A. (1989). *A Visual Introduction to Dynamical Systems Theory for Psychology*. Santa Cruz, CA : Aerial Press (Science Frontier Express Series).
- Asperger, H. (1944). "Die 'autistischen Psychopathen' im Kindesalter." *Archiv für Psychiatrie und Nervenkrankheiten*, 117, 76–136.
- Atlan, H. (1979). *Entre le cristal et la fumée*. Paris : Seuil.
- Auyeung, B., & Baron-Cohen, S. (2009). "Fetal testosterone and autistic traits." *British Journal of Psychology*, 100(Pt 1), 1–22.
- Baron-Cohen, S., Leslie, A. M., & Frith, U. (1985). "Does the autistic child have a 'theory of mind'?" *Cognition*, 21(1), 37–46.
- Baron-Cohen, S. (2003). *The Essential Difference : The Truth about the Male and Female Brain*. New York : Basic Books.

- Berthoz, A. (2009). *La simplicité*. Paris : Odile Jacob.
- Berthoz, A. (2013). *La décision*. Paris : Odile Jacob.
- Bettelheim, B. (1969). *The Empty Fortress : Infantile Autism and the Birth of the Self*. New York : Free Press. (French translation : *La forteresse vide*, Paris : Gallimard, 1969.)
- Bick, E. (1968). "The Experience of the Skin in Early Object Relations." *International Journal of Psycho-Analysis*, 49, 484.
- Binswanger, L. (1947/1971). *Introduction à l'analyse existentielle*. Paris : Éditions de Minuit.
- Bourgeron, T. (2007). "The Possible Interplay of Synaptic and Clock Genes in Autism Spectrum Disorders." *Cold Spring Harbor Symposia on Quantitative Biology*, 15(4), 432–437.
- Botte, M. C., & Canévet, G. (1988). *Psychoacoustique et perception auditive*. Paris : Éditions Inserm.
- Brentano, F. (1873/1944). *La psychologie au point de vue empirique* (M. de Gandillac, Trans.). Paris : Aubier-Montaigne.
- Bridgeman, B. (2010). "How the Brain Makes the World Appear Stable." *i-Perception*, 1, 69–72.
- Brown, R., & Hobson, R. P. (1997). "Are There 'Autistic-Like' Features in Congenitally Blind Children?" *Journal of Child Psychology and Psychiatry*, 38(6), 693–703.
- Bruneau, N., Roux, S., & Adrien, J. L. (1999). "Auditory Associative Cortex Dysfunction in Children with Autism : Evidence from Late Auditory Evoked Potentials (N1 Wave-T Complex)." *Clinical Neurophysiology*, 110, 1927–1934.
- Bryson, S. E. (1990). "Autism : A Developmental Spatial Neglect Syndrome?" In J. T. Enns (Ed.), *The Development of Attention : Research and Theory* (pp. 405–427). Amsterdam : Elsevier.
- Bullinger, A. (1989). "Locomotion, posture et manipulation manuelle chez l'enfant autiste." In G. Lelord, J.-P. Muh, M. Petit, & D. Sauvage (Eds.), *Autisme et troubles du développement global de l'enfant*. Paris : Expansion scientifique française.
- Charney, D. S., & Buxbaum, J. D. (Eds.). (2013). *Neurobiology of Mental Illness* (4th ed.). Oxford : Oxford University Press.
- Courchesne, E. (1988). "Physioanatomical Considerations in Down Syndrome." In L. Nadel (Ed.), *The Psychobiology of Down Syndrome* (pp. 291–314). Cambridge, MA : MIT Press.
- Courchesne, E. (1991). "Neuroanatomic Imaging in Autism." *Pediatrics*, 87 (Suppl.), 781–790.
- Courchesne, E., et al. (1988). "Hypoplasia of Cerebellar Vermal Lobules VI and VII in Autism." *New England Journal of Medicine*, 318, 1349–1354.
- Courchesne, E., & Pierce, K. (2005). "Brain Overgrowth in Autism during a Critical Time in Development." *International Journal of Developmental Neuroscience*, 23(2–3), 153–170.
- Constantino, J., & Zhang, Y. (2010). "Sibling Recurrence and the Genetic Epidemiology of Autism." *American Journal of Psychiatry*, 167(11), 1349–1356.
- Crespi, B. (2013). "Developmental Heterochrony and the Evolution of Autistic Perception, Cognition and Behaviour." *BMC Medicine*, 11, 119.
- Delattre, P. (1979). "Le problème de la justification des modèles dans le cadre du formalisme des systèmes de transformation." In *Élaboration et justification des modèles, applications en biologie* (Vol. 1, p. 98). Paris : Maloine.
- Damasio, A. R., & Maurer, R. G. (1978). "A Neurological Model for Childhood Autism." *Archives of Neurology*, 35, 777–786.
- DSM-IV. (1996). *Manuel diagnostique et statistique des troubles mentaux*. Paris : Masson.
- DSM-5. (2012). "Diagnostic Criteria for Autism Spectrum Disorder." *Journal of the American Academy of Child & Adolescent Psychiatry*, 51(4), 368–383.
- Ferenczi, S. (1919/1982). *Psychanalyse* (Œuvres complètes). Paris : Payot.
- Fitzgerald, M. (2004). *Autism and Creativity : Is There a Link between Autism in Men and Exceptional Ability?* Hove : Brunner-Routledge.
- Fombonne, E. (2007). "Epidemiological Surveys of Pervasive Developmental Disorders." In F. R. Volkmar (Ed.), *Autism and Pervasive Developmental Disorders* (pp. 33–68). New York : Cambridge University Press.
- Frith, U. (1989). *Autism : Explaining the Enigma*. Oxford : Blackwell.
- Gepner, B., Lainé, F., & Tardif, C. (2010). "Désordres de la constellation autistique : un monde trop rapide pour un cerveau déconnecté?" *Psychiatrie, Sciences Humaines, Neurosciences*.
- Gibson, J. J. (1966). *The Senses Considered as Perceptual Systems*. Boston : Houghton Mifflin.

- Gibson, J. J. (1977). "The Theory of Affordances." In R. Shaw & J. Bransford (Eds.), *Perceiving, Acting and Knowing : Toward an Ecological Psychology*. Hillsdale, NJ : Lawrence Erlbaum.
- Gibson, J. J. (1979). *The Ecological Approach to Visual Perception*. Hillsdale, NJ : Lawrence Erlbaum.
- Guo, H., Hu, Z., Zhao, J., et al. (2011). "Genetics of Autism Spectrum Disorders." *Journal of Central South University (Medical Sciences)*, 36(8), 703–711.
- Guy, B. (2011). "Penser ensemble le temps et l'espace." *Philosophia Scientiae*, 15(3).
- Haag, G. (1984). "Autisme infantile précoce et phénomènes autistiques, réflexions psychanalytiques." *Psychiatrie de l'enfant*, 27(2), 293–354.
- Haag, G. (1985). "La mère et le bébé dans les deux moitiés du corps." *Neuropsychiatrie de l'enfance et de l'adolescence*, 33(2–3), 107–114.
- Haag, G. (1990). "Approche psychanalytique de l'autisme et des psychoses de l'enfant." In P. Mazet & S. Lebovici (Eds.), *Autisme et psychoses de l'enfant*. Paris : PUF.
- Haag, G. (2000). "Mise en perspective des données psychanalytiques et des données développementales." *Neuropsychiatrie de l'enfance et de l'adolescence*, 48, 432–440.
- Hochmann, J. (2009). *Histoire de l'autisme*. Paris : Odile Jacob.
- Houzel, D. (1988). "Autisme et conflit esthétique." *Journal de psychanalyse de l'enfant*, 5, 98–115.
- Houzel, D. (1994). "Les modèles topologiques." In D. Widlöcher (Ed.), *Traité de psychopathologie*. Paris : PUF.
- Hobson, R. P. (1989). "Beyond Cognition : A Theory of Autism." In G. Dawson (Ed.), *Autism : Nature, Diagnosis and Treatment* (pp. 22–48). New York : Guilford.
- Husserl, E. (1929/1969). *Méditations cartésiennes*. Paris : Vrin.
- Husserl, E. (1935/1962). *La crise des sciences européennes et la phénoménologie transcendantale*. Paris : Gallimard.
- Husserl, E. (1950). *Idées directrices pour une phénoménologie* (P. Ricoeur, Trans.). Paris : Gallimard.
- Husserl, E. (2001). *Sur l'intersubjectivité* (Vols. I–II ; N. Depraz, Trans.). Paris : PUF.
- Ioan, J. (2005). *Asperger's Syndrome and High Achievement : Some Very Remarkable People*. London : Jessica Kingsley.
- Kanner, L. (1943). "Autistic Disturbances of Affective Contact." *Nervous Child*, 2, 217–250.
- Klin, A. (1993). "Auditory Brainstem Responses in Autism : Brainstem Dysfunction or Peripheral Hearing Loss ?" *Journal of Autism and Developmental Disorders*, 23, 15–35.
- Labruyère, N. (2006). *Approche neuropsychologique de l'autisme infantile : entre théorie de l'action et théorie de l'esprit* (Doctoral dissertation, Université Lyon 2).
- Lacan, J. (1966). "Le stade du miroir." In *Écrits* (p. 94). Paris : Le Seuil.
- Laplanche, J. (1987). *Nouveaux fondements pour la psychanalyse*. Paris : PUF.
- Laznik, M.-C. (2013). "Pulsion invocante avec les bébés à risque d'autisme." In G. Crespín (Ed.), *La voix : des hypothèses psychanalytiques à la recherche scientifique* (Cahiers de PréAut, No. 10).
- Lécuyer, R., & Streri, A. (Eds.). (2002). "Débats actuels sur la cognition chez le bébé." *Intellectica*, 34(1).
- Lefort, R., & Lefort, R. (2003). *La distinction de l'autisme*. Paris : Seuil.
- Le Moigne, J.-L. (1977). *La théorie du système général*. Paris : PUF.
- Le Moigne, J.-L. (1999). *La modélisation des systèmes complexes*. Paris : Dunod.
- Mahler, M. (1968/1973). *Psychose infantile*. Paris : Payot.
- Mahler, M. (1970). *On Human Symbiosis and the Vicissitudes of Individuation*. New York : International Universities Press.
- Manneville, P. (1991). *Structures dissipatives, chaos et turbulence*. Saclay : Aléas.
- Markram, K., & Markram, H. (2010). "The Intense World Theory — A Unifying Theory of the Neurobiology of Autism." *Frontiers in Human Neuroscience*, December 21, 2010.
- Marr, D. (1982). *Vision*. San Francisco : Freeman.
- Maturana, H. R., & Varela, F. J. (1973/1980). *Autopoiesis : The Organization of the Living*. In *Autopoiesis and Cognition*. Dordrecht : Reidel.
- Meltzer, D. (1975/1984). *Explorations dans le monde de l'autisme*. Paris : Payot.
- Morin, E. (1977). *La Méthode : La Nature de la Nature*. Paris : Le Seuil.

- Morin, E. (1990). *Introduction à la pensée complexe*. Paris : ESF.
- Mottron, L. (2006). *L'autisme, une autre intelligence*. Brussels : Mardaga.
- Ornitz, E. M., & Ritvo, E. R. (1968). "Perceptual Inconstancy in Early Infantile Autism." *Archives of General Psychiatry*, 18(1), 76–98.
- Ornitz, E. M. (1985). "Neurophysiology of Infantile Autism." *Journal of the American Academy of Child Psychiatry*, 24, 251–262.
- Pennington, B., & Ozonoff, S. (1996). "Executive Functions and Developmental Psychopathology." *Journal of Child Psychology and Psychiatry*, 37(1), 51–87.
- Petitot, J. (2002). "Eidétique morphologique de la perception." In J. Petitot, F. J. Varela, B. Pachoud, & J.-M. Roy (Eds.), *Naturaliser la phénoménologie : essais sur la phénoménologie contemporaine et les sciences cognitives*. Paris : CNRS Éditions.
- Pezard, L., & Nandrino, J.-L. (2001). "Paradigme dynamique en psychopathologie : la théorie du chaos, de la physique à la psychiatrie." *L'Encéphale*, 27, 260–268.
- Proust, J. (1998). "Intentionnalité." In O. Houdé et al. (Eds.), *Vocabulaire de sciences cognitives* (p. 213). Paris : PUF.
- Ramus, F. (2012, October 18). *L'hypothèse neurodéveloppementale de l'autisme* [Conference presentation]. Laboratoire des sciences cognitives et psycholinguistique, ENS/EHESS/CNRS, Paris.
- Reardon, S. (2013). "Bacterium Can Reverse Autism-Like Behaviour in Mice." *Nature News*, December 2013.
- Rinehart, N., et al. (2001). "A Deficit in Shifting Attention Present in High-Functioning Autism but Not Asperger's Disorder." *Autism*, 5(1), 67–80.
- Riva, D., et al. (2013). *Neurobiology, Diagnosis and Treatment in Autism*. See also "Advances in Autism Genetics : On the Threshold of a New Neurobiology," *Nature Reviews Genetics*, 9(5), 341–355 (2008).
- Rumelhard, G. (1995). "Histoire didactique du concept de régulation en biologie." In *La régulation en biologie : approche didactique, conceptualisation, modélisation*. Paris : Éditions INRP.
- Rutter, M. L., & Kreppner, J. M. (2001). "Specificity and Heterogeneity in Children's Responses to Profound Institutional Privation." *British Journal of Psychiatry*, 179, 97–103.
- Rogers, S. J., & Pennington, B. F. (1991). "A Theoretical Approach to the Deficits in Infantile Autism." *Development and Psychopathology*, 3, 137–162.
- Rosenthal, V. (2011). "Synesthésie et intermodalité." *Intellectica*, 55.
- Schank, R. (1975). *Conceptual Information Processing*. New York : American Elsevier.
- Schmitz, C., Martineau, J., Barthélémy, C., & As-saiante, C. (2003). "Motor Control and Children with Autism : Deficit of Anticipatory Function?" *Neuroscience Letters*, 348(1), 17–20.
- Seron, X., & Jeannerod, M. (Eds.). (1998). *Neuropsychologie humaine*. Brussels : Mardaga.
- Sherrington, C. (1906/1952). *The Integrative Action of the Nervous System*. Cambridge : Cambridge University Press.
- Spitz, R. (1965). *The First Year of Life*. New York : International Universities Press.
- Thom, R. (1990). *Apologie du logos*. Paris : Hachette.
- Trevarthen, C. (1989). "Les relations entre autisme et développement socio-culturel." In G. Lelord et al. (Eds.), *Autisme et troubles du développement global de l'enfant* (pp. 56–80). Paris : Expansion scientifique française.
- Tustin, F. (1972/1977). *Autisme et psychose de l'enfant*. Paris : Seuil.
- Varela, F. J. (1989). *Autonomie et connaissance : essai sur le vivant*. Paris : Seuil.
- Varela, F., Thompson, E., & Rosch, E. (1993). *L'inscription corporelle de l'esprit : sciences cognitives et expérience humaine*. Paris : Seuil.
- Virole, B. (1992). "Morphogenèse des stéréotypies motrices dans l'autisme infantile." *Sémiotiques*, 3, 31–62.
- Virole, B. (1994). "Typologie dynamique des stéréotypies motrices." *Neuropsychiatrie de l'enfance et de l'adolescence*, 42(4–5), 203–211.
- Virole, B. (1996/2006). *Psychologie de la surdit  * (3rd ed.). Brussels : De Boeck.
- Virole, B. (2006). "L'utilisation de la langue des signes avec des enfants autistes." *Perspectives Psychiatriques*, 45(3).
- Virole, B. (2011). *La complexit   de soi*. Paris : Charrielle   ditions.
- Virole, B. (2013). "La technique des jeux vid  o en psychoth  rapie." In S. Tisseron (Ed.), *Subjectivation et empathie dans les mondes num  riques*. Paris : Du-nod.

- Virole, B. (2015). *Éloge de la pensée autiste*. Paris : Éditions des archives contemporaines / Gordon and Breach. ISBN 9782813001818.
- Wells, A. J. (2002). "Gibson's Affordances and Turing's Theory of Computation." *Ecological Psychology*, 14(3), 141–180.
- Zilbovicius, M., et al. (2000). "Temporal Lobe Dysfunction in Childhood Autism : A PET Study." *American Journal of Psychiatry*, 157, 1988–1993.

[1] : The author's clinical work with autism began in 1984 as a psychotherapist at the Perray-Vaucluse psychiatric hospital, followed by work in day hospitals, pediatric hospital settings, and private practice, with a specialization in autistic children presenting comorbid hearing and visual impairments.

[2] : For Leo Kanner, infantile autism presented the following traits : (1) social isolation ("aloneness"); (2) a need for sameness; (3) repetitive behaviors; (4) atypical language; (5) specific talents. Hans Asperger described, in 1944, a form characterized by (1) a disturbance of contact (limited empathy and anticipation); (2) difficulties of communication and social adaptation; (3) intellectual achievements associated with uneven capacities.

[3] : On the history of autism and its relationship to psychoanalysis, see Hochmann, J. (2009). *Histoire de l'autisme*. Paris : Odile Jacob. More generally, autism has occupied a borderline position with respect to every current of psychoanalytic thought. Within the Lacanian current, attempts to understand autism have centered on the notions of the foundations of subjectivity in relation to symbolization (Lefort, 2003), and on the topology of the spaces within which the autistic subject moves.

[4] : For a review of this question, see "Débats actuels sur la cognition chez le bébé," ed. R. Lécuyer & A. Streri, *Intellectica*, 2002/1, No. 34.

[5] : Laplanche, J. *Nouveaux fondements pour la psychanalyse*. Paris : PUF, 1987, p. 129.

[6] : Psychoanalysis has inspired institutional therapies in day hospitals, where the "group envelope" of the care team is supposed, if not to "cure" autism, then at least to allow for more harmonious development. Used with discernment, and combined with educational care, these approaches have proven genuinely effective. Fetishized, however, they lead to a confusion of professional specialties, degrading into a pseudo-consensual interpretive discourse that conflates effects of meaning with reality, and functioning in a pathological mode (idealization of a master analyst or doctrine, the constitution of a "bad

object" — a particular parent, administration, or school of thought...).

[7] : The analysis of home videos of infants who later developed autism, and of therapy sessions with infants at risk for autism, has shown that these infants can respond positively to adapted prosodic inflections ("motherese"), thereby allowing for very early therapeutic interventions (Laznik, 2013). See the practices of the PREAUT association — www.preaut.fr.

[8] : Focused, exogenous attention — consisting of repeatedly detecting an element within the perceptual field over an extended duration (concentration) — remains intact in autistic individuals when the perceptual objects are physical objects unrelated to social life (no human representations, voices, or faces). Voluntary, endogenous attention, triggered by a cue appearing in peripheral vision, is deficient relative to non-autistic subjects, including for non-social stimuli. Autistic individuals show greater difficulty shifting from focal attention (foveal vision) on a small object to global attention (peripheral vision) on a scene containing several objects or a large object (Damasio & Maurer, 1978). They are slower to detect a global target when it is preceded by a local target. Attentional filtering, which requires the inhibition of distractors, is deficient. In Asperger-type autism, an attentional deficit resembling the impulsive manifestations of hyperactive children is observed.

[9] : The ADI (Autism Diagnostic Interview) is a semi-structured interview. Its questions distinguish qualitative impairments from developmental delays by assessing behaviors relative to mental age, identifying those that would be considered deviant at any age. The PEP-R is an inventory of behaviors and competencies designed to identify uneven and singular learning profiles.

[10] : For an introduction to complex dynamical systems, see Abraham, F. A. (1989). *A Visual Introduction to Dynamical Systems Theory for Psychology*. Science Frontier Express Series. On modeling issues, see Le Moigne, J.-L. (1999). *La modélisation des systèmes complexes*. Paris : Dunod. For a mathematical presentation, see Manneville, P. (1991). *Structures dissipatives, chaos et turbulence*. Saclay : Aléas.

[11] : On the question of neuromodulation, see "Neuroanatomie fonctionnelle et le problème des relations structures-fonctions," Le Moal, M., Tassin, J.-P., & Baron, J.-C., in *Neuropsychologie humaine* (X. Seron & M. Jeannerod, Eds.). Brussels : Mardaga, 1998.

[12] : For Alain Berthoz (2013), the capacity to make decisions cannot be localized to a single neuroanatomical

structure but is a fundamental property of the nervous system as a complex system.

[13] : Phenomena of synesthesia (confusions between the senses) do not arise at the level of isolated sensory modules, nor from "wiring" errors, but at a global, integrative level, delocalized across neuroanatomical structures. See, for a review, *Synesthésie et intermodalité*, Rosenthal, V., 2011.

[14] : Fechner's law, according to which sensation increases as the logarithm of stimulus intensity, is a typical expression of a regulation operating through the compression of the input signal, protecting the organism against excessively strong physical intensities.

[15] : At a singular point, a dynamical system is always structurally unstable : a small variation in control parameters can lead to qualitatively different regimes (bifurcations of developmental trajectories).

[16] : In 1992, the author proposed a typology and interpretation of the morphodynamics of motor stereotypies as the instantiation of elementary catastrophes linked to experiences of individuation (Virole, 1992).

[17] : This subsection owes much to the entire body of work of Jean Petitot. See in particular "Eidétique morphologique de la perception," in *Naturaliser la phénoménologie : essais sur la phénoménologie contemporaine et les sciences cognitives*, ed. J. Petitot, F. J. Varela, B. Pachoud, & J.-M. Roy. Paris : CNRS Éditions, 2002.

[18] : The semiotic constitution of the manual signs of sign languages provides a fine illustration of this : the morphology of many object-signs corresponds to an iconic imitation of the way the object is grasped (Virole, 1996, 2006, 2009).

[19] : This conception is closely related to enaction : "cognition, far from being the representation of a pre-given world, is the joint emergence of a world and a mind from the history of the various actions a being performs in the world" (Varela, 1993).

[20] : Husserl distinguishes the kinesthetic posture, which pertains to the *Leib* (lived body), from the anatomical posture, which pertains to the *Körper* (physical body).

[21] : Husserl writes : "Instead of 'I,' perhaps I should always say 'self'... In the reflection that continues without interruption, I find the self continuously identical across all its acts and states, which are given there in temporal extension as lived experiences of immanent time." For Husserl, the self is "the uninterrupted identical." See *Sur*

l'intersubjectivité (N. Depraz, Trans.), Vol. II, PUF, p. 503.

[22] : "The jubilant assumption of his specular image by the child at the infans stage, still sunk in his motor incapacity and nursling dependence, would seem to exhibit in an exemplary situation the symbolic matrix in which the I is precipitated in a primordial form, before it is objectified in the dialectic of identification with the other, and before language restores to it, in the universal, its function as subject." (Lacan, J., "Le stade du miroir," *Écrits*, 1966, p. 94.)

[23] : The trajectories of avatars in video games serve as highly appreciated spatiotemporal integrators for autistic individuals. In these universes, time is projected onto space in sequences bounded by save points. Digital interfaces are not palliative gadgets for communication difficulties, nor mere recreational activities, but cognitive systems endowed with a form of intelligence, allowing autistic individuals to exercise their intentions to act within a framework that is reactive, emotionally neutral, constant, and sensorially stimulating. They allow for the deployment of a compensatory intentionality within an immutable objective world. The images of virtual worlds render present the processes of objective synthesis (2½D or pseudo-3D images, and the centering of foveal vision through the activation of peripheral vision). See Virole, 2013.

Translator's note : This is a scientific English translation of the French original by Benoît Virole. Clinical and diagnostic terminology follows standard usage in English-language autism research (ASD, DSM-5, theory of mind, executive function, central coherence, joint attention). Psychoanalytic and phenomenological vocabulary (object relations, adhesive identification, precurrence, objective synthesis, the Leib/Körper distinction, the mirror stage) follows established English usage in these respective fields. Figures 1 through 6, referenced in the original as visual diagrams, are described in bracketed captions here in place of the images themselves.