Unraveling the Dark Paradox of 'Blindsight'

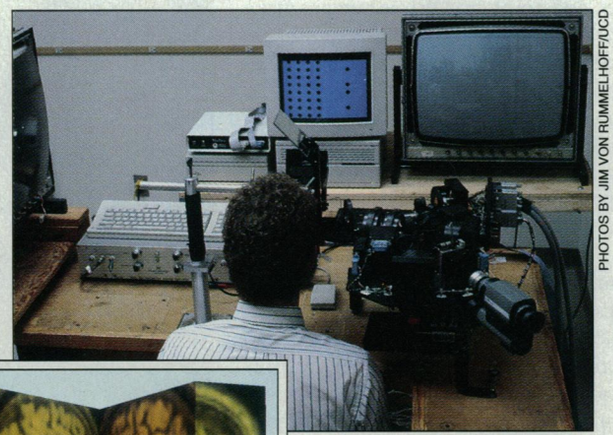
Look at a point straight ahead of you, and imagine a vertical line passing through that point. Then imagine that your visual field ended at that line—that you could see nothing to the left of it. Many people experience that kind of partial blindness after damage to the primary visual cortex, an important processing area in the brain. Researchers have long been intrigued, however, by the fact that in some of these people, the blank part of their visual field isn't as blind as it initially seems. Carefully designed experiments show that some people who are unaware of seeing anything in that blind area can nevertheless be induced to look at, point to, or base simple judgments on images presented there.

Those who study this phenomenon call it "blindsight" and have attributed it to alternative neural pathways by which visual information from the blind area can bypass the damaged primary visual cortex and still reach higher areas of the brain. But now Michael Gazzaniga and his colleagues at the University of California, Davis, have come along with a new proposal that could turn this model on its head. In at least some cases, they argue in this issue of *Science* (p. 1489), information is not bypassing the damaged primary visual cortex, but instead is being relayed through islands of functional brain tissue there that have escaped damage.

At the very least, this finding sends a warning to blindsight researchers to check their subjects for islands of healthy visual cortex before interpreting their findings. Gazzaniga doesn't stop there, however. He has gone on to suggest that his group's finding may provide a general explanation for blindsight. "We would put our bet down that when you do get the phenomenon, it is [primary] visual cortex supporting it," he says. But many of those who study blindsight aren't buying the generalization. "I don't quarrel with his observations at all," says blindsight researcher Alan Cowey of Oxford University. "The mistake I think he has made is to jump to the conclusion that everybody else has been studying this artifact."

One fact on which everyone agrees is that the primary visual cortex is an essential element in the normal visual pathway. In that pathway, information from the two eyes is blended in the thalamus, then passed on to the primary visual cortex, where it is laid out in the form of a map of the visual field. The primary visual cortex is itself split between the two hemispheres of the brain—the left part contains the map for the right part of the visual field, and vice versa.

When light from a particular part of the visual field falls on the retina, the signal travels to the equivalent spot in the map before being relayed to higher parts of the brain. If part of the primary visual cortex is destroyed—for example, by stroke, injury, or surgery—the corresponding part of the visual field disappears from view. Because such damage usually affects only one brain hemisphere, this type of blindness usually blocks out at most half of a person's visual field, while the other half retains normal vision.



PHOTOS BY JIM VON RUMMELHOFF/UCD

The vision thing. Mark Wessinger demonstrates apparatus for measuring blindsight. Computer image shows brain with a lesion in the primary visual cortex.

Beginning in the early 1970s, several labs began finding an odd sort of residual vision in the blind areas of some patients. When an image is flashed in the blind part of their visual field, they are able to point to where the image appeared, or to guess correctly when it appeared. Usually they insist that they saw nothing, and are merely guessing, but their scores are better than would be expected from an uninformed guess, suggesting the visual processing areas of the brain have received some signal from the blind area.

But how does that visual information get through? Blindsight researchers have assumed that the signal must be taking alternative routes to bypass the damaged area of the visual cortex. Gazzaniga and his colleagues, psychophysicist Robert Fendrich and graduate student Mark Wessinger, tested that assumption using a two-pronged approach to take the closest look yet at the primary visual cortex of a blindsight subject.

They used a new technique to finely map the parts of one subject's blind area in which he had blindsight. Such fine mapping had never been accomplished, because it depends on the subject fixing his gaze absolutely unwaveringly and reproducibly during each testing session—something that is virtually impossible. Gazzaniga's group got around the problem with a new eye-tracking device that shifts the entire visual scene to follow even the slightest movements of the subject's eyes. When their map was complete, it revealed a well-delineated island within the blind area that showed blindsight. But when tested outside that island, the man showed no blindsight at all.

When the team compared that information with magnetic resonance images of the man's primary visual cortex, they found an island of living tissue in an area that corresponded roughly with the location of the blindsight. That correlation led them to conclude that their subject's blindsight was probably due to visual signals being relayed through that spared bit of primary visual cortex; if blindsight relied on completely separate neural pathways, reasoned Gazzaniga, it shouldn't be so patchy, because those pathways should serve the entire visual field.

"It's a very plausible explanation; they have done the experiment [very] carefully," says Richard Andersen, a visual scientist at the Massachusetts Institute of Technology (MIT). "I think Gazzaniga's argument is solid," adds vision researcher Peter Schiller, also of MIT. But Schiller says it doesn't eliminate all other interpretations. For example, he says, he has been studying a neural pathway from the thalamus directly to higher brain regions, a pathway that is itself patchy and doesn't evenly cover the visual field. That pathway, rather than spared patches of primary visual cortex, he says, could be responsible for the blindsight the Gazzaniga team observes. Both Schiller and Andersen add that findings from just one patient can hardly be the basis for sweeping changes in the interpretation of blindsight.

That point is echoed strongly by other blindsight researchers, who cite evidence they say proves Gazzaniga's notion wrong. Alain Ptito of the Montreal Neurological Institute points to his studies in people who have had hemispherectomies, a radical treatment for epilepsy in which an entire half of the cerebral cortex is removed. Some of these patients show blindsight, and they can't possibly have spared islands of primary visual

cortex, he says—because they have no cortex on that side at all. Oxford University psychologist (and codiscoverer of blindsight) Lawrence Weiskrantz adds that monkeys show blindsight even when they have had both halves of their primary visual cortex completely removed.

Gazzaniga is not convinced by either argument. First of all, he insists, "you absolutely cannot cross-reference monkey and human work." As evidence of the difference between the species, he notes that all monkeys that lack a primary visual cortex show blindsight, while the phenomenon is rare in humans with similar damage. As for the hemispherectomized patients, all those that have been studied had their surgery as children, and so he argues that they constitute special cases, because young brains have a great ability to reorganize and compensate for lost functions. Test someone who had a hemisphere removed as an adult, he posits, and you won't find blindsight.

Indeed, Gazzaniga's hypothesis makes many predictions like that, which can be tested with further experiments. And those experiments are proceeding. Ptito says he is about to begin testing a group of patients who had hemispherectomies as adults. And psychophysicist Keith Ruddock of Imperial College, London, and his colleagues recently found that magnetic resonance imaging of a much-studied patient known as "GY" revealed no spared regions in primary visual cortex—a result that would seem to contradict Gazzaniga's hypothesis.

Gazzaniga is reluctant to comment on work he hasn't seen, but he says he would like to test GY in his blindsight set-up to make the experiment complete. Such subject-swapping, many agree, would also help to standardize such important parameters as what kinds of tests are used for blindsight, how the questions are asked, and how the images are presented to the patients, all of which have been shown to be critical to the phenomenon.

Whatever the outcome of the subject swapping, most agree that Gazzaniga's study is an important contribution to the field. "When you claim there is blindsight [in the absence of any primary visual cortex] there is a chance that it may not actually be the case," says MIT's Andersen. "It raises a caution for anyone doing this research."

–Marcia Barinaga

Additional Reading

A. Cowey and P. Stoerig, "The Neurobiology of Blindsight," *Trends in Neurosciences*, **14**, 140 (1991)

L. Weiskrantz, "Unconscious Vision: The Strange Phenomenon of Blindsight," *Sciences*, **32**, 22 (1992)

A. Ptito, F. Lepore, M. Ptito, M. Lassonde, "Target Detection and Movement Discrimination in the Blind Field of Hemispherectomized Patients," *Brain*, **114**, 497 (1991)

ASTRONOMY

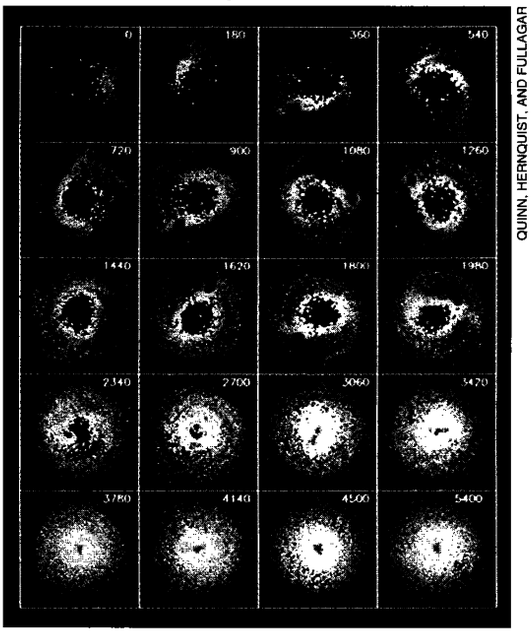
Tracing the Milky Way's Rough-and-Tumble Youth

Our galaxy's graceful spiral form seems the very picture of serenity. For decades, astronomers have thought it had a history to match: an uneventful progression from a primordial gas cloud to an orderly swirl of stars. But as observers take a closer look at the ages, chemistry, and motions of the stars that make up the Milky Way, they are seeing subtle signs of turmoil. And now several groups have concluded that our galaxy had a violent youth, tangling with and gobbling up one or several smaller galaxies in its neighborhood.

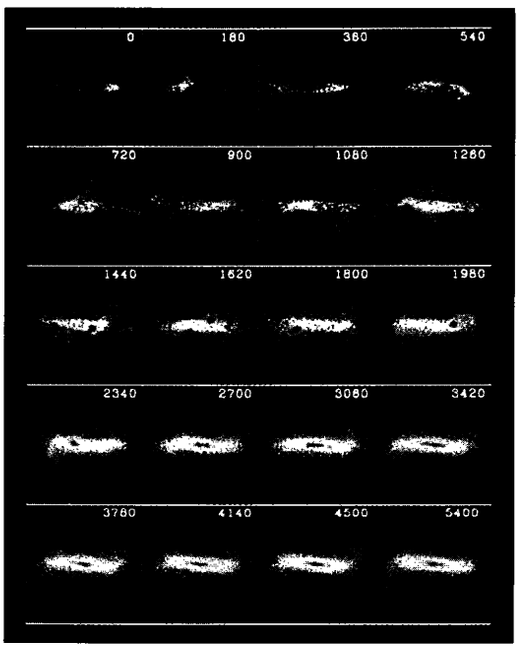
One line of evidence comes from studies of globular clusters, dense knots of stars that are scattered in a vast spherical halo around the familiar disk of the galaxy. Some of them, say Robert Zinn of Yale University and Sidney van den Bergh of the Dominion Astrophysical Observatory in Canada, show every sign of being stolen goods, wrested away from other galaxies as the Milky Way collided with them. Meanwhile, other investigators are reporting signs that these ancient collisions have left their mark on stars within the disk of the Milky Way itself.

If these findings are confirmed, they would add to the challenge facing the classical picture of how the galaxy took shape. Proposed 30 years ago by Olin Eggen of Cerro Tololo Interamerican Observatories, Donald Lynden-Bell of Cambridge University, and Allan Sandage of the Carnegie Institution, that picture holds that the galaxy formed from a single cloud of gas that collapsed in isolation. First born, in this picture, was a spherical halo of stars—precursors of today's globular clusters. The galaxy's flat disk and its central bulge came later, in the last stages of the collapse. Earlier this year, Yale astronomer Young-Wook Lee cast doubt on this "outside-in" picture when he reported that the oldest stars in the galaxy reside in the bulge, not in the halo (*Science*, 7 August, p. 746). Similarly, notes astronomer Peter Quinn of the Mount Stromlo and Siding Spring Observatories in Australia, "a subdivision of globular clusters is not what you would expect from a monolithic collapse." Instead it suggests that at least some of the clusters were added by collisions well after the core of the galaxy formed.

As a theoretical possibility, "inside-out" formation isn't new to astronomers; they've been debating it since 1978, when the scenario was proposed by Zinn himself and by Carnegie's Leonard Searle. And in 1984, Alexander Rodgers of the Australian National University and George Paltoglou of the University of Maryland presented some



QUINN, HERNQUIST, AND FULLAGAR



Milk shake. In a computer simulation, top and side views show in million-year intervals how a collision inflates the Milky Way's disk.