

Understanding the Embodiment of Perception

Obviously perception is embodied. After all, if creatures were entirely disembodied, how could physical processes in the environment, such as the propagation of light and sound, be transduced into a neurobiological currency capable of generating experience? Is there, however, any deeper, more subtle sense in which perception is embodied? Perhaps. Alva Noë's (2004) theory of enactive perception provides one proposal. Where it is commonly thought that

(CAH) Perceptual experience is caused, in part, by sensorimotor skills,

Noë suggests the more radical hypothesis that

(COH) Perceptual experience is constituted, in part, by sensorimotor skills.

Clearly these two hypotheses offer distinct conceptions of what the embodiment of perception amounts to. In this paper, I will argue that the two principal lines of argumentation in Noë (2004) fail to support (COH) over (CAH).

Three features of these hypotheses bear comment. First, in investigating these hypotheses, we assume that perceptual experience is, in some sense, other than mere peripheral stimulation of the sense organs and something more than sensation. Perceiving might, thus, involve the application of concepts to sensations or the organization of sensations into concepts. Second, we suppose that there is an empirical question at stake here. Thus, to make this an interesting empirical question, rather than a matter of stipulation, we will want some characterization of "perceptual experience" that is conceptually independent of our characterization of sensorimotor skills. In other words, it would be uninteresting to find that perceptual experiences are constituted, in part, by sensorimotor skills if one simply means by a "perceptual experience" an experience that is constituted, in part, by sensorimotor skills. Third, by a sensorimotor skill, we CAH mean a capacity to perform in a certain way, rather than

merely possessing some theoretical understanding or understanding of what is practically involved. It is one thing to know what a poker face is; it is another to have the skill to keep a poker face during games of poker. Other refinements could surely be added, but these three provide us with a reasonable starting point for evaluating some of the evidence for (COH) and (CAH).¹

A thorough evaluation of (COH) and (CAH) would require looking at evidence bearing on each sense modality. As a bit of *a priori* scientific methodology, we should not simply assume that all sense modalities involve sensorimotor skills or that they all involve sensorimotor skills in the same way. Scientific familiarity with the sense modalities reinforces this point. Vision seems to make the greatest demands on sensorimotor skills. For example, the eye is in constant motion due to a variety of factors. Two or three times per second the eye moves from one fixation point to another. The eye saccades. During these saccades we do not experience blurring or distortion. Nor do we experience distortions when we move our heads normally. There are sensorimotor mechanisms for preventing distorted perceptions in these cases. This stability through saccades and head motions might be contrasted with the distortion introduced when a person moves her eye with her finger. There are no compensatory mechanisms for handling the finger-induced eye motions. Exactly how different vision and, say, olfaction may ultimately turn out to be, the point remains that there are good reasons not to assume that both

¹ For example, one might want to consider (COH) as part of a case for extended cognition, according to which cognitive processes literally extend beyond the boundaries of the brain. To do this, however, one would have to make the case that sensorimotor skills extend beyond the brain and that, if sensorimotor skills do so extend, that the extended part is itself a cognitive process.

make the same demands on sensorimotor skills.

This last caveat in place, space limitations require that the present discussion be limited to vision, the modality Noë uses to make his case. So, how does the available evidence in vision bear on (COH) and (CAH)? The evidence from saccades shows, at most, that sensorimotor skills are causally relevant to perceptual experience. This much is common fare in textbooks on sensation and perception. Why should we suppose that the sensorimotor mechanisms or skills, in part, constitute the perceptual experience? Noë believes he has two principal reasons.

1. Cases of congenital cataracts.

The first source of evidence Noë cites involves the consequences of removing congenital cataracts in adult patients. Consider the reports to which Noë draws our attention. One is Gregory and Wallace's description of patient, S. B.:

S. B.'s first visual experience, when the bandages were removed, was of the surgeon's face. He described the experience as follows: He heard a voice coming from in front of him and to one side: he turned to the source of the sound and saw a "blur." He realized that this must be a face. Upon careful questioning, he seemed to think that he would not have known that this was a face if he had not previously heard the voice and known that voices came from faces (Gregory & Wallace, 1963, p. 366, cited in Noë, 2004, p. 5).

Oliver Saks provides another example Noë cites:

Virgil told me later that in this first moment he had no idea what he was seeing. There was light, there was movement, there was color, all mixed up, all meaningless, a blur. Then out of the blur came a voice that said, "Well?" Then, and only then, he said, did he finally realize that this chaos of light and shadow was a face—and, indeed, the face of his surgeon (Saks, 1995, p. 114, cited in Noë, 2004, p. 5).

Clearly, immediately after surgery, the adult patients continue to have some sort of deficit.

The existence of this deficit does not, of course, tell us about its nature. They have

sensations, but there still appears to be something missing. What's missing? One possibility, the one Noë favors, is that the deficit arises because these patients have not integrated their sensations with their sensorimotor skills. They are what Noë described as “experientially blind” (Noë, 2004, p. 4). Another possibility is that development with cataracts in place leads to a deficit in sensory processing. These possibilities, of course, are not mutually exclusive. Growing up with cataracts may well cause deficits in both the production of sensations and in the integration of sensations with the normal sensorimotor apparatus.

Noë considers these two hypotheses, but is surprisingly dismissive of the possibility of sensory processing deficits. After providing an articulate statement of the problem, Noë offers the following comments: “This objection has some force. In section 1.3 I turn to an example of putative experiential blindness [that of inverting lens] that is not vulnerable to this criticism. Taken together the two examples make a strong case for experiential blindness and so for (COH)” (Noë, 2004, p. 7). While conceding some force to the argument in one sentence, two sentences later he appears to think it helps make for a “strong case” for experiential blindness. Yet, if the evidence does not show that there is some deficit of sensorimotor integration, it is unclear how it can support (COH). Generally, data that accords with either of two interpretations is not taken to support one or the other. The situation here is worse for Noë than just the logic of the situation indicates. It is not just that the logic of the situation allows for the existence of sensory processing deficits, there is, in fact, experimental evidence indicating that congenital cataracts lead to deficits in sensory processing. Perhaps there are sensorimotor integration problems and sensorimotor problems as well, but there is evidence for sensory deficits.

Several recent studies have indicated that humans with dense congenital cataracts suffer

deficits in grating acuity, linear letter acuity, spatial contrast sensitivity, temporal contrast sensitivity, peripheral vision, stereo vision, perception of global form, and perception of global motion (Birch, Stager, Leffler, & Weakley, 1998, Bowering, Maurer, Lewis, & Brent, 1993, Ellemberg, Lewis, Maurer, Brar, & Brent, 2002, Ellemberg, Lewis, Maurer, & Brent, 2000, Ellemberg, Lewis, Maurer, Liu, & Brent, 1999, Lewis, Ellemberg, Maurer, Wilkinson, Wilson, Dirks, & Brent, 2002, Lewis, Maurer, & Brent, 1995, Mioche & Perenin, 1986, Tytla, Lewis, Maurer, & Brent, 1993, Tytla, Maurer, Lewis, & Brent, 1988). None of these deficits have been described as any kind of failure to co-ordinate vision with sensorimotor skills. Another source of evidence is the study of the effects of binocular lid suture on neonate non-human animals. These interventions, like congenital cataracts, allow the projection of diffuse light onto the retina, while blocking the highest frequency spatial patterns in the stimulus. In a 1982 review, Murray Sherman and Peter Spear reported that striate cortex shows any number of abnormalities.² Striate cortex is less responsive than normal to visual stimulation. In deprived cats, fewer cells respond to standard stimuli and even those cells that do respond have lower peak response rates. In addition, among cells that remain responsive, a large proportion have abnormal receptive fields. In normal cats, a large fraction of cortical cells have excitatory and inhibitory regions that make them sensitive to the orientations of edges, bars, and lines. In bilaterally lid sutured cats, however, the inhibitory portions of the receptive fields of these cortical cells are weak or absent.³ This will likely make the perceptual experience of borders faint or fuzzy, suggesting a sensory

² Sherman & Spear, (1982).

³ White, Coppola, and Fitzpatrick, (2001), report similar results in binocularly lid sutured ferrets.

explanation of some of the perceptual deficits found in Gregory and Wallace's patient, S. B., and Sacks's patient Virgil. Further, in normal cats, many of the cells in striate cortex are sensitive to the direction of motion of edges, bars, and lines. In bilaterally lid sutured cats, many of the responsive cortical cells lose their sensitivity to the direction of motion. This might explain any failure of sensorimotor co-ordination found in patients after bilateral congenital cataract removal. Finally, bilaterally lid sutured cats also lose sensitivity to binocular disparity, thereby losing an important depth cue. Again, this purely sensory deficit might explain the difficulty patients might have in negotiating the three dimensional world.

Suppose, however, that in fact these post-operative cataract patients are experientially blind in Noë's sense. That is, suppose that they do not or cannot integrate their sensations with patterns of movement and thought. Does this suffice to establish (COH)? No. There still remain two ways in which this data falls short. Noë's conception of experiential blindness is a kind of perceptual deficit that he sets alongside sensory blindness. But, there are, of course, many kinds of perceptual blindness. There is achromotopsia (color-blindness) and this of several types. There is akinetopsia, motion blindness. There is face agnosia and form agnosia. A person that is color blind is not entirely without perception, only without perception of some color distinctions. A person who is motion blind is not completely blind, only blind to motion. Similarly for face and form agnosia. And, indeed, the same for Noë's experiential blindness. As a conceptual possibility, it appears to be possible that some humans might perceive things, only without these perceptions being integrated into patterns of personal movement and thought. The only thing that might preclude this conceptual possibility is if Noë's concept of perceptual experience simply includes being constituted, in part, by sensorimotor skills. But, then, Noë's claim that

perceptual experience is constituted, in part, by sensorimotor skills just becomes some sort of logical truth, rather than an empirical hypothesis.

To see the second problem, we again suppose, as Noë wishes us to, that these cataract patients are experientially blind; they fail to integrate sensory stimulation with patterns of movement and thought. What is responsible for this failure? What explains it? Noë wants us to conclude that the failure is due to the loss of a constituent of perception, namely, sensorimotor skills. No sensorimotor skills, no perceptual experience. That is, he thinks that (COH) explains the failure of integration; he thinks that (COH) explains the experiential blindness. But, (CAH) seems to offer just as good an explanation of the putative experiential blindness as does (COH). According to (CAH), the reason post-operative cataract patients are experientially blind is that their sensorimotor skills are not yet causally connected to their sensory apparatus in the proper way. It's not that these sensorimotor skills are essential to perception because they are constitutive, in part, of perception; it is that these sensorimotor skills are essential to perception because they have an important causal role in shaping perceptions and normal causal connections have been disrupted.

So, it appears that Noë's appeal to post-operative congenital cataract patients fails to establish (COH) in three ways. First, the cataract patients may well suffer from sensory blindness, in addition, to experiential blindness. Second, even if cataract patients are experientially blind, this still fails to establish (COH) for two reasons. First, these patients might be experientially blind, but not totally blind as (COH) requires. Second, (COH) provides no better explanation of experiential blindness than does (CAH).

2. Distorting lens

For over a century, psychologists have been interested in the effects of long-term use of distorting lens of one sort or another. Noë wishes to appeal to them in support of (COH). Noë is correct in his claim that these experiments provide cases of experiential blindness that overcome the first problem we noted above regarding possible sensory deficits in cataract patients.

Nevertheless, he fails to recognize the gaps between the discovery of experiential blindness and (COH). Consider, then, a description of the effects of wearing spherical prism lens,

During visual fixations, every movement of my head gives rise to the most unexpected and peculiar transformations of objects in the visual field. The most familiar forms seem to dissolve and reintegrate in ways never before seen. At times, parts of figures run together, the spaces between disappearing from view: at other times, they run apart, as if intent on deceiving the observer. Countless times I was fooled by these extreme distortions and taken by surprise when a wall, for instance, suddenly appeared to slant down the road, when a truck I was following with my eyes started to bend, when the road began to arch like a wave, when houses and trees seem to topple down, and so forth. I felt as if I were living in a topsy-turvy world of houses crashing down on you, of heaving roads, and of jellylike people (Kohler, 1964, p. ???, cited in Noë, 2004, p. 8).

What appears to be going on here is a failure to integrate sensations into sensorimotor skills, i.e., we appear to have experiential blindness. This is the point of Noë's example.

Nevertheless, as we saw in the previous section, the existence of experiential blindness does not suffice to establish (COH). Experiential blindness as Noë defines it is a particular kind of perceptual deficit. But, one can have this deficit without being entirely without perception. In the case of this subject, K, even Noë admits that "K is not completely blind, to be sure; he recognizes the trucks, the trees, and so forth. But nor is he completely able to see. His visual world is distorted, made unpredictable, and topsy-turvy" (Noë, 2004, p. 8). If K is able to synthesize the sensations caused by trucks, trees, people and so forth, into forms that enable him

to recognize them as trucks, trees, and people, then there seems to be some basis for saying that he perceives the trucks, trees, people, and so forth. Thus, K perceives things, even though experientially blind. K perceives things even through the loss of his sensorimotor skills. This interpretation, however, is inconsistent with (COH).

As in our discussion of the cataract patients, we can ask why those who wear distorting lens of one form or another are experientially blind. Why is it that those who wear distorting lens have their sensations decoupled from their sensorimotor apparatus? Noë proposes (COH), that a constitutive element of perception is lost. But, (CAH) seems to offer as good an explanation of experiential blindness as does (COH). (CAH) proposes that perceptions are influenced by two types of factors, the input from the retina and input from the mechanisms that compensate for bodily motions. The lens, however, disrupt the relationship that normally exists between the retinal input and the motion compensating mechanisms. The lens distort the co-ordination of retinal signals and bodily signals. There is no need to adopt (COH) for this explanation.

3. Conclusion

The upshot of the foregoing is that the evidence from congenital cataract patients and from observations of individuals wearing distorting lens fails to support the radical hypothesis that perception is constituted, in part, by sensorimotor skills. Rather, they are perfectly consistent with what mainstream cognitive psychology has long believed, namely, that perception is causally influenced by sensorimotor skills.

References

Birch, E. E., Stager, D., Leffler, J., & Weakley, D. (1998). Early treatment of congenital

- unilateral cataract minimizes unequal competition. *Investigative Ophthalmology and Visual Science*, 39, 1560-1566.
- Bowering, E. R., Maurer, D., Lewis, T. L., & Brent, H. P. (1993). Sensitivity in the nasal and temporal hemifields in children treated for cataract. *Investigative Ophthalmology and Visual Science*, 34, 3501-3509.
- Elleberg, D., Lewis, T. L., Maurer, D., & Brent, H. P. (2000). Influence of monocular deprivation during infancy on the later development of spatial and temporal vision. *Vision Research*, 40, 3283-3295.
- Elleberg, D., Lewis, T. L., Maurer, D., Liu, C. H., & Brent, H. P. (1999). Spatial and temporal vision in patients treated for bilateral congenital cataracts. *Vision Research*, 39, 3480-3489.
- Elleberg, D., Maurer, D., Brar, S., & Brent, H. P. (2002). Better perception of global motion after monocular than after binocular deprivation. *Vision Research*, 42, 169-179.
- Fine, I., Smallman, H. S., Doyle, P., & Macleod, D. I. A., (2002). Visual function before and after the removal of bilateral congenital cataracts in adulthood. *Vision Research*, 42, 191-201.
- Gregory, R. L., and Wallace, J. G. (1963). Recovery from early blindness: A case study. *Experimental Psychology Society*. Monograph No. 2.
- Hubel, D., & Wiesel, T. (1963). Receptive fields of cells in striate cortex of very young, visually inexperienced kittens. *Journal of Neurophysiology*, 26, 994-1002.
- Hubel & Wiesel, (1965). Comparison of effects of unilateral and bilateral eye closure on cortical unit responses in kittens. *Journal of Neurophysiology*, 28, 1029-1040.
- Noë, A. (2004). *Action in Perception*. Cambridge, MA: MIT Press.
- Lewis, T. L., Elleberg, D., Maurer, D., Wilkinson, F., Wilson, H. R., Dirks, M., & Brent, H. P. (2002). Sensitivity to global form in glass patterns after early visual deprivation in humans. *Vision Research*, 42, 939-948.
- Lewis, T. L., Maurer, D., & Brent, H. P. (1995). The development of grating acuity in children treated for unilateral or bilateral congenital cataract. *Investigative Ophthalmology and Visual Science*, 36, 2080-2095.
- Mioche, L., & Perenin, M. (1986). Central and peripheral residual vision in humans with bilateral deprivation amblyopia. *Experimental Brain Research*, 62, 259-272.
- Sacks, O. (1995). *An Anthropologist on Mars: Seven Paradoxical Tales*. New York: Knopf.
- Tytla, M. E., Lewis, T. L., Maurer, D., & Brent, H. P. (1993). Stereopsis after congenital cataract deprivation in the monkey. III. Reversal of anatomical effects in the visual cortex. *Investigative Ophthalmology and Visual Science*, 34, 1767-1773.
- Tytla, M. E., Maurer, D., Lewis, T. L., & Brent, H. P. (1988). Contrast sensitivity in children treated for congenital cataract. *Clinical Vision Sciences*, 2, 251-264.