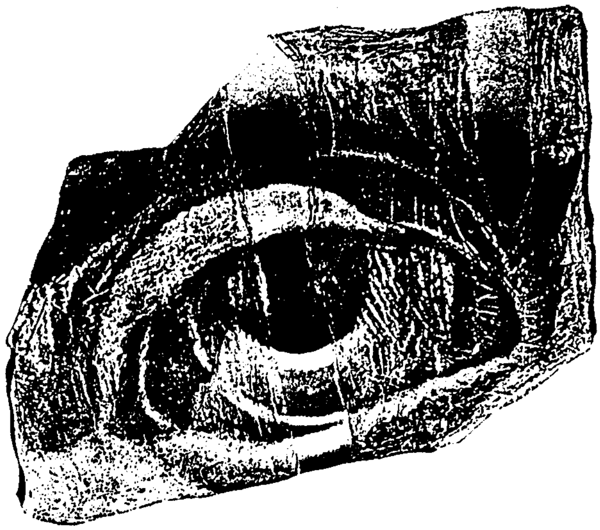
UNCONSCIOUS VISION

The Strange Phenomenon of Blindsight

by LAWRENCE WEISKRANTZ



Irina Nakhova, #13, 1992

AT AGE FOURTEEN a boy I will call Daniel began having migraine headaches of such searing intensity that they often reduced him to retching within fifteen minutes. They came on every month or two and tormented him for as long as two days; mercifully, sleep usually intervened to overcome some of the agony. As a prelude to each attack, a flashing oval of white light, spangled with colored lights around its edges, appeared before Daniel's eyes. The oval gradually expanded and then blackened into a blind spot just as the pain seized the right side of his head.

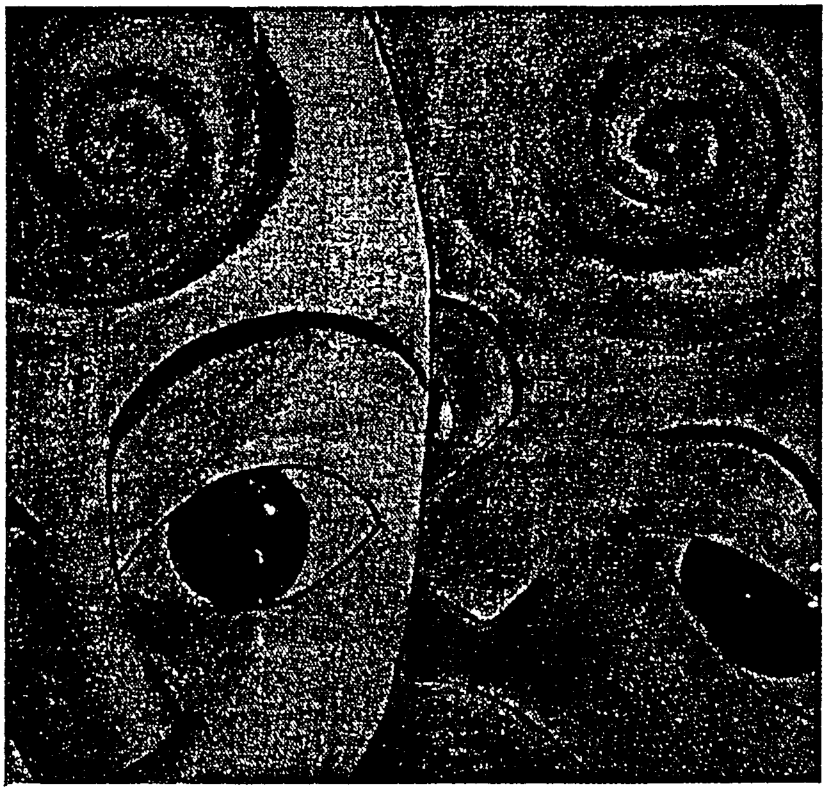
As Daniel reached his twenties, the headaches doubled in frequency and became a serious threat to career and family life. Efforts to treat him with drugs and minor surgery failed miserably. Finally, his physicians at the National Hospital in London decided to embark on a radical course. They surgically removed a section of his visual cortex, the center for the control of vision in the brain, and in so doing, excised a benign tumor in a blood vessel, the apparent source of the migraines. The operation was a remarkable success: it virtually eliminated the migraines and granted Daniel's life a measure of normality—albeit without the left half of his vision.

That happy ending in 1973 turned out to be just the beginning of an odyssey into the enigmatic workings of the brain. Six weeks after the surgery the ophthalmologist Michael D. Sanders of the National Hospital held out his hand in Daniel's left, blinded field. Astonishingly, Daniel reached out and touched it. On more pointed testing Sanders and I, along with the National Hospital psychologist Elizabeth K. Warrington, discovered to our amazement that Daniel's "blind" field was not blind at all in the usual sense. He could tell, for instance, whether a line of light projected into his blind field was vertical or horizontal. He could distinguish between an X and an O flashed to his left. When objects were placed in his blind field, he made virtually no errors locating them, though he could not tell us what they were. Yet throughout the tests he insisted he had only guessed at the answers after we had urged him to do so. When we gave him the results of the testing, he was incredulous that he had done so well. "I couldn't see anything, not a darn thing," Daniel told us. All he would allow was a "feeling" about an object in some, but not all, the tests.

We named the extraordinary phenomenon blindsight. Since its discovery in Daniel, other investigators have noted similar capacities in a number of people who have suffered damage to their visual cortexes. Through all manner of examinations in the past two decades—including some ingenious tests devised only recently—blindsight has held up as a robust and quite real phenomenon. Moreover, its physiological basis is starting to become clear. Studies of blindsight have afforded a look at the intricate pathways of vision in the brain, revealing that there is much more capacity and complexity than has ever been suspected. The fruits of those studies may help some blind people regain at least part of their vision. Already, repeated testing of the kind we gave Daniel has led to marked improvements in the visual capabilities of a number of otherwise blind people.

But the most tantalizing windfall from the studies of blindsight is the possibility that they will yield clues to how consciousness arises in the brain. One striking, indeed defining, characteristic of the phenomenon is that people who exhibit it are not aware of what their eyes have taken in nor of what their brains have processed. The overarching conundrum is, Why? Solving that riddle of blindsight promises to help give form and substance to the conscious—and the unconscious—mind.

THE MOST intriguing attribute of blindsight—the absence of consciousness—is also partly to blame for its relatively recent recognition. As it turns out, however, for more than a century monkeys that have lost vision as a result of removal of their visual cortexes have been observed displaying behavior that resembles blindsight. Blind people had been asked whether they could perform the tasks the monkeys could, but the matter had always been dropped when the answer was no.



Francesco Clemente, Head, *1990*

One of the first suggestions for residual vision in monkeys was published in 1886 by David Ferrier, the Scottish neurologist who mapped much of the cerebral cortex. Ferrier reported an incredible experiment involving the occipital lobe, the region of the brain containing the visual cortex:

> I removed the greater portion of both occipital lobes at the same time without causing the slightest appreciable impairment of vision. One of these animals within two hours of the operation was able to run about freely, avoiding obstacles, to pick up such a minute object as a raisin without the slightest hesitation or want of precision, and to act in accordance with its visual experience in a perfectly normal manner.

Ferrier was taken to task by some of his colleagues for making such an extreme statement, particularly because it was unclear at the time exactly where the visual cortex was located. Still, the scientific community slowly began to develop a consensus that monkeys without visual cortexes could respond at least crudely to light. In 1942 the psychologist Heinrich Klüver of the University of Chicago trained monkeys to respond to different levels and patterns of light; then he surgically removed their entire visual cortexes. After the surgery the monkeys still responded appropriately to the overall quantity of light in the visual field, though not, Klüver reported, to varying patterns of light. (They could discriminate gray paper from white.) Other investigators noted in the ensuing decade that monkeys without visual cortexes seemed to follow moving stimuli with their eyes.

In the mid-1960s the psychologist Nicholas K. Humphrey, then at the University of Cambridge, substantially expanded on the earlier observations by teaching monkeys to reach out and touch objects to get a food reward. After he made surgical lesions in both occipital lobes, the monkeys—now blind—could still detect small objects with exquisite sensitivity. At about the same time one of my initial efforts in the field showed that Klüver had been too quick to conclude that blind monkeys could not discriminate patterns of light.

The finding came about quite by chance on a visit to Bellevue Hospital in New York, where I wanted to conduct tests on a monkey with no visual cortexes. It was a Sunday and the gray paper normally used as a target in testing could not be had because the stores were closed. So my wife and I simulated gray paper by cutting up pieces of black paper and white paper and combining them in a salt-and-pepper pattern that gave off the same amount of light as a gray target does. To our surprise, the blind monkey had an easier time detecting the salt-and-pepper pattern than it did a striped pattern that gave off the same quantity of light. The monkey, it appeared, was responding to the degree of "edginess" in the pattern as well as to the quantity of light.

Those findings stimulated other research into the monkey visual cortex. In 1972 the husband and wife neurologists Pedro and Tauba Pasik of the Mount Sinai School of Medicine in New York showed that with specific training, monkeys could indeed learn to discriminate patterns. And by 1980 it became clear they could perceive extraordinarily fine detail: They could discriminate a gray target from a grating of equal brightness with lines separated by only three minutes of arc. They could tell the difference between two lines whose orientations varied by as little as eight degrees. One study even suggested monkeys without visual cortexes could discriminate color, though others have since refuted it.

My further investigations of residual vision in monkeys in collaboration with the neuroscientist Alan Cowey, who is now at the University of Oxford, showed that its potential is even greater when the visual cortex is removed from only one occipital lobe, causing blindness in only half the field of vision. When such an animal is trained to detect small, brief flashes of light, its blind field quickly becomes more sensitive to stimuli and often shrinks.

What physiological mechanism could account for residual vision in monkeys? Branches of the optic nerve that wend their way to a certain region of the midbrain are strong candidates. That mechanism would go along with the prevailing evidence that residual vision is not merely an extension or part of the capabilities of the visual cortex. Instead, it seems to be a different kind of vision altogether, controlled by a separate part of the brain. In extended studies of a blind monkey named Helen, for instance, Humphrey observed that even though she was sensitive to the presence of an object, she could not identify it until she touched it. "After six years," he wrote, "she still does not know a carrot when she sees one nor apparently can she recognize my face, despite an excellent ability to locate visual events in her environment and to avoid obstacles by vision alone."

HUMPHREY'S OBSERVATIONS on Helen bring to mind a metaphor suggested in the late nineteenth century by the American psychologist and philosopher William James. He proposed that peripheral vision acts as a sentinel, scanning for objects in the visual field and calling in the fovea in the center of the retina to identify what it finds. James was unaware of the visual pathway to the midbrain; perhaps the midbrain region is the mastermind of his sentinel. In his time little was known about the wiring of the brain, but James believed, on the basis of the human clinical evidence, that the occipital lobes are indispensable for vision in people. Indeed, it soon became established that most of the million or so fibers in the optic nerve that trails off the back of each eyeball end up in the visual cortex through a connection in the thalamus. It seemed impossible that the visual mechanism could work if most of its connections with the eye were eliminated. James's view on the necessity of the occipital lobes still reflects the conventional clinical wisdom.

But about 150,000 of the fibers in each human optic nerve travel to the midbrain and other areas outside the visual cortex. And although they make up only a small part of the optic nerve, those branches measure about five times the size of the acoustic nerve. Thus as investigators began solving the mystery of residual vision in monkeys, the lack of any evidence for a similar capability in people became more and more bothersome, given the similarity of the two visual anatomies.

Some workers suggested the branch of the optic nerve leading to the midbrain might be merely vestigial in people. Surely the discrepancy between simian and human experience could be explained as an evolutionary shift: the seat of vision had simply moved from the lower brain—the hindbrain and midbrain regions—to the cortex. Others attributed the discrepancy to the pool of human research subjects, which was necessarily limited to people—mainly war veterans—who had accidentally suffered damage to their occipital lobes. The extent of the lesion can rarely be determined in such cases.

But even with limited data on people it was clear that, in contrast with the plasticity and adaptability of the visual capacity in monkeys, blindness in people tends to be much more severe and persistent. Lesions in the visual cortex never lead to absolute blindness in monkeys. As often as not, however, they do so in people. It turns out that the arrangement of the human brain makes it unlikely that the visual cortex could be damaged without affecting the surrounding cortex: the human visual cortex is almost entirely buried inside the cerebral cortex. In contrast, the monkey visual cortex is exposed at the surface of the brain, and it can readily be destroyed without harming nearby tissue. Thus perhaps residual vision had been observed in monkeys but not in people because of differential destruction.

All such considerations, however, remained unsatisfying. For example, even when, in addition to the visual cortex, surrounding areas of the cortex in monkeys are damaged, the animals still retain some residual vision. Not until the psychologist Ernst Pöppel, now at the University of Munich, and colleagues at the Massachusetts Institute of Technology tried a radically different—some might say foolhardy—approach was the mask of consciousness lifted to reveal the first glimmerings of blindsight in people. In 1973, shortly before we tested Daniel, Pöppel and his colleagues asked four war veterans, partly blinded by gunshot wounds and protesting that they could see nothing, to move their eyes anyway to the position of a light flashed in their blind fields—in effect, to take wild guesses. Pöppel noted that the veterans had a tendency, albeit a weak one, to move their eyes to the correct position. That result inspired my colleagues and me to suggest guessing when we tested Daniel.

DANIEL OFFERED a rare chance to study a person whose brain damage was known to be confined to the visual cortex. Elizabeth Warrington and I conducted extensive, repeated testing of his blindsight for ten years. He retained his abilities throughout that time and, in fact, showed a steady improvement in his sensitivity to various stimuli. Three years after his surgery, for unknown reasons, Daniel spontaneously regained half his lost vision, which happens occasionally in cases of blindness caused by brain injury.

During our tests Daniel sat with his head steadied by a chin rest and kept his eyes fixed on a point in front of him. We verified his gaze by recording his eye position. In tests of his ability to locate objects he pointed with his finger to a position in his blind field; in the other tests he just answered orally. We varied the intensity, size and duration of light flashes and the background illumination to explore how the variations affected his blindsight. As a matter of routine, we also included blanks randomly mixed among the test runs to ensure that he was not reacting to auditory or other cues.

Daniel did well locating target flashes in certain areas of his blind field, but in other areas his results were no better than chance alone would yield. In one test, in which he guessed whether one target was closer than another to the center of the field, he gave thirty correct answers out of thirty when the targets were separated by certain distances. Other tests, in which he had only to say whether or not a light was present, elicited his best overall performance: at least 80 percent correct in most regions of the blind field and better than 90 percent correct in other locations. Curiously, when the intensity of the flashes was high, Daniel remarked that although he could see nothing, he sometimes felt that something was coming at him: "It is like a billiard cue coming out or going back and when that happens I say 'yes.'" He tended to be more accurate when he noted that effect, but his highest score still came when he said he was purely guessing.

Since our exploration of Daniel's blindsight began, at least a dozen other people have displayed similar capabilities. From person to person, however, the capabilities are far from uniform, most likely because of variations in the location of the brain damage. For that reason, single cases investigated in depth are often more revealing than group studies, and they may be a greater help in mapping the functional geography of the brain. The psychologist Anthony J. Marcel of the Medical Research Council's Applied Psychology Unit in Cambridge, England, for example, has noted that two of his partly blind patients, while reaching for objects in their blind fields, adjusted their grasp for size and orientation before they touched the objects. Even more astounding, the same two people seemed able to pick up the meanings of words flashed into their blind fields; they could reliably guess which of two words displayed in their sighted fields was related to a word hidden in their blind fields. And the psychologist Petra Stoerig of the University of Munich together with Alan Cowey reported last April in the journal *Brain* that some people can actually discriminate lights of different colors in their blind fields.

The subjective experiences of the tested subjects are also fascinating clues to blindsight. Like Daniel, the others denied seeing anything in their blind fields, though sometimes they reported specific feelings about the target object. In general, the feelings came on when the target was moving vigorously or when it was high-intensity light. Daniel sometimes described his feeling as being like "moving my hand in front of my eyes when the room is totally dark." When O's and X's were displayed, he might say he felt a "smoothness" with an O and a "jaggedness" with an X. A black object seemed to him to be far away whereas a white square felt close. Furthermore, he occasionally felt that something was approaching him when a light was turned on in his blind field, and felt that something was receding when the light was turned off. Nevertheless, in the many tests for which he had no feeling at all, he still guessed accurately.

Almost ten years after his surgery Daniel reported that parts of his blind field had taken on a wavy quality, especially when targets were moving or when they appeared suddenly. Apparently the sensation is unlike anything in normal visual experience; Daniel could not find the words to describe it. When a square was projected into those regions of his blind field, he sensed "a sharp movement" and, on another occasion, "a corner-shaped wave." A triangle gave rise to "thin" waves, whereas a triangle with curved sides generated "thicker and quicker" waves. Yet he insisted he was not seeing anything.

When blindsight began getting attention in scientific circles, we were not surprised to see a number of skeptics come forward. Some vigorously objected to the line of inquiry altogether, arguing that the observations were metaphysical and inadmissible to science. Others said the phenomenon must surely be an artifact of the testing technique or some other spurious factors. To meet those criticisms we have done extensive testing to verify that blindsight in people is a genuine residual vision, not the outcome of processing by the sighted part of the visual field.

First, blindsight has been found in one person who lost both visual cortexes and in a number of people from whom the entire cerebral hemisphere was removed in surgery. Second, tests have been done on the natural blind spot, where the optic nerve attaches to the back of the retina, to rule out the possibility that light from an object may somehow scatter into the sighted field. People who can detect lights flashed in their blind fields cannot see a light projected on the natural blind spot. Finally, we have taken care to design our studies to rule out the possibility that our subjects are getting cues to the correct answers from their sighted fields or from some other sensory pathway.

Critics who are bothered by testing that relies on oral answers may be heartened by techniques that bypass patients' introspections. For example, the neurologist Robert O. Rafal and his colleagues at the University of California at Davis are gauging the speed of eyeball movements as a test of blindsight. They have observed that people with blindsight move their eyes toward a light in the good part of their fields much more slowly when there is a light on in their blind field.

What, then, is the neural basis for the distinction between conscious vision and blindsight? Some investigators have postulated that blindsight and its analogues are products of automatic control circuits that are regulated by structures in the midbrain and hindbrain. Those areas govern involuntary, unconscious actions.

To date, workers have identified nine branches of the optic nerve that connect with regions of the brain other than the visual cortex. One relatively large branch, which runs to a midbrain area called the superior colliculus, looks like a good candidate for mediating blindsight. In people with normal vision, the region responds electrically to light projected in all parts of the visual field, and investigators have constructed a map showing how the region corresponds to different locations in the visual field. In one experiment, the neurophysiologists Charles W. Mohler and Robert H. Wurtz at the National Institutes of Health made a small lesion in the visual cortex of a monkey and then put the animal through the standard training for regaining some vision. Knowing the section of the superior colliculus that mapped to the recovered portion of the visual field, the investigators made an incision in that section to recreate the original blind region. That time the blindness was permanent even with further training. Yet when the same midbrain lesion was made in another monkey with a fully functioning visual cortex, there was no blindness. Thus the visual region of the midbrain appears to serve as a kind of backup for the visual cortex.

Studies of people also strongly suggest blindsight and normal vision are supervised by different sections of the brain. Daniel's vision in his blind field was not merely a degraded version of his normal vision; at the periphery of vision, for instance, he was better at detecting objects and worse at recognizing shapes in his blind field than in his sighted field. What is more, he tired much faster during the testing of his sighted field than of his blind field. Psychophysical experiments are usually tiring for both subject and investigator, yet Daniel declined the usual rest during the testing of his blind field. "But I am not *doing* anything in my blind field," he said. "I am just guessing."

Pathways other than the superior colliculus may also play a role in blindsight. Other branches of the optic nerve, such as the accessory optic nuclei, travel to various areas of the midbrain and the thalamus. Some of the great bulk of the optic nerve that reaches the visual cortex also transmits information to nonvisual cortical regions close by. Those regions, some tests suggest, continue to process vision in the absence of the primary visual cortex. If they too contribute to blindsight, awareness cannot be a general property of the cortex but must be confined to specific structures such as the visual cortex.

Intriguingly, some evidence suggests that when the brain damage takes place at an early age the midbrain region can take on not just extra visual tasks but the awareness of them as well. Presumably the plasticity of the young brain allows it to reorganize its functioning. In 1935, for instance, the psychologist D. G. Marquis of Yale University recounted the case of an eleven-year-old boy from whom the occipital lobe had been surgically removed to eliminate a cyst the boy had had since before birth. To his doctors' amazement, the boy had normal vision after the surgery, except for a slight constriction in his right field and a patchy sensitivity to color.

When pressed, the alternative pathways can replace some lost vision in adults too. First of all, the testing given Daniel and other blindsight subjects improved their guessing accuracy. Furthermore, it is possible that the testing was at least partly responsible for the dramatic reduction of Daniel's blind field nine years after surgery. For it has been established that monkeys can regain significant chunks of vision with special training. More modest improvements have been reported for people who have undergone the same sort of training as Daniel. It seems to work by recruiting intact pathways, in either the cortex or the midbrain or in both.

If INVESTIGATORS CAN disentangle the physiological underpinnings of blindsight from those of normal conscious vision, they will undoubtedly make a lasting contribution to the mind–body problem, which has plagued philosophers for millennia. One measure of the more general importance of the phenomenon of blindsight is its apparent analogues with unconscious processing by the other senses, including hearing and touch, and by other regions of the brain. Jacques Paillard and his colleagues at the Institute of Neurophysiology and Psychology in Marseilles, for instance, found echoes of blindsight in a woman with a brain defect that left her no sensation of touch in her right arm, no matter how forceful the contact. Yet when the woman was blindfolded, she could point roughly to the area of contact. She said she felt nothing and had guessed at the spot only because the investigators had encouraged her to do so. Paillard noted that she could also judge the direction of movements traced on her arm. And she improved with practice, just as people with blindsight do. Indeed, he named the phenomenon blindtouch.

People with defects in language comprehension and memory also have shown remarkable, unconscious residual capacity. Severe amnesiacs, who cannot remember experiences much longer than a minute, seem to remember words even when they say they cannot. A typical test begins with a long list of words. A few minutes after being shown the list, the amnesiac will deny having seen the words and fail to pick them out even when asked to choose them from among words on another list. Yet when given a few letters of a word and asked to guess the rest of it, the amnesiac is much more accurate if the word was on the initial list. That kind of test probe can summon words as well as pictures for weeks or sometimes months. Even learned mental skills such as doing arithmetic and solving jigsaw puzzles can be retained.

Another odd defect of memory occurs in a variety of agnosia in which the memory for familiar faces is lost. Agnosiacs with that kind of memory loss act as if they do not know a friend, but when they see a familiar face the electrical conductance of their skin is distinctly different from that when they see a stranger. And when they try to memorize the names and professions that go with familiar faces, agnosiacs learn faster when the information they are given matches reality.

WHAT CAN BE SAID of these curious findings so far? At least this: Some of the mundane questions of everyday life—What do you remember? What did you see?—are rather more complicated than they appear. In every major realm of cognition, it seems, there are robust hidden processes that are relatively immune to neurological brain dysfunction. Furthermore, the strength and duration of such covert reactions can be comparable to that of normal functioning. The central defect in the cases we have seen and described appears to be a lack of awareness of those reactions. Intriguingly, cases of excess awareness also exist, such as blind people who deny being blind even though they are constantly bumping into things. Such people typically make excuses such as "The light is too dim in here" to account for their difficulties. There has even been evidence of covert *nondenial* in such cases: although the person denies he is blind, tests suggest he is at some level aware of it. The hopes for a treatment for any of these conditions lie in elucidating the neurology of awareness. ●

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