Affective blindsight?

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Recent studies suggest that the amygdala, which resides in the temporal lobe of the human brain, plays a crucial role in processing the emotional valence conveyed by human facial expression. Moreover, functional imaging reveals that activation of the amygdala occurs when subjects are presented with facial expressions, most notably those that signal fear[1] or anger[2], even when the viewer denies having seen the fearful face[1]. The latter condition can occur under conditions of backward masking whereby the brief presentation of an emotional facial expression is rapidly followed by the presentation of a neutral expression. Although activation of the amygdala as a result of viewing emotional stimuli (masked or unmasked) does not unequivocally identify its role in the recognition of facial expression, a recent study by de Gelder, Vroomen, Pourtois and Weiskrantz hints that the amygdala response might be capable of mediating discrimination among facial expressions without conscious awareness[3].

The study by de Gelder *et al.* was prompted by a report by Morris *et al.*[4] who suggested that 'unseen' facial expression might be processed via a subcortical route that need not involve primary visual (striate) cortex. The superior colliculus receives direct input from the retina and is involved in early visual processes. The amygdala is a midbrain structure that is centrally involved in emotional processes and receives input from the superior colliculus via the pulvinar. Morris *et al.* demonstrated positive covariations of cerebral blood flow (as measured by PET imaging) in the pulvinar, superior colliculus and amygdala in response to masked facial expressions of anger that had been previously associated with an aversive stimulus. It is assumed that visual masking is a result of interference between the induction of neural activity by the stimulus and the mask, which occurs within the relatively slow response time of primary visual cortex neurons. Neurons in the superior colliculus are capable of responding to much more rapid changes in visual input and hence produce quite distinct responses to the facial expression and neutral mask. However, such responses fail to elicit conscious experience.

Interestingly, this secondary visual pathway, involving the midbrain and posterior thalamus, is precisely that which has been implicated in the phenomenon of blindsight. Blindsight refers to the residual visual ability of patients who, following damage to striate cortex, can detect, discriminate and localize visual stimuli presented in their blind visual field despite denying any accompanying conscious visual experience[5]. Blindsight may therefore be functionally akin to visual masking. Although the findings with blindsight patients so far have suggested that such residual ability applies only to the processing of relatively simple stimulus properties, de Gelder *et al.* set out to discover whether a blindsight subject (GY) could discriminate facial expression while remaining unaware of the faces to which he responded.

In the latest experiment, GY was presented with short video clips of an actress showing sad, happy, fearful or angry expressions, both to his intact and to his blind fields. GY could distinguish between these expressions (performance was significantly better than that expected from random responding) even when the video clips were presented to his blind field. However, he could not discriminate similar expressions when these were presented as still rather than moving images. Nevertheless, by recording event related potentials, de Gelder *et al.* did show that even these stationary images evoked neural responses in extrastriate cortex, although there was no suggestion that these responses differed according to facial expression. These data do, however, indicate that the visual stimuli elicited responses in cortical areas beyond striate cortex, although they cannot indicate whether or not the collicular–amygdala route was activated.

There is ample evidence that abstract properties associated with unseen visual stimuli can influence behaviour, for example, unseen digits or number words affect subsequent number judgements[6]. However, apart from one recent report[7] there has been little previous evidence that blindsight subjects can discriminate between even simple shapes, let alone something as complex as facial expression. So how did GY demonstrate 'affective blindsight'?

One explanation could be that, because in the initial experiment the same stimuli were presented to GY's intact and blind visual fields, GY could have implicitly learned associations between individual facial expressions and other simple visual properties of each stimulus, which could subsequently be used to discriminate between the four video clips. This explanation can be ruled out, however, because de Gelder *et al.*'s second experiment used four new video clips presented solely to the blind field. There was therefore no opportunity to learn such associations. One factor that might restrict the ability of blindsight subjects to discriminate shape is the relatively limited number of axons projecting from the colliculus to the pulvinar and the concomitant limitation of spatial resolution of the visual pathways available in blindsight. It could therefore be telling that de Gelder *et al.* could only find evidence for discrimination of expression using moving stimuli. Considerably more information about a face can be conveyed through a limited capacity channel when the face moves. Perhaps more importantly, though, it is well known that socially and biologically significant features, including facial expression[8], can be extracted from extremely limited sources of moving visual information. It has even been shown that 7-month-old infants can discriminate facial expression from the motion of just a few points of light attached to a moving face[9]. Perhaps GY can indeed make such ecological perceptions with the limited visual pathways remaining to him.

If, as the evidence of de Gelder *et al.* suggests, GY can covertly discriminate facial expression in his blind field, then we might ask how he does it. Morris *et al.*[4] showed in normal subjects that unseen faces excite a subcortical route to the amygdala. There is, however, a problem in invoking this pathway as the explanation of GY's abilities. Although the amygdala is implicated in the recognition of facial expression it has long been thought to be more involved in the perception of some emotions than others[10]. In their neuroimaging study Blair *et al.*[2] failed to find any systematic variation in amygdala response with the degree of anger in expressions, although they did find a systematic effect of fear and sadness. Neurological studies have found that patients with amygdala damage have some difficulty recognizing a range of emotions, although they showed particular problems with fear and anger and were near normal in their ability to recognize happiness and sadness[11]. There are several regions that appear to be involved in the processing of facial expression, including orbitofrontal and anterior cingulate cortex. The pattern of GY's performance, where happy and sad are discriminated well but anger and fear are often misidentified, may reflect the distributed nature of the representation of human facial expression.

In the conditioned fear paradigm[4] it is not clear whether the response of the amygdala is a result of the emotional quality of the facial expression or of the aversive nature of the event associated with that visual stimulus. The amygdala has been implicated in mediating fear responses following conditioning to a previously neutral stimulus. It is conceivable that the amygdala is chiefly involved in, for example, the generation of an autonomic response to the aversive stimulus. In neuroimaging studies that have required subjects to view masked

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facial expressions, whenever amygdala activation has been demonstrated in the absence of conditioned fear, subjects have not been required to make a forced-choice response about the nature of the unseen expression. That is, they were not engaged in the sort of guesswork undertaken by blindsight patients. It is plausible that GY, a much-practised observer, is able to monitor his autonomic responses and use them to mediate above-chance performance in the discrimination of facial expression. However, the differential responses of the amygdala to different facial expressions[2] is consistent with its role in the processing of at least some facial expressions. The rapidity with which the responses to unmasked fear-conditioned stimuli desensitize[12] leaves open the possibility that repeated presentation could mitigate against GY's performance. Moreover, it remains an interesting possibility that an improvement in performance might have been obtained had GY been asked to make a reflexive response, such as a key press, which is less likely than verbalization to invoke reflective conscious processes. The genuine guesses of an uninformed conscious system might potentially interfere with the stimulus-driven responses of the putative collicular circuit. We will have to wait for further experiments to answer this question.

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Affective blindsight: are we blindly led by emotions?

Response to Heywood and Kentridge (2000)

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The recent findings that facial expression can be recognized in the absence of awareness by blindsight patients suggests that, as the saying goes, we might indeed be blindly led by emotions. Although we are entirely in agreement with the comments made by Heywood and Kentridge [Heywood, C.A. and Kentridge, R.W. (2000) Affective blindsight? *Trends Cognit. Sci*. 4, 125–126][1] we would like to take this opportunity to discuss some of the questions that they raised and to describe our most recent data that may clarify some of the important issues.

As Heywood and Kentridge remark, the finding of covert discrimination by a blindsight subject of facial expressions presented to his blind field ('affective blindsight') raises the question of how this performance is achieved. An fMRI approach should provide new evidence with regard to the actual pathways sustaining affective blindsight, but it is worth noting that behavioral experiments can also help to clarify the neural basis of this phenomenon; for example, by determining which stimulus categories and attributes can be processed in the absence of striate cortex. Indeed, our most recent results indicate that blindsight is found only for facial expression and that covert discrimination of other facial attributes such as personal identity, gender and facial speech are not observed[2].

This pattern is consistent with the explanation suggested by Heywood and Kentridge that the biological or ecological salience of a stimulus is more important than the degree of visual complexity *per se* when deciding whether a given stimulus will support blindsight. However, if this were the only critical factor one might expect facial speech to support blindsight. Indeed, natural language, certainly when taken at the level of basic phoneme and syllable discrimination, is an integral part of our basic biological make-up. So it was something of a surprise that we were unable to find any indication of a capacity for discriminating or identifying facial speech in blindsight patients. One possible explanation rests upon the size of the stimuli used. There is evidence that spatial resolution is poor in blindsight, and so stimulus size is likely to be crucial. Perhaps discrimination of facial speech was not found because the lower part of the face contains relatively small stimulus features. It remains to be seen whether a very large lip-reading stimulus would support blindsight.

More importantly though, this negative result does seem to pose problems for Heywood and Kentridge's suggestion that movement might be one of the critical factors in explaining the findings. This suggestion was based upon our earlier finding that, although moving images supported affective blindsight, stationary images did not. This is consistent with findings that demonstrate that discriminating between two patterns of biological movement can be done on the basis of very limited or very impoverished input. But if movement is important, why does facial speech not support blindsight? In facial speech, one has a stimulus that is socially and biologically significant and for which discrimination can be done on the basis of the same kind of impoverished information consisting of a small number of moving dots[3].

Whatever the outcome of that particular debate we do now have some preliminary evidence suggesting that stationary images of facial expressions can support affective blindsight (de Gelder *et al.*, unpublished data). In our experiment, we measured the impact of a face presented to the blind field on the response to a facial stimulus presented to the intact, seeing field. The results showed that incongruency between the expressions presented to the two hemifields significantly delayed judgement of the facial expression in the seeing field.

This is an illustrative example that covert processing can often only be found with an indirect rather than a direct method, in which subjects are required to 'guess' the identity of stimuli they patently deny seeing. As Heywood and Kentridge suggest – in line with some recent findings about qualitative differences between overt and covert processes – the superior sensitivity of indirect methods for uncovering covert processing or residual processing abilities might be due to an absence of conflict between overt, reflexive answering and covert responding. We addressed

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