In the Eye of the Beholden: Tracking Developmental Psychopathology

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One area of agreement between poetry and neuroscience is that the eyes are the window to the soul. In fact, the eyes are the springboard for filial attachment and lifelong socialization. Human newborns prefer to look at faces looking at them than looking away,[1] and by 3 months of age, they are drawn to the eye region when viewing speaking faces.[2] Indeed, the importance of mutual gaze for early social development[3] and for social adaptation through the life span[4] highlights the fact that much of our socialization happens through processes mediated by the eyes.

Mutual gaze is also one of the key reflections of the forces of socialization affecting brain specialization. Lesion[5] and neurofunctional[6] studies indicate that both spontaneous fixation on eyes and reactivity to mutual gaze are mediated by the amygdala, which empowers a salience-detection system critical for survival (and for social adaptive functioning). It is, therefore, little wonder that nature selected the human eye to have the largest contrast between the white sclera and the darker iris relative to lower species, which are less dependent on distal social signals for survival.[7]

What happens when spontaneous orientation to the eyes of others does not happen? Maybe if this disruption occurs early enough in development, the result is autism.[8] From a clinical standpoint, there is mounting evidence that infants with autism fail to spontaneously orient to people, and failure to look at what others are looking at, a key mechanism of social and language learning,[9] is a hallmark of the condition. This is the context for the puzzle and its illumination in the study by de Jong and colleagues[10] in this issue of the *Journal*. On the one hand, children with autism show a deficit in following the gaze of others—deficits in joint attention; on the other hand, various experimental studies show that these children can adequately respond to the directional cues inherent in gaze shifts (as the eyes move in one or another direction). How can we reconcile these conflicting observations?

In an innovative fashion, Jong and colleagues raise the possibility and demonstrate it in their experiments that the ability to follow the directional cue of gaze shifts is intact in children with autism when the face stimuli are neutral. Yet, when faces are emotionally expressive, the children with autism fail to display the facilitation or potentiation effect generated by expressive faces. Typical individuals respond more quickly to shifts in other people's gaze when their face is emotional. Thus, the investigators conclude that impaired gaze following in autism results from impaired emotion processing, not from failure to extract direction from the gaze-shift cue. This is one example in which past experiments may have sacrificed ecological validity (the fact that in the real world, the faces that we encounter are dynamic and emotional) for experimental rigor (the use of neutral, static, or cropped faces to avoid experimental confounds). Thus, this study marks a strong return of more naturalistic paradigms in autism research.[11] It strongly raises the possibility that abstracting a social phenomenon from the social reality may render it, at times, invalid. Interestingly, results in previous gaze-cueing studies may have foreshadowed this conclusion. For example, in one experiment,[12] the performance of toddlers with autism was not only comparable to that of typical controls, it was faster. This unexpected finding raised the possibility that the children with autism were able to more quickly disengage from the faces than their typical peers, for

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whom the eyes may have been more than a directional cue (like an arrow): they were signals of a person, thus more difficult from which to disengage.

The study by Dadds and colleagues[13] builds on the premise that people with amygdala damage fail to process fear because they fail to fixate on other people's eyes. This, in turn, suggests an important role for the amygdala in regulation of spontaneous orientation to the eye region of the face. In an interesting clinical application, they hypothesized that because children with high psychopathic traits have difficulties recognizing fear, they may also fail to spontaneously orient to the eyes of others. The results of their experiments corroborate this hypothesis and suggest a role for amygdala dysfunction in this condition.

This study makes use of eye-tracking technology to measure the visual fixation patterns to the social stimuli presented to their subjects. This application of eye-tracking technology has been underused in psychiatry and represents a cost-effective, noninvasive, and relatively inexpensive way of quantifying social behavior. As clinicians, some of us have had for years the wish to be able to quantify our experiences when interacting with patients. Often felt intuitively, these experiences give us some avenue of understanding into how a patient is "processing us." The notion that a clinician is also a "walking laboratory of social engagement," shaped in the image of the social interaction partner (the patient), is a powerful force driving clinical insights and conclusions. Eye-tracking methodology can be used to add some quantification and precision to our intuitive measures of the human experience. In this context, Dadds and colleagues[13] help us contemplate the untapped potential of eye-tracking studies to shed light onto a wide range of childhood disorders insofar as their impact on social engagement is concerned.

Of great interest is also the lesson that seems to be embedded in this study. From autism to psychopathy, a similar mechanism—spontaneous orientation to the eyes of others—may be at play. In developmental psychopathology, development itself may be the key for developmental disorders.[14] Thus, a similar mechanism, affecting children's development at different times of the ontogenetic edifice, may lead to different disruptive effects with varied outcomes.

Taken together, this pair of studies reminds us of two key principles in experimental clinical research with children: do not forget real life (our clinical reality) and do not forget development (our best instructor).

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