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SCIENCE DIRECT
Brain and Cognition 61 (2006) 5–13
Brain and Cognition
www.elsevier.com/locate/b&c

Tactile sensitivity in Asperger syndrome

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Accepted 9 December 2005
Available online 24 February 2006

Abstract

People with autism and Asperger syndrome are anecdotally said to be hypersensitive to touch. In two experiments, we measured tactile thresholds and suprathreshold tactile sensitivity in a group of adults with Asperger syndrome. In the first experiment, tactile perceptual thresholds were measured. Two frequencies of vibrotactile stimulation were used: 30 and 200 Hz. The results demonstrated significantly lower tactile perceptual thresholds in the Asperger group at 200 Hz but not at 30 Hz, thus confirming tactile hypersensitivity but only for one class of stimulus. A second experiment investigated whether self-produced movement affected the perception of touch in a group of adults with Asperger syndrome. A suprathreshold tactile stimulus was produced either by the participant (self-produced condition) or by the experimenter (externally produced condition) and participants were asked to rate the perception of the tactile stimulation. The results demonstrated that, while both Asperger and control groups rated self-produced touch as less tickly than external touch, the Asperger group rated both types of tactile stimulus as significantly more tickly and intense than did the control group. This experiment confirms the finding of tactile hypersensitivity, but shows that the perceptual consequences of self-produced touch are attenuated in the normal way in people with Asperger syndrome. An abnormality in this process cannot therefore account for their tactile hypersensitivity.

Keywords: Perception; Sensory; Somatosensory; Autism; Tickling; Self; Other; Methods of limits; Hypersensitivity; Weak central coherence

1. Introduction

"*To be just lightly touched appeared to make my nervous system whimper, as if the nerve ends were curling up. If anyone hit on the terrible idea of tickling me, I died. It was so way beyond unbearable unbearableness that I simply died—or that's what it felt like.*" (Gerland, 1997, p. 38). "*I pulled away when people try to hug me, because being touched sent an overwhelming wave of stimulation through my body…Small itches and scratches that most people ignored were torture…When my mother scrubbed my hair, my scalp hurt. I also had problems with adapting to new clothing on my body.*" (Grandin, 1996). Despite these vivid autobiographical reports by individuals with autistic disorder, there is a surprising lack of empirical research on the sensitivity to touch in autism.

Hans Asperger, in his first description of autism, drew attention to the hypersensitivity of the senses, especially touch, smell and taste (Asperger, 1944; Talay-Ongan & Wood, 2000). Since then, hypersensitivity to touch has been reported extensively, mainly anecdotally, in people with autism. As shown in the examples above, people with autistic disorder and their carers report that they are intolerant of certain textures and find wearing certain materials aversive (see also Rogers, Hepburn, & Wehner, 2003; Willey, 1999). On questionnaires evaluating sensory perception, for example Dunn's Sensory Profile questionnaire, parents report that their autistic children overreact to cold, heat, pain, tickle and itch and avoid being touched by other people (Dunn, 2001; Kientz & Dunn, 1997).

A related concept is tactile defensiveness, which is characterised by behaviours such as rubbing, scratching, negative expressions, withdrawal, or avoidance in response to

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doi:10.1016/j.bandc.2005.12.013

tactile stimulation (Royeen, 1986). Tactile defensiveness is elevated in several developmental disorders including autism and is associated with enhanced response and slower habituation rates to a repeated tactile stimulus (Baranek & Berkson, 1994; Baranek, Foster, & Berkson, 1997). Thus, a lack of habituation in the neural pathways that normally occurs after being exposed repeatedly to a sensory stimulus is a possible explanation of tactile hypersensitivity. Some evidence for a failure to show response habituation to repeated stimulation in the visual and auditory domain has indeed been reported in autism (Barry & James, 1988).

Hypersensitivity may be the result of changes at one or more sensory processing stages, ranging from peripheral receptors in the skin, spinal synapses, the brain's perceptual system, through to cognitive or emotional processes. However, it is unknown at what level the hypersensitivity reported in autism occurs. The theory of weak central coherence (Booth, Charlton, Hughes, & Happé, 2003; Frith, 1989; Happé, 1996, 1999; Jolliffe & Baron-Cohen, 2001), proposes that in autism information processing is biased such that individual stimuli are well analysed but not integrated sufficiently into a coherent meaningful Gestalt. Thus, hypersensitivity could be due to impaired top-down modulation of incoming stimuli (Frith, 2003; U. Frith, 2003). Top-down modulation in the brain normally acts as a filter so that expected stimuli do not have to be processed as thoroughly as new stimuli. Such filters normally function to prevent informational overload. If this aspect of information processing was impaired in autism then incoming stimuli would all be processed as unexpected, resulting in enhanced sensitivity. This account might explain why there is a lack of habituation.

Other accounts of perceptual abilities in autism (Mottron & Burack, 2001; Plaisted, 2001; Plaisted, Saksida, Alcantara, & Weisblatt, 2003) suggest that there is enhanced processing of detailed stimuli (Bonnel et al., 2003; Plaisted, O'Riordan, & Baron-Cohen, 1998), or an over-development of low-level perceptual operations which causes detection, discrimination, and other low-level tasks to be enhanced (Mottron, Burack, Iarocci, Belleville, & Enns, 2003), without implications for global processing. These accounts too suggest a mechanism for hypersensitivity.

The main problem of these theories is that they would predict hypersensitivity to all perceptual stimuli. However, several studies have shown that enhanced discrimination, which may be a type of hypersensitivity, does not apply wholesale to all stimuli even within the same modality. In the visual domain, studies have revealed a specific deficit in the processing of magnocellular properties of motion stimuli (Milne et al., 2002; Spencer et al., 2000), which is not accompanied by a deficit in processing of parvocellular properties of form (Spencer et al., 2000). Spencer et al. (2000) interpreted these results as demonstrating a specific deficit of dorsal (but not ventral) stream functioning in autism. An alternative explanation for these results pertains to the "complexity" of the visual stimuli. In a recent study on sensitivity to visual motion stimuli in autism, Bertone, Mottron, Jelenic, and Faubert (2003) suggest that first-order (simple) and second-order (complex) neural processes need to be distinguished. Second order, or complex, stimuli are those requiring additional integration of information (central coherence), while first order, or simple, stimuli do not. Bertone et al. (2003) showed a dissociation in motion direction identification thresholds in autism according to the complexity of the visual motion stimuli. While individuals with autism had similar identification thresholds as control subjects for simple motion, they were less sensitive than controls for complex motion, which requires integration. Although there are not known to be separate neural pathways in the processing of tactile stimulation, in the domain of touch, many of the anecdotal reports are suggestive of hypersensitivity to certain tactile stimuli and not others.

In this study, we examined the perception of touch in individuals with Asperger syndrome (AS) and normal control (NC) participants. In the first experiment, we examined sensitivity to vibrotactile stimuli at two different frequencies (30 and 200 Hz). These two frequencies were chosen because they are known to stimulate different mechanoreceptors in the skin. High-frequency vibration (200 Hz) stimulates Pacinian corpuscles and activates FAII fibres, whereas lower-frequency vibration (30 Hz) stimulates Meissner corpuscles and activates SAI fibres. We wished to explore whether hypersensitivity would be found in people with autism within one or both of these neural systems. The first experiment was therefore designed to investigate whether people with AS have lower tactile perception thresholds to vibratory tactile stimulation, and to investigate the generality of any effect across different submodalities of stimulation.

2. Experiment 1 method

2.1. Participants

A group of participants with a diagnosis of AS ($N = 10$; 3 females) and a group of NC participants ($N = 9$; 7 females) took part in Experiment 1. Each participant in the AS group had previously received a diagnosis of Asperger syndrome from an independent clinician according to standard criteria (DSM-IV, APA 1994). All participants were right handed. Participants were questioned about their general health and were excluded if they were on medication or had a history of psychiatric or neurological illness. The mean age of the participants was 32.2 (±12.9) years in the AS group and 26.9 (±9.5) years in the NC group. There was no significant difference between the ages of the two groups ($t = 1.01$; $p = .33$). Assessments of Verbal, Performance, and Full-Scale IQ were carried out on seven of the AS participants using the eleven IQ subtests of the Wechsler Adult Intelligence Scale (WAIS-IIIUK; Wechsler, 1999a). Due to time constraints, two AS participants were assessed using a shortened form of the WAIS, and one was tested using the Wechsler Abbreviated Scale of Intelligence