NIH Public Access
Author Manuscript
Int J Eat Disord. Author manuscript; available in PMC 2014 January 01.

Published in final edited form as:
Int J Eat Disord. 2013 January ; 46(1): 12–22. doi:10.1002/eat.22062.

Perception of Affect in Biological Motion Cues in Anorexia Nervosa

Nancy Zucker, PhD[1,2], **Ashley Moskovich, MA**[2], **Cynthia M. Bulik, PhD**[3,4], **Rhonda Merwin, PhD**[1], **Katherine Gaddis**[2], **Molly Losh, PhD**[5], **Joseph Piven, MD**[3], **Henry R. Wagner, PhD**[1], and **Kevin S. LaBar, PhD**[2]

[1]Department of Psychiatry and Behavioral Sciences, Duke University School of Medicine, Durham, NC

[2]Department of Psychology and Neuroscience, Duke University, Durham, NC

[3]Department of Psychiatry, UNC School of Medicine, University of North Carolina, Chapel Hill, NC

[4]Department of Nutrition, School of Public Health, University of North Carolina, Chapel Hill, NC

[5]Department of Communication and Language, Northwestern University, Evanston, IL

Abstract

Objective—Nonverbal motion cues (a clenched fist) convey essential information about the intentions of the actor. Individuals with anorexia nervosa (AN) have demonstrated impairment in deciphering intention from facial affective cues but it is unknown whether such deficits extend to deciphering affect from body motion cues.

Method—We examined the capacities of adults with AN (AN; *n*=21) or those weight restored for >= 12 months (WR; *n*=20) to perceive affect in biological motion cues relative to healthy controls (HC; *n*=23).

Results—Overall, individuals with AN evidenced greater deficit in discriminating affect from biological motion cues than WR or HC. Follow-up analyses showed that individuals with AN differed especially across two of the five conditions—deviating most from normative data when discriminating sadness and more consistently discriminating anger relative to WR or HC.

Discussion—Implications of these findings are discussed in relation to some puzzling interpersonal features of AN.

Keywords

anorexia nervosa; eating disorders; social cognition; social perception; motion perception

Anorexia nervosa (AN) is a disorder in which the preoccupying and determined pursuit of a low body weight not only impacts health, but also interferes with acquisition of alternative sources of reinforcement. For instance, although not a defining feature of the illness, there is accumulating evidence of social isolation, as well as impairment in interpersonal functioning

Disclosures
Parts of this manuscript were presented at the annual meeting of the American Academy of Neuropsychopharmacology (December, 2010) and at the International Conference of Eating Disorders (May, 2011).

All authors reported no biomedical financial interests or potential conflicts of interest. We thank all participants for their time and effort.

that predates illness onset and persists with remission of eating disorder symptomatology(1–3). Critically, the ability to form and sustain close interpersonal bonds has implications for the formation of a therapeutic alliance, persistence in treatment, and the prevention of symptom relapse in both adolescents and adults with AN (4). In fact, recent formulations of AN have posited that treatments for AN should emphasize the establishment of intimate interpersonal bonds as such social connection may be one of the few sources of reinforcement that effectively competes with the valued aspects of the ill state. Thus understanding barriers to the formation of intimate attachments is critical to design interventions for those with AN who struggle in the interpersonal realm.

Several theoretical models have been proposed to explain the social impairment in AN (5, 6). A model proposed by Zucker et al.(7) examines interpersonal deficits from a neurodevelopmental framework, positing that basic deficits in social perception may compromise capacities to accurately interpret the affect of others, a necessary substrate of empathic responding. Specifically, hypotheses from this model propose that given well-documented cognitive deficits in AN, such as aberrant set-shifting and local biases in visually-guided attention,(8, 9) individuals with AN would demonstrate impairment in the integration of the complex social signals that constitute typical human interaction. As all human interaction also involves movement and constant change, the model of Zucker et al.(7) proposes that deficits in social perception would most consistently emerge in complex social contexts that involve the integration of multiple inputs over time.

Rather than the *integration* of complex signals, alternative models propose that certain individuals with AN may have *biased* attention: focusing attention on certain elements of a social interaction to the exclusion of other elements (6). Such constrained focus would contribute to a biased interpretation of a social encounter. For example, Cserjesi, Vermeulen, Lenard, and Luminet (10) report that individuals with AN demonstrate an attention bias towards angry faces with decreased attention directed to faces expressing positive affect. Oldershaw et al.(6) reviewed attention bias of social-affective stimuli (words and faces) as part of a broader conceptualization of socio-affective functioning in AN. The bulk of the evidence supported aberrant patterns of visually-guided attention in AN that persisted with weight restoration. Thus, difficulty integrating complex social signals and/or biases towards certain affective features, may contribute to inaccurate interpretation of the intentions of others. Such deficits may have an added subjective consequence in that individuals with AN may consequently experience interpersonal relationships as confusing, rejecting, or both. Understanding the interplay of these factors necessitates studying perception of dynamic social cues among individuals with AN.

Dynamic motion of the body (e.g., a clenched waving fist) offers valuable information concerning motivational and related emotional states of the actor (11, 12). Two lines of evidence support that those with AN may evidence difficulties in deciphering affect from motion cues. According to Decety and Meyer (13), empathy and related complex processes of social perception are partially based on the inter-subjectivity of another's experience with our own. More specifically, *viewing* an action may evoke a *simulation* of that action in the observer, establishing the foundation that permits empathic understanding to occur. Thus, individuals sense as well as perceive in order to comprehend the experiences of others. By definition, individuals with AN have disturbance in the experience of their bodies. According to this framework, such disturbance may be associated with aberrant capacities to interpret meaning from the bodies of others.

Second, there is a limited, though increasing, body of research documenting overlap in a subgroup of those with AN and symptoms of autism spectrum disorder (ASD). Children and adults with autism spectrum disorders have shown impairment both in recognizing emotion and in detecting biological motion. Children with ASD have also shown significant impairment in their ability to differentiate biological motion from inanimate motion. They are able to detect biological formations of shapes that are not in motion, but these shapes move in a coherent pattern, they struggle to translate this visual motor information into social information (14). For example, a child with autism may be able to identify large shapes in the form of a child as a child, but when these shapes are moving as though a person is skipping, he cannot interpret the meaning of such motion. A similar finding was reported when children with autism were asked to identify affect from biological motion cues (i.e. pointlight walkers). In pointlight walkers, biological motion stimuli are created by affixing points of light to body joints of individuals dressed completely in black. When these individuals are filmed performing various body movements (e.g. jumping, dancing), an individual cannot see their body, rather they just see the moving points of light and have to use this information to make judgments about type of movement or affect, depending on the task. Children with autism recognize the motion in the points of light, but the mechanisms involved in integrating these local biological motion patterns into global, coherent, biological activity are impaired (15). Given cited evidence of overlap among those with AN and ASD, and evidence of impairment in performance on tasks that require capacities for global integration in AN, individuals with AN may have difficulty deciphering affect from motion cues.

Accurately interpreting the affect conveyed by body movement is essential in understanding others and guiding responses in social situations. Thus, interpretation of nonverbal body cues serves as a bedrock for relationship formation and related interpersonal tasks. Body movements influence the interpretation of facial affect (2) and overall intention of the actor (3–5). Such signals have been shown to be processed as early in the visual temporal sequence as facial affect, signifying the critical role of such cues in conveying meaning.(12). In contrast to facial affect(16), individuals with AN have demonstrated a relative attentional bias towards the bodies of others, and thus these signals may be disproportionately influential in social decision making. However, it is unclear whether those with AN would evidence impairment in interpreting body motion as has been shown with facial affect(17). If so, this finding might help explain deficits in interpersonal functioning and guide intervention development.

The aim of the current study was to assess the capacity of individuals with AN to decipher affective cues conveyed by biological motion. We use Pointlight Walkers (Pointlight) as the study stimuli to discern perception of body motion (18). In Pointlight displays, the human form is reduced to small patches of light located at each of the major joints of the body (e.g., ankle, knee). When these points are set in motion, the spatiotemporal pattern they create can quickly and easily convey a wealth of information about the actor and/or the action being portrayed (18). We hypothesize that individuals with AN would be impaired in their ability to decipher affect from motion cues relative to healthy controls. Given increasing emphasis on the delineation of neurocognitive features that manifest premorbidly and persist beyond the acute phase of illness, we incorporated neuropsychological measures of visually guided attention and intelligence both to better characterize our sample and to include as nuisance variables into our statistical models. Combined, these strategies are a first attempt to characterize biological motion perception in individuals with AN.

Method

Participants

The sample comprised 64 adult females who agreed to participate in a study on relationships from a sample of 164 adult females who were screened for eligibility. Of the 99 eligible individuals based on the screen, 74 were eligible based on diagnostic interview with 68 agreeing to participate and 64 completers. Participants were classified into three groups based on a case-control design as follows: current diagnosis of AN/AN-NOS (AN: *n* = 21), weight-restored with a prior diagnosis of AN (WR: *n* = 20), and no history of AN or other eating disorder (CN: *n* = 23). Individuals were recruited via printed advertisements placed within a 50-mile radius of the hosting university, and via electronic advertisements posted to parent forums and web-sites devoted to eating disorders. Notices were sent to a mailing list of healthcare providers known to specialize in the care of individuals with eating disorders within a 60-minute traveling distance, to all therapists in two university-based eating disorder programs, and were posted in clinic waiting rooms.

Inclusion criteria—To be eligible, individuals had to have met diagnostic criteria for AN based on the Diagnostic and Statistical Manual for Mental Disorders IV either currently or in the past (19). However, given the number of individuals on birth control pills (*n*=10 or 14.7%), it was impossible to discern whether amenorrhea was present and thus this feature was relaxed for diagnosis, consistent with prior studies (20). In addition, individuals were not actively experiencing psychosis or related thought disorder, were not actively abusing substances, and were not diagnosed with a learning disability based on self-report of prior learning history. Participants participated in an initial telephone interview of eating disorder history which included questions regarding exclusionary criteria (substance use, prior diagnosis of learning disability), followed by a face-to-face structured diagnostic interview of eating disorder symptoms over the past 3 months. Figure 1 shows the recruitment scheme and number of eligible participants. *Thus, the current AN group is most accurately conceptualized is a combination of AN and AN-NOS, with the subtype that continues to menstruate.*

BMI determination—Weight-loss and maintenance of an unhealthy low-weight are defining features of AN. While the DSM-IV provides an example of a low-weight threshold (e.g., less than 85% of ideal body weight), the determination of ideal body weight for a given individual is complex and is most precisely determined based on an individual's weight history. An individual's ideal body weight is the weight range associated with optimal health. Definition of a "less than ideal" body weight would then be defined as less than the lowest point of this healthy weight range. Unfortunately, body mass index (BMI) itself is proving to be inadequate in indexing physical recovery, given recent studies demonstrating that other physiological parameters such an insulin or degree of body fat and not body mass was predictive of return of menstruation (21, 22). To determine weight thresholds for the current study, we combined information from a variety of sources: medical record abstraction (when further clarification was needed), a weight history, structured diagnostic interview, and self-report measures. Based on this information, we derived current and past lowest-BMI per individual and defined weight-restoration as being at an individual's ideal body weight for ≥ 6 months. For example, one individual began to show medical compromise at a BMI of 20.2 based on chart review. Thus, this strategy led to deviations from the classic distinction of a body mass index of 18.5 or less as underweight, but more accurately reflects the weight history and optimal functioning of a given individual. This approach is less arbitrary and more accurately reflects the inter-individual clinical complexity of AN.

Procedure

Participants completed a battery of neuropsychological measures and measures of social perception as part of a study of social cognition and interpersonal functioning in AN. Tasks were administered in a fixed order with neuropsychological measures interspersed with measures of social perception and diagnostic interviews to maintain interest and decrease participant burden. Testing sessions were kept to a maximum of 3 hours and participants were offered breaks between every task. A battery of self-report measures was completed prior to the testing day to characterize the premorbid and current functioning of our sample. Measures assessed premorbid features that may influence interpersonal functioning (e.g., features of the autism spectrum and obsessive compulsive spectrum), a continuous measure of psychiatric symptoms frequently found to be comorbid in those with AN, as well as interview and questionnaire measures of eating disorder symptoms.

Assessment Measures: Psychopathology

Eating Disorder Examination—(EDE; 23, 24, 25). The EDE is a widely implemented and favored structured diagnostic interview of eating disorder psychopathology (23, 26, 27). Behavioral and attitudinal features are assessed according to DSM-IV clinical diagnostic criteria. While the interview largely focuses on symptoms over the past 28 days, behavioral diagnostic criteria (e.g., binge-eating, self-induced vomiting, driven exercise, laxative abuse) are assessed for presence, frequency, and total days of occurrence over the past three months in accordance with the duration criteria specified in DSM-IV. Attitudinal items are rated according to a seven-point scale ranging from 0–6 with higher scores reflecting more severe eating disorder psychopathology and resulting in four subscales - Restraint, Eating Concern, Shape Concern, and Weight Concern – and are computed as the mean of constituent scale items. Discriminant validity, internal consistency, and concurrent validity are well documented for the EDE (25).

Eating Disorder Examination Questionnaire—(EDE-Q; 28). The EDE-Q is a 41-item self-report version of the EDE (24). Normative data collected from a large sample of young adult women (n = 5231) between 18 and 42 years of age produced the following subscale means: Restraint (M = 1.30, SD = 1.40), Eating Concern (M = 0.76, SD = 1.06), Weight Concern (M = 1.79, SD = 1.51), Shape Concern (M = 2.23, SD = 1.65). Good convergence between the EDE and EDE-Q has been documented among community and clinical samples, though inconsistencies have been reported (e.g., (29–31). We administered both measures to ensure we captured the breadth of eating pathology, as some individuals may not disclose certain forms of pathology during a live interview.

Brief Symptom Inventory—(BSI; 32). The BSI is a shortened form of the revised version of the Symptom Checklist-90 (SCL-90R; 33, 34), a self-report measure of symptom levels reflecting psychopathology and was administered to capture comorbid symptomatology in a continuous fashion. The BSI consists of 49-items that form nine symptom dimensions (i.e., Somatization, Obsession-Compulsion, Interpersonal Sensitivity, Depression, Anxiety, Hostility, Phobic Anxiety, Paranoid ideation, and Psychoticism). Participants indicate level of distress over the past seven days using a Likert scale ranging from "*0 = Not at all*" to "*4 = Extremely.*" Norms derived for adult females indicate a GSI mean of 0.35 (SD = .37) for nonpatients (n = 358) and a mean of 1.40 (SD = 0.72) for psychiatric outpatients (n=577; 35). Depression means were M = 0.36 (SD = 0.56) for female nonpatients and M =1.90 (SD = 1.05) for female psychiatric outpatients. Anxiety means were M = 0.44 (SD = 0.54) and M = 1.82 (SD = 1.02) for female nonpatients and female psychiatric outpatients respectively. The BSI has shown good internal consistency and reliability for the nine dimensions with alpha coefficients ranging from 0.71 to 0.85, and test-retest reliability coefficients ranging from 0.68 to 0.91 (32, 36). Good convergent, construct, and predictive validity have been reported (32).

Autism-Spectrum Quotient—(AQ; 37). The AQ is a 50-item self-report measure assessing autism traits among individuals of normal intelligence with items that use a 4-point Likert scale. Higher scores reflect greater pathology. The following is a sample item: "I prefer to do things with others rather than on my own." Five subscale scores can also be derived, but were not included in the current manuscript. Mean scores for the Total AQ score have been reported as 38.1 (*SD* = 4.4) for adult females with Asperger syndrome or high-functioning autism and 15.4 (*SD* = 5.7) for female healthy controls (37). Hambrook and colleagues (38) reported that adult women with AN (*M* = 23.2, *SD* = 7.3) had significantly higher scores on the AQ than adult female healthy controls (*M* = 15.3, *SD* = 5.5). The AQ has shown moderate discriminative validity (39), and moderate to high internal consistency for the total score with Chronbach's alpha ranging between 0.67–0.82 (38, 40, 41). Furthermore, the AQ has demonstrated good test-retest and inter-rater reliability (37). A study investigating the use of the AQ as a screening tool recommended a cut-off of 26 be used to distinguish those with a probable diagnosis of Asperger's Syndrome (39).

Assessment Measures: Neuropsychology

Group Embedded Figures Test(42)—The GEFT measures individual differences in field-dependence or local processing, a default pattern of visually guided attention characterized by a bias towards detail. Participants are asked to locate and trace a simple figure embedded within a complex figure as quickly and accurately as possible. Responses are timed and are scored by summing the total number of correct items completed within the time limit (ranging from 0–18). Higher scores reflect better ability to locate details in complex figures. Preliminary norms using a sample of college students indicate a mean score of 10.8 (*SD* = 4.2) among females (*n* = 242) (42).

Wechsler Abbreviated Scale of Intelligence—(WASI; 43). The WASI is a shortened version of the Wechsler Adult Intelligence Scale—Third Edition (44). Full Scale IQ can be estimated from the four WASI subtests (vocabulary, similarities, matrix reasoning, and block design). The WASI has demonstrated good convergent validity with the WAIS-III and high levels of internal consistency for the Full Scale IQ estimate (43).

Assessment Measures: Motion Perception

Pointlight Walkers—Pointlight; (45). Individuals were presented a series of 23 short films that depicted a pointlight walker display. After each clip, participants verbally indicate their response from 5 emotional choices (*Happy, Sad, Afraid, Angry, Neutral*. Participants were instructed to respond as quickly as possible although time was not limited. Raw scores from the Pointlight were transformed to normative scores obtained via a prior validation sample (46). These normative scores were formed due to the complexity of labeling emotional experience. Rather than one correct answer, there are degrees of correctness in labeling the affect presented via biological motion cues. Prior work has characterized this diversity by creating standardized scores based on modal responses from the standardization sample. Thus, a score of "1" was given to any item that matched the most frequent normative response and various proportional scores were given for the remaining responses. Five constituent subscales for each emotion (Happy; Sad; Afraid; Angry; Neutral) were derived by summing scale items for which the modal response for a particular item was the emotion in question (i.e., the Happy subscale was comprised of all items for which the modal response was happy).

Statistical Method

Statistical analyses were based on a series of ordinary least-squares regression models regressing the various outcome measures on a three-level proxy variable denoting diagnostic status (Group). Evaluated outcomes included the Pointlight scale averaged over the five constituent emotion subscales. In instances where the estimated main effect for group was significant (based on a Wald-based Type 3 statistic), differences between diagnostic conditions were tested using a series of two-level contrasts. In instances where the main effect was not significant, contrasts are presented but not discussed. In some analyses, the five subscales were simultaneously modeled using a within-subjects design focusing on the interaction between level of subscale (i.e., type of emotion) and Group to determine if the outcome measure differed depending on emotion type. Because of complexities in interpreting the five-by-three interaction term in the latter model, we also estimated a series of simplified models stratified on subscale. For the latter, each of the five Pointlight subscales was in turn regressed on the group proxy variable; as above, in instances where the estimated main effect of group was significant, contrasts between conditions were subsequently tested using a series of two-level contrasts. Although both analytic approaches yielded similar results, the stratified analyses are presented for purposes of clarity. In addition, we examined the influence of individual differences in default patterns of visually guided attention (number of correct items derived from the GEFT) in our models as a covariate of interest. The purpose of this covariate was to rule out lower-level attentional-mode differences on affective motion perception as independent from the clinical background of these groups.

Results

Sample demographic characteristics are presented in Table 1. Our sample was predominately Caucasian (81.3%). The majority of participants were single/not married with 17.2% currently married. Groups did not differ on age or IQ. By design, groups differed in BMI: those with AN had a significantly lower weight than WR or CN, while WR was not different from CN.

Table 2 presents eating disorder pathology. WR continued to endorse eating disordered attitudes as measured by the EDE-Q with scores still significantly different from CN on all subscales (though also significantly different from AN). Comparison of our CN group with normative scores revealed very small to small effect sizes, with the CN group in this study having higher Restraint scores of very small effect (d = .19) relative to Mond et. al (47), but being healthier than the normative sample on Weight Concern, Eating Concern, and Shape Concern (d = .36 –.45). Thus, our CN group can be considered representative of normative group values. While the clinical groups did not differ in lowest BMI or age of onset, the effect size of the latter was of medium to large effect (d = .51, an average difference of 2.2 years). Our failure to find statistical significance may be due to sample size. The WR group can thus not be considered a fully recovered group, but rather a group that has regained weight to a healthy level, but still maintains at least statistically significant eating disordered attitudes. As such, they represent a more stringent test of the impact of weight restoration (rather than full AN recovery) on biological motion perception.

Table 3 presents comorbid secondary psychopathology. Again, the WR group endorsed lingering psychopathology with differences from CN on continuous measures of depressive, anxious, and obsessive-compulsive symptoms. These findings are consistent with many reports of the long-term outcome of AN (48). The clinical groups scores on the AQ was consistent with the findings of Hambrook and colleagues being higher than controls (38), significantly lower than normative scores of adults with high-functioning autism, and lower than the recommended cut-off screening score of 26. Thus, our clinical groups demonstrate similar patterns of affective and personality symptoms relative to the existing literature on the course of AN.

Analyses of differences in the Pointlight outcome measures are presented in Table 4 and Figure 1. The main effect for Group in the Pointlight total score (averaged over the five subscales) was significant (Table 4: $\chi^2_{(df=2)}$=6.01; p<0.05); contrasts between levels of the group proxy indicated that average total Pointlight scores were significantly decreased overall in AN participants relative to WR and CN. The main effects for Group in models re-estimated after stratifying on the Pointlight subscales were significant among two of the five conditions: Sad (Table 4: $\chi^2_{(df=2)}$=13.54; p<0.01) and Angry (Table 4: $\chi^2_{(df=2)}$=6.40; p=0.04). Post-hoc evaluation of scores revealed that AN were significantly higher on the Angry subscale and lower on the Sad subscale relative to both WR and CN (Table 4 and Figure 1). Group differences between subscale scores were not significant on remaining emotion subscales (Happy, Afraid, or Neutral; Table 4 and Figure 1). A subsequent exploratory analysis of the frequency of angry ratings indicate that those with AN ($\underline{x}$ = 25.57) did not simply endorse anger more frequently than controls ($\underline{x}$ = 24.35), $\chi^2_{(df=2)}$=13.54; p<0.01) $\underline{p}$ > .4. To further probe the interpretation of these findings, we examined the role of BMI as an explanatory variable for group differences. Adding BMI as a covariate in regression models weakened effects such that the overall regression comparing emotion identification as a function of group membership was no long statistically significant ($\chi^2_{(df=2)}$=3.83; p>0.10), while contrasts comparing AN to CN remained marginally significant, ($\chi^2_{(df=1)}$=3.80; p=0.051). As a final indication of the importance of illness severity in explaining aberrations in motion perception, we examined the association of Pointlight score to self-reported dietary restraint, finding a significant negative association: r = .-.30, p < .02.

Discussion

It has been theorized that those with AN would have difficulties integrating complex social signals when attempting to decipher the affect of others. We examined one facet of this capacity: namely accuracy in deciphering emotion from body motion cues relative to other groups. In a sample of 64 adult females grouped based on current diagnosis of AN/AN-NOS, history of AN/AN-NOS and presently weight-restored, or no history of AN, individuals with current AN had greater difficulty deciphering affect from biological motion cues. In a series of exploratory analyses examining the meaning ascribed to particular motion patterns, individuals with AN were less accurate in indentifying sadness relative to normative data and were more consistent with normative data when deciphering anger portrayed via body motion. We further examined what aspect of the ill state was related to body motion perception and did an exploratory analysis examining the role of BMI on study findings. The addition of BMI into regression models weakened these effects such that the main effect of group was no longer significant and subsequent exploratory contrasts marginally significant between AN and CN. Combined these results suggest that is the reduced BMI of the ill state of AN that particularly comprises capacities to integrate complex social signals.

Deciphering affect from motion demands capacities to integrate perceptual streams of information with their global context. This specific neurocognitive task was hypothesized to be impaired in AN given documented neurocognitive deficits in visually-guided attention and executive control. Initial analyses were supportive of this hypothesis. Yet, exploratory analyses that examined patterns of affect recognition indicated that those currently ill were not universally impaired suggesting that certain alternations of the ill state may influence the meaning and/or capacity to decipher complex social cues. Findings were weakened by the addition of BMI, an imperfect index of diagnostic severity. Findings thus suggest that the state of starvation may impact capacities to integrate complex social signals.

The state of starvation may impact vigilance and accuracy in deciphering specific social cues consistent with prior work on attention bias. Given the ego-syntonic presentation (49) and seeming surge of energy and locomotion associated with the ill state (50), it is easy to forget that individuals with AN are in a state of physical threat to person. Consequently, individuals with AN evidence physical adaptations to starvation that would facilitate survival (51). As with any starved animal, the ability to perceive threat would be advantageous as it would facilitate escape and adaptive protective mechanisms in vulnerable organisms. Further, sadness, as a motivational state of loss, would be of little advantage to rapidly decipher as starved organisms are incapable of caring for other needy organisms. Thus, we postulate that the current results are interpretable as biological adaptations to starvation and may not be unique to AN. This is not to say that a potential mechanism of such alterations in social perception is due to changes in body experience combined with the state of starvation as a critical event in one's learning history. Thus, patterns of perception in the starved state may be influenced by perception-action relationships cited earlier. This viewpoint also does not detract from the fact that the findings may have important clinical implications for the management of AN.

To our knowledge, this is the first study of biological motion perception in AN. Thus, we cannot examine our findings relative to other studies of body affect perception in those with eating disorders. We can, however, examine the implications of such findings in autism spectrum disorders, so findings from that body of work can guide relevant investigations in AN. In autism spectrum disorders, findings of aberrations of biological motion perception have been linked to theories of perceptual processing such as weak central coherence (52) or enhanced perceptual functioning (53). Such theories posit that biases in visually-guided attention or superiorities in the rapid detection of fine-grained visual details are alternatively construed as perceptual assets, as barriers to processing global configurations, or as biases that can be modified (52, 53). Research conducted with more nuanced task manipulations has provided evidence to update these theories revealing that individuals with autism spectrum disorders can process global configurations under certain conditions(54). As motion perception demands such global integration, poor performance on this task was linked to these theories. In AN, accumulating evidence is highlighting a slightly different perceptual pattern. Those with AN have not consistently exhibited superiority in local processing but evidence worse performance on tasks that require global processing (55). Current findings are consistent with these results – at least in the actively ill state. Further, given findings in autism of modification of perceptual processing with modified direction or deliberate training, current findings suggest the need to examine alterations in perceptual biases longitudinally as the state of starvation may reasonably be expected to alter the motivational salience of visual stimuli and with implications with alterations in visually guided attention even with weight restoration(56).

Our study has several limitations bearing mention. We did not compare our active AN group to a truly recovered group. While our sample had restored their weight to within normal limits, advanced definitions of recovery have stressed the importance of incorporating sustained psychological change as part of a definitive sustained state of illness remission (57). Such comparisons would have been optimal in examining the persistence of social cognitive deficits. However, our design was able to examine the impact of being underweight on motion perception. We did not employ a structured diagnostic interview of secondary psychopathology. Balancing participant burden, the current debate about dimensional vs. dichotomous views of psychopathology, we opted for self-report measures of secondary psychopathology that index the past week. While our study has face validity, we may have missed some clinically informed diagnoses that would enhance our characterization of our samples and potentially account for the findings. Further, future studies should examine the role of other psychopathological symptoms (e.g. depression, anxiety) in contributing to deficits in motion perception.

Nonverbal gestures convey essential information about human emotions and related intentions. Findings reveal that those with AN have difficulty identifying body motion as indicative of sadness but are more accurate in identifying biological motion connoting anger relative to individuals with AN who have been weight restored or healthy controls. Results may have implications for adaptive interpersonal functioning in AN. Detecting anger, particularly when others do not, may be adaptive in dangerous contexts. However, during the course of typical interpersonal interaction, such detection may promote bias and negatively impact the interpretation of social encounters. Critically, such bias has been demonstrated in AN towards threatening facial affect (6). While the current study is not a threshold-detection study, future research should examine this hypothesis. Results also have immediate clinical relevance in the education of families about changes in social cognition that may accompany the starved state. Such education may help family members appreciate that aberrations in deciphering emotion may be due to biological changes rather than characterological motivations. Educating families about adaptations in social cognition that accompany starvation may help family members better understand and empathize with the phenomenology of AN as a starved state in which the ability to decipher emotional cues of others is compromised. Understanding how perception of others changes as a function of starvation will be pivotal to designing treatments sensitive to the phenomenology of the ill state.

Acknowledgments

This research was supported by NIH grants (Zucker; R01-MH-078211 and K23-MH-070418), and a Duke Institute for Brain Sciences Research Incubator Award awarded to Drs. LaBar and Zucker.

References

1. Godart NT, Flament MF, Lecrubier Y, Jeammet P. Anxiety disorders in anorexia nervosa and bulimia nervosa: co-morbidity and chronology of appearance. European Psychiatry. 2000 Feb; 15(1):38–45. [PubMed: 10713801]
2. Troop NA, Bifulco A. Childhood social arena and cognitive sets in eating disorders. British Journal of Clinical Psychology. 2002 Jun.41:205–211. [PubMed: 12034006]
3. Rastam M, Gillberg C, Wentz E. Outcome of teenage-onset anorexia nervosa in a Swedish community-based sample. European Child & Adolescent Psychiatry. 2003; 12(Suppl 1):I78–I90. [PubMed: 12567219]
4. Stewart W. The Role of Perceived Loneliness and Isolation in the Relapse from Recovery in Patients with Anorexia and Bulimia Nervosa. Clinical Social Work Journal. 2004; 32(2):185–196.
5. Rieger E, Van Buren DJ, Bishop M, Tanofsky-Kraff M, Welch R, Wilfley DE. An eating disorder-specific model of interpersonal psychotherapy (IPT-ED): Causal pathways and treatment implications. Clinical Psychology Review. 2010 Jun; 30(4):400–410. [PubMed: 20227151]
6. Oldershaw A, Hambrook D, Stahl D, Tchanturia K, Treasure J, Schmidt U. The socio-emotional processing stream in Anorexia Nervosa. Neuroscience and Biobehavioral Reviews. 2011 Jan; 35(3): 970–988. [PubMed: 21070808]
7. Zucker NL, Losh M, Bulik CM, LaBar KS, Piven J, Pelphrey KA. Anorexia nervosa and autism spectrum disorders: guided investigation of social cognitive endophenotypes. Psychological Bulletin. 2007 Nov; 133(6):976–1006. [PubMed: 17967091]
8. Roberts ME, Tchanturia K, Stahl D, Southgate L, Treasure J. A systematic review and meta-analysis of set-shifting ability in eating disorders. Psychological Medicine. 2007 Aug; 37(8):1075–1084. [PubMed: 17261218]
9. Lopez C, Tchanturia K, Stahl D, Treasure J. Weak central coherence in eating disorders: A step towards looking for an endophenotype of eating disorders. Journal of Clinical and Experimental Neuropsychology. 2009; 31(1):117–125. [PubMed: 18608648]
10. Cserjesi R, Vermeulen N, Lenard L, Luminet O. Reduced capacity in automatic processing of facial expression in restrictive anorexia nervosa and obesity. Psychiatry Research [Article]. 2011 Jul; 188(2):253–257.

11. de Gelder B, Hadjikhani N. Non-conscious recognition of emotional body language. Neuroreport. 2006 Apr; 17(6):583–586. [PubMed: 16603916]
12. Meeren HKM, van Heijnsbergen C, de Gelder B. Rapid perceptual integration of facial expression and emotional body language. Proceedings of the National Academy of Sciences of the United States of America. 2005 Nov; 102(45):16518–16523. [PubMed: 16260734]
13. Decety J, Meyer M. From emotion resonance to empathic understanding: A social developmental neuroscience account. Development and Psychopathology. 2008 Fal;20(4):1053–1080. [PubMed: 18838031]
14. Rutherford M, Pennington BF, Rogers SJ. The perception of animacy in young children with autism. Journal of autism and developmental disorders. 2006; 36(8):983–992. [PubMed: 16897392]
15. Blake R, Turner LM, Smoski MJ, Pozdol SL, Stone WL. Visual recognition of biological motion is impaired in children with autism. Psychological Science. 2003; 14(2):151–157. [PubMed: 12661677]
16. Watson KK, Werling DM, Zucker NL, Platt ML. Altered social reward and attention in anorexia nervosa Frontiers in Psychology. 2010
17. Kucharska-Pietura K, Nikolaou V, Masiak M, Treasure J. The recognition of emotion in the faces and voice of anorexia nervosa. International Journal of Eating Disorders. 2004 Jan; 35(1):42–47. [PubMed: 14705156]
18. Johansson G. Visual-perception of biological motion and a model for its analysis. Perception & Psychophysics. 1973; 14(2):201–211.
19. American Psychiatric Association. Diagnostic and Statistical Manual for Psychiatric Disorders. 4th ed. Washington, DC: American Psychiatric Press; 1994.
20. Roberto CA, Steinglass J, Mayer LE, Attia E, Walsh BT. The clinical significance of amenorrhea as a diagnostic criterion for anorexia nervosa. Int J Eat Disord. 2008 Sep; 41(6):559–563. [PubMed: 18454485]
21. Dei M, Seravalli V, Bruni V, Balzi D, Pasqua A. Predictors of recovery of ovarian function after weight gain in subjects with amenorrhea related to restrictive eating disorders. Gynecological Endocrinology. 2008; 24(8):459–464. [PubMed: 18850384]
22. Misra M, Prabhakaran R, Miller KK, Tsai P, Lin A, Lee N, et al. Role of cortisol in menstrual recovery in adolescent girls with anorexia nervosa. Pediatric Research. 2006 Apr; 59(4):598–603. [PubMed: 16549537]
23. Wilson, GT. Assessment of binge eating. In: Fairburn, CG.; Wilson, GT., editors. Binge eating: Nature, assessment, and treatment. New York: Guilford Press; 1993. p. 227-249.
24. Fairburn, C.; Cooper, Z., editors. The Eating Disorder Examination. 12th edition. New York: Guilford Press; 1993.
25. Fairburn, C.; Cooper, Z. The Eating Disorder Examination. 12th edition. Wilson, CGFGT., editor. New York: Guilford Press; 1993.
26. Rosen, JC.; Srebnik, D. The assessment of eating disorders. In: McReynolds, P.; Rosen, JC.; Chelune, G., editors. Advances in Psychological Assessment. New York: Plenum; 1991.
27. Garner, DM. Measurement of eating disorder psychopathology. In: Fairburn, CG.; Brownell, KD., editors. Eating disorders and obesity: A comprehensive handbook. 2nd ed. New York: Guilford Press; 2002. p. 141-146.
28. Fairburn C, Beglin S. Assessment of eating disorders: Interview or self-report questionnaire? Int J Eat Disord. 1994; 16:363–370. [PubMed: 7866415]
29. Fairburn CG, Beglin S. Assessment of eating disorder psychopathology: Interview or self-report questionnaire? International Journal of Eating Disorders. 1994; 16:363–370. [PubMed: 7866415]
30. Mond JM, Hay PJ, Rodgers B, Owen C, Beumont RJV. Validity of the Eating Disorder Examination Questionnaire (EDE-Q) in screening for eating disorders in community samples. Behaviour Research and Therapy. 2004 May; 42(5):551–567. [PubMed: 15033501]
31. Binford RB, Le Grange D, Jellar CC. Eating Disorders Examination Versus Eating Disorders Examination-Questionnaire in Adolescents with Full and Partial-Syndrome Bulimia Nervosa and Anorexia Nervosa. International Journal of Eating Disorders. 2005 Jan; Vol 37(1):44–49. [PubMed: 15690465]

32. Derogatis, LR. BSI Brief Symptom Inventory. Administration, Scoring, and Procedures Manual. 4th. Minneapolis, MN: National Computer Systems; 1993.
33. Derogatis, LR. Brief Symptom Inventory. Baltimore, MD: Clinical Psychometric Research; 1975.
34. Derogatis, LR. The SCL-R-90 Manual I: Scoring, Administration and Procedures for the SCL-90. Baltimore, MD: Clinical Psychometric Research; 1977.
35. Derogatis, LR. SCL-90-R administration, scoring, and procedures manual. Baltimore, MD: Clinical Psychometric Research; 1983.
36. Derogatis, LR.; Spencer, PM. The Brief Symptom Inventory: Administration, scoring and procedures manual. Baltimore: Clinical Psychometric Research; 1982.
37. Baron-Cohen S, Wheelwright S, Skinner R, Martin J, Clubley E. The autism-spectrum quotient (AQ): evidence from Asperger syndrome/high-functioning autism, males and females, scientists and mathematicians.[erratum appears in J Autism Dev Disord 2001 Dec;31(6):603]. Journal of Autism & Developmental Disorders. 2001 Feb; 31(1):5–17. [PubMed: 11439754]
38. Hambrook D, Tchanturia K, Schmidt U, Russell T, Treasure J. Empathy, systemizing, and autistic traits in anorexia nervosa: A pilot study. British Journal of Clinical Psychology. 2008; 47:335–339. [PubMed: 18208640]
39. Woodbury-Smith MR, Robinson J, Wheelwright S, Baron-Cohen S. Screening adults for asperger syndrome using the AQ: A preliminary study of its diagnostic validity in clinical practice. Journal of Autism and Developmental Disorders. 2005 Jun; 35(3):331–335. [PubMed: 16119474]
40. Austin EJ. Personality correlates of the broader autism phenotype as assessed by the Autism Spectrum Quotient (AQ). Personality and Individual Differences. 2005 Jan; 38(2):451–460.
41. Hurst RM, Mitchell JT, Kimbrel NA, Kwapil TK, Nelson-Gray RO. Examination of the reliability and factor structure of the autism spectrum quotient (AQ) in a non-clinical sample. Personality and Individual Differences. 2007; 43:1938–1949.
42. Witkin, HA.; Oltman, PK.; Raskin, E.; Karp, SA. Group Embedded Figures Test Manual. Redwood City, CA: Mind Garden, Inc; 2002.
43. Wechsler, D. WASI: Wechsler Abbreviated Scale of Intelligence. San Antonio, TX: The Psychological Corporation; 1999.
44. Wechsler, D. Wechsler Adult Intelligence Scale-third edition: Administration and scoring manual. San Antonio, TX: Psychological Corporation; 1997.
45. Johansso G. Visual-perception of biological motion and a model for its analysis. Perception & Psychophysics. 1973; 14(2):201–211.
46. Couture SM, Penn DL, Losh M, Adolphs R, Hurley R, Piven J. Comparison of social cognitive functioning in schizophrenia and high functioning autism: more convergence than divergence. Psychological Medicine. 2010 Apr; 40(4):569–579. [PubMed: 19671209]
47. Mond JM, Hay PJ, Rodgers B, Owen C. Eating Disorder Examination Questionnaire (EDE-Q): Norms for young adult women. Behaviour Research and Therapy. 2006 Jan; 44(1):53–62. [PubMed: 16301014]
48. Crane AM, Roberts ME, Treasure J. Are obsessive-compulsive personality traits associated with a poor outcome in anorexia nervosa? A systematic review of randomized controlled trials and naturalistic outcome studies. International Journal of Eating Disorders. 2007 Nov; 40(7):581–588. [PubMed: 17607713]
49. Vitousek K, Watson S, Wilson GT. Enhancing motivation for change in treatment-resistant eating disorders. Clinical Psychology Review. 1998 Jun; 18(4):391–420. [PubMed: 9638355]
50. Holtkamp K, Herpertz-Dahlmann B, Hebebrand K, Mika C, Kratzsch J, Hebebrand J. Physical activity and restlessness correlate with leptin levels in patients with adolescent anorexia nervosa. Biological Psychiatry. 2006 Aug; 60(3):311–313. [PubMed: 16376860]
51. Wang T, Hung CCY, Randall DJ. The comparative physiology of food deprivation: From feast to famine. Annual Review of Physiology. 2006; 68:223–251.
52. Happe F, Frith U. The weak coherence account: Detail-focused cognitive style in autism spectrum disorders. Journal of Autism and Developmental Disorders. 2006 Jan; 36(1):5–25. [PubMed: 16450045]

53. Mottron L, Dawson M, Soulieres I, Hubert B, Burack J. Enhanced perceptual functioning in autism: An update, and eight principles of autistic perception. Journal of Autism and Developmental Disorders. 2006 Jan; 36(1):27–43. [PubMed: 16453071]

54. Wang LX, Mottron L, Berthiaume C, Dawson M. Local bias and local-to-global interference without global deficit: A robust finding in autism under various conditions of attention, exposure time, and visual angle. Cognitive Neuropsychology. 2007 Jul; 24(5):550–574. [PubMed: 18416507]

55. Lopez C, Tchanturia K, Stahl D, Treasure J. Central coherence in eating disorders: a systematic review. Psychological Medicine. 2008; 38(10):1393–1404. [PubMed: 18447964]

56. Harrison A, Tchanturia K, Treasure J. Attentional Bias, Emotion Recognition, and Emotion Regulation in Anorexia: State or Trait? Biological Psychiatry. 2010 Oct; 68(8):755–761. [PubMed: 20591417]

57. Couturier J, Lock J. What is recovery in adolescent anorexia nervosa? International Journal of Eating Disorders. 2006 Nov; 39(7):550–555. [PubMed: 16791851]

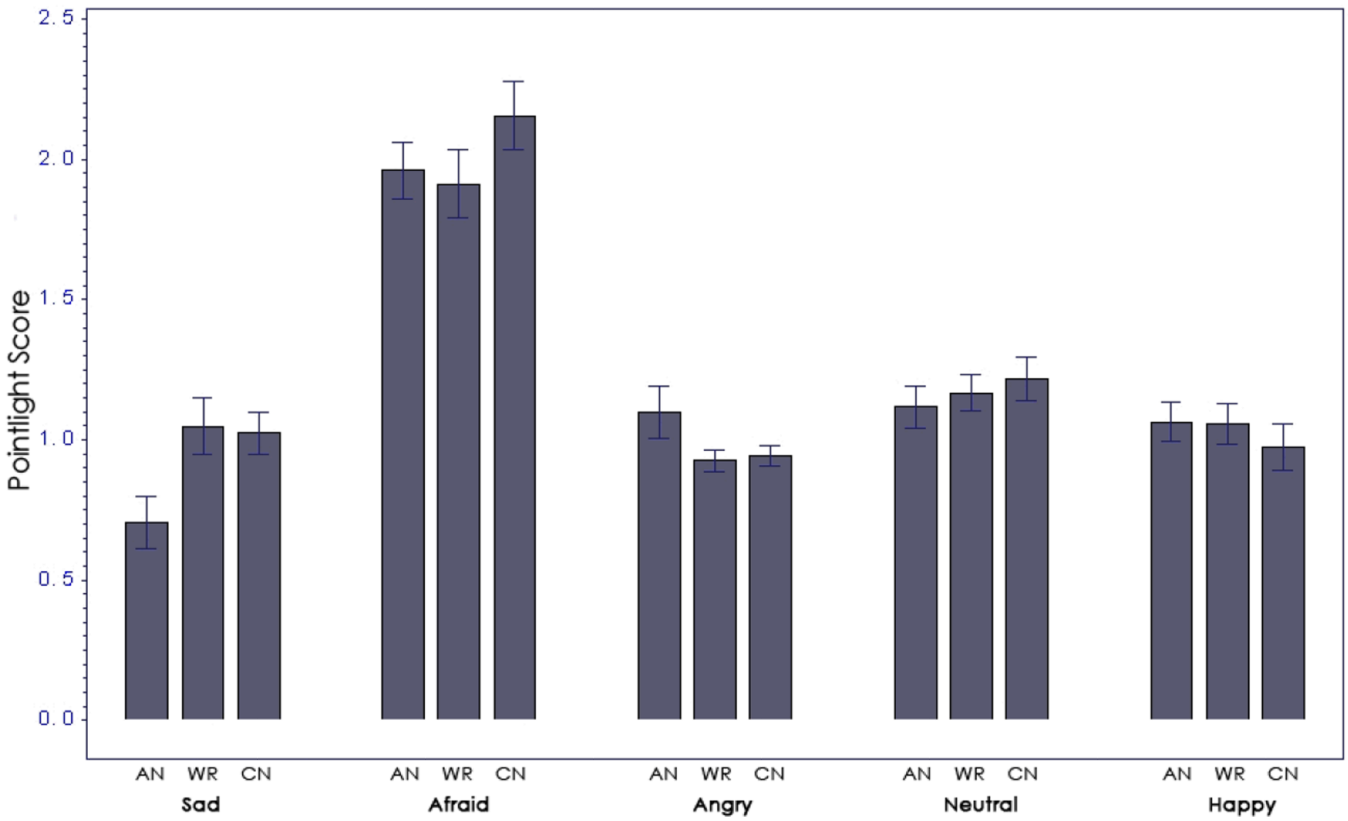

Figure 1.
Pointlight emotion subscale scores by group membership. Error bars represent +/− one standard error unit. AN = current diagnosis of anorexia nervosa; WR = prior diagnosis of anorexia nervosa but currently weight-restored; CN = no history of anorexia nervosa.

Table 1

Demographic Information

Demographic Variable	AN (*n* = 21)	WR (*n* = 20)	CN (*n* = 23)	Total (*n* = 64)
Age				
Mean (SD)	25.6 (8.7)	27.4 (10.1)	26.6 (9.8)	27.2 (9.4)
18–25	8 (38.1%)	12 (60%)	14 (60%)	34 (53.1%)
26–30	8 (38.1%)	2 (10%)	3 (13%)	13 (20.3%)
31–35	1 (4.8%)	2 (10%)	3 (13%)	6 (9.4%)
36–40	2 (9.5%)	2 (10%)	1 (4.3%)	5 (7.8%)
41–45	1 (4.8%)	1 (5%)	0 (0%)	2 (3.1%)
Over 45	1 (4.8%)	1 (5%)	2 (8.7%)	4 (6.3%)
Race/Ethnicity				
White/Caucasian	21 (100%)	18 (90%)	13 (56.5%)	52 (81.3%)
Black/African American	0 (0%)	1 (5%)	5 (21.7%)	6 (9.4%)
Asian	0 (0%)	1 (5%)	1 (4.3%)	2 (3.1%)
Hispanic/Latino	0 (0%)	0 (0%)	1 (4.3%)	1 (1.6%)
Mixed Race	0 (0%)	0 (0%)	1 (4.3%)	1 (1.6%)
Other/Not Classified	0 (0%)	0 (0%)	2 (8.7%)	2 (3.1%)
Current Relationship Status				
Married	2 (9.5%)	5 (25%)	4 (17.4%)	11 (17.2%)
Divorced	1 (4.8%)	0 (0%)	0 (0%)	1 (1.6%)
Separated	1 (4.8%)	0 (0%)	0 (0%)	1 (1.6%)
Engaged	0 (0%)	1 (4.8%)	0 (0%)	1 (1.6%)
Single, partnered	4 (19%)	5 (25%)	11 (47.8%)	20 (31.3%)
Single, not partnered	13 (61.9%)	9 (45%)	8 (34.8%)	30 (46.9%)
Years of Education				
Mean (SD)	15.1 (2.4)	16.3 (3.3)	15.8 (3.2)	15.7 (3.0)
12 or less	3 (14.3%)	3 (15%)	2 (8.7%)	8 (12.7%)
13–16	13 (61.9%)	7 (35%)	12 (52.2%)	32 (50%)
17–18	5 (23.8%)	9 (47.4%)	9 (39.1%)	23 (36.5%)
Not Reported	0 (0%)	1 (5%)	0 (0%)	1 (1.6%)
Verbal IQ				
Mean (SD)	121.0 (11.6)	125.6 (12.0)	120.6 (13.9)	122.2 (12.6)

Note. Groups were not significantly different on age, years of education, or verbal IQ. % (Percentage of each group as indicated). AN = current diagnosis of anorexia nervosa; WR = prior diagnosis of anorexia nervosa but currently weight-restored; CN = no history of anorexia nervosa.

Table 2

Associated Eating Disorder Psychopathology by Group

		Weight History		Presence of Lifetime Threshold Symptoms			Current Self-Reported Eating Disorder Attitudes (EDE-Q)			
Group	Age of Eating Disorder Onset	Lowest BMI	Current BMI	Lifetime Binge Eating	Lifetime Driven Exercise	Lifetime Laxative Use	Restraint	Weight Concern	Shape Concern	Eating Concern
AN (n=21)	17.52 (4.43)	14.92 (1.56)^{a}†	17.43 (1.33)^{a}†	33.3%	80.9%	57.1%	4.00 (1.53)^{a}†	4.49 (1.06)^{a}†	4.82 (0.98)^{a}†	3.65 (1.39)^{a}†
WR (n=20)	15.30 (4.33)	15.14 (1.69)^{a}†	21.44 (2.12)^{b}†	39.3%	64.7%	37.5%	2.18 (1.29)^{b}†*	2.57 (1.24)^{b}†	2.64 (1.23)^{b}†	1.39 (1.21)^{b}†*
CN (n=23)		20.51 (2.13)^{b}†	22.73 (3.80)^{b}†				0.91 (0.91)^{c}†*	0.94 (0.87)^{c}†	1.25 (0.94)^{c}†	0.25 (.32)^{c}†*

Note. Mean (standard deviation). % (Percentage of each group as indicated). Different superscripts connote differences between groups. Two members from AN, two members from WR, and one from CN were missing lowest BMI information. Two members from WR were missing current BMI information. BMI = body mass index. AN = current diagnosis of anorexia nervosa; WR = prior diagnosis of anorexia nervosa but currently weight restored; CN = no history of anorexia nervosa; EDE-Q.

* = $p < .01$.

† = $p < .001$.

NIH-PA Author Manuscript

NIH-PA Author Manuscript

Table 3

Associated Psychopathology by Group

		Brief Symptom Inventory (BSI)*							EATATE**	
Group	Autism Spectrum Quotient*	Somat	Obsess Comp	Depress	Anxiety	Hostility	Paranoid Ideation	Global Severity	Impuls	# OCPD Child-hood Traits
AN (n=21)	22.28 (6.43)[a]	1.28 (0.85)[a]	2.06 (0.88)[a]	2.24 (0.87)[a]	1.86 (0.90)[a]	0.84 (0.80)[a]	1.36 (0.94)[a]	1.65 (0.70)[a]	2.55 (2.11)[a]	2.43 (1.66)[a]
WR (n=20)	19.94 (6.38)[a,b]	0.58 (0.45)[b]	1.52 (0.84)[a]	1.02 (0.90)[b]	1.17 (1.00)[b]	0.61 (0.46)[a,b]	0.71 (0.78)[b]	0.92 (0.60)[b]	2.05 (1.32)[a]	2.15 (1.69)[a]
CN (n=23)	16.20 (4.67)[b]	0.25 (0.32)[b]	0.62 (0.51)[b]	0.36 (0.40)[c]	0.36 (0.58)[c]	0.31 (0.27)[b]	0.31 (0.29)[b]	0.33 (0.26)[c]	0.61 (1.20)[b]	0.57 (0.99)[b]

Note. Mean (standard deviation). Different superscripts connote differences between groups. Somat = Somatization BSI dimension; Obsess Comp = Obsessive Compulsive BSI dimension; Depress = Depression BSI dimension; Impuls = EATATE Impulsivity score. AN = current diagnosis of anorexia nervosa; WR = prior diagnosis of anorexia nervosa but currently weight restored; CN = no history of anorexia nervosa.

* = $p < .05$.

** = $p < .01$.

NIH-PA Author Manuscript
NIH-PA Author Manuscript

Table 4

Social Perception as a Function of Group

Model 1: All Emotions								
						Wald Statistics For Type 3 Analysis		
Parameter	**df**	**Est**	**SE**	**−95%CI**	**+95%CI**	**df**	**χ^2**	**p**
Intercept	1	0.26	0.01	0.23	0.28			
Group Embedded Figures Test	1	0.00	0.00	0.00	0.00	1	5.74	0.02
Condition: AN	1	−0.02	0.01	−0.04	0.00	2	6.01	0.05
Condition: WR	1	−0.01	0.01	−0.03	0.01			
Condition: CN	0	0.00	0.00	0.00	0.00			
Contrast Estimate Results								
AN vs. WR		−0.01	0.01	−0.03	0.01		1.89	0.17
AN vs. CN		−0.02	0.01	−0.04	0.00		6.01	0.01
WR vs. CN		−0.01	0.01	−0.03	0.01		1.16	0.28

Model 2: Sad								
						Wald Statistics For Type 3 Analysis		
Parameter	**df**	**Est**	**SE**	**−95%CI**	**+95%CI**	**df**	**χ^2**	**p**
Intercept	1	0.99	0.18	0.64	1.34			
Group Embedded Figures Test	1	0.00	0.01	−0.02	0.03	1	0.04	0.84
Condition: AN	1	−0.39	0.13	−0.64	−0.14	2	13.54	0.001
Condition: WR	1	0.06	0.12	−0.18	0.30			
Condition: CN	0	0.00	0.00	0.00	0.00			
Contrast Estimate Results								
AN vs. WR		−0.45	0.13	−0.71	−0.19		11.64	0.001
AN vs. CN		−0.39	0.13	−0.64	−0.14		9.29	0.002
WR vs. CN		0.06	0.12	−0.18	0.30		0.25	0.62

NIH-PA Author Manuscript
NIH-PA Author Manuscript

Model 3: Angry

Parameter	df	Est	SE	−95%CI	+95%CI	Wald Statistics For Type 3 Analysis		
						df	χ^2	p
Intercept	1	1.00	0.13	0.75	1.25			
Group Embedded Figures Test	1	0.00	0.01	−0.02	0.01	1	0.24	0.63
Condition: AN	1	0.20	0.09	0.02	0.38	2	6.40	0.04
Condition: WR	1	−0.02	0.09	−0.19	0.15			
Condition: CN	0	0.00	0.00	0.00	0.00			
Contrast Estimate Results								
AN vs. WR		0.22	0.10	0.03	0.41		5.27	0.02
AN vs. CN		0.20	0.09	0.02	0.38		4.67	0.03
WR vs. CN		−0.02	0.09	−0.19	0.15		0.05	0.83

Note: All models controlled for the number of correct items on the Group Embedded Figures Test. Only statistically significant models are presented (comparisons of Happy, Afraid, and Neutral are not presented). AN = current diagnosis of anorexia nervosa; WR = prior diagnosis of anorexia nervosa but currently weight-restored; CN = no history of anorexia nervosa.