Research Article

SELECTIVE IMPAIRMENT IN VISUAL PERCEPTION OF BIOLOGICAL MOTION IN OBSESSIVE-COMPULSIVE DISORDER

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Obsessive-compulsive disorder (OCD) is associated with a variety of well-documented cognitive deficits such as deficits in memory and executive functioning, but little is known about basic perceptual concomitants of OCD. This study investigated global, configural processing in OCD using dynamic (moving) and static stimuli with minimal demands on cognitive function. Twenty OCD patients and 16 age- and education-matched healthy control subjects were tested on four perceptual tasks: two motion tasks involved detection and discrimination of human activity portrayed by point-light animations ("biological" motion). The other two tasks involved detection of coherent, translational motion defined by random-dot cinematograms and detection of static global shape defined by spatially distributed contours. OCD patients exhibited impaired performance on biological motion tasks; in contrast, their performance on tasks of coherent motion detection and global form perception were comparable to those of healthy controls. These results indicate that OCD patients have a specific deficit in perceiving biological motion signals, whereas their perception of non-biological coherent motion and static global shape is intact. Because efficient social interactions depend on accurate and rapid perception of subtle socially relevant cues, deficits in biological motion perception may compromise social functioning in people with OCD. Depression and Anxiety 25:E15–E25, 2008. *Published 2007 Wiley-Liss, Inc.*†

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INTRODUCTION

Obsessive-compulsive disorder (OCD) is characterized by recurrent thought, intrusive visual imagery, impulses (obsession) and repetitive, ritualistic behaviors (compulsion), symptoms that obviously interfere with effective mental functioning and daily social behavior [American Psychiatric Association, 1994].

When studied using standard neuropsychological tests, patients with OCD exhibit a variety of cognitive and perceptual impairments, including deficits in memory and executive functioning [Boone et al., 1991; Head et al., 1989; Nelson et al., 1993; Savage et al., 1999; Shin et al., 2004]. Patients with OCD also have difficulty with high-level perceptual tasks, such as the Rey-Osterrieth

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Complex Figure Test, that involves encoding and then reproducing visual patterns [Savage et al., 1999].

Brain imaging studies of OCD patients point to functional and structural abnormalities in the fronto-subcortical neural circuitry that includes the orbitofrontal cortex, the anterior cingulate cortex, the caudate nucleus, and the basal ganglia. For example, significantly higher metabolic activity has been observed in the fronto-subcortical circuitry in patients with OCD, and this elevated activation is hypothesized to arise from disinhibition that, in turn, may be related to psychopathological symptoms such as inability to control reflexive responses and to regulate social behavior [Baxter et al., 1987, 1988; Benkelfat et al., 1990; Insel, 1992; Kang et al., 2004; Machlin et al., 1991; McGuire et al., 1994; Shin et al., 2006; Swedo et al., 1989]. As for the neural bases of perceptual impairments in OCD, there is no direct evidence implicating deficits in the sensory visual pathways. However, there are prominent neural connections linking visual cortical areas with the fronto-subcortical circuitry. For example, eye movement control is governed by fronto-subcortical circuitry that includes the frontal eye field (FEF), the supplementary eye field (SEF), the basal ganglia, and the superior colliculus. It is known that patients with OCD have deficits in smooth-pursuit eye movement (SPEM), in which the FEF and the SEF are significantly involved [Clementz et al., 1996; Lencer et al., 2004; Sweeney et al., 1992]. It is also well known that the FEF and SEF receive inputs from motion-sensitive visual areas MT (V5) and MST, suggesting the possible involvement of those areas in SPEM dysfunction. Another brain region involved in aspects of visual perception is the superior temporal cortex (STC), which has been implicated in perception of biological activity. The STC, too, is interconnected with the orbitofrontal cortex, the caudate, and the amygdala in the fronto-subcortical circuitry. Studies of the STC show that in OCD patients this structure exhibits unusually high levels of cerebral blood flow [Cottraux et al., 1996] and partial volumetric reduction [Choi et al., 2006]. It is natural to wonder, then, whether these abnormalities in STC might have consequences for the perceptual functions putatively subserved by that visual area.

Considered together, these neurobiological results led us to wonder to what extent visual motion perception might be compromised in patients with OCD. Movements of objects within the visual scene provide rich perceptual information about the three-dimensional shapes of those objects and their relative positions within the visual field [Sekuler et al., 2002]. Indeed, in some situations, motion can show the presence of objects that would otherwise be camouflaged [Regan, 2000]. In addition, motion information also can specify the actions and intentions of other people, an ability termed biological motion perception in the vision literature [Blake and Shiffrar, 2007: review]. This aspect of motion perception undoubtedly guides our everyday social interactions, and individuals with impaired biological motion perception could be seriously disadvantaged socially. Indeed, Kim and colleagues have previously reported a link between impaired perception of biological motion and reduced social functioning in schizophrenic patients [Kim et al., 2005]. Moreover, strong evidence exists for impaired biological motion perception in autism [Blake et al., 2003]; autism is also characterized by serious social dysfunction. Both schizophrenia and autism are associated with abnormal neural circuitry that includes the superior temporal and orbitofrontal cortices [e.g. Baron-Cohen et al., 2000; Shenton et al., 1992], which are also implicated in OCD.

There are similarities among autism, schizophrenia, and OCD. For example, autistic individuals, like OCD patients [Borkowska et al., 2003], often show increased obsessive and compulsive behaviors [Hollander et al., 2003; Russell et al., 2005]; likewise, more than a third of individuals diagnosed with schizophrenia experience clinically significant obsessive or compulsive symptomatology [Berman et al., 1995; Bland et al., 1987; Lysaker et al., 2004]. Furthermore, schizophrenia, autism, and OCD are all characterized by impaired social skills that compromise the quality of life for those individuals [Bystritsky et al., 2001]. Moreover, the extent of social impairment seems to be correlated with the severity of OCD symptoms [e.g. Koran et al., 1996]. Longitudinal studies show that residual social dysfunction and poor quality of life continue even after completion of symptom-focused treatments [Bystritsky et al., 1999; Hollander et al., 1996].

Given these similarities among these mental disorders as well as impaired visual motion perception in autism and schizophrenia, we felt it worthwhile to investigate motion perception in OCD patients using well-established visual stimuli and tasks. Finding deficits, if any, in visual motion perception in OCD will be meaningful in that it will aid to understand underlying common perceptual processing among major mental illness. In addition, related with social functioning and quality of life, it could be used to develop interventions for OCD patients aimed at achieving progress in those domains.

In this study, motion perception was assessed using two distinct types of visual stimuli: (1) random-dot cinematograms (RDC) that index visual sensitivity to simple, translational motion [Newsome et al., 1989], and (2) point-light (PL) animations that portray the kinematics of human activity [Johansson, 1973]. Successful performance in tasks deploying RDCs and PL animations requires integration of motion information over space and time. However, the hierarchical, pendular movements defining PL biological motion sequences are fundamentally different from the translational movements characteristic of RDCs. Moreover, converging lines of evidence from neurophysiological experiments in animals and from brain imaging studies in humans indicate that the neural events critical for

perception of coherent, translational motion differ from those involved in perception of biological motion [Blake and Shiffrar, 2007; Grossman et al., 2000; Sekuler et al., 2002]. It is entirely possible, therefore, that OCD patients could perform normally in one motion task but abnormally in the other.

In addition to the RDC and PL motion tasks, we also included in our test battery another task that assessed detection of visual shape defined by a set of unconnected contours appearing within a larger field of similar-sized contours unrelated to the shape of the test object [Kovacs and Julesz, 1993]. Successful performance on this task requires integration of spatially distributed, local features appearing among distracting elements, a characteristic common also to the two motion tasks we employed. However, this shape detection task employs stationary features and, hence, does not require motion information. This global form task is every bit as challenging as the motion tasks we employed and, thus, provides a reasonable marker for potential differences between OCD patients and normal controls in their abilities to sustain attention and remain motivated during these somewhat boring psychophysical tests. For all of the visual tasks used in this study, we have good evidence concerning the neural mechanisms underlying normal performance on those tasks; that evidence will be reviewed in the discussion.

MATERIALS AND METHODS

SUBJECTS

Twenty outpatients (eight females) were recruited from the Seoul National University Hospital Obsessive-Compulsive Disorder Clinic, Seoul, Korea. All patients met the DSM-IV criteria for OCD. Eighteen out of 20 were under medication at the time of testing: sertraline ($n = 4$, average daily dose = 175 mg), citalopram ($n = 6$, 40 mg), fluoxetine ($n = 5$, 52 mg), fluvoxamine ($n = 2$, 200 mg), risperidone ($n = 5$, 0.8 mg), olanzapine ($n = 1$, 10 mg), clonazepam ($n = 14$, 0.7 mg), valproic acid ($n = 1$, 375 mg), and lamotrigine ($n = 1$, 50 mg). The Yale-Brown Obsessive-Compulsive Scale [Y-BOCS; Goodman et al., 1989a,b] was used to assess symptom severity.

Sixteen age- and education-matched healthy controls (five females) were recruited from the community. Control subjects were assessed for a history of mental illness or neurological disorders, and none were receiving any kind of psychotropic medications. All subjects had normal or corrected-to-normal vision, gave written informed consent, and were paid for their participation. The Seoul National University Institutional Review Board approved the study protocol. Demographic information is summarized in Table 1.

TABLE 1. Summary of demographic information

	OCD ($N = 20$)	Control ($N = 16$)
Age (years)	24.3 (6.2)[a]	23.2 (5.8)
Education (years)	13.9 (2.1)	14.6 (5.8)
Sex (M/F)	12/8	11/5
IQ	111.7 (11.5)	113.9 (11.9)
Obsession (YBO)[b]	11.5 (5.3)	N/A
Compulsion (YBC)[b]	10.8 (4.6)	N/A
Total (Y-BOCS)[b]	22.4 (9.1)	N/A
Handedness (L/R/Bi)	0/20/0	0/16/0

[a]Mean (*SD*).
[b]The Yale-Brown Obsessive-Compulsive Scale.
OCD, obsessive-compulsive disorder; L, left; R, right.
N/A, not applicable.

STIMULI AND PROCEDURE

All four visual tasks were presented on a TFT-LCD monitor (LG Electronics, Seoul, Korea) controlled by a Macintosh computer (Apple Inc., Cupertino, CA); two tasks were controlled by Matlab© (Mathworks Inc., Natick, MA) and the Psychophysics Toolbox [Brainard, 1997], and the other two tasks were run from application programs. All experiments were conducted in a dimly lit room at a viewing distance of 57 cm.

Two of the tasks involved tests of perception of biological motion involving a human activity portrayed by PL animations created in the following way. Twenty-four familiar human activities such as running and jumping were captured by video-recording an adult engaged in each of those activities. Video clips were digitized and stored in a computer where they were converted into PL animations portraying these various biological motion activities. To produce successive frames of these animations, small dots were placed on the joints of the body (ankles, knees, hips, wrists, elbows, and shoulders) and the top of the head in each and every frame (12 dots/frame). These successive frames were then compiled into an animation that, when played at the appropriate frame rate, produced smooth apparent motion among the dots. Twenty-four scrambled motion sequences were also created by randomizing the spatial locations and temporal phases of the 12 dots in each of the biological animations. This scrambling procedure perturbed the hierarchical, pendular motions characterizing biological motion, yet maintained the same local motions of individual dots as those used in the unperturbed sequences (Fig. 1B). Unless otherwise stated, the entire cluster of 12 dots defining a human figure fell within a square region subtending approximately 7° on a side.

Task 1: Biological motion detection. This task used a two-alternative, temporal forced-choice procedure to assess detectability of biological motion superimposed within a field of moving "noise" dots [details are given in Ikeda et al., 2005]. On each trial, the observer viewed two successive, 1-sec intervals (separated by a 0.5-sec blank interval) in both of which a variable number of moving black dots appeared centered on a fixation point. In one of the intervals, a subset of the dots defined a PL biological motion activity and in the other interval the scrambled version

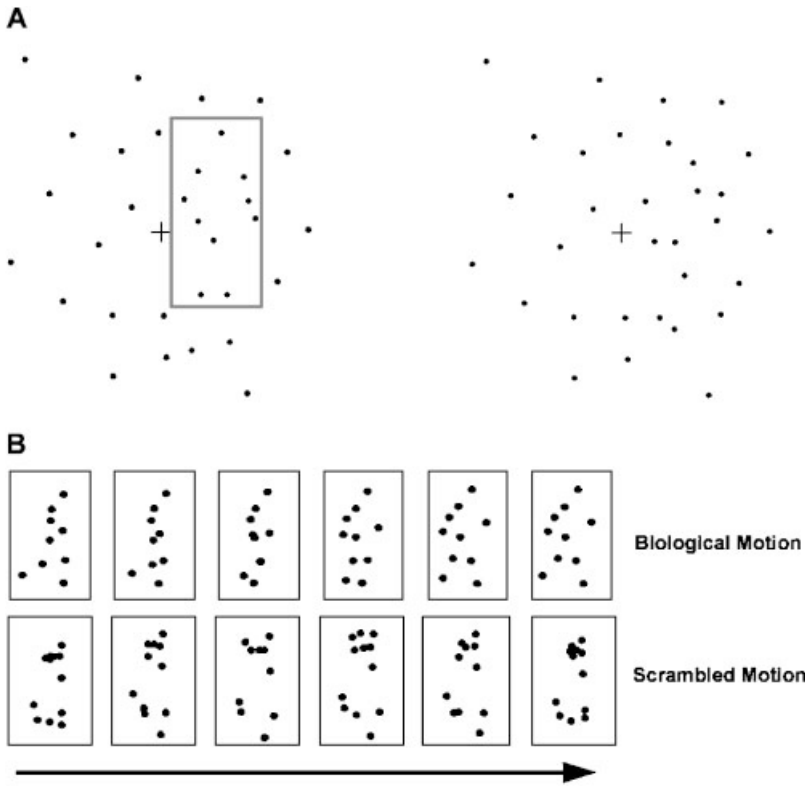

Figure 1. Biological motion tasks. (A) An example of biological motion detection task (Task 1). (B) An example of biological motion sequence and its scrambled (non-biological) counterpart (Task 2).

of that animation was presented; the interval, first or second, containing the biological sequence was randomly determined in each trial. In both intervals, a fixed number of "noise" dots was also presented, with the motions of those dots corresponding to the motions of the dots defining the biological sequence presented on that trial (e.g. dots defining a "walking" figure were also used to produce the noise dots that masked detectability of that figure). The locations of individual noise dots were spatially randomized within the virtual rectangular region within which all dots appeared. The dots defining the biological figure and those defining the scrambled set corresponding to that figure did not always fall at exactly the same location relative to fixation but, instead, appeared anywhere within 80 pixels of fixation. This spatial jittering maneuver made it impossible to monitor just a small subset of dots to judge which interval contained the biological sequence; the task required global integration of dots over space and time, and differentiation of those dots from the noise dots [Bertenthal et al., 1984]. The difficulty of the task, therefore, varied directly with the number of noise dots presented. The size of each dot was 5-arc min, and the average speed within a sequence was 4°/sec. The entire array of dots appeared within a virtual square region approximately 11° on a side.

On each trial, the subject indicated which of the two intervals contained a biological motion sequence, guessing if necessary. Auditory feedback was provided after each incorrect response. The number of noise dots presented on a given trial was governed by the subject's performance on the previous trial(s), according to a two-up/one-down staircase rule that converges onto the noise level producing approximately 71% correct detection performance. The staircase was terminated after 16 reversals in the direction of the staircase, and the threshold was calculated as the average number of noise dots over the last six reversals of the staircase. Previous studies confirm that these staircase parameters provide reliable measures of detection performance [Grossman et al., 2004].

Task 2: Discriminating biological motion from non-biological motion. The forced-choice procedure used in Task 1 required comparison of stimulation in two successive intervals. One might worry, therefore, that the task placed demands on working memory, which could disadvantage people with OCD. To get around this possibility, we employed a discrimination task comparable to one used in previous studies on schizophrenia, autism, and healthy subjects including young children [Blake et al., 2003; Friere et al., 2006; Grossman and Blake, 1999; Kim et al., 2005].

For this task, subjects viewed a single, 1-sec motion sequence that portrayed either biological motion or scrambled biological motion; on no trials were noise dots presented. Immediately following each presentation, the subject pressed one of two pre-assigned computer keys to indicate whether that sequence portrayed biological motion or scrambled motion, guessing if necessary; auditory feedback informed the subject about the correctness of his/her response. The entire experiment consisted of a series of 100 trials in which half were biological and half were scrambled, with the order randomized for each subject. Nineteen out of 20 patients and all normal controls finished this task; the one remaining patient did not participate in this task for reasons of her own, but participated in the other three tasks.

To index performance on this discrimination task, we counted the number of "hits" ("biological" response to biological motion sequence) and "false alarms" ("biological" response to scrambled motion sequence), and used these hit-rates and false alarm-rates to compute an unbiased measure of sensitivity, d' [Macmillan and Creelman, 1991]. To reiterate, subjects viewed a simple, 12-dot animation and responded immediately following offset of the stimulus, thus obviating potential limitations in working memory capacity.

Task 3: Coherent motion detection. This task measured the detectability of weak, translational motion within a field of dynamic noise dots, a widely used task in studies of motion perception [Braddick et al., 2001; Chen et al., 2003b; Hiris and Blake, 1995; Newsome et al., 1989; Rizzo and Nawrot, 1998; Wattam-Bell, 1994]. We used a two-alternative, spatial forced-choice procedure wherein both stimulus alternatives are present simultaneously; this procedure, like the discrimination procedure used in Task 2, places minimal demands on working memory (Fig. 2).

On each trial, two arrays of moving dots (black dots against a white background) were presented on either side of a central fixation point. Each array of dots fell within a virtual circular region 6° in diameter, and the nearest borders of the two arrays were separated by 3° on either side of the fixation point. Each of the two arrays always comprised 100 dots, and all dots on both halves of the display were the same size (3-arc min) and moved at the same speed (1.2°/sec). Dots in one of the two arrays moved entirely randomly with respect to direction ("noise" dots only), and dots in the other array consisted of noise dots together with a variable percentage of "signal" dots, all of which moved upward; the left/right location of the signal-plus-noise dots varied randomly over trials. On each trial, the subject indicated by key press which dot array—left or right—contained signal dots (coherent motion), guessing if necessary. The percentage of "upward signal" dots was varied over trials according to a two-up/one-down staircase procedure, which started with 50% signal dots. The staircase was terminated after 10 reversals in the direction of the staircase, and the average percentage of coherent signal dots over the last four reversals of the staircase provided the estimate of threshold.

Figure 2. Coherent motion task (Task 3). One of the two displays has a group of dots moving coherently upward.

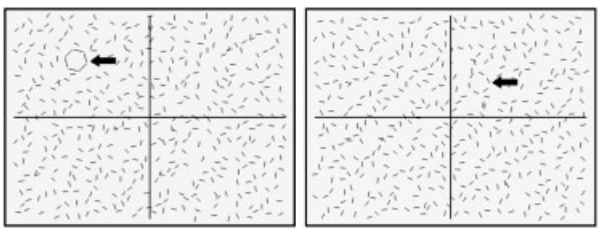

Figure 3. Static global form task (Task 4). Left: an example of an easy trial (i.e., jitter = 0). Right: an example of a more difficult trial.

Task 4: Global form perception. This task employed a four-alternative, forced-choice task, and it measured the ability to group small, stationary line elements into a larger, global form (Fig. 3). The screen was divided into four, equal-sized quadrants, and was filled with short lines most of which were oriented randomly. Each line subtended a visual angle approximately 30 min length × 2 min width. In one of the four quadrants, a group of six lines formed a quasi-circular shaped "figure" within a randomly selected region of the quadrant, with equal probability of this "figure" appearing in any quadrant. All line elements in the screen were identical in color and size, leaving only the spatial arrangement of the locally distributed lines as a cue for detecting the target. Task difficulty was manipulated by changing the clarity of the target. For this, we introduced "jitter" in the orientation of each line forming the quasi-circular shape. Jitter comprised an angular deviation among target contours defining the shape. Therefore, larger degrees of jitter degraded the clarity of the target. The visual angle of the target was approximately 2.5°. Displays remained visible until the subject responded.

The subject's task was to indicate the quadrant in which the target appeared by pressing one of four computer keys. The key assignment was spatially matched to the arrangement of quadrants in the screen. The test began with trials of easy detection (jitter = 0) so that subjects quickly became accustomed to the task. For this reason, we did not provide practice trials

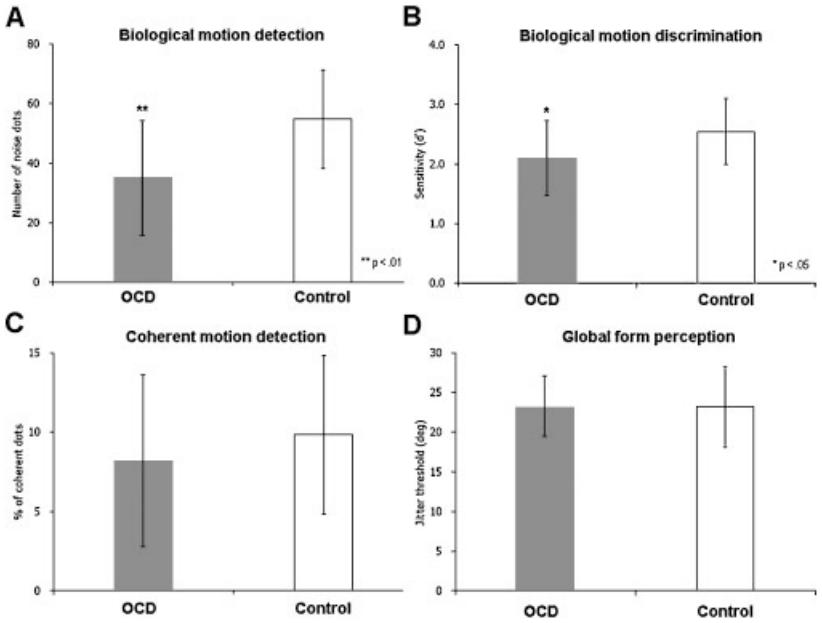

Figure 4. Mean (*SD*) performance on the four visual tasks.

before the actual test. We used a one-up/one-down staircase procedure to adjust the degree of jitter over trials to find the level of jitter guaranteeing 71% accuracy in each individual's performance. Visual feedback (correct location of the target) was provided after the subject's response on each trial. The total number of trials was 100; we estimated the threshold by calculating the mean and standard deviation of the jitter from the last eight trials of the staircase. Elapsed time for completing the task was also recorded.

RESULTS

TASK 1: BIOLOGICAL MOTION DETECTION

To reiterate, this task involved distinguishing biological from scrambled biological motion in PL animations to which were added variable amounts of noise dots undergoing the same pendular motions as the dots defining the normal and scrambled figures. Over trials, the number of noise dots was varied to find the noise level at which performance was approximately 71% correct (i.e., approximately midway between chance and perfect on this two-alternative forced-choice task).

Mean (*SD*) number of noise dots was 35.05 (19.19) in OCD patients, and 54.72 (16.64) in controls (Fig. 4A). This difference was statistically significant ($t(34) = 3.24$, $P = .003$), indicating that OCD patients had more difficulty detecting biological motion within noise dots compared to normal controls. OCD patients' symptom was not significantly correlated with the performance in this task (YBO: $r = .23$, $P = .35$; YBC: $r = .38$, $P = .11$; YBOC: $r = .33$, $P = .17$).

We did not record reaction times on a trial-by-trial basis (recall that on each trial the subject made his/her response only after comparing the two successive, briefly presented animations). We did, however, measure the total elapsed time for each test session, and there was no significant difference between the two groups on this measure ($t(34) = .29$, $P = .78$). We think it is unlikely, therefore, that the patients' overall poorer performance arose from uncertainty that led to prolonged decisions and, therefore, more errors.

TASK 2: DISCRIMINATING BIOLOGICAL FROM NON-BIOLOGICAL MOTION

This task, arguably much easier than the forced-choice task, simply involved judging whether a given PL animation portrayed a normal biological figure or a scrambled one. Mean (*SD*) d' values in OCD patients and controls are shown in Figure 4B. OCD patients were significantly worse than normal controls in discriminating biological motion from scrambled motion ($d' = 2.09$ (.63) vs. 2.54 (.55)), and this difference was statistically significant ($t(33) = 2.21$, $P = .034$). Correlation between d' values and patients' symptom was not significant (YBO-d': $r = .30$, $P = .22$; YBC-d': $r = .042$, $P = .87$; YBOC-d': $r = 27$, $P = .26$). In this task, as in the previous one, there was no significant difference in elapsed time between groups ($t(33) = .18$, $P = .86$).

TASK 3: COHERENT MOTION DETECTION

This task required detecting a subset of dots moving in one, given direction among noise dots that could move in all possible directions. The RDC animations were created in a way that made it impossible to make the judgment based on the movements of a given individual dot, thereby forcing observers to base their

judgments on the global coherence of motion defined over space and time. Moreover, observers had to compare two successive motion sequences to determine their response on each trial, thus replicating the trial structure of the two-alternative forced-choice biological motion task. Mean (*SD*) threshold (minimum coherence level) in OCD patients was 8.15 (5.47), and it was 9.84 (4.99) in normal controls. This difference was not statistically significant ($t(34) = .96$, $P = .34$) (Fig. 4C). Thus, OCD patients' performance in the task requiring non-biological, global motion processing was comparable to that of controls.

We also examined whether the stimulus exposure duration possibly affected subjects' performance, because motion sequences in this task were presented until subjects responded. First, difference in mean elapsed time for finishing this task was not statistically significant ($t(34) = 1.36$, $P = .18$). Next, elapsed time was not significantly correlated with the performance ($r = -.37$, $P = .10$). The correlation between symptoms and elapsed time was not significant (YBO: $r = -.08$, $P = .76$; YBC: $r = .24$, $P = .33$; YBOC: $r = .08$, $P = .76$). We do not believe, therefore, that exposure duration influenced the pattern of results in this task.

TASK 4: GLOBAL FORM PERCEPTION

Unlike the other three tasks, this one involved judging in which of four possible locations a target figure appeared, with the target defined by a set of spatially distributed contours. Over trials, the relative orientations of the target contours were jittered (i.e., varied), thereby distorting to varying degrees the clarity of the target figure. As shown in Figure 4D, the mean (*SD*) jitter threshold values in the two groups were equivalent: (23.05 (4.01) in OCD and 23.18 (5.09) in controls, $t(34) = .09$, $P = .93$). Difference in mean elapsed time for completing the task was not significant between groups ($t(34) = .82$, $P = .42$). Correlation between the jitter threshold and elapsed time in the control group was significant ($r = .88$, $P < .001$), whereas it was not significant in the OCD group ($r = .40$, $P = .08$). Elapsed time was not correlated with symptoms (YBO: $r = .15$, $P = .54$; YBC: $r = .17$, $P = .50$; YBOC: $r = .17$, $P = .48$).

DISCUSSION

To our knowledge, this is the first study of OCD patients that has compared the perception of globally defined visual stimuli using static and moving displays. The results showed that OCD patients, relative to normal controls, experienced difficulty in perceiving PL animations portraying the kinematics of human activity (biological motion), whereas their ability to perceive non-biological global motion and static global form was comparable to that of healthy controls. Before discussing the implications of these findings, we wish to consider several alternative, non-perceptual accounts of this selective deficit in biological motion perception.

First, most of the OCD patients in our study were medicated, and the possible effects of medication on biological motion perception are unknown. In the case of schizophrenia, psychotropic medications do not seem to affect perceptual and cognitive abilities in any specific manner [Allen et al., 1997; see also Chen et al., 2003a]. In a previous study by Kim et al. performance of patients with schizophrenia was not correlated with the dosage of antipsychotic medication. Although specific pharmacological effects on visual perception in OCD are not known, it seems unlikely that the medication in these patients would produce such specific deficits on biological motion perception but not coherence detection or shape discrimination.

Turning to a second alternative account of our results, several earlier studies on cognitive function in OCD indicate that patients with this disorder exhibit a "local-bias" in the allocation of attention during cognitive tasks such as the Rey-Osterrieth Complex Figure Test [Savage et al., 1999]. Could such a "local-bias" also affect OCD patients' perception of biological motion animations, perhaps because they focus on individual dots and not on the spatiotemporal pattern among dots? We find this unlikely, as the other two tasks on which OCD patients performed normally—motion coherence and shape from proximity—also required integration of local stimulus elements distributed over space and embedded in distracting "noise" elements. If anything, the biological motion animations used in Task 2 should have been the least susceptible to "local-bias" because those animations were presented without noise elements at all. Yet, OCD patients performed poorly on this task.

A third alternative interpretation of our results arises from the special nature of the PL animations we used. One could argue that these kind of biological motion animations represent a particularly complex class of visual stimuli, embodying hierarchical, pendular motions that are much more difficult to perceive than the simpler, translational motion signals defining RDCs. In fact, however, converging lines of evidence indicate that perception of biological motion is remarkably efficient and can be accomplished with very brief exposure durations and in the presence of massive levels of random motion masking noise[1] [Blake and Shiffrar, 2007]. In fact, PL animations are highly perceptually salient and "complex" only in that the motion vectors defining those animations portray kinematics and not just rigid transformations. We do acknowledge, however, that it could be useful

[1]The noise dots used in Task 1 were local motion signals sampled from the biological motion sequence being masked; those noise dots, too, underwent pendular motions and not random motion of the sort used in the RDC task. Biological motion sequences embedded in large amounts of completely random noise remain very easy to perceive.

to examine whether OCD patients show deficits on other motion tasks involving complex, non-biological motion sequences. Whatever the outcome from such a study, however, our results show definitively that OCD patients have specific deficits in perceiving biological motion.

Interestingly, the correlation between performance on either of the biological motion tasks and YBOCS scores in the OCD group was not significant: symptom severity, in other words, does not predict the extent of perceptual deficit. This was also true in previous studies of autistic children [Blake et al., 2003] and schizophrenic adults [Kim et al., 2005]. Of course, symptom severity in OCD can change over time and with medication. In all events, impaired biological motion perception could be regarded as a potential marker for existence of OCD, just as a working memory deficit is a hallmark symptom in schizophrenia. It is also intriguing to note that impaired social functioning, of which perception of people's action is an important component, can persist even after other OCD symptoms have subsided [Bystritsky et al., 1999; Hollander et al., 1996; Koran et al., 1996]. It would be revealing to assess perception of biological motion in asymptomatic OCD patients.

In the following section, we turn to consideration of possible structural and/or functional abnormalities within the human brain that might account for the deficits experienced by OCD patients in perceiving this unique class of visual stimuli.

POSSIBLE NEURAL SUBSTRATE OF IMPAIRED BIOLOGICAL MOTION PERCEPTION IN OCD

On the basis of decades of neurophysiological and anatomical work, we now know that visual processing is accomplished within a hierarchically organized series of visual areas that are broadly organized into dorsal and ventral streams, both of which originate within primary visual complex [Maunsell and Newsome, 1987; Van Essen and Felleman, 1991]. Performance on different visual tasks presumably relies on neural events within different neural areas, dependent on the nature of the visual stimuli and the task demands. With this broad conceptualization in mind, what can we conclude about the neural concomitants of the tasks used in our study and, therefore, about the possible neural bases of the deficits in biological motion perception exhibited by OCD patients?

Considering first the task involving spatial integration of stationary line elements (Task 4), it is widely believed [e.g. Gilbert, 1993] that neural interconnections among orientation-selective neurons within primary visual cortex (V1) subserve integration of contour information in targets like those used in our global form task. Because OCD patients seem to have no problems on this contour integration task, we see no reason to single out area V1 as a possible site of neurological damage in these patients. It is true that area V1 also contains movement-sensitive neurons whose responses are selective for direction of motion [e.g. Movshon and Newsome, 1996]. However, those neurons do not have the requisite response selectively to distinguish the PL motions of dots defining biological and scrambled motion. For that matter, direction-selective V1 neurons are also probably not the substrate for psychophysical discrimination of direction in RDC animations [e.g. see Movshon and Newsome, 1992]. To account for performance on tasks employing RDCs and PL animations, we must consider extrastriate cortical areas forming the dorsal stream pathway [Maunsell and Newsome, 1987].

Most prominent within the dorsal stream pathway are visual areas MT and MST, both of which have been implicated in perception of motion in RDC stimuli [Britten et al., 1996; Celebrini and Newsome, 1994]. Could impaired perception of biological motion arise from deficits within MT/MST in OCD patients? Based on circumstantial evidence, this possibility is not implausible a priori. It is known, for example, that OCD patients show deficits in SPEMs [Clementz et al., 1996; Lencer et al., 2004]. The neural circuit involved in oculomotor function includes the FEF, the SEF, and the basal ganglia, all of which receive significant input from areas MT/MST [Born and Tootell, 1992; Maunsell and Newsome, 1987; Newsome and Pare, 1988; Van Essen and Maunsell, 1983]. For several reasons, however, we are disinclined to believe that the pattern of results found in our OCD patients point to deficits within areas MT/MST. For one thing, our OCD patients showed normal visual sensitivity to motion portrayed in RDC animations, and there is overwhelming evidence that perception of coherent motion associated with viewing these kinds of stimuli depends crucially on neural events within areas MT/MST. For another, neurons in areas MT/MST respond equally well to biological motion sequences and to non-biological, scrambled motion, implying that those areas are not involved in explicitly representing biological motion [although that they are undoubtedly involved in registering motion signals used by subsequent processing stages; Grossman et al., 2000; Howard et al., 1996].

For the reasons summarized above, we do not believe that visual areas MT/MST are responsible for the compromised abilities of OCD patients to perceive biological motion. Rather, we are inclined to look elsewhere in the dorsal processing stream, to an area within the posterior region of the superior temporal sulcus (STS). Here we find a key brain region that may be implicated in OCD. The STS is associated with auditory and visual perception that is rich in socially relevant information such as interpretation of the actions and intentions of other people [Baron-Cohen et al., 2000]. This brain region is also connected with the orbito-frontal cortex, the caudate, and the amygdala [Amaral and Price, 1984; Yeterian and Pandya,

1998], all of which have been implicated in the neural basis of OCD. The STS and its surrounding regions are strongly and selectively activated by biological motion signals [Jellema and Perrett, 2003], and damage to the STS region leads to an impaired biological motion recognition but a spared ability to perceive other types of visual motion [Schenk and Zihl, 1997a,b]. It is tempting, therefore, to speculate that deficient biological motion perception studying our OCD patients may be traced to dysfunction within the STC. Earlier studies of patients with OCD found significantly higher regional cerebral blood flow levels [e.g. Cottraux et al., 1996] and partial volumetric reduction within areas along the superior temporal gyrus [Choi et al., 2006]. It remains unclear to us, however, why these particular brain abnormalities would adversely impact perception of biological motion; future studies must be carried out to pursue this particular question.

Interestingly, similar perceptual deficits have also been observed in other clinical populations: patients with schizophrenia [Kim et al., 2005] and children with autism [Blake et al., 2003]. Both patients with autism and with schizophrenia showed impaired recognition of biological motion (comparable to our Task 2), but intact perception of global form (comparable to our Task 4). In schizophrenia, it is noteworthy that similar volumetric reduction was observed in anterior portion of the left superior temporal gyrus [Shenton et al., 1992]. Although such structural evidence has not been reported in people with autism, the behaviorally observed deficits and structural findings suggest that compromised functioning within the STS area is also implicated in patients with autism and schizophrenia. Therefore, STS deficit and deficits in biological motion perception are not specific to OCD and may be a rather general concomitant of mental disorders. Exactly why STS would be so widely involved remains unknown, and it is possible that deficits in STS are secondary to other causes such as an abnormal early experience.

However, differences in perceptual deficits between OCD and other clinical populations do exist: OCD patients in our study showed a deficit in only biological motion tasks whereas patients with schizophrenia [Chen et al., 2003b] and autism [Milne et al., 2002] were deficient in perceiving global, coherent motion stimuli. Therefore, deficits in biological motion perception are manifested more specifically in OCD, strongly suggesting a role of STC, whereas these other clinical populations may have more extensive impairment in regions along the dorsal visual pathway, including visual areas MT/MST and STS. These possibilities can and should be tested using functional imaging methods.

To close on a speculative note, autistic children and schizophrenic patients both show deficits in tasks that tap the "Theory of Mind (ToM)", which is related to social functioning. The neural network that putatively supports ToM includes the amygdala, the STS, and the orbitofrontal cortex [Baron-Cohen et al., 2000]. Thus, abnormal functioning of the STS and its surrounding regions involved in biological motion perception may co-occur with deficits of ToM (and possibly impairment in general social functioning). Indeed, in our previous study [Kim et al., 2005], performance in biological motion task was correlated with social functioning in patients with schizophrenia. A next logical step would be to test the relationship between biological motion perception and social functioning in OCD patients.

CONCLUSIONS

We found that OCD patients have a specific deficit in perceiving biological motion signals, whereas their perception of non-biological, coherent motion and static global form is intact. Further studies are needed to investigate structural and/or functional abnormality in implicated brain regions (e.g. STS). Considering that efficient social interactions depend on accurate and rapid perception of subtle socially relevant cues, deficits in biological motion perception may compromise social functioning in people with OCD.

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