The Physical Basis of Out-of-Body Vision

To the Editor:

Some time ago I suggested in this Journal some ways to advance our understanding of out-of-body vision (Krishnan, 1988). I would like to suggest three more strategies.

First, a number of people who have had an out-of-body experience (OBE) spontaneously have claimed that when the experience occurred in conditions of darkness, they could see the environment clearly and in color, just as they could in daylight in the normal state (Green, 1968). That is not how we normally see in the dark. In total darkness we cannot see anything at all. But if there is just enough light to stimulate the photosensitive elements in the retina, they adapt gradually to the low intensity of light. It takes about an hour for the eyes to become fully dark-adapted, and even then we do not see objects in any detail or in normal colors. They may be seen as different shades of gray or they may take on a greenish or bluish shade (Vernon, 1971).

Since out-of-body experiencers (OBErs) have not reported these characteristics of normal dark vision, the question arises whether they were hallucinating or using some unknown physical perceptual processes. The cases of OBEs in the dark that have been cited in the literature are not very helpful in deciding this question, because they generally do not seem to have been verified. Therefore, in future research it would be important to verify claims of clear out-of-body sight in darkness.

If it is confirmed that OBErs do see objectively in the dark and are not hallucinating, then we can examine whether or not this form of perception resembles normal dark-adapted vision. That may help us address questions such as whether it is visible light or some other form of electromagnetic radiation that serves as the carrier of information in out-of-body vision, what may be the receptors sensitive to the information carrier, and what could be the related afferent neural pathways. I give below, by way of illustration, some examples of the kind of inferences that can be made about the information-carrying radiation involved in out-of-body sight.

If the OBEr's vision is like that of someone whose eyes are dark-adapted, that would suggest that visible light could be the information carrier and that the receptors could be like the rods and cones of the retina. On the other hand, if OBErs are able to see correctly in complete darkness, that would imply that the receptors involved are not of the retinal kind and that the carrier of information is not visible light,

though it might be some other form of electromagnetic radiation. Finally, if subjects cannot see at all in total darkness but are able to perceive in dim light in color and as clearly as we do normally in daylight, that would indicate that visible light is necessary for out-of-body sight but that the light receptors differ in photosensitivity from those in the retina.

My second suggestion derives from the finding that several experiencers have reported "seeing" events in their immediate vicinity but outside their visual field (Sabom, 1982). Such cases may mean only that the *eyes* may not play a part in out-of-body sight; they do not rule out the involvement of other parts of the visual system. Indeed, one of the features of out-of-body vision supports this view. It usually takes some practice to learn a new way of doing a familiar task. For example, a righthanded person will not be able to write with the left hand clearly and quickly at first try, but only after some days or weeks of practice. But it appears from verified OBEs that the subjects do not have to learn to see without the use of the eyes; they readily begin to see just as one would when one opens one's closed eyes (Sabom, 1982). That, I should think, would not be possible without some of the deeper elements of the visual system vitally concerned with sight coming into play.

If out-of-body sight does not occur independently of the visual system, what neural structures might underlie it? One hint comes from a speculation made by Stanislav Grof and Joan Halifax-Grof (1976) about near-death experiences. They pointed out that the cellular elements of the old subcortical parts of the brain are less sensitive to lack of oxygen than those in the newer cortex, and therefore they may survive longer than those in the cortex after the cessation of the heartbeat. That means that even when the electroencephalograph is flat, brain activity could still be going on in the old brain.

Grof and Halifax-Grof suggested that if consciousness is associated with the subcortical regions, then NDEs could be the conscious concomitants of the old brain processes. It is noteworthy in this connection that the pioneering neurosurgeon Wilder Penfield (1975) held the view that the neural substratum most essential for consciousness lay in the older brainstem. I think the suggestion offered by Grof and Halifax-Grof makes a good case for investigating the role of the old subcortical brain in out-of-body sight.

Another hint of the neural structures underlying out-of-body vision comes from experiments with persons whose primary visual cortex has been damaged. These patients may insist that they cannot see a thing,

and yet when they are asked to touch or look at a light placed in front of them they are able to do so accurately. In other words, they know, or as they put it, they "feel," the direction in which they should look. Experiments on baby hamsters, some of them with their primary visual cortex removed and others with their midbrain visual center destroyed, suggest that in mammals, including human beings, the midbrain visual region functions to locate *where* an object is and the primary visual cortex to see *what* it is (Nathan, 1983). The implication of this finding for out-of-body vision is that, since OBErs have no difficulty recalling after the experience the location of objects and individuals in their immediate environment, it is possible that the midbrain visual region participates in out-of-body sight.

Finally, as pointed out above, experiencers do not appear to have to learn to see extraocularly. Why then can't they evoke this form of vision at will? One possibility worth examining is that the receptors involved in out-of-body vision may not ordinarily be sensitive to register information, but only become sensitive under special circumstances. That is, out-of-body sight manifests only when the sensitivity of its receptors rises to a certain level, and it disappears when their sensitivity falls below that level.

This proposal is not a farfetched one. It is known that individuals sometimes develop unusual abilities under certain circumstances, even though the underlying mechanism is far from clear. For example, many psychics have dated the development of their "powers" to the time when they were gravely ill or were involved in a serious accident. Deficiency of adrenal cortical hormones increases sensory acuity to an extraordinary extent (Luce, 1973). Yet another example comes from Alfred Binet's experiments on hysterical subjects with anesthetic skin areas. He found that when he lightly pressed a steel disk with a design in relief against an anesthetic area on the back of the neck of one of these subjects she experienced a visual image of the design vivid enough for her to draw (McGurdy, 1961).

In the case of out-of-body vision, what may make the involved receptors sensitive? I suggest that all situations in which the experience is known to occur are marked by sensory deprivation, in the general sense of reduced input of patterned information to the brain. Out-of-body sight may be one of the ways in which the subject's need for information or stimulation is satisfied (Krishnan, 1985). It seems useful therefore to investigate whether sensory deprivation, or the stress that it causes, has biochemical or other concomitants that can alter receptor sensitivity.

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