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AUTISM, MOVEMENT, TIME AND THOUGHT E-MOTION MIS-SIGHT AND OTHER TEMPORO- SPATIAL PROCESSING DISORDERS IN AUTISM

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ABSTRACT

In this chapter we propose a new approach of autism called *E-Motion mis-sight and other temporospatial processing disorders*. According to our view, subjects with autistic spectrum disorders (ASD) present more or less disabilities, delays and deviances, to perceive and integrate environmental world’s sensory events *online* and to produce *real-time* sensorymotor coupling and adequate verbal and nonverbal outputs, from the beginning of their life. In other words, the environmental world is going and changing too fast for persons with ASD.

In the first paragraph, we present some biographical self-reports, clinical considerations and neuropsychological arguments, which open windows on the peculiar visual and visuo-motor world of autistic persons. In the second paragraph, we expose the available experimental results in favour of physical and biological motion integration disorders in autistic population, and we expose a first synthesis of our approach. In the third

paragraph, we review some results demonstrating other temporo-spatial processing disorders in ASD, and suggest some possible underlying neurobiological mechanisms of our *E-Motion mis-sight and other temporospatial processing disorders* hypothesis of autism, based on putative *multi-system temporal dissynchronization and functional disconnectivity*. We think that this approach, which is compatible with the major contemporary theories of ASD, may have new implications for the comprehension and new applications for the rehabilitation of these disorders. In the last paragraph, we propose some psychological and philosophical perspectives of our approach concerning with the *integration of movement and time in thought*, and the mind-brain relationships in autism in particular, and in human being in general.

Keywords: Autism; Asperger syndrome; autistic spectrum disorders; autistic continuum; high and low functioning autism; severity of autism; developmental neuropsychology; early signs of autism; self-reports of autistic adults; movement perception; visuo-postural coupling; sensorymotor integration; environmental movement; physical movements; biological motion; rapid movements; facial processing; facial movements; emotional expression; non emotional expression; verbal and emotional communication; *E-Motion mis-sight*; *mis-developmental cascades*; speech sounds processing; phonemes categorization; proprioceptive perception; online perception; real-time processing; temporal processing; temporospatial processing; slowing down; imitation; facial expression recognition; reeducation; neural synchronization; neural connectivity; *dissynchronization*; *disconnectivity*; *autistic personality*; *mind-brain dissociation*; thought in pictures; *thought in movement*; consciousness; intention; attention; mind-brain relationships.

INTRODUCTION

The american author of science fiction, Philip K. Dick, wrote in his book *Martian time-sleep* (1964) :

- (...) *There is a new theory of autism*, Dr. Glaub said. *It comes from Berghölzlei, in Switzerland. I wanted to talk with you about it because it may offer new perspectives for your son.*

- *I doubt*, Steiner answered. Dr Glaub did not seem to hear him and continued.

- *It supposes an impairment of duration perception in autistic individual, so that his environment is so accelerated that he cannot face it ; indeed, he is unable to perceive it adequately, exactly as if we were watching an accelerated TV program, in which objects would speed along so fast that they become invisible and in which sound is like an incomprehensible gibberish...do you understand ? Only a sharp gobbledygook. Following this new theory, we could place the autistic child in a room where slowed down filmed sequences are screened...do you catch it ? Sound and image would be slowed down, and they would be displayed so slowly that neither you nor me perceive any slight movement nor any human speech sound.*

- *Fascinating*, Steiner said with a tired voice. (...) ¹

Reality sometimes passes fiction. Fiction stays sometimes beyond reality. Fiction sometimes precedes and sometimes induces reality. Sometimes it is the opposite. In the book, Mars is a suburbs of the Earth, and autistic persons are concentrated in a camp on the red planet : hard fiction ; and Steiner, the desperate father of an autistic boy, commits suicide : black reality. Sometimes, fiction sticks to reality...

The aim of this chapter is to demonstrate that numerous persons with autistic spectrum disorders suffer a distortion of their *online* and *real-time* integration of environment. In other words, the environmental world is going too fast for persons with autism. Thus the idea born in Philip K. Dick's imagination some 40 years ago, despite it is science fiction, has something to do with the everyday life of autistic persons. Indeed, we will expose various biographical, clinical and experimental arguments showing that at least some children, adolescents and adults with autism suffer visual-motion processing disorders, as well as other spatio-temporal processing disorders occurring in auditory and proprioceptive modalities.

We will also try to show that this new developmental and neuropsychological approach has a major advantage in comparison with other contemporary theories of autism, in that it may open, in a relatively short future, new perspectives for the reeducation of perceptual and sensorimotor impairments of autistic persons, as well as for the rehabilitation of their emotional and verbal communication disorders.

But first we wish to draw a few general views on autism and treatment of autistic persons.

Kanner described the syndrome of infantile autism in 1943. Despite substantial progress since 60 years in the therapeutics of infants, children, adolescents and adults with autism and related disorders all around the world, we must admit that it still remains overall disappointing, insufficient in terms of quality and quantity. These insufficiencies are essentially due to a persistent lack of comprehension of the underlying physiopathogenic and psychopathologic mechanisms of autistic spectrum disorders (Tardif & Gepner, 2003 ; Gepner, 2005).

The enigma of autism itself persists for numerous *complexity reasons*, some of which are briefly summarized here :

- i) There is not *One Autism*, but a constellation of developmental disorders related to autism, named *autistic spectrum disorders* (ASD) (Rapin, 2002), also named *the great continuum of autistic disorders* by Grandin (1995).
- ii) There is not *One Cause* of autistic disorders, but a multiplicity of genetic, epigenetic and environmental (ante-, peri- and post-natal) risk factors affecting brain epigenesis (Changeux, 1983) and self-organization of brain and psychism (Bourguignon, 1981) of people with ASD (Gepner & Soulayrol, 1994).
- iii) Between these risk factors affecting the development of CNS and their behavioral consequences on one individual, there are numerous physiopathological mechanisms involving different levels of organization and integration (neurobiological, neurophysiological, neuropsychological) and

¹ Free translation of *Glissement de temps sur Mars* (Dick, 1964), the french version of the original *Martian time-sleep*, by the authors.

affecting numerous interconnected neurofunctional systems, territories and pathways (Waterhouse et al., 1996 ; Gepner, 2001).

- iv) There are also various psychological reactions (depression, anxiety, distress...) that influence personality and behavior of individuals with autism in one or another direction.
- v) Finally, there are various neurodevelopmental disorders associated with autism (mental retardation, epilepsy), as well as there exists clinical and neuropsychological overlap between autism and other neuropsychological or neuropsychiatric disorders, such as attention deficit with/without hyperactivity, language learning impairments – dysphasia, dyslexia - and obsessive compulsive disorders (e.g., Volkmar & Pauls, 2003 ; Tardif et Gepner, 2003).

Thus, improvement in the treatment and reeducation of persons suffering an autism spectrum disorder may emerge only after an increased comprehension of causes and mechanisms of ASD in general, and of their consequences on one individual in particular. We logically assume that a better comprehension of neurobiological, neurophysiological, neuropsychological and psychopathological mechanisms involved in ASD will soon or later lead to the conception of new, more specific, relevant and efficient treatments and intervention programs.

These general considerations about the persisting enigma of these very complex neurodevelopmental disorders were certainly a strong motivating background when we started our research on autism, some 15 years ago. As Milne et al. (2005) say in their recent review of motion perception disorders in autism, our group was the first to investigate motion perception in children with autism, particularly in those having low-functioning autism (LFA). These children that we meet in child day-care psychiatric units and in special schools force us not only to understand their neuropsychological impairments, but also to help them here-and-now in the best way and help their bewildered parents (Tardif & Gepner, 2003).

I. HOW WAS BORN THE IDEA OF MOTION *MIS-SIGHT* IN AUTISM?

Several arguments lead us to suppose that at least some individuals with autism may suffer visual-motion processing disorders affecting attention to visual-motion, and/or motion perception, and/or visuo-motor integration of motion. We called this visual-motion integration disorder : *E-Motion mis-sight* (Gepner, 2001, 2005; Gepner, Lainé & Tardif, 2005).

These arguments come from (1) clinical observations, (2) developmental psychopathology, (3) self-reports from adults with high-functioning autism, (4) adult neuropsychology, and (5) experimental cognitive neuropsychology.

(1) Clinical observations

What fascinates and/or disturbs almost everyone who ‘encounters’ persons with autism for the first time is the frequent contrast between their physical presence and psychological

absence. When observing them carefully, we discover rapidly that the 'autistic world' they live and move in is obviously different from our world.

Whatever is our grid of interpretation, we promptly come to think that they feel, process and interpret the world differently than we do. People exhibiting so peculiar manners and behaviors cannot be like us.

Sometimes, with some adults having an Asperger syndrome, or with some relatives of first or second degree of individuals with autism, we still feel subtle differences between them and us in facial speech, 'eye speech', empathy, emotional expression or social contact.

But what exactly is the difference? Who are the autistic persons, how do they (dys-)function? Since Kanner (1943), this question is still passionately debated. As a matter of fact, Kanner noted in almost all of the eleven children with autism several behavioral peculiarities, that are directly or indirectly related to movement processing (movement perception and integration, or action) and dynamic or static world's processing impairments, e.g., gaze and face avoidance; attraction or aversion for moving, spinning and rolling objects; attraction for details of objects, static forms, puzzles; motor clumsiness, awkwardness; sensorymotor (e.g., oculomotor, oculomotor, imitative) disorders; manual, gestural and postural stereotypes ... some of them having adaptative or compensatory functions.

All of these symptoms plead for a possible dissociation between movement and static vision, with a movement perception and integration deficit and a static over-vision (Gepner, 2001, for a review), as well as for a sensorymotor decoupling (Gepner & Mestre, 2002a). It should be noted that more than thirty years ago, Ornitz and Ritvo (1968) and Ornitz (1974) have already examined precisely the autistic perceptual inconstancy and suggested the existence of a deficit in sensorymotor integration, i.e., reciprocal modulation between sensory inputs and motor outputs, in children with autism.

(2) Developmental psychopathology

Given that physical and biological movement is ubiquitous from birth, and that it is crucial for the development of motor imagery, posture, gait and motor activity, as well as for that of imitation, and verbal and emotional interactions (Gepner, 1997, 2001 for review), it is not difficult to imagine the various possible drastic developmental consequences of early motion processing disorders in a baby.

Since two decades, several studies using family home movies identified a number of very early signs of autism in babies aged 0 to 24 months, in various aspects of development, i.e. perceptive (vision, audition, proprioception) and sensorimotor behaviors, verbal and non verbal communication, and socialization (Sauvage, 1988). Here, we describe early signs of autism that can be related to a possible visual-motion perception and/or visuo-motor integration deficit. Note that these early signs are sometimes very subtle, and that they are not systematically observed in babies who will present later an autistic syndrome (e.g. a desintegrative disorder).

In the first weeks of life, autistic babies may exhibit anomalies of gaze contact and ocular pursuit of moving objects or persons. Around 3 months of life, autistic babies show a deficit in attention to familiar persons, and poor facial expressions. Around 6 months, visual contact disorders may persist, with « empty gaze », squint, impression of blindness. At the same time, babies may exhibit atypical interests for their hands, details of objects, static forms, and a lack

of interest for moving games and objects. Between 6 and 12 months of life, autistic babies may show a deficit of imitation of facial expressions, a lack of interest for people ; they withdraw from social interaction ; simultaneously, they exhibit new self-stimulating sensory and sensorimotor behaviors, like fingers and hand flapping in front of their eyes. In the second year, autistic children may show a lack of visual attention (peripheric gaze) and joint attention, and peculiar interests for light sources, reflected light, shadows, wind in trees...(Sauvage, 1988).

In the domain of motor development, autistic babies may show disturbances in some or all of the milestones of development, including lying, righting, sitting, crawling and walking (Teitelbaum et al., 1998). Besides, they frequently exhibit postural adjustments disorders, a lack or a delay in anticipating attitudes, as well as in oculo-manual coordination, a lack of environment's exploration, and stereotyped behaviors like swinging, rocking, swaying (Sauvage, 1988 ; Leary and Hill, 1996 for a review).

To summarize, the first signs of autism concern with visual development, with a dissociation between movement vision (poor, deficient, aversive) and static vision (normal or even overdeveloped, with an attraction for details, as also found later in children and adults with HFA and Asperger syndrome, see Frith, 1989 ; Happé, 1999 ; Mottron, 2004). Secondly, while autistic babies may show a developmental delay (that we could name 'negative signs of autism'), they also show atypical self-stimulating visual and visuomotor behaviors, i.e., a deviant developmental trajectory (that we could name 'productive signs of autism'), some of which probably have an adaptative and/or compensatory value. Third, it is possible to consider the progression of autistic symptomatology during childhood as a succession of *mis-developmental cascades*, in which impairment of visual behaviors will secondarily disturb visuo-motor development, as well as communicative and social interactions between autistic child and his human and physical environment (Gepner, 2001 ; 2004 ; 2005 ; see also Figure 1 below). Finally, it is to be noted that all the autistic symptoms related to audition, which also widely contribute to language developmental disorders in autism, are missing in this description of early autistic signs. We will consider these elements below (see § III and Figure 2).

(3) Self-reports from adults with autism and other testimonies

Some adults with high-functioning autism gave us important testimonies about their inner world, some of which are directly related to movement processing, speed of changes, and compensatory strategies of sensory limitations.

For example, Donna Williams, a well-known adult with high-functioning autism wrote : *"The constant change of most things never seemed to give me any chance to prepare myself for them. Because of this I found pleasure and comfort in doing the same things over and over again. I always loved the saying, 'Stop the world, I want to get off'. Perhaps I'd been caught up in the spots and the stars at a time when other children kept developing and so I had been left behind. The stress of trying to catch up and keep up often became too much, and I found myself trying to slow everything down and take some time out... One of the ways of making things seem to slow down was to blink or to turn the lights on and off really fast. If you blinked really fast, people behaved like in old frame-by-frame movies, like the effect of strobe lights without the control being taken out of your hands"* (Williams, 1992, p. 39-40).

At the end of her book, Donna Williams added some 'outlines of language in my world' :

- *“Continuity. The reassurance that things will stay the same long enough to grasp an undeniable guaranteed place within the complex and moving situation around me.*

- *Blinking compulsively. To slow things down and make them seem like a more detached, and therefore less frightening, frame-by-frame film. Switching lights on and off very fast had an element of this, too.*

- *Switching lights on and off. Similar to above, but the clicking sound is an impersonal and graspable connection with things outside oneself, like bells and music. It gives the pleasure of sensation which almost all touch denies and provides security. The more patterned and predictable, the more reassuring”* (Williams, *ibid.*, p.189-190).

The main point for our topic is the difficulty for Donna to handle the constant change of most things and her behavioral strategies to slow down these things in order to catch them and prepare herself for them.

Another famous adult with Asperger syndrome, Temple Grandin, wrote : *“Minor sensory processing deficits heightened my attraction to certain stimulation (e.g. airport’s doors), whereas a greater sensory processing defect might cause another child to fear and avoid the same stimulus. Some of the problems autistics have with making eye contact may be nothing more than an intolerance for the movement of the other person’s eyes. One autistic person reported that looking at people’s eyes was difficult because the eyes did not stay still (...) Distorted visual images may possibly explain why some children with autism favor peripheral vision. They may receive more reliable information when they look out of the corners of their eyes. One autistic person reported that he saw better from the side and that he didn’t see things if he looked straight at them”* (Grandin, 1995, p. 73-75). As a spokeswoman of people with autism, Temple Grandin emphasizes peculiar visual behaviors of individuals with autism as a continuum between aversion and attraction for movement, according to the degree of sensory processing defect.

Listen also to what Raymond, the high-functioning autistic adult of the film *Rain Man* played by Dustin Hoffman, says to his brother (Tom Cruise) : *« Daddy says that I am an excellent driver !!! »*. In fact, everyone who watched the film remembers the poor performance of Raymond in driving a car : at a speed of a walking man, he drives his father’s car into the sidewalk of a casino in Las Vegas. This naïve sentence of *Rain Man* reminds us that individuals with autism could not become excellent pilots of racing cars.

When we turn to more anonymous persons, we can learn more about this problem of *motion mis-sight*. The mother of a low-functioning autistic adolescent told me : *« My daughter always had a problem with movements. She is afraid when people come close to her, she avoids all the situations in which things go quickly. Myself, when I have to drive a car, I never exceed 70 km/h, because over this speed I feel uncomfortable, so everyone horns at me. My mother never drove a car because she feels the same discomfort but to a worse extent, that is, she cannot exceed 40 km/h. For the same reason, she never watches TV and never goes to cinema, because everything goes too fast... »*. This kind of trans-generational example of visual-motion processing impairment is particularly interesting for our purpose, and may be not unique. One has to ask to parents whether their child presented this kind of peculiar visual behavior during their early development, otherwise one cannot learn it.

(4) Adult neuropsychology

Zihl, von Cramon and Mai (1983) reported the case of a woman aged 43 who suffered bilateral cerebral lesions affecting the lateral temporo-occipital cortex and the underlying white matter, which selectively affected her movement vision. The authors report that : « *The visual disorder complained of by the patient was a loss of movement vision in all three dimensions. She had difficulty, for example, in pouring tea or coffee into a cup because the fluid appeared to be frozen, like a glacier. In addition, she could not stop pouring at the right time since she was unable to perceive the movement in the cup when the fluid rose. Furthermore the patient complained of difficulties in following a dialogue because she could not see the movements of the face, and especially the mouth of the speaker. In a room where more than two other people were walking she felt very insecure and unwell, and usually left the room immediately, because 'people were suddenly here or there but I have not seen them moving'. The patient experienced the same problem but to an even more marked extent in crowded streets or places, which she therefore avoided as much as possible. She could not cross the street because of her inability to judge the speed of a car. 'When I'm looking at the car first, it seems far away. But then, when I want to cross the road, suddenly the car is very near'. She gradually learned to 'estimate' the distance of moving vehicles by means of the sound becoming louder* » (Zihl et al., 1983, p. 315).

By reporting this remarkable case, we obviously do not want to let the reader suppose that subjects with autism suffer a comparable brain damage as that affecting this 43 year-old woman. Neither do we pretend that this woman became autistic after her brain damage. However, this case study lets us measure the extremely important impact of a selective disturbance of movement vision affecting a young – previously normal - woman on her perceptual, sensorimotor, communicative and social behaviors. Because of her brain damage, her life became totally different in several points. Because of her sensory impairments, she started to avoid physical and social events, to isolate herself. We will see below what this woman and individuals with autism have in common. But it is already possible, based on this study case, to imagine the various developmental consequences of a visual-motion processing disorder affecting a baby from the very beginning of his life.

(5) Experimental cognitive neuropsychology

Autism spectrum disorders (ASD) are known as neurodevelopmental disorders marked by social interaction and verbal and non verbal communication impairments (Kanner, 1943; WHO, 1992; APA, 1994; Rapin, 2002 ; Volkmar & Pauls, 2003 ; Tardif & Gepner, 2003). Since human face is the primary and most powerful source of information mediating emotional and verbal communication as well as social interaction, it is not surprising that face processing has often and regularly been studied in autistic population for the past twenty-five years (Dawson et al., 2005, for a review). Indeed, a growing body of data demonstrated that individuals with ASD generally process various aspects of faces in a different way than typically developing and/or mentally retarded control subjects. Peculiarities have been shown in the processing of facial identity (Langdell, 1978; Volkmar et al., 1989; Boucher & Lewis, 1992; Davies et al., 1994; Klin et al., 1999; Rondan et al., 2003 ; Deruelle et al.; 2004) and emotional facial expression (Hobson, 1986a, 1986b; Hobson et al., 1988; Celani et al., 1999),

in lip-reading (de Gelder et al., 1991) and eye direction detection or interpretation (Gepner et al., 1996; Baron-Cohen et al., 1995). Studies using fMRI confirmed facial processing peculiarities in subjects with ASD (Critchley et al., 2000; Schultz et al., 2000).

Following this line of neuropsychological exploration, an important question was to know whether each aspect of face processing is impaired separately, or based on more general and basic impairments affecting the processing of environmental physical and/or social world. In order to answer to this question, two previous studies conducted by our group, concerning with various aspects of face processing (identity, expressions of emotion, eye direction detection and lip-reading) in young children and adolescents with autism, revealed that the aspects of faces which were the most difficult to process were specifically related to facial configuration, visuo-auditory association (as already shown with simple visuo-auditory stimuli by Martineau et al., 1992), and facial dynamics, i.e., lips' movements, eyes' movements and emotional facial expressions (Gepner et al., 1994; Gepner et al., 1996). These results suggested that difficulties of children with autism in processing faces were neither related to impairments in recognition of facial identity *per se*, nor to impairments in recognizing emotional aspects of faces *per se*, but rather to anomalies in the processing of facial movements (as well as of facial configuration and visuo-auditory cues).

In conclusion, all the arguments reviewed in this paragraph pushed us to investigate directly how subjects with ASD process visual movements, physical movements as well as biological ones.

II. PREVIOUS AND RECENT WORKS ON PHYSICAL AND BIOLOGICAL MOTION PROCESSING

In this paragraph, we review the available data related to physical and biological movements' processing disorders in subjects with ASD, and we propose a first synthetic neuropsychological view of autism called *E-Motion mis-sight*.

(1) Physical movements processing

As Milne et al. (2005) said, our group was the first, ten years ago, to assess directly visual-motion processing in children with ASD. In this first study, Gepner, Mestre, Masson & de Schonen (1995) explored postural reactivity to visually perceived environmental movements in children with autism and normal control children.

Vision is known to be an important source of information used in postural control. One of the most probant hypotheses in this context is the 'ex-proprioceptive' role of optic flow (Lee & Aronson, 1974). Since the body's displacements generate a global motion of the visual scene (i.e., an optic flow) across the retina, and this optical flow specifies the kinematic properties of the ongoing movements, Gibson (1979) established that these optical flow fields due to ego-motion serve to regulate and control a subject's self-orientation. It has been clearly demonstrated thirty years ago that humans make postural readjustments in response to an optical flow (Lee & Aronson, 1974). A number of studies have shown that young infants react posturally to movements in their visual environment as soon as they are able to stand

unaided (Butterworth & Hicks, 1977), and even earlier (Jouen, 1988), confirming that visual proprioception plays a major role in the control of stance.

Since children with autism have sometimes very poor verbal and/or motor performance, it was necessary to explore their visual-motion processing with a reflex-like paradigm, requiring no voluntary verbal nor motor answers, e.g., postural reactivity to motion vision.

In our first experiment, children with autism and matched control children of the same age were standing on a force platform, in front and at a viewing distance of 3.8 m of a screen, in a dark room. The force platform capted the lateral and antero-posterior movements of their center of gravity. On the screen were displayed circularly symmetric frequency modulated gratings, animated by contraction and expansion movements, that simulated a tunnel moving in the fore-aft axis more or less rapidly. Results of this study revealed that, contrary to normal control children, children with autism have a very poor postural reactivity to this kind of environmental movements (Gepner et al., 1995). This weak visuopostural coupling in autistic children may account for their motor disturbances (poor motor control, poor motor imitation, motor clumsiness...), and is a good example of perception-action coupling disorder in this population.

In a replication and extension study, we showed that postural reactivity of children with autism was particularly poor when *speed of movement* was high (slow movements inducing a small postural reactivity), whereas children with Asperger syndrome (the mildest autistic spectrum disorder) were reacting normally, and even maybe overreacting, to the same kind of stimuli. In other terms, visuo-postural coupling is deficient in children with autism, and conversely, children with Asperger rather show a visuo-postural over-coupling. Thus, visuo-postural coupling (and more generally, sensorimotor coupling) may be a good neuropsychological marker of autism, and possibly a good predictor of the severity of ASD (Gepner & Mestre, 2002a). Besides, the question of *speed of movement* was raised with this last study.

Indeed, speed of movement seems to be critical for children with ASD : Gepner (1997) showed that children with autism are impaired in the perception of moving small squares in central vision, and that their performance is all the less so as speed of moving points is high and direction is complex (i.e., less foreseeable).

As far as direction of movement is concerned, Bertone et al. (2003) also showed that high-functioning subjects with autism exhibit a deficit in the perception of second order radial, translational and rotational direction of movement.

Other works by Spencer et al. (2000) and Milne et al. (2002) showed that, when presented with a random dot kinematogram animated by lateral displacements, children with autism need higher motion coherence to detect movement than normal control children, i.e., they overall show less optokinetic nystagmus (OKN) than their controls. Mestre et al. (2002), using the same motion coherence paradigm but with a video-oculographic measure of OKN, showed that children with autism show particularly higher motion coherence thresholds than normal control children when speed of motion is high, thus confirming a rapid visual-motion integration deficit in autism. A reasonable interpretation of these results is a lack of spatio-temporal integration of singular points in a global coherent motion (a form of central coherence deficiency). These results are interpreted by all these authors as an argument of a dorsal stream deficiency, an interpretation being also confirmed by Pellicano et al. (2005).

Interesting finding on pursuit eye movements deficits in HFA children (Takarae et al., 2004) is also to be reported, which suggests a disturbance in the extrastriate areas that extract

motion information, or in the transfer of visual motion information to the sensorimotor areas that transform visual information into appropriate oculomotor commands. However, another group did not find smooth pursuit eye movements deficits in children with pervasive developmental disorders (Kemner et al., 2004).

(2) Biological motion processing

Our group was also the first to introduce a new paradigm, i.e., *facial motion*, in order to further investigate and reinterpret the vast literature on facial recognition in autism. Indeed, we showed that children with autism, comparatively to normally developing children, have relatively good performance in emotional and non emotional facial recognition tasks when facial expressions are displayed slowly on video (Gepner, Deruelle and Grynfeldt, 2001). We thus argued that children with ASD are impaired in rapid facial movements processing, and that this impairment could explain secondarily gaze avoidance and poor performance in emotional and non-emotional facial processing in these children (Gepner, 2004).

In a replication and extension study (Tardif et al., submitted), we demonstrated that children with autism recognize significantly more emotional and non-emotional facial expressions and exhibit more facial-vocal imitation when facial expressions and their corresponding vocal sounds are slowed down naturally or artificially than when they are displayed at normal speed or statically. Therefore, slowing down the speed of facial and vocal events enhances imitative and cognitive abilities in children with autism. Given that deviant and/or delayed imitation has been very well documented in individuals with autism for more than thirty years (see Smith & Bryson, 1994 and Williams et al., 2004, for reviews), even from the very beginning of their life (Zwaigenbaum et al., 2005), and particularly in the domain of facial expressions' imitation (e.g., Hertzog et al., 1989; Loveland et al., 1994; Rogers et al., 2003), this study by Tardif et al. is of particular interest for reeducational perspectives, and suggests the existence of a key-link between visual-motion processing disorders and deficits of imitation in autism (Gepner, 2001, 2005; Gepner et al., 2005).

Blake et al. (2003) showed that children with autism are impaired in the recognition of human movements (e.g., walking, running or jumping) displayed through animated lighting points, and that their performance was correlated to the severity of their autistic syndrome.

Finally, at the intersection of physical and biological motion processing, Castelli et al. (2002) showed in a TEP study that adults with high-functioning autism or Asperger syndrome have a hypoactivation of median prefrontal cortex and superior temporal sulcus (STS) when they have to attribute mental states to animated shapes, and a diminution of connectivity between extrastriatal cortex and STS.

(3) First synthesis

All these studies confirm that subjects with ASD have visual-motion perception and integration disorders, that we have named previously *visual-motion mis-sight* in autism (Gepner, 2001).

In particular, Gepner & Mestre (2002b) reached the conclusion that children with autism have a rapid visual-motion integration deficit. According to this hypothesis, some autistic individuals having major movement-processing disorders from early in their lives will avoid

rapid physical and biological movements (considered as aversive stimuli), thus disrupting secondarily social interaction. Some of these individuals, or some autistic persons having minor motion-processing disorders, will search for, habituate themselves to, and learn to handle and cope with such kinds of stimuli. To summarize, rapid visual-motion processing deficit constitutes a core neuropsychological marker of autism and secondarily accounts for the deficit in social interaction (Gepner & Mestre, 2002b; see also Gepner, 2004).

In order to integrate the various developmental consequences (*mis-developmental cascades*) of this disorder, we proposed to name it *E-Motion mis-sight* (see Figure 1).

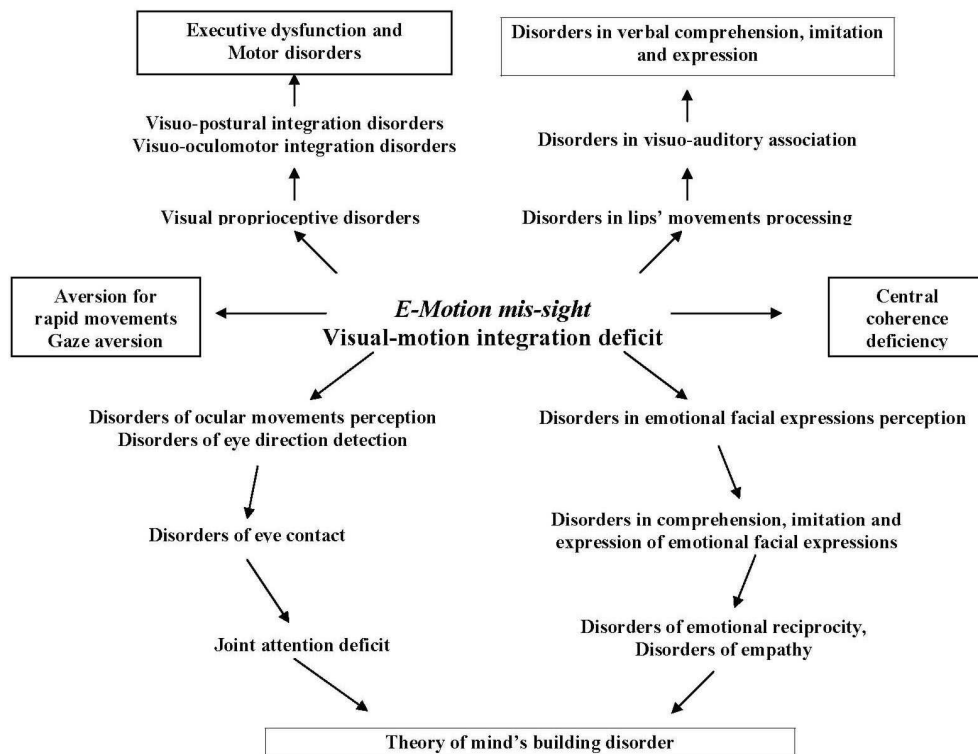


Figure 1. Cascades of neurodevelopmental consequences of a visual-motion integration deficit in autism

from Gepner, Lainé & Tardif (2005)

From a neurophysiological point of view, and given i) the implication of the magnocellular system pathway in processing visual motion (Livingstone & Hubel, 1988), ii) the role of visual inputs, and especially the dynamic ones, for visuo-motor control through mossy fibres via the pontine nuclei to the cerebellum (Stein & Glickstein, 1992), iii) the role of cerebellum in integrating multisensory inputs, and especially its role in speed and temporal coding of these inputs (e.g. Johnson & Ebner, 2000), iv) its role in motor control through real-

time fine-tuning of movement (e.g. Ito, 1984), and v) the co-contributing roles of cerebellum and basal ganglia for motor control as well as for learning (Doya, 2000), through their projections not only on the motor and premotor cortices but also on the prefrontal, temporal and parietal cortices (Middleton & Strick, 2000), we thus previously suggested (Gepner and Mestre, 2002a, 2002b) that visual magnocellular system, visuo-cerebellar pathways and cerebello-premotor-motor pathways might be centrally involved in the neurophysiopathology of at least a subclass of autistic spectrum disorders, and could explain the strange visuo-motor reactivity, as well as the strange cognitive style and higher-order cognitive peculiarities observed in this population. The discovery of hypoplastic or hyperplastic cerebellar vermal lobules VI and VII involving Purkinje cells in autistic adults (Courchesne et al., 1994), which supposes hypo- and/or hyperconnectivity according to a neuromimetic model (Cohen, 1994), gives a strong support to this suggestion. In the same line, it was found that a specific damage of the cerebellar vermal lobules VI and VII is responsible for a deficit in the accuracy of ocular saccades (Lewis and Zee, 1993), and thus may negatively impact visual motion integration.

Therefore, several neurofunctional systems and networks, including visual magnocellular system, dorsal stream and cerebellum, as well as superior temporal sulcus, which is involved in facial movements processing (Allison et al., 2000), are most probably involved in the physical and biological motion processing deficits in autism and need to be further investigated.

III. OTHER TEMPORAL PROCESSING DISORDERS IN AUTISM

Even though *E-Motion mis-sight* may account for some of the major sensorymotor, behavioral and communicative disorders manifested by children with ASD, it is however unable to explain autistic symptoms occurring in other sensory modalities (e.g. auditory or proprioceptive). A crucial question coming next was thus to know whether this visual-motion integration deficit reflects, or results from, a more primary and pervasive neuropsychological deficit. A plausible candidate-deficit concerns with *temporal processing* in various sensory modalities.

(1) Other arguments for temporo-spatial processing disorders in autism

In order to explore the effectiveness of a temporal processing deficit in autism in other sensory modalities, we tested, within a same group of 22 children and adolescents with ASD, the ability to extract a relevant information among a noisy stimulus *on line*, through three types of tasks :

a) oculomotor reactivity to visual-motion of a coherent pattern of lighting points, via the measurement of optokinetic nystagmus, b) speech flow perception and segmentation through categorization of simple and complex phonemes, and c) proprioception and motor anticipation in a bimanual load lifting task, through electromyographic and kinematic index. Results of this study were as follows (Gepner & Massion (directed by), 2002).

As already mentioned above (see Mestre et al., 2002), the group of subjects with ASD showed very weak oculomotor reactivity (i.e., a reduced occurrence of slow phase tracking eye

movements) to visual motion of a coherent pattern, and higher motion coherence thresholds (i.e., the necessity of higher percentage of points going in a direction out of the whole points, for inducing OKN) as compared to normal children of the same mean age, as already shown previously (Spencer et al., 2000 ; Milne et al., 2002). This deficit, which supposes a defect in rapid temporal analysis of visual motion stimuli embedded in noise, is a strong argument for a degraded temporo-spatial integration in the visual modality.

Secondly, the same group of autistic subjects showed a deficit in speech phoneme categorization. Indeed, compared to normal children who categorize an ambiguous phoneme such as MNA (made of an algorithmic superimposition of MA and NA) in a MA or a NA response randomly, autistic children over-categorize MNA in a NA response. This deviant over-categorization specifically appears in autistic subjects when speech phonemes are displayed at normal speed, whereas their phoneme category perception is normalized when phonemes are slowed down 2 times. This phoneme categorization deficit may partly be due to a difficulty in processing rapid speech flow, and thus to a temporal integration deficit in the auditory modality (Tardif et al., 2002). A similar temporal processing deficit has been found in children with language-learning impairments (Tallal, 1976); it has been related to a deficit in visual magnocellular system (Talcott et al., 2000) and/or auditory magnocellular system (Stein, 2001), and has also been found to be ameliorated by slowing down the speech flow (Tallal et al., 1996; Merzenich et al., 1996; Habib et al., 1999). As already said, autism and language learning impairments very probably share common physiopathologic mechanisms and neurofunctional pathways (Gepner & Mestre, 2002b). From a neurophysiological point of view, since superior temporal sulcus is involved not only in facial movement processing (Allison et al., 2000), but also in voice processing (Belin et al., 2000), and that it was found hypoactivated in adults with HFA in response to vocal sounds (Gervais et al., 2004), this region is of strong interest in the pathophysiology of ASD.

Finally, it also appeared that a subgroup of the same autistic subjects present a deficit in motor anticipation in a bimanual load-lifting task (Schmitz et al., 2002), a result confirmed by another study (Schmitz et al., 2003). This task requires the rapid processing of proprioceptive inputs, the correct use of an internal representation, and the precise timing adjustment of the muscular events. Compared to control children who use a feedforward mode of control to stabilize their forearm while lifting an object placed on it, autistic children mostly use a feedback mode of control, which results in slowing down their movement. In other words, autistic children are reacting instead of predicting. This deficit of accurate timing of anticipatory control could partly result from an impaired processing of proprioceptive inputs at least during the learning phase of the task, and thus from a temporo-spatial integration deficit in the proprioceptive modality. In this context also, impairments in the structure and/or function of cerebellum, which is crucial for temporal processing of sensory inputs and motor outputs (Massion, 1993, 1997), plays a major role

According to these three series of results, subjects with ASD have deficiencies in the temporal processing of visual, auditory and proprioceptive stimuli *on line* (Gepner and Massion (directed by), 2002). Altogether, these results suggest that subjects with ASD have a deficit in the temporo-spatial processing of sensory inflow which is necessary to detect and integrate visual motion, code and parse language or program postural adjustments (see Figure 2). To summarize, *the world is going or changing too fast* for at least some autistic persons. This view could account for the sensory and social avoidance of autistic subjects (when sensory inflow is aversive), for the desynchronization and discontinuity in their perception-

action coupling and sensory-motor tuning, as well as for their mis-understanding of, and disorders in their action and interaction with, the physical and human world.

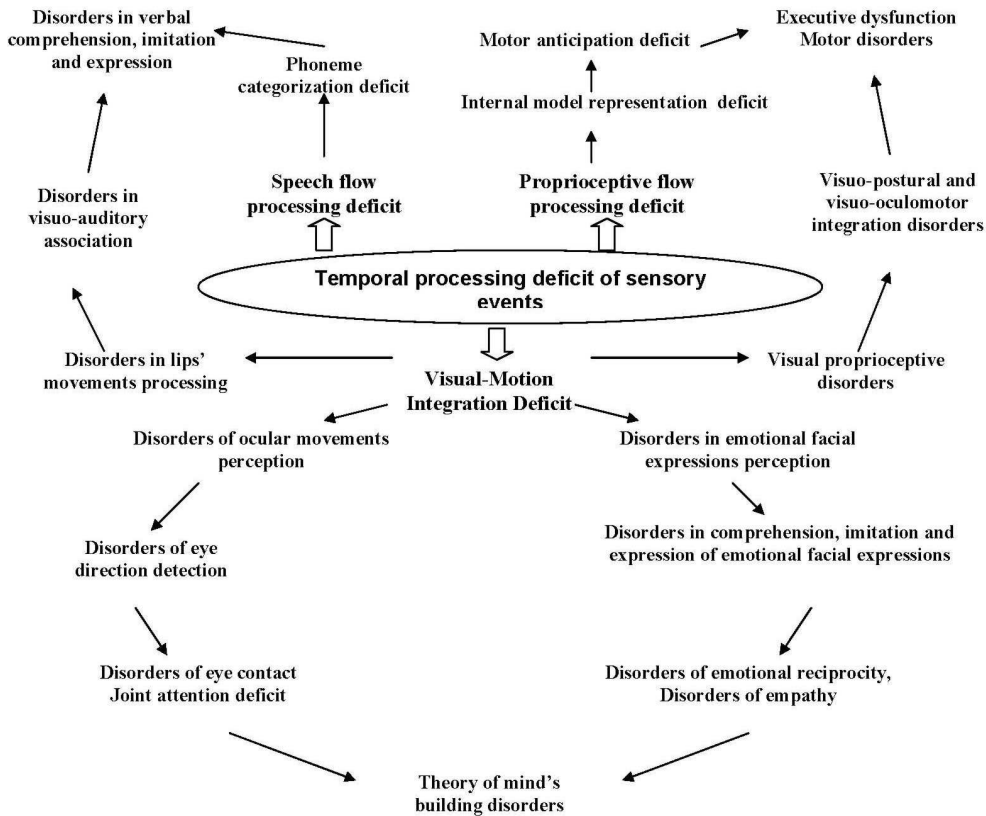


Figure 2. Cascades of neurodevelopmental consequences of a temporal processing deficit in autism

from Gepner, Lainé & Tardif (2005)

In conclusion, our temporal processing disorder hypothesis of autism has the strong advantage to account for the vast majority of developmental disorders seen in ASD, and to be compatible with the major contemporary approaches and theories of autism, such as the weak central coherence theory, the deficit of theory of mind, the theories of imitation deficit and executive dysfunction.

(2) Second synthesis: *E-Motion mis-sight* and other temporo-spatial processing disorders in autism

We showed that the various deficits affecting visual-motion, vocal sounds and proprioceptive processing in a same group of subjects with autism (Gepner & Massion (directed by), 2002), may be related to a more basic impairment, i.e., a deficit in the temporal processing of multisensory events. In order to take into account all the neuropsychological previous findings, we called our synthetic approach *E-Motion mis-sight and other temporo-spatial processing disorders* in autism. According to this approach, subjects with autism present more or less disabilities to perceive and integrate the environmental world's sensory events *online* and to produce *real-time* sensorymotor coupling, postural adjustments and adequate verbal and nonverbal outputs (Gepner et al., 2005).

At a neurobiological level, we can now extend our view on the neurophysiological pathways and neurofunctional processes which are most probably involved in our theory of visual-motion and other temporal processing disorders in autism: i) magnocellular system which conveys information on movement (optic flow), global form and low spatial frequency from retina to lateral geniculate nucleus and prestriate cortex (Livingstone & Hubel, 1988); ii) dorsal stream, which receives informations from magnocellular system and distributes them from prestriate cortex to temporal, parietal, prefrontal and frontal cortices, as well as to cerebellum and to thalamic mesencephalic and pontic structures (Boisacq-Schepens & Crommelinck, 1994). Among these connections, prestriate-cerebellar pathways are crucial since cerebellum plays a major role in speed and temporal coding of multisensory inputs (Johnson & Ebner, 2000) and has been found hypo- and/or hyperplastic in autism (Courchesne et al., 1994). Also important are the connections between prestriate cortex and superior temporal sulcus (STS), since STS is known for its role in biological motion processing (Allison et al., 2000); iii) cerebello-premotor-motor cortices loops, which are responsible with basal ganglia for real-time fine-tuning of motor outputs (Ito, 1984; Doya, 2000), and projections from cerebellum to prefrontal, parietal and temporal cortices which are responsible for motor and cognitive learning (Middleton & Strick, 2000).

This neurophysiological view of ASD has the strong advantage to take into account the whole complexity of CNS connectivity as well as a vast majority of neurological, anatomic and neuroimaging disorders found in ASD in the past; this view may thus bring interpretation guidelines for future research.

At another neurofunctional level, we proposed that this temporal processing anomaly may be related to a deficit in *temporal coding* (Gepner & Massion (directed by), 2002) of sensory inputs and motor outputs, in which cerebellum would play a central and crucial role (see above). More precisely, and according to Welsh et al. (2005), disturbances in inferior olive structure (and consequently in olivocerebellar pathways) seen in autism (Bailey et al., 1998; Kemper & Bauman, 1993) would disrupt the ability of inferior olive neurons to become electrically synchronized and to generate coherent rhythmic output, thus impairing the ability of individuals with autism to process rapid information, and therefore slowing their overall cognitive processing speed. According to this hypothesis, anomalous temporal synchronization of neural networks within each sensory modalities may consequently produce a deficit of temporal binding between multi sensory modalities.

Thus, the so-called *E-Motion mis-sight and other temporal processing disorders hypothesis* of autism (Gepner et al., 2005) may be connected to the *temporal binding deficit hypothesis* of autism (Brock et al., 2002) according which autistic individuals would suffer a deficit in synchronization of high frequency (30-100 Hz) gamma activity between local neural networks, and leading to the well accepted *central coherence deficiency theory* of autism (Frith, 1989; Happé, 1999). Interestingly, schizophrenic individuals show a reduced frequency of gamma-oscillation when viewing a Gestalt (Spencer et al., 2004), and the authors assume that this deficit in neural synchronization would be responsible for impaired attentional, perceptive and/or cognitive acts in these patients. In a recent study, however, individuals with autism showed an overall *increased* gamma-activity whilst identifying the presence or absence of an illusory Kanizsa shape (Brown et al., 2005).

We propose here that, according to the type of stimuli they are exposed to, subjects with autism will suffer desynchronization (e.g. for rapidly moving or changing sensory stimuli) and/or hypersynchronization (e.g. for static and permanent sensory stimuli) of neural networks. Besides, hypersynchronization may appear during development as an excessive compensatory mechanism of a previous desynchronization. In particular, hypersynchronization within and/or between local neural networks would fit well with the very frequent association between autism and clinical and/or infraclinical epilepsy (e.g., Hughes & Melyn, 2005).

Therefore, we suggest that a *temporal dissynchronization* (hypo- and/or hypersynchronization) within/between key neural networks and pathways, including especially visual (and auditory) magnocellular systems (Gepner & Mestre, 2002a, 2002b; Deruelle et al., 2004), dorsal stream (Spencer et al., 2000; Pellicano et al., 2005; Villalobos et al., 2005), cerebellum (Courchesne et al., 1994; Courchesne, 1997), mirror neurons (Oberman et al., 2005) and superior temporal sulcus (Gervais et al., 2004), leading to a *functional disconnectivity* (hypo- and/or hyper-connectivity, according to the neuromimetic model of Cohen, 1994) within/between these structures and numerous others, i.e. in sum, a *multi-system temporal dissynchronization and functional disconnectivity*, may be a crucial neuropsychological mechanism of ASD, responsible for attentional, perceptive, sensorymotor and/or cognitive impairments in this population. As an example, it was shown that adults with Asperger's syndrome have a delayed cortical activation from occipital cortex to STS, inferior parietal lobe and inferior frontal lobe, when imitating still pictures of lip forms (Nishitani et al., 2004), i.e., a deficit in cortical connectivity.

Recent works also confirm or suggest hypo-connectivity and/or over-connectivity in the brain of subjects with ASD (Chung et al., 2004; Courchesne & Pierce, 2005; Wickelgren, 2005).

This disconnectivity may be partly due to mutations of the X-linked genes encoding neuroligins NLGN3 and NLGN4, which are normally responsible for cell-adhesion and synaptogenesis (Jamain et al., 2003).

From a psychophysical point of view, since visual-motion is composed of temporal and spatial frequency informations ($V=TF/SF$, in which V represents speed of motion, TF represents temporal frequency and SF represents spatial frequency of a visual pattern), a visual-motion processing disorder may be due to a temporal and/or a spatial frequency processing disorder.

We showed above that a temporal processing disorder is most probably involved in ASD. Recently, a spatial frequency processing impairment has also been found in autism by Deruelle et al. (2004). These authors demonstrated that, contrary to normal control children, children with autism and Asperger syndrome rely more on high spatial frequency than on low spatial frequency information when presented with facial identity recognition tasks. Therefore, subjects with ASD do not only suffer temporal processing disorders, but rather temporo-spatial processing disorders. For example, it has been shown in two fMRI studies conducted in adults with HFA or Asperger syndrome that faces and voices were processed like objects and non vocal sounds respectively (Schultz et al., 2000; Gervais et al., 2004). Again our *E-Motion mis-sight* and other temporo-spatial processing disorders theory of autism may bring an explanation to these two results. Faces and voices are perceived oddly by subjects with ASD, may be not as human patterns *per se*, but possibly because of an impaired processing of dynamic and/or temporospatial characteristics of faces and voices. This interpretation has however to be further explored.

When interpreting with such a temporo-spatial processing hypothesis the results of Tardif et al. (submitted), which demonstrate that slowing down facial movements and corresponding vocal sounds enhances emotional and non emotional facial expression recognition and induces facial and vocal imitation in children with autism, we suggest that slowing down sensory incoming events would increase time for signal processing, temporal synchronization and sensorymotor coupling/tuning. Throughout time, slowing down may act on temporal processing like a (re)-synchronizing factor thus reinforcing perceptual and cognitive integration. Anyhow, the finding by Tardif et al. strenghtens *a posteriori* our temporospatial processing deficit theory of autism (Gepner and Massion (dir. By) 2002; Gepner et al., 2005). It is also the first evidence of the interest of slowing down the speed of visual and auditory environmental events around children with autism. If this finding was confirmed, it may potentially have two important consequences in the future, for diagnosis and reeducation. First, based on these new neuropsychological findings, it could be useful to build new instruments of assessment aimed at detecting the earliest signs in babies and young children with ASD; secondly, our finding should logically lead clinicians and care-takers to slow down environmental world around this population, by ecological and artificial means, and may therefore have a direct consequence for the reeducation of verbal and emotional communication impairments in children with autism, particularly if applied early during their development.

IV. PSYCHOLOGICAL, PHILOSOPHICAL AND ETHICAL PERSPECTIVES

In this last paragraph, we try to explore the possible consequences of this *E-Motion mis-sight* and other temporospatial processing disorders approach on the development and functioning of thought, and give our personal views on what could be called an « autistic personality ».

In this paragraph also, we propose an unorthodox approach of autism as an example of mind-brain dissociation, and propose a continuum between thought, language and act, in terms of degrees of energy and materiality.

(1) Autistic continuum and continuity of thought

When she describes her way of thinking, the famous autistic adult Temple Grandin says : « *I think in pictures. Words are like a second language to me. I translate both spoken and written words into full-color movies, complete with sound, which run like a VCR tape in my head. When somebody speaks to me, his words are instantly translated into pictures... When I read, I translate written words into color movies or I simply store a photo of the written page to be read later. When I retrieve the material, I see a photocopy of the page in my imagination. It is likely that Raymond, the autistic savant depicted in the movie Rain Man, used a similar strategy to memorize telephone books, maps, and other information. He simply photocopied each page of the phone book into his memory. When he wanted to find a certain number, he just scanned pages of the phone book that were in his mind. To pull information out of my memory, I have to replay the video. Pulling facts up quickly is sometimes difficult, because I have to play bits of different videos until I find the right tape. This takes time... When I am unable to convert text to pictures, it is usually because the text has no concrete meaning. Some philosophy books and articles are simply incomprehensible... All of my thinking is still in visual images, though it appears that thinking may become less visual as one moves along the continuum away from classic Kanner's syndrome... Another indicator of visual thinking is the remarkable ability many autistic people exhibit in solving jigsaw puzzles, finding their way around the city, or memorizing enormous amounts of information at a glance. My own thought patterns are similar to those described by A.R. Luria in *The Mind of a Mnemonist*. This book describes a man who worked as a newspaper reporter and could perform amazing feats of memory. Like me, the mnemonist had a visual image for everything he had heard or read* » ...

When reading this extraordinary description by Temple Grandin, one can understand the link between her sensory processing peculiarities and her way of thinking. That is, her sensory processing guides and influences the building and functioning of her thought.

What we logically propose here is that, parallel to a continuum in the severity of autism, which is correlated to the severity of the underlying sensory processing disorders, there is a correlated continuum of psychological consequences of these disorders.

For example, Temple Grandin, which has an Asperger syndrome, describes her way of thinking in dynamic or static pictures (photos). Following her description, and given our neuropsychological theory of autism, we can imagine that another person affected by a more severe autism, with more severe sensory processing disorders, will think in static pictures only, and not in dynamic ones.

We previously proposed that thinking in static pictures, i.e., without the continuity inherent to movement, may constitute a psychological marker of autism (Gepner, 2001). Thought of individuals with moderate to severe autism would function with a partial or total lack of continuity, like a strobe thought, a fractionated and fragmented thought. According to our view, there would be a continuum from *static thought* (thought in static pictures) to *dynamic thought* (thought in dynamic pictures), and thought in words and pure symbols. There would be a parallel between the degree of *E-Motion mis-sight* and the way of thinking.

The great french philosopher Henri Bergson wrote in « *L'évolution créatrice* (1941) : « *We take quasi-instant views of the reality, and since they characterize this reality, we just have to thread them along an abstract, uniform, invisible future, which is situated in the heart of the cognition's apparatus... Perception, intellection, language generally proceed like it.*

Whether it concerns with thinking the future, express it, or even perceiving it, we do not make anything else than activating a sort of inner cinematograph ».

For all the reasons mentioned above, we came to think that autistic persons have a problem with their inner cinematograph, and our view has a possible important heuristic impact on the relation between thought, time and movement.

We previously proposed a neuro-psychodynamic view of autistic thought (Gepner, 2001). According to this sketch, thought (thought on self, on others, on the world) is deeply inscribed in movement, i.e., in perceived movement, in memory of movement (Berthoz, 1997), in anticipated movement, and in reflex or voluntarily-produced movement. Following this, an early disorder of movement and time integration would logically schakle and fetter the developmental course of thought and consciousness of self and others. *Motion* (action of moving), *motility* (ability to move physically), *instinctual impulse*², as well as *emotion* (psychic movement, movement of one's being, movement of one's soul) : all these kinds of movement proceed from a same dynamic essence. Being unable to integrate adequately physical and human movement in their inner world, people with autism will be disrupted and disturbed in *feeling and expressing e-motion* and *thinking in movement*. Motion, the dynamics of the world, and continuity, which is co-substantial and inherent to motion, are wrongly integrated in the thought of autistic persons. Therefore, schematically, thought of autistic persons may function in separated transient pictures with no continuity between them.

In the same line, how do we suppose that functions a mind when the neural substrates are dissynchronized and disconnected, and cannot process the environmental world online ? How works a mind when the brain cannot *thread* here-and-now *the quasi-instant views of the reality along a uniform, invisible future*, as writes Bergson ? What means a temporal consciousness, the consciousness of time and duration when the brain does not function *on time*, on real-time ? Such a mind is likely to function more or less out of the ordinary time, in a peculiar and difficult to imagine dissynchronized, discontinuous, distorted and sometimes frightening time.

Living in this discontinuous time-and-space, and trying to survive in this temporally discontinuous and spatially fractionated world, numerous persons with autism self-stimulate themselves by moving their body or a part of it. That is, what appears as typical autistic symptoms, may be in fact a desperate compensatory strategy for feeling alive, dynamic, and for inscribing time and movement in their own body.

(2) Towards an “autistic personality”?

In this paragraph, we would like to ask some questions about the existence of an ‘autistic personality’. Does an ‘autistic personality’ exist ? Do autistic traits of personality, or traits of autistic personality, exist ? If so, what would be the quantitative and qualitative links and differences between pathologic autism and autistic personality ? Within the framework of autistic spectrum disorders, is there a seat for an ‘autistic personality’ ? Is there a continuum between the most severe autism and an ‘autistic personality’ ? Autistic spectrum disorders are known as neurodevelopmental disorders. At one extremity of the spectrum, we find severe

² *triebregung* in german, about which Sigmund Freud (1915) said : « We can only have in mind an instinctual impulse whose ideational representation is unconscious ».

and low-functioning autism. At the other extremity, we find mild and high-functioning autism and Asperger syndrome. But beyond this extremity, is it possible to find a milder autistic condition, based or not on very mild neurodevelopmental disorders, that we could call 'autistic personality' ?

One can reasonably consider that *personality* i) is the subjective psychological identity of a person, made of propensities, tendencies, potentialities ; ii) is the resultant of genetic, epigenetic, and environmental (educational, moral) factors and influences, and iii) that it is expressed via character, unconscious and conscious motivations, intentions, choices, but also through language, voice (frequency, loudness), facial expression, and of course, through behavior.

Given this, what could be an 'autistic personality' ? Let us consider that it could be made, at least, of aloneness, isolation or independence, abstract interests, contemplative tendency, and poor social sense. Let us then propose a scenario according which, from the first months of his life, a child has such a personality. If no big emotional trauma, no major accident nor other risk factors happen in his life, he may grow up with his autistic personality. If a trauma or a neurological illness occurs early in his life, and if this baby had genetic vulnerability or genetic abnormalities or other risk factors, this baby's personality may decompensate, and may evolve in one or another autistic spectrum disorder. This view has been previously proposed by Gepner & Soulayrol (1994), who used the concepts of epigenesis and self-organization in order to explain how the brain and psychism of an individual integrate intrinsic and extrinsic pathogenic factors (i.e., genetic, epigenetic and environmental factors) and how these conditions provoke the emergence of an autistic syndrome.

Although this scenario is speculative, it is anyhow similar to the well accepted psychopathological argument connecting a personality disorder with a mental illness, e.g., schizoïd personality and schizophrenia, obsessive personality and obsessive-compulsive disorders (OCD), or paranoïac personality and paranoïac psychosis.

(3) Mind-brain dissociation in autism?

Referring to his professor Babinski, who emphasized that all the organic or functional impairments of the central nervous system are translated in dissociated manifestations, the great neuro-psychiatrist Henri Baruk wrote : « *A long observation of mentally disturbed patients showed us that even with delirious, incoherent or incomprehensible patients, if one knows how to arouse their confidence and consider them with sympathy, one will discover that persists a deep personality, sensitive, which lost logical means to express itself, but which stays alive, vibrates and suffers... This deep personality that we studied for long time in our work on Moral Psychiatry (1945), it is the patient's soul* » (Baruk, 1985).

In what consists autism, this peculiar condition of life, revealed by a relative dissociation between self and others ? What are the psychological implications of our *E-Motion mis-sight and other temporospatial integration disorders* and *multi-system temporal dissynchronization and functional disconnectivity* approach of autism ?

According to this approach, persons with autism present at least the following disorders : temporal dissynchronization within/between various sensory modalities, temporal coherence deficit and central coherence deficit, sensorymotor decoupling, anticipation disorders, imitation disorders; verbal, nonverbal and emotional expression disorders; they present also

attention disorders (attention deficit and/or overfocused attention, according to the type of stimuli), and some degree of compensatory sensorymotor overcoupling.

In sum, *world coherence by simultaneity* and *consciousness by synchronization* are compromised in autism. Following this, we proposed that, at an extreme degree of autism, there exists a dissociation between mind and brain (Gepner, 2003; see also François, 1997). In the hardest way, autistic condition probably constitutes an extreme of mind-brain-body dissociation. In what meaning ?

Through Facilitated Communication (a still controversial method used in several physical and mental handicaps around the world), Pierre, an autistic adult, wrote about his movements and gestures : « *I had a big problem with synchronization and control of movement. I was like paralyzed. Shaking hand was very difficult. I used to compose the movement in my mind. I sent it frame-by-frame like in a silent film. I perceived my gestures like jerky, like frames arriving not enough rapidly. There was sometimes a very long time between my intention of movement and its realization. The mental impulse wants me to move, my body starts to vibrate like an electric pricking, and then everything stops. My body does not follow my will. I felt like I pushed a wall. I spoke to my body as if it was an alien person* » (in Vexiau, 1996). We were amazed by the similarity between this description and what an adult with Parkinson disease could say, i.e., by the striking difficulty of this adult to execute his will, to control his body with his mind, to connect his ideation with action ; indeed, there are several reports revealing similar motor impairments in these two neuropsychiatric disorders (Vilensky et al., 1981 ; Maurer & Damasio, 1982 ; Jones & Prior, 1985 ; Kohen-Raz et al., 1992 ; Hallet et al., 1993; Leary & Hill, 1996, for a review).

What else characterizes this mind-brain dissociation in autism?

As we saw in the previous paragraphs, world is going too fast for the brain of persons with autism. Therefore autistic persons have a *different consciousness of duration*, and need more time to negotiate (“*The stress of trying to catch up and keep up often became too much, and I found myself trying to slow everything down and take some time out...*”, Donna Williams (1992) claims). In that meaning, the fictive theory of autism proposed by Philip K. Dick (1964) in his “Martian time sleep” was a premonition fourty years ago. Some persons with autism have probably few or even no consciousness and idea of duration, with sometimes a strong degree of dissociation between their perception and integration of space and time. Time is most probably lived by some autistic persons like fractionated, erratic and distorted instants, without continuity and coherence, and space is lived like a puzzle of static pictures with no in-between relations. And if some other autistic persons succeed, and sometimes excel, in rassembling pieces of the puzzle, or if some others desperately produce continuity and a kind of reassurance through their motor stereotypes, all of them still live in their own -more or less open- private world.

The mind-brain dissociation hypothesis in autism supposes an increased relative autonomy of mind on the one hand, and brain-body on the other (Gepner, 2003). In our view, mind and brain of autistic persons function in relatively separated dimensions, with poor reciprocal influences between them. Mind integrates and controls poorly the cerebral and somatic activities. Mind can function at a more or less high level or proportion in a pure abstract world. “*Perhaps I'd been caught up in the spots and the stars at a time when other children kept developing and so I had been left behind*”, Donna Williams (1992) says.

(4) Consciousness and degrees of materiality

If this view has some reality, it has a lot to do with the work and philosophy of Sir John Eccles and his *interactionist dualism* (Eccles, 1994).

According to this famous and relatively forgotten nobelized scientist, the possibility exists that mind and brain interact together through quantic processes, and particularly that intention and attention, considered by Eccles and Karl Popper as immaterial psychic conscious events, increase the probability of exocytosis of a synaptic vesicle in the inter-synaptic space. As so, intention and attention control, or at least act on, brain and body, by inducing neural recruitment.

A very important recent finding came to credit this view : Lutz et al. (2004) demonstrated that buddhist monks have an increased gamma-activity during meditation comparatively to normal control adults, i.e., during meditative state, trained people exhibit more neural synchronization between distant groups of neurons than untrained people. In other terms, a voluntary mental activity (in the experiment of Lutz et al., monks meditated on *compassion*), influences cerebral organization and functioning. Varela and colleagues had already shown that a conscious act, such as the perception of a gestalt, is correlated to an increased gamma-activity in normal adults (Rodriguez et al., 1999 ; Varela et al., 2001). But what Lutz et al. (2004) demonstrate is the first clear correlation, to our knowlege, between a peculiar complex mental activity, meditation, itself related to a high degree of consciousness (attention, intention, concentration and motivation), on the one hand, and neural synchronization, on the other. Moreover, these authors demonstrate that the more the monks have mental practice, the more their brain is synchronized. This result is the first major argument in favour of the influence of mental or psychic activity on the brain.

Of course, we do not rule out the reciprocal influences of the brain activity on thought and mind, which are nevertheless still widely ignored, despite an abundant scientific and philosophic literature on the topic. We ourselves tried to contribute to the investigation on the *gap between brain and mind* with the concept of self-organization (Gepner & Soulayrol, 1994). However, pursuing a new line of theoretical and experimental research opened half a century ago by Eccles (1951), we propose to consider carefully his *interactionist dualism* as a plausible alternative to the dominant monist and materialistic interpretation of mind–brain relationships, according which consciousness is a mere by-product of brain. By postulating quantic mechanical effects at the core of brain functioning (Beck & Eccles, 1992), this approach puts cartesian dualism on the agenda of modern neurosciences and modern physics. Besides the three dimensions of space and the one of time, which set the stage of matter and brain functioning, there may be another one, more subtle, purely psychic “fifth dimension”. In this perspective, mind and brain distribute themselves on different discrete levels of energy or matter in such a way that, in a human life time, they confound themselves in case of normal neuro-psychic functioning, but dissociate and keep a relatively autonomous functioning in case of neuro-psychic illness (Gepner, 2003).

We know, since the first results by Rizzolatti et al. (1996), that thinking to something, seeing the same thing, or acting it, share common neural processes. In other words, there is a correspondance between thought, word and act. We suppose that what differs between these human brain-psychic activities is the degree of mental and neural activity devoted to the process, with an increasing neural recruitment and neural synchronization from thought to word and from word to act, i.e., an increasing degree of energy or matter put in the process.

In autism, the neural dissynchronization leads to an impaired temporal perception and integration of the world. Reciprocally, ideas, feelings, attention, intention, cannot be adequately converted in normal brain activity.

As a final conclusion, we hope that our neuropsychological approach can help clinicians, researchers and parents to understand more about physio-psychopathogeny of autistic spectrum disorders. We also hope that it can bring new ideas for the reeducation of persons with ASD. We finally hope that our views on mind-brain relationships can open new perspectives for thought, communication and care in human being.

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