The Case of the Colorblind Painter

Early in March 1986 I received the following letter:

> I am a rather successful artist just past 65 years of age. On January 2nd of this year I was driving my car and was hit by a small truck on the passenger side of my vehicle. When visiting the emergency room of a local hospital, I was told I had a concussion. While taking an eye examination, it was discovered that I was unable to distinguish letters or colors. The letters appeared to be Greek letters. My vision was such that everything appeared to me as viewing a black and white television screen. Within days, I could distinguish letters and my vision became that of an eagle—I can see a worm wriggling a block away. The sharpness of focus is incredible. BUT—I AM ABSOLUTELY COLOR BLIND. I have visited ophthalmologists who know nothing about this color-blind business. I have visited neurologists, to no avail. Under hypnosis I still can't distinguish colors. I have been involved in all kinds of tests. You name it. My brown dog is dark grey. Tomato juice is black. Color TV is a hodge-podge.…

Had I ever encountered such a problem before, the writer continued; could I explain what was happening to him—and could I help?

This seemed an extraordinary letter. Colorblindness, as ordinarily understood, is something one is born with—a difficulty distinguishing red and green, or other colors, or (extremely rarely) an inability to see any colors at all, due to defects in the color-responding cells, the cones, of the retina. But clearly this was not the case with my correspondent, Jonathan I. He had seen normally all his life, had been born

with a full complement of cones in his retinas. He had become colorblind, after sixty-five years of seeing colors normally—totally colorblind, as if "viewing a black and white television screen." The suddenness of the event was incompatible with any of the slow deteriorations that can befall the retinal cone cells and suggested instead a mishap at a much higher level, in those parts of the brain specialized for the perception of color.

Total colorblindness caused by brain damage, so-called cerebral achromatopsia, though described more than three centuries ago, remains a rare and important condition. It has intrigued neurologists because, like all neural dissolutions and destructions, it can reveal to us the mechanisms of neural construction—specifically, here, how the brain "sees" (or makes) color. Doubly intriguing is its occurrence in an artist, a painter for whom color has been of primary importance, and who can directly paint as well as describe what has befallen him, and thus convey the full strangeness, distress, and reality of the condition.

Color is not a trivial subject but one that has compelled, for hundreds of years, a passionate curiosity in the greatest artists, philosophers, and natural scientists. The young Spinoza wrote his first treatise on the rainbow; the young Newton's most joyous discovery was the composition of white light; Goethe's great color work, like Newton's, started with a prism; Schopenhauer, Young, Helmholtz, and Maxwell, in the last century, were all tantalized by the problem of color; and Wittgenstein's last work was his Remarks on Colour. And yet most of us, most of the time, overlook its great mystery. Through such a case as Mr. I.'s we can trace not only the underlying cerebral mechanisms or physiology but the phenomenology of color and the depth of

its resonance and meaning for the individual.

On getting Mr. I.'s letter, I contacted my good friend and colleague Robert Wasserman, an ophthalmologist, feeling that together we needed to explore Mr. I.'s complex situation and, if we could, help him. We first saw him in April 1986. He was a tall, gaunt man, with a sharp, intelligent face. Although obviously depressed by his condition, he soon warmed to us and began talking with animation and humor. He constantly smoked as he talked; his fingers, restless, were stained with nicotine. He described a very active and productive life as an artist, from his early days with Georgia O'Keeffe in New Mexico, to painting backdrops in Hollywood during the 1940s, to working as an Abstract Expressionist in New York during the 1950s and later as an art director and a commercial artist.

We learned that his accident had been accompanied by a transient amnesia. He had been able, evidently, to give a clear account of himself and his accident to the police at the time it happened, late on the afternoon of January 2, but then, because of a steadily mounting headache, he went home. He complained to his wife of having a headache and feeling confused, but made no mention of the accident. He then fell into a long, almost stuporous sleep. It was only the next morning, when his wife saw the side of the car stove in, that she asked him what had happened. When she got no clear answer ("I don't know. Maybe somebody backed into it") she knew that something serious must have happened.

Mr. I. then drove off to his studio and found on his desk a carbon copy of the police accident report. He had had an accident, but somehow, bizarrely, had lost his memory of it. Perhaps the report would jolt his memory. But lifting it up,

he could make nothing of it. He saw print of different sizes and types, all clearly in focus, but it looked like “Greek” or “Hebrew” to him.[1] A magnifying glass did not help; it simply became large “Greek” or “Hebrew.” (This alexia, or inability to read, lasted for five days, but then disappeared.)

Feeling now that he must have suffered a stroke or some sort of brain damage from the accident, Jonathan I. phoned his doctor, who arranged for him to be tested at a local hospital. Although, as his original letter indicates, difficulties in distinguishing colors were detected at this time, in addition to his inability to read, he had no subjective sense of the alteration of colors until the next day.

That day he decided to go to work again. It seemed to him as if he were driving in a fog, even though he knew it to be a bright and sunny morning. Everything seemed misty, bleached, greyish, indistinct. He was flagged down by the police close to his studio: he had gone through two red lights, they said. Did he realize this? No, he said, he was not aware of having passed through any red lights. They asked him to get out of the car. Finding him sober, but apparently bewildered and ill, they gave him a ticket and suggested he seek medical advice.

Mr. I. arrived at his studio with relief, expecting that the horrible mist would be gone, that everything would be clear again. But as soon as he entered, he found his entire studio, which was hung with brilliantly colored paintings, now utterly grey and void of color. His canvases, the abstract color paintings he was known for, were now greyish or black and white. His paintings—once rich with associations, feelings, meanings—now looked unfamiliar and meaningless to him. At this point the magnitude of his loss overwhelmed him. He had spent his life as a painter; now even his art was

without meaning, and he could no longer imagine how to go on.

The weeks that followed were very difficult. “You might think,” Mr. I. said, “loss of color vision, what’s the big deal? Some of my friends said this, my wife sometimes thought this, but to me, at least, it was awful, disgusting.” He knew the colors of everything, with an extraordinary exactness (he could give not only the names but the numbers of colors as these were listed in a Pantone chart of hues he had used for many years). He could identify the green of van Gogh’s billiard table in this way unhesitatingly. He knew all the colors in his favorite paintings, but could no longer see them, either when he looked or in his mind’s eye. Perhaps he knew them, now, only by verbal memory.

It was not just that colors were missing, but that what he did see had a distasteful, “dirty” look, the whites glaring, yet discolored and off-white, the blacks cavernous—everything wrong, unnatural, stained, and impure.[2]

Mr. I. could hardly bear the changed appearances of people (“like animated grey statues”) any more than he could bear his own appearance in the mirror: he shunned social intercourse and found sexual intercourse impossible. He saw people’s flesh, his wife’s flesh, his own flesh, as an abhorrent grey; “flesh-colored” now appeared “rat-colored” to him. This was so even when he closed his eyes, for his vivid visual imagery was preserved but was now without color as well.

The “wrongness” of everything was disturbing, even disgusting, and applied to every circumstance of daily life. He found foods disgusting due to their greyish, dead appearance and had to close his eyes to eat. But this did not help very much, for the mental image of a tomato was as black as its appearance. Thus, unable to rectify even the

inner image, the idea, of various foods, he turned increasingly to black and white foods—to black olives and white rice, black coffee and yogurt. These at least appeared relatively normal, whereas most foods, normally colored, now appeared horribly abnormal. His own brown dog looked so strange to him now that he even considered getting a Dalmatian.

He encountered difficulties and distresses of every sort, from the confusion of red and green traffic lights (which he could now distinguish only by position) to an inability to choose his clothes. (His wife had to pick them out, and this dependency he found hard to bear; later, he had everything classified in his drawers and closet—grey socks here, yellow there, ties labeled, jackets and suits categorized, to prevent otherwise glaring incongruities and confusions.) Fixed and ritualistic practices and positions had to be adopted at the table; otherwise he might mistake the mustard for the mayonnaise, or, if he could bring himself to use the blackish stuff, ketchup for jam.[3]

As the months went by, he particularly missed the brilliant colors of spring—he had always loved flowers, but now he could only distinguish them by shape or smell. The blue jays were brilliant no longer; their blue, curiously, was now seen as pale grey. He could no longer see the clouds in the sky, their whiteness, or off-whiteness as he saw them, being scarcely distinguishable from the azure, which seemed bleached to a pale grey. Red and green peppers were also indistinguishable, but this was because both appeared black. Yellows and blues, to him, were almost white.[4]

Mr. I. also seemed to experience an excessive tonal contrast, with loss of delicate tonal gradations, especially in direct sunlight or harsh artificial light; he made a comparison

here with the effects of sodium lighting, which at once removes color and tonal delicacy, and with certain black-and-white films—"like Tri-X pushed for speed"—which produce a harsh, contrasty effect. Sometimes objects stood out with inordinate contrast and sharpness, like silhouettes. But if the contrast was normal, or low, they might disappear from sight altogether.

Thus, though his brown dog would stand out sharply in silhouette against a light road, it might get lost to sight when it moved into soft, dappled undergrowth. People's figures might be visible and recognizable half a mile off (as he himself said in his original letter, and many times later, his vision had become much sharper, "that of an eagle"), but faces would often be unidentifiable until they were close. This seemed a matter of lost color and tonal contrast, rather than a defect in recognition, an agnosia. A major problem occurred when he drove, in that he tended to misinterpret shadows as cracks or ruts in the road and would brake or swerve suddenly to avoid these.

He found color television especially hard to bear: its images always unpleasant, sometimes unintelligible. Black-and-white television, he thought, was much easier to deal with; he felt his perception of black-and-white images to be relatively normal, whereas something bizarre and intolerable occurred whenever he looked at colored images. (When we asked why he did not simply turn off the color, he said he thought that the tonal values of "decolored" color TV seemed different, less "normal," than those of a "pure" black-and-white set.) But, as he now explained, in distinction to his first letter, his world was not really like black-and-white television or film—it would have been much easier to live with had it been so. (He sometimes wished he could wear

miniature TV glasses.)

His despair of conveying what his world looked like, and the uselessness of the usual black-and-white analogies, finally drove him, some weeks later, to create an entire grey room, a grey universe, in his studio, in which tables, chairs, and an elaborate dinner ready for serving were all painted in a range of greys. The effect of this, in three dimensions and in a different tonal scale from the "black and white" we are all accustomed to, was indeed macabre, and wholly unlike that of a black-and-white photograph. As Mr. I. pointed out, we accept black-and-white photographs or films because they are representations of the world—images that we can look at, or away from, when we want. But black and white for him was a reality, all around him, 360 degrees, solid and three-dimensional, twenty-four hours a day. The only way he could express it, he felt, was to make a completely grey room for others to experience—but of course, he pointed out, the observer himself would have to be painted grey, so he would be part of the world, not just observing it. More than this: the observer would have to lose, as he himself had, the neural knowledge of color. It was, he said, like living in a world "molded in lead."

Paintings done by Mr. I. shortly before his accident.

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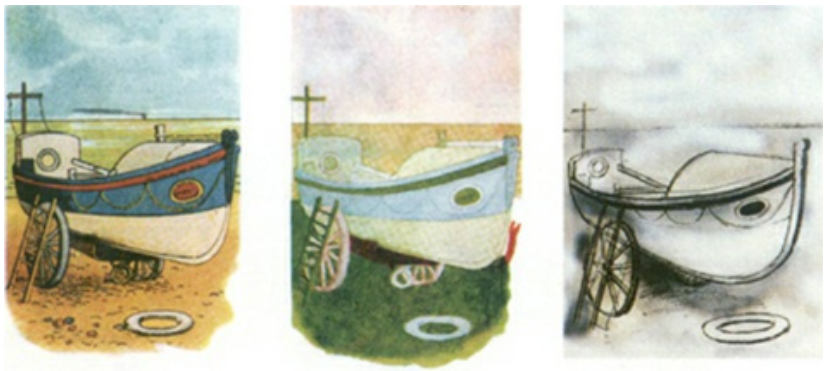
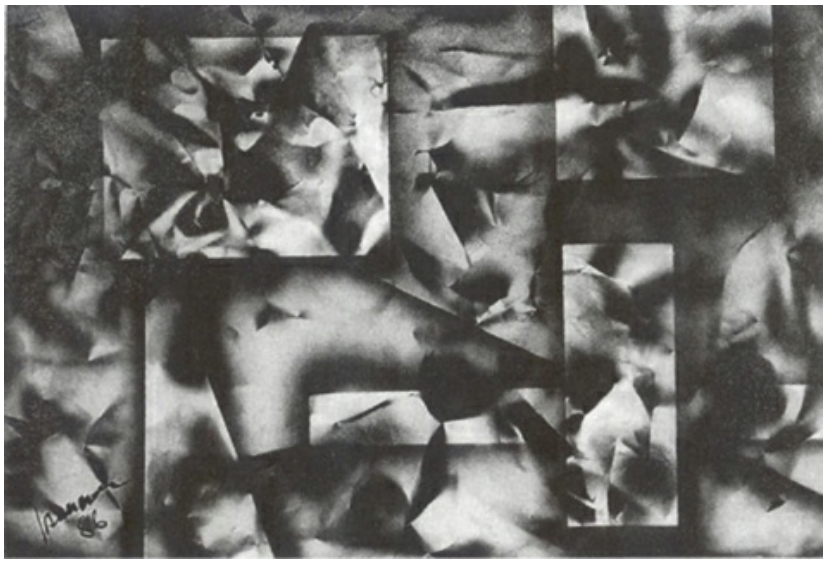
A painting of flowers done four weeks after Mr. I.’s accident. The underlying outlines are clear, but camouflaged by a random application of color.

Mr. I. painted pieces of grey fruit to show us the “leaden” universe into which he had fallen.



A test painting from Mary Collin's Colour-Blindness (left), as reproduced by someone with red-green colorblindness, and by Mr. I. (right).

The sunset scene of which Mr. I. could see virtually nothing (–an effect simulated by a black-and-white photocopy of it.)



A black-and-white painting done about two months after Mr. I.'s accident …

... and a painting done two years later–Mr. I. at this time was experimenting with adding single colors, even though he could not see them.

Subsequently, he said neither “grey” nor “leaden” could begin to convey what his world was actually like. It was not “grey” that he experienced, he said, but perceptual qualities for which ordinary experience, ordinary language, had no equivalent.

Mr. I. could no longer bear to go to museums and galleries or to see colored reproductions of his favorite pictures. This was not just because they were bereft of color, but because they looked intolerably wrong, with washed-out or “unnatural” shades of grey (photographs in black and white, on the other hand, were much more tolerable). This was especially distressing when he knew the artists, and the perceptual debasement of their work interfered with his sense of their identity—this, indeed, was what he now felt was happening with himself.

He was depressed once by a rainbow, which he saw only as a colorless semicircle in the sky. And he even felt his occasional migraines as “dull”—previously they had involved brilliantly colored geometric hallucinations, but now even these were devoid of color. He sometimes tried to evoke color by pressing the globes of his eyes, but the flashes and patterns elicited were equally lacking in color. He had often dreamed in vivid color, especially when he dreamed of landscapes and painting; now his dreams were washed-out and pale, or violent and contrasty, lacking both color and delicate tonal gradations.

Music, curiously, was impaired for him too, because he had previously had an extremely intense synesthesia, so that different tones had immediately been translated into color, and he experienced all music simultaneously as a rich tumult of inner colors. With the loss of his ability to generate colors, he lost this ability as well—his internal “color-organ” was out

of action, and now he heard music with no visual accompaniment; this, for him, was music with its essential chromatic counterpart missing, music now radically impoverished.[5]

A certain mild pleasure came from looking at drawings; he had been a fine draftsman in his earlier years. Could he not go back to drawing again? This thought was slow to occur to him, and it only took hold after being suggested repeatedly by others. His own first impulse was to paint in color. He insisted that he still "knew" what colors to use, even though he could no longer see them. He decided, as a first exercise, to paint flowers, taking from his palette what tints seemed "tonally right." But the pictures were unintelligible, a confusing welter of colors to normal eyes. It was only when one of his artist friends took black-and-white Polaroids of the paintings that they made sense. The contours were accurate, but the colors were all wrong. "No one will get your paintings," one of his friends said, "unless they are as colorblind as you."

"Stop pushing it," said another. "You can't use color now." Mr. I. reluctantly allowed all his colored paints to be put away. It's only temporary, he thought. I'll be back to color soon.

These first weeks were a time of agitation, even desperation; he was constantly hoping that he would wake up one fine morning and find the world of color miraculously restored. This was a constant motif in his dreams at the time, but the wish was never fulfilled, even in his dreams. He would dream that he was about to see in color, but then he would wake and find that nothing had changed. He constantly feared that whatever had happened would happen again, this time depriving him of all his sight completely. He

thought he had probably had a stroke, caused by (or perhaps causing) his accident in the car, and feared that there could be another stroke at any moment. In addition to this medical fear, there was a deeper bewilderment and fear that he found almost impossible to articulate, and it was this that had come to a head in his month of attempted color painting, his month of insisting that he still "knew" color. It had gradually come upon him, during this time, that it was not merely color perception and color imagery that he lacked, but something deeper and difficult to define. He knew all about color, externally, intellectually, but he had lost the remembrance, the inner knowledge, of it that had been part of his very being. He had had a lifetime of experience in color, but now this was only a historical fact, not something he could access and feel directly. It was as if his past, his chromatic past, had been taken away, as if the brain's knowledge of color had been totally excised, leaving no trace, no inner evidence, of its existence behind.[6]

By the beginning of February, some of his agitation was calming down; he had started to accept, not merely intellectually, but at a deeper level, too, that he was indeed totally colorblind and might possibly remain so. His initial sense of helplessness started to give way to a sense of resolution—he would paint in black and white, if he could not paint in color; he would try to live in a black-and-white world as fully as he could. This resolution was strengthened by a singular experience, about five weeks after his accident, as he was driving to the studio one morning. He saw the sunrise over the highway, the blazing reds all turned into black: "The sun rose like a bomb, like some enormous nuclear explosion," he said later. "Had anyone ever seen a

sunrise in this way before?"

Inspired by the sunrise, he started painting again—he started, indeed, with a black-and-white painting that he called Nuclear Sunrise, and then went on to the abstracts he favored, but now painting in black and white only. The fear of blindness continued to haunt him but, creatively transmuted, shaped the first "real" paintings he did after his color experiments. Black-and-white paintings he now found he could do, and do very well. He found his only solace working in the studio, and he worked fifteen, even eighteen, hours a day. This meant for him a kind of artistic survival: "I felt if I couldn't go on painting," he said later, "I wouldn't want to go on at all."

His first black-and-white paintings, done in February and March, gave a feeling of violent forces—rage, fear, despair, excitement—but these were held in control, attesting to the powers of artistry that could disclose, and yet contain, such intensity of feeling. In these two months he produced dozens of paintings, marked by a singular style, a character he had never shown before. In many of these paintings, there was an extraordinary shattered, kaleidoscopic surface, with abstract shapes suggestive of faces—averted, shadowed, sorrowing, raging—and dismembered body parts, faceted and held in frames and boxes. They had, compared with his previous work, a labyrinthine complexity, and an obsessed, haunted quality—they seemed to exhibit, in symbolic form, the predicament he was in.

Starting in May—it was fascinating to watch—he moved from these powerful but rather terrifying and alien paintings toward themes, living themes, he had not touched in thirty years, back to representational paintings of dancers and

racehorses. These paintings, even though still in black and white, were full of movement, vitality, and sensuousness; and they went with a change in his personal life—a lessening of his withdrawal and the beginnings of a renewed social and sexual life, a lessening of his fears and depression, and a turning back to life.

At this time, too, he turned to sculpture, which he had never done before. He seemed to be turning to all the visual modes that still remained to him—form, contour, movement, depth—and exploring them with heightened intensity. He also started painting portraits, although he found that he could not work from life, but only from black-and-white photographs, fortified by his knowledge of and feeling for each subject. Life was tolerable only in the studio, for here he could reconceive the world in powerful, stark forms. But outside, in real life, he found the world alien, empty, dead, and grey.

This was the story Bob Wasserman and I got from Mr. I. —a story of an abrupt and total breakdown of color vision, and his attempts to live in a black-and-white world. I had never been given such a history before, I had never met anyone with total colorblindness before, and I had no idea what had happened to him—nor whether his condition could be reversed or improved.

The first thing was to define his impairments more precisely with various tests, some quite informal, making use of everyday objects or pictures, whatever came to hand. For instance, we first asked Mr. I. about a shelf of notebooks—blue, red, and black—by my desk. He instantly picked out the blue ones (a bright medium blue to normal eyes) —“they’re pale.” The red and the black were

indistinguishable—both, for him, were “dead black.”

We then gave him a large mass of yarns, containing thirty-three separate colors, and asked him to sort these: he said he could not sort them by color, but only by grey-scale tonal values. He then, rapidly and easily, separated the yarns into four strange, chromatically random piles, which he characterized as 0–25 percent, 25–50 percent, 50–75 percent, and 75–100 percent on a grey-tone scale (though nothing looked to him purely white, and even white yarn looked slightly “dingy” or “dirty”).

We ourselves could not confirm the accuracy of this, because our color vision interfered with our ability to visualize a grey scale, just as normally sighted viewers had been unable to perceive the tonal sense of his confusingly polychromatic flower paintings. But a black-and-white photograph and a black-and-white video camera confirmed that Mr. I. had indeed accurately divided the colored yarns in a grey scale that basically coincided with their own mechanical reading. There was, perhaps, a certain crudeness in his categories, but this went with the sense of sharp contrast, the paucity of tonal gradations, that he had complained of. Indeed, when shown an artist’s grey scale of perhaps a dozen gradations from black to white, Mr. I. could distinguish only three or four categories of tone.[7]

We also showed him the classic Ishihara color-dot plates, in which configurations of numerals in subtly differentiated colors may stand out clearly for the normally sighted, but not for those with various types of colorblindness. Mr. I. was unable to see any of these figures (although he was able to see certain plates that are visible to the colorblind but not to normally sighted people, and thus designed to catch pretended or hysterical colorblindness).[8]

We happened to have a postcard that could have been designed for testing achromatopes—a postcard of a coastal scene, with fishermen on a jetty silhouetted against a dark red sunset sky. Mr. I. was totally unable to see the fishermen or the jetty, and saw only the half-engulfed hemisphere of the setting sun.

Though such problems arose when he was shown colored pictures, Mr. I. had no difficulty describing black-and-white photographs or reproductions accurately; he had no difficulty recognizing forms. His imagery and memory of objects and pictures shown to him were indeed exceptionally vivid and accurate, though always colorless. Thus, after being given a classic test picture of a colored boat, he looked intensely, looked away, and then rapidly reproduced it in black and white paint. When asked the colors of familiar objects, he had no difficulties in color association or color naming. (Patients with color anomia, for instance, can match colors perfectly but have lost the names of colors, and might speak, uncertainly, of a banana being "blue." A patient with a color agnosia, by contrast, could also match colors, but would evince no surprise if given a blue banana. Mr. I., however, had neither of these problems.)[9] Nor did he (now) have any difficulties reading. Testing up to this point, and a general neurological examination, thus confirmed Mr. I.'s total achromatopsia.

We could say to him at this point that his problem was real —that he had a true achromatopsia and not a hysteria. He took this, we thought, with mixed feelings: he had half hoped it might be merely a hysteria, and as such potentially reversible. But the notion of something psychological had also distressed him and made him feel that his problem was "not real" (indeed, several doctors had hinted at this). Our

testing, in a sense, legitimized his condition, but deepened his fear about brain damage and the prognosis for recovery.

Although it seemed that he had an achromatopsia of cerebral origin, we could not help wondering whether a lifetime of heavy smoking could have played a part; nicotine can cause a dimming of vision (an amblyopia) and sometimes an achromatopsia—but this is predominantly due to its effects on the cells of the retina. But the major problem was clearly cerebral: Mr. I. could have sustained tiny areas of brain damage as a result of his concussion; he could have had a small stroke either following, or conceivably precipitating, the accident.

The history of our knowledge about the brain's ability to represent color has followed a complex and zigzag course. Newton, in his famous prism experiment in 1666, showed that white light was composite—could be decomposed into, and recomposed by, all the colors of the spectrum. The rays that were bent most ("the most refrangible") were seen as violet, the least refrangible as red, with the rest of the spectrum in between. The colors of objects, Newton thought, were determined by the "copiousness" with which they reflected particular rays to the eye. Thomas Young, in 1802, feeling that there was no need to have an infinity of different receptors in the eye, each tuned to a different wavelength (artists, after all, could create almost any color they wanted by using a very limited palette of paints) postulated that three types of receptors would be enough.[10] Young's brilliant idea, thrown off casually in the course of a lecture, was forgotten, or lay dormant, for fifty years, until Hermann von Helmholtz, in the course of his own investigation of vision, resurrected it and gave it a new precision, so that we now

speak of the Young-Helmholtz hypothesis. For Helmholtz, as for Young, color was a direct expression of the wavelengths of light absorbed by each receptor, the nervous system just translating one into the other: “Red light stimulates the red-sensitive fibres strongly, and the other two weakly, giving the sensation red.”[11]

In 1884, Hermann Wilbrand, seeing in his neurological practice patients with a range of visual losses—in some predominantly the loss of visual field, in others predominantly of color perception, and in still others predominantly of form perception—suggested that there must be separate visual centers in the primary visual cortex for “light impressions,” “color impressions,” and “form impressions,” though he had no anatomical evidence for this. That achromatopsia (and even hemi-achromatopsia) could indeed arise from damage to specific parts of the brain was first confirmed, four years later, by a Swiss ophthalmologist, Louis Verrey. He described a sixty-year-old woman who, in consequence of a stroke affecting the occipital lobe of her left hemisphere, now saw everything in the right half of her visual field in shades of grey (the left half remained normally colored). The opportunity to examine his patient’s brain after her death showed damage confined to a small portion (the fusiform and lingual gyri) of the visual cortex—it was here, Verrey concluded, that “the centre for chromatic sense will be found.” That such a center might exist, that any part of the cortex might be specialized for the perception or representation of color, was immediately contested and continued to be contested for almost a century. The grounds of this contention go very deep, as deep as the philosophy of neurology itself.

Locke, in the seventeenth century, had held to a

"sensationalist" philosophy (which paralleled Newton's physicalist one): our senses are measuring instruments, recording the external world for us in terms of sensation. Hearing, seeing, all sensation, he took to be wholly passive and receptive. Neurologists in the late nineteenth century were quick to accept this philosophy and to embed it in a speculative anatomy of the brain. Visual perception was equated with "sense-data" or "impressions" transmitted from the retina to the primary visual area of the brain, in an exact, point-to-point correspondence—and there experienced, subjectively, as an image of the visual world. Color, it was presumed, was an integral part of this image. There was no room, anatomically, it was thought, for a separate color center—or indeed, conceptually, for the very idea of one. Thus when Verrey published his findings in 1888, they flew in the face of accepted doctrine. His observations were doubted, his testing criticized, his examination regarded as flawed—but the real objection, behind these, was doctrinal in nature.

If there was no discrete color center, so the thinking went, there could be no isolated achromatopsia either; thus Verrey's case, and two similar ones in the 1890s, were dismissed from neurological consciousness—and cerebral achromatopsia, as a subject, all but disappeared for the next seventy-five years.[12] There was not to be another full case study until 1974.[13]

Mr. I. himself was actively curious about what was going on in his brain. Though he now lived wholly in a world of lightnesses and darknesses, he was very struck by how these changed in different illuminations; red objects, for instance, which normally appeared black to him, became lighter in the long rays of the evening sun, and this allowed him to infer

their redness. This phenomenon was very marked if the quality of illumination suddenly changed, as, for example, when a fluorescent light was turned on, which would cause an immediate change in the brightnesses of objects around the room. Mr. I. commented that he now found himself in an inconstant world, a world whose lights and darks fluctuated with the wavelength of illumination, in striking contrast to the relative stability, the constancy, of the color world he had previously known.[14]

All of this, of course, is very difficult to explain in terms of classical color theory—Newton's notion of an invariant relationship between wavelength and color, of a cell-to-cell transmission of wavelength information from the retina to the brain, and of a direct conversion of this information into color. Such a simple process—a neurological analogy to the decomposition and recomposition of light through a prism—could hardly account for the complexity of color perception in real life.

This incompatibility between classical color theory and reality struck Goethe in the late eighteenth century. Intensely aware of the phenomenal reality of colored shadows and colored afterimages, of the effects of contiguity and illumination on the appearance of colors, of colored and other visual illusions, he felt that these must be the basis of a color theory and declared as his credo, "Optical illusion is optical truth!" Goethe was centrally concerned with the way we actually see colors and light, the ways in which we create worlds, and illusions, in color. This, he felt, was not explicable by Newton's physics, but only by some as-yet unknown rules of the brain. He was saying, in effect, "Visual illusion is neurological truth."

Goethe's color theory, his Farbenlehre (which he regarded as the equal of his entire poetic opus), was, by and large, dismissed by all his contemporaries and has remained in a sort of limbo ever since, seen as the whimsy, the pseudoscience, of a very great poet. But science itself was not entirely insensitive to the "anomalies" that Goethe considered central, and Helmholtz, indeed, gave admiring lectures on Goethe and his science, on many occasions—the last in 1892. Helmholtz was very conscious of "color constancy"—the way in which the colors of objects are preserved, so that we can categorize them and always know what we are looking at, despite great fluctuations in the wavelength of the light illuminating them. The actual wavelengths reflected by an apple, for instance, will vary considerably depending on the illumination, but we consistently see it as red, nonetheless. This could not be, clearly, a mere translation of wavelength into color. There had to be some way, Helmholtz thought, of "discounting the illuminant"—and this he saw as an "unconscious inference" or "an act of judgement" (though he did not venture to suggest where such judgement might occur). Color constancy, for him, was a special example of the way in which we achieve perceptual constancy generally, make a stable perceptual world from a chaotic sensory flux—a world that would not be possible if our perceptions were merely passive reflections of the unpredictable and inconstant input that bathes our receptors.

Helmholtz's great contemporary, Clerk Maxwell, had also been fascinated by the mystery of color vision from his student days. He formalized the notions of primary colors and color mixing by the invention of a color top (the colors of which fused, when it was spun, to yield a sensation of

grey), and a graphic representation with three axes, a color triangle, which showed how any color could be created by different mixtures of the three primary colors. These prepared the way for his most spectacular demonstration, the demonstration in 1861 that color photography was possible, despite the fact that photographic emulsions were themselves black and white. He did this by photographing a colored bow three times, through red, green, and violet filters. Having obtained three "color-separation" images, as he called them, he now brought these together by superimposing them upon a screen, projecting each image through its corresponding filter (the image taken through the red filter was projected with red light, and so on). Suddenly, the bow burst forth in full color. Maxwell wondered if this was how colors were perceived in the brain, by the addition of color-separation images or their neural correlates, as in his magic-lantern demonstrations.[15]

Maxwell himself was acutely aware of the drawback of this additive process: color photography had no way of "discounting the illuminant," and its colors changed helplessly with changing wavelengths of light.

In 1957, ninety-odd years after Maxwell's famous demonstration, Edwin Land—not merely the inventor of the instant Land camera and Polaroid, but an experimenter and theorizer of genius—provided a photographic demonstration of color perception even more startling. Unlike Maxwell, he made only two black-and-white images (using a split-beam camera so they could be taken at the same time from the same viewpoint, through the same lens) and superimposed these on a screen with a double-lens projector. He used two filters to make the images: one passing longer wavelengths (a red filter), the other passing shorter wavelengths (a green

filter). The first image was then projected through a red filter, the second with ordinary white light, unfiltered. One might expect that this would produce just an overall pale-pink image, but something "impossible" happened instead. The photograph of a young woman appeared instantly in full color—"blonde hair, pale blue eyes, red coat, bluegreen collar, and strikingly natural flesh tones," as Land later described it. Where did these colors come from, how were they made? They did not seem to be "in" the photographs or the illuminants themselves. These demonstrations, overwhelming in their simplicity and impact, were color "illusions" in Goethe's sense, but illusions that demonstrated a neurological truth—that colors are not "out there" in the world, nor (as classical theory held) an automatic correlate of wavelength, but, rather, are constructed by the brain.

These experiments hung, at first, like anomalies, conceptless, in midair; they were inexplicable in terms of existing theory, but did not yet point clearly to a new one. It seemed possible, moreover, that the viewer's knowledge of appropriate colors might influence his perception of such a scene. Land decided, therefore, to replace familiar images of the natural world with entirely abstract, multicolored displays consisting of geometric patches of colored paper, so that expectation could provide no clues as to what colors should be seen. These abstract displays vaguely resembled some of the paintings of Piet Mondrian, and Land therefore terms them "color Mondrians." Using the Mondrians, which were illuminated by three projectors, using long-wave (red), middle-wave (green), and short-wave (blue) filters, Land was able to prove that, if a surface formed part of a complex multicolored scene, there was no simple relationship between the wavelength of light reflected from a surface and its

perceived color.

If, moreover, a single patch of color (for example, one ordinarily seen as green) was isolated from its surrounding colors, it would appear only as white or pale grey, whatever illuminating beam was used. Thus the green patch, Land showed, could not be regarded as inherently green, but was, in part, given its greenness by its relation to the surrounding areas of the Mondrian.

Whereas color for Newton, for classical theory, was something local and absolute, given by the wavelength of light reflected from each point, Land showed that its determination was neither local nor absolute, but depended upon the surveying of a whole scene and a comparison of the wavelength composition of the light reflected from each point with that of the light reflected from its surround. There had to be a continuous relating, a comparison of every part of the visual field with its own surround, to arrive at that global synthesis—Helmholtz's "act of judgement." Land felt that this computation or correlation followed fixed, formal rules; and he was able to predict which colors would be perceived by an observer under different conditions. He devised a "color cube," an algorithm, for this, in effect a model for the brain's comparison of the brightnesses, at different wavelengths, of all the parts of a complex, multicolored surface. Whereas Maxwell's color theory and color triangle were based on the concept of color addition, Land's model was now one of comparison. He proposed that there were, in fact, two comparisons: first of the reflectance of all the surfaces in a scene within a certain group of wavelengths, or waveband (in Land's term, a "lightness record" for that waveband), and second, a comparison of the three separate lightness records for the three wavebands

(corresponding roughly to the red, green, and blue wavelengths). This second comparison generated the color. Land himself was at pains to avoid specifying any particular brain site for these operations and was careful to call his theory of color vision the Retinex theory, implying that there might be multiple sites of interaction between the retina and the cortex.

If Land was approaching the problem of how we see colors at a psychophysical level by asking human subjects to report how they perceived complex, multicolored mosaics in changing illuminations, Semir Zeki, working in London, was approaching the problem at a physiological level, by inserting microelectrodes in the visual cortex of anesthetized monkeys and measuring the neuronal potentials generated when they were given colored stimuli. Early in the 1970s, he was able to make a crucial discovery, to delineate a small area of cells on each side of the brain, in the prestriate cortex of monkeys (areas referred to as V4), which seemed to be specialized for responding to color (Zeki called these "color-coding cells").[16] Thus, ninety years after Wilbrand and Verrey had postulated a specific center for color in the brain, Zeki was finally able to prove that such a center existed.

Fifty years earlier, the eminent neurologist Gordon Holmes, reviewing two hundred cases of visual problems caused by gunshot wounds to the visual cortex, had found not a single case of achromatopsia. He went on to deny that an isolated cerebral achromatopsia could occur. The vehemence of this denial, coming from such a great authority, played a major part in bringing all clinical interest in the subject to an end.[17] Zeki's brilliant and undeniable demonstration startled the neurological world, reawakening attention to a subject it had for many years dismissed. Following his 1973 paper, new

cases of human achromatopsia began appearing in the literature once again, and these could now be examined with new brain-imaging techniques (CAT, MRI, PET, SQUID, etc.) not available to neurologists of an earlier era. Now, for the first time, it was possible to visualize, in life, what areas of the brain might be needed for human color perception. Though many of the cases described had other problems, too (cuts in the visual field, visual agnosia, alexia, etc.), the crucial lesions seemed to be in the medial association cortex, in areas homologous to V4 in the monkey.[18] It had been shown in the 1960s that there were cells in the primary visual cortex of monkeys (in the area termed V1) that responded specifically to wavelength, but not to color; Zeki now showed, in the early 1970s, that there were other cells in the V4 areas that responded to color but not to wavelength (these V4 cells, however, received impulses from the V1 cells, converging through an intermediate structure, V2). Thus each V4 cell received information regarding a large portion of the visual field. It seemed that the two stages postulated by Land in his theory might now have an anatomical and physiological grounding: lightness records for each waveband being extracted by the wavelength-sensitive cells in V1, but only being compared or correlated to generate color in the color-coding cells of V4. Every one of these, indeed, seemed to act as a Landian correlator, or a Helmholtzian "judge."

Color vision, it seemed—like the other processes of early vision: motion, depth, and form perception—required no prior knowledge, was not determined by learning or experience, but was, as neurologists say, a "bottom-up" process. Color can indeed be generated, experimentally, by magnetic stimulation of V4, causing the "seeing" of colored

rings and halos—so-called chromatophenes.[19] But color vision, in real life, is part and parcel of our total experience, is linked with our own categorizations and values, becomes for each of us a part of our life-world, of us. V4 may be an ultimate generator of color, but it signals to, it converses with, a hundred other systems in the mind-brain; and perhaps it can also be modulated by these. It is at higher levels that integration occurs, that color fuses with memories, expectations, associations, and desires to make a world with resonance and meaning for each of us.[20]

Mr. I. not only presented a rather "pure" case of cerebral achromatopsia (virtually uncontaminated by additional defects in the perception of form, motion, or depth), but was a highly intelligent and expert witness as well, one who was skilled at drawing and reporting what he saw. Indeed, when we first met, and he described how objects and surfaces "fluctuated" in different lights, he was, so to speak, describing the world in wavelengths, not in colors. The experience was so unlike anything he had ever experienced, so strange, so anomalous, that he could find no parallels, no metaphors, no paints or words to depict it.

When I phoned Professor Zeki to tell him of this exceptional patient, he was greatly intrigued and wondered, in particular, how Mr. I. might do with Mondrian testing, such as he and Land had used with normally sighted people and with animals. He at once arranged to come to New York to join us—Bob Wasserman, my ophthalmologist colleague; Ralph Siegel, a neurophysiologist; and myself—in a comprehensive testing of Jonathan I. No patient with achromatopsia had ever been examined in this way before.

We used a Mondrian of great complexity and brilliance,

illuminated either by white light or by light filtered through narrow-band filters allowing only long wavelengths (red), intermediate wavelengths (green), or short wavelengths (blue) to pass. The intensity of the illuminating beam, in every case, was the same.

Mr. I. could distinguish most of the geometric shapes, though only as consisting of differing shades of grey, and he instantly ranked them on a one-to-four grey scale, although he could not distinguish some color boundaries (for example, between red and green, which both appeared to him, in white light, as black). With rapid, random switching of the filters, the grey-scale value of all the shapes dramatically changed—some shades previously indistinguishable now became very different, and all shades (except actual black) changed, either grossly or subtly, with the wavelength of the illuminating beam. (Thus a green area would be seen by him as white in medium-wavelength light, but as black in white or long-wavelength light.)

All Mr. I.'s responses were consistent and immediate. (It would have been very difficult, if not impossible, for a normally sighted person to make these instant and invariably "correct" estimations, even with a perfect memory and a profound knowledge of the latest color theory.) Mr. I., it was clear, could discriminate wavelengths, but he could not go on from this to translate the discriminated wavelengths into color; he could not generate the cerebral or mental construct of color.

This finding not only clarified the nature of the problem, but also served to pinpoint the location of the trouble. Mr. I.'s primary visual cortex was essentially intact, and it was the secondary cortex (specifically the V4 areas, or their connections) that bore virtually the whole brunt of the

damage. These areas are very small, even in man; yet all our perception of color, all our ability to imagine or remember it, all our sense of living in a world of color, depend crucially on their integrity. A mischance had devastated these bean-sized areas of Mr. I.'s brain—and with this, his whole life, his life-world, had been changed.

The Mondrian testing had demonstrated damage in these areas; we wondered now if we could see this, using brain scans. But CAT and MRI scans were entirely normal. This could have been because the scanning techniques of the time had a resolution inadequate to visualize what may have been only a patchy damage to V4; it could have been that the damage sustained was metabolic only, not structural; or it could have been that the main damage was not in V4 itself, but in the structures (the so-called "blobs" in V1 or the "stripes" in V2) leading up to it.[21]

It has been stressed—by both Zeki and Francis Crick—that these small structures, the blobs and stripes, are intensely active metabolically and may be unusually vulnerable to even temporary reductions of oxygen. Crick, in particular (with whom I discussed the case in great detail), wondered whether Mr. I. could have suffered from carbon monoxide poisoning, which is known to cause changes in color vision through its effects on the oxygenation of the blood to the color areas. Mr. I. might have been exposed to carbon monoxide through a leaky exhaust in his car, Crick speculated—perhaps due to the accident, conceivably even causing it.[22]

But all this was in a sense academic. Mr. I.'s achromatopsia, after three months, remained absolute, and he had persisting impairments of contrast vision, too.[23] Whether these would clear eventually we could not say—

some cases of acquired cerebral achromatopsia improve with time, but others do not. We still did not know what had caused the damage to Mr. I.'s brain, whether it was a toxin such as carbon monoxide, or the impact of the car accident, or the result of an impairment of blood flow to the visual areas of the brain. It was possible that if it had been caused by a stroke, there might be more such strokes. The prognosis had to remain uncertain, although his situation by now seemed to be stable.

We were, however, able to offer a little practical help. Mr. I. had consistently seen the boundaries of the Mondrian patches most clearly when these were illuminated by medium-wavelength light, and Dr. Zeki therefore suggested we give him a pair of green sunglasses, transmitting only this waveband in which he saw most clearly. A pair of glasses was specially made, and Mr. I. took to wearing them, especially in bright sunlight. The new glasses delighted him, for although they did nothing to restore his lost color vision, they did seem noticeably to enhance his contrast vision and his perception of form and boundaries. He could even enjoy color TV with his wife again. (The dark-green glasses, in effect, rendered the color set monochromatic—though he continued to prefer his old black-and-white set when alone.)

The sense of loss following his accident was overwhelming to Jonathan I., as it must be to anyone who loses color, a sense that interweaves itself in all our visual experiences and is so central in our imagination and memory, our knowledge of the world, our culture and art. This sense of loss, in relation to the natural world, has been remarked upon in every case. For the nineteenth-century physician thrown from his horse, flowers had "lost more than half their

beauty," and entering his garden, abruptly bereft of color, was not short of shocking. This sense of loss and of shock was doubled and redoubled for Mr. I., for he had not only lost the beauty of the natural world, and the world of people, and of the innumerable objects whose colors are part of daily life, but he had also lost the world of art, he felt—the world that, for fifty years or more, had absorbed his profoundly visual and chromatic talents and sensibilities. The first weeks of his achromatopsia were thus weeks of an almost suicidal depression.[24]

In addition to his sense of loss, Jonathan I. found his changed visual world, at first, abhorrent and abnormal. This, too, is the experience of most people in his position: the concussed physician thrown from his horse found his vision "perverted," one of Damasio's patients found her grey world "dirty." Why, one must wonder, do all subjects with a cerebral achromatopsia express themselves in such terms—why should their experience seem so abnormal? Mr. I. was seeing with his cones, seeing with the wavelength-sensitive cells of V1, but unable to use the higher-order, color-generating mechanism of V4. For us, the output of V1 is unimaginable, because it is never experienced as such and is immediately shunted on to a higher level, where it is further processed to yield the perception of color. Thus the raw output of V1 never appears in awareness for us. But for Mr. I. it did—his brain damage had made him privy to, indeed trapped him within, a strange in-between state—the uncanny world of V1—a world of anomalous and, so to speak, prechromatic sensation, which could not be categorized as either colored or colorless.[25]

Mr. I., with his heightened visual and aesthetic sensibilities, found these changes particularly intolerable. We

know too little about what determines emotion and aesthetic appeal in relation to color, and indeed in relation to seeing generally—and this is a matter of individual experience and taste.[26]

Color perception had been an essential part not only of Mr. I.'s visual sense, but his aesthetic sense, his sensibility, his creative identity, an essential part of the way he constructed his world—and now color was gone, not only in perception, but in imagination and memory as well. The resonances of this were very deep. At first he was intensely, furiously conscious of what he had lost (though "conscious," so to speak, in the manner of an amnesiac). He would glare at an orange in a state of rage, trying to force it to resume its true color. He would sit for hours before his (to him) dark grey lawn, trying to see it, to imagine it, to remember it, as green. He found himself now not only in an impoverished world, but in an alien, incoherent, and almost nightmarish one. He expressed this soon after his injury, better than he could in words, in some of his early, desperate paintings.

But then, with the "apocalyptic" sunrise, and his painting of this, came the first hint of a change, an impulse to construct the world anew, to construct his own sensibility and identity anew. Some of this was conscious and deliberate: retraining his eyes (and hands) to operate, as he had in his first days as an artist. But much occurred below this level, at a level of neural processing not directly accessible to consciousness or control. In this sense, he started to be redefined by what had happened to him—redefined physiologically, psychologically, aesthetically—and with this there came a transformation of values, so that the total otherness, the alienness of his V1 world, which at first had such a quality of horror and nightmare, came to take on,

for him, a strange fascination and beauty.

Immediately after his accident, and for a year or more thereafter, Jonathan I. insisted that he still "knew" colors, knew what was right, what was appropriate, what was beautiful, even if he could no longer visualize them in his mind. But, thereafter, he became somewhat less sure, as if now, unsupported by actual experience or image, his color associations had started to give way. Perhaps such a forgetting—a forgetting at once physiological and psychological, at once strategic and structural—may have to occur, to some extent, sooner or later, in anyone who is no longer able to experience or imagine, or in any way to generate, a particular mode of perception. (Nor is it necessary that the primary damage be cortical; it may occur, after months or years, even in those who are peripherally or retinally blind.)[27]

There was a lessening concern with what he had lost, and indeed with the whole subject of color, which at first had so obsessed him. Indeed, he now spoke of being "divorced" from color. He could still speak fluently about it, but there seemed to be a certain hollowness to his words, as if he were drawing only from past knowledge and no longer understood it.

Nordby writes:

> Although I have acquired a thorough theoretical knowledge of the physics of colours and the physiology of the colour receptor mechanisms, nothing of this can help me to understand the true nature of colours.[28]

What was true for Nordby was now true for Jonathan I., too. He had in some ways started to resemble a person born colorblind, even though he had lived in a color world for the first sixty-five years of his life.

At once forgetting and turning away from color, turning away from the chromatic orientation and habits and strategies of his previous life, Mr. I., in the second year after his injury, found that he saw best in subdued light or twilight, and not in the full glare of day. Very bright light tended to dazzle and temporarily blind him—another sign of damage to his visual systems—but he found the night and nightlife peculiarly congenial, for they seemed to be "designed," as he once said, "in terms of black and white."

He started becoming a "night person," in his own words, and took to exploring other cities, other places, but only at night. He would drive, at random, to Boston or Baltimore, or to small towns and villages, arriving at dusk, and then wandering about the streets for half the night, occasionally talking to a fellow walker, occasionally going into little diners: "Everything in diners is different at night, at least if it has windows. The darkness comes into the place, and no amount of light can change it. They are transformed into night places. I love the nighttime," Mr. I. said. "Gradually I am becoming a night person. It's a different world: there's a lot of space—you're not hemmed in by streets, by people.... It's a whole new world."

Mr. I., when he was not traveling, would get up earlier and earlier, to work in the night, to relish the night. He felt that in the night world (as he called it) he was the equal, or the superior, of "normal" people: "I feel better because I know then that I'm not a freak … and I have developed acute night vision, it's amazing what I see—I can read license plates at night from four blocks away. You couldn't see it from a block away."[29]

One wonders whether his night vision might, with time, have taken on heightened function in compensation for the

damage to his color system—there might, at this stage, also have been a heightening of movement sensitivity, perhaps of depth sensitivity, too, possibly going with an increased dependence on and use of the intact M system.[30]

Most interesting of all, the sense of profound loss, and the sense of unpleasantness and abnormality, so severe in the first months following his head injury, seemed to disappear, or even reverse. Although Mr. I. does not deny his loss, and at some level still mourns it, he has come to feel that his vision has become "highly refined," "privileged," that he sees a world of pure form, uncluttered by color. Subtle textures and patterns, normally obscured for the rest of us because of their embedding in color, now stand out for him.[31] He feels he has been given "a whole new world," which the rest of us, distracted by color, are insensitive to. He no longer thinks of color, pines for it, grieves its loss. He has almost come to see his achromatopsia as a strange gift, one that has ushered him into a new state of sensibility and being. In this his transformation is exceedingly similar to that of John Hull, who, after two or three years of experiencing blindness as an affliction and curse, came to see it as "a dark, paradoxical gift," a "concentrated human condition … one of the orders of human being."

Once, about three years after his injury, an intriguing suggestion was made (by Israel Rosenfield), that Mr. I. try to regain his color vision. Since the mechanism for comparing wavelengths was intact, and only V4 (or its equivalent) was damaged, it might be possible, at least in theory, Rosenfield thought, to "retrain" another part of the brain to perform the requisite Landian correlations, and thus to achieve some restoration of color vision. What was striking was Mr. I.'s response to this suggestion. In the first months after his

injury, he said, he would have embraced such a suggestion, done everything possible to be "cured." But now that he conceived the world in different terms, and again found it coherent and complete, he thought the suggestion unintelligible, and repugnant. Now that color had lost its former associations, its sense, he could no longer imagine what its restoration would be like. Its reintroduction would be grossly confusing, he thought, might force a welter of irrelevant sensations upon him, and disrupt the now-reestablished visual order of his world. He had been for a while in a sort of limbo; now he had settled—neurologically and psychologically—for the world of achromatopia.

In terms of his painting, after a year or more of experiment and uncertainty, Mr. I. has moved into a strong and productive phase, as strong and productive as anything in his long artistic career. His black-and-white paintings are highly successful, and people comment on his creative renewal, the remarkable black-and-white "phase" he has moved into. Very few of them know that his latest phase is anything other than an expression of his artistic development, that it was brought about by a calamitous loss.

Though it has been possible to define the primary damage in Mr. I.'s brain—the knocking out of an essential part of his color-constructing system—we are still totally ignorant of the "higher" changes in brain function that must have occurred in its train. Jonathan I. did not lose just his perception of color, but imagery, and even dreaming in color. Finally he seemed to lose even his memory of color, so that it ceased to be part of his mental knowledge, his mind.

Thus, as more and more time elapsed without color vision, he came to resemble someone with an amnesia for color—or,

indeed, someone who had never known it at all. But, at the same time, a revision was occurring, so that as his former color world and even the memory of it became fainter and died inside him, a whole new world of seeing, of imagination, of sensibility, was born.[32]

There is no doubt of the reality of these changes—although it may have required a subject as gifted and as articulate as Jonathan I. to bring them out with such clarity. Neuroscience, at this point, can say nothing about the cerebral basis of such "higher" changes. The physiological investigation of color, thus far, has terminated in the color systems of early vision, the Landian correlations that occur in V1 and V4. But V4 is not an end point, it is only a way station, projecting in its turn to higher and higher levels—eventually to the hippocampus, so essential for the storage of memories; to the emotional centers of the limbic system and amygdala; and to many other parts of the cortex. The cessation of information flow from V4 to the memory systems of the hippocampus and prefrontal cortex, for example, might in part explain Mr. I.'s "forgetting" of color. We do not have the tools at the moment to map the subtle, higher-level neural consequences of such a sensory loss, but a history such as Jonathan I.'s shows how crucial it is to do this.

Work in the last decade has shown how plastic the cerebral cortex is, and how the cerebral "mapping" of body image, for example, may be drastically reorganized and revised, not only following injuries or immobilizations, but in consequence of the special use or disuse of individual parts. We know, for instance, that the constant use of one finger in reading Braille leads to a huge hypertrophy of that finger's representation in the cortex. And with early deafness and the

use of sign language, there may be drastic remappings in the brain, large areas of the auditory cortex being reallocated for visual processing. Similarly, it seemed, with Mr. I.: if entire systems of representation, of meaning, had been extinguished inside him, entirely new systems had been brought into being.

On the ultimate question—the question of qualia: why a particular sensation may be perceived as red—the case of Jonathan I. may not be able to help us at all. After describing "the celebrated phaenomenon of colours," Newton drew back from all speculation about sensation and would hazard no hypothesis as to "by what modes or action light produceth in our minds the phantasms of colours." Three centuries later, we still have no hypothesis, and perhaps such questions can never be answered at all.

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[1] I asked Mr. I. later if he knew Greek or Hebrew; he said no, there was just the sense of an unintelligible foreign language; perhaps, he added, "cuneiform" would be more accurate. He saw forms, he knew they had to have meaning, but could not imagine what this meaning might be.

[2] Similarly, a patient of Dr. Antonio Damasio, with achromatopsia from a tumor, thought everything and everyone looked "dirty," even finding new-fallen snow unpleasant and dirty.

[3] In 1688, in Some Uncommon Observations about Vitiated Sight, Robert Boyle described a young woman in her early twenties whose eyesight had been normal until she was eighteen, when she developed a fever, was "tormented with blisters," and, with this, "deprived of her sight." When she was presented with something red, "she look'd attentively upon it, but told me, that to her, it did not seem Red, but of another Colour, which one would guess by her description to be a Dark or Dirty one." When "tufts of Silk that were finely Color'd" were given to her, she could only say that "they seem'd to be a Light-colour, but could not tell which." When asked whether the meadows "did not appear to her Cloathed in Green," she

said they did not, but seemed to be “of an odd Darkish colour,” adding that when she wished to gather violets, “she was not able to distinguish them by the Colour from the surrounding Grass, but only by the Shape, or by feeling them.” Boyle further observed a change in her habits, that she liked now to walk abroad in the evenings, and this “she much delighteth to do.”

A number of accounts were published in the nineteenth century—many collected in Mary Collins’s Colour-Blindness—one of the most vivid (besides that of an achromatopic house painter) being that of a physician who, thrown from his horse, suffered a head injury and concussion. “On recovering sufficiently to notice objects around him,” George Wilson recorded in 1853,

> he found that his perception of colours, which was formerly normal and acute, had become both weakened and perverted.… All coloured objects … now seem strange to him.… Whilst formerly a student in Edinburgh he was known as an excellent anatomist; now he cannot distinguish an artery from a vein by its tint.… Flowers have lost more than half their beauty for him, and he recalls the shock which he received on first entering his garden after his recovery, at finding that a favourite damask rose had become in all its parts, petals, leaves, and stem, of one uniform dull colour; and that variegated flowers had lost their characteristic tints.

[4] One sees interesting similarities, but also differences, from the vision of those with congenital achromatopsia. Thus Knut Nordby, a congenitally colorblind vision researcher, writes:

> I only see the world in shades that colour-normals describe as black, white and grey. My subjective spectral sensitivity is not unlike that of orthochromatic black and white film. I experience the colour called red as a very dark grey, nearly black, even in very bright light. On a grey-scale the blue and green colours I see as mid-greys, somewhat darker greys if they are saturated, somewhat lighter greys when unsaturated. Yellow typically appears to me as a rather light grey, but is usually not confused with white. Brown usually appears as a dark grey and so does a very saturated orange.

[5] Only one sense could give him any real pleasure at this time, and this was the sense of smell. Mr. I. had always had a most acute, erotically charged sense of smell—indeed, he ran a small perfume business on the side, compounding his own scents. As the pleasures of seeing were lost, the pleasures of smell were heightened (or so it seemed to him), in the first grim weeks after his accident.

[6] The question of "knowing" color is very complex and has paradoxical aspects that are difficult to dissect. Certainly Mr. I. was intensely aware of a profound loss with the change in his vision, so clearly some sort of comparison with past experience was possible for him. Such a comparison is not possible if there is a complete destruction of the primary visual cortex on both sides, say from a stroke, as in Anton's syndrome. Patients with this syndrome become totally blind, but make no complaint or report of their blindness. They do not know they are blind; the whole structure of consciousness is completely reorganized—instantly so—at the moment they are stricken.

Similarly, patients with massive strokes in the right parietal cortex may lose not only the sensation and use but the very knowledge of their left sides, of everything to the left, and indeed of the very concept of leftness. But they are "anosognosic"—they have no knowledge of their loss; we may say their world is bisected, but, for them, it is whole and complete.

[7] One anomaly showed itself in the yarn-sorting test; he ranked bright saturated blues as "pale" (as he had complained that the blue sky seemed almost white). But was this an anomaly? Could we be sure that the blue wool was not, under its blueness, rather washed-out or pale? We had to have hues that were otherwise identical—identical in brightness, saturation, reflectivity, so we obtained a set of carefully produced color buttons known as the Farnsworth-Munsell test and gave this to Mr. I. He was unable to put the buttons in any order, but he did separate out the blue ones as "paler" than the rest.

[8] Further testing with the Nagel anomaloscope and the Sloan achromatopsia cards confirmed Mr. I.'s total colorblindness. With Dr. Ralph Siegel, we did tests of depth and motion perception (using Julesz random-dot stereograms and moving random-dot fields)—these were normal, as were tests of his ability to generate structure and depth from motion. There was, however, one interesting anomaly:

Mr. I. was unable to “get” red and green stereograms (bicolor anaglyphs), presumably because color vision is needed to segregate the two images. We also obtained electroretinograms, and these were quite normal, indicating that all three cone mechanisms in the retina were intact, and that the colorblindness was indeed of cerebral origin.

9 In 1877, Gladstone, in an article entitled “On the Colour Sense of Homer,” spoke of Homer’s use of such phrases as “the wine-dark sea.” Was this just a poetic convention, or did Homer, the Greeks, actually see the sea differently? There is indeed considerable variation between different cultures in the way they will categorize and name colors—individuals may only “see” a color (or make a perceptual categorization) if there is an existing cultural category or name for it. But it is not clear whether such categorization may actually alter elementary color perception.

10 “As it is almost impossible to conceive each sensitive point of the retina to contain an infinite number of particles, each capable of vibrating in perfect unison with every possible undulation,” Young wrote, “it becomes necessary to suppose the number limited, for instance to the three principal colours, red, yellow, and blue.”

The great chemist John Dalton, just five years earlier, had provided a classic description of red-green colorblindness in himself. He thought this was due to a discoloration in the transparent media of the eye—and, indeed, willed an eye to posterity to test this. Young, however, provided the correct interpretation—that one of the three types of color receptor was missing. (Dalton’s eye still resides, pickled, on a shelf in Cambridge.)

Lindsay T. Sharpe and Knut Nordby discuss this and many other aspects of the history of colorblindness research in “Total Colorblindness: An Introduction.”

11 In 1816, the young Schopenhauer proposed a different theory of color vision, one that envisaged not a passive, mechanical resonance of tuned particles or receptors, as Young had postulated, but their active stimulation, competition, and inhibition—an explicit “opponens” theory such as Ewald Hering was to create seventy years later, in apparent contradiction of the Young-Helmholtz theory. These opponens theories were ignored at the time, and continued to be ignored

until the 1950s. We now envisage a combination of Young-Helmholtz and opponens mechanisms: tuned receptors, which converse with one another, are continually linked in an interactional balance. Thus integration and selection, as Schopenhauer divined, start in the retina.

[12] There is no mention of it in the great 1911 edition of Helmholtz's Physiological Optics, though there is a large section on retinal achromatopsia.

[13] There were, however, brief mentions of achromatopsia in these intervening years, which were ignored, or soon forgotten, for the most part. Even Kurt Goldstein, although philosophically opposed to notions of isolated neurological deficits, remarked that he had seen several cases of pure cerebral achromatopsia without visual field losses or other impairments—an observation thrown off casually in the course of his 1948 book, Language and Language Disturbances.

[14] A perhaps similar phenomenon is described by Knut Nordby. During his first school year, his teacher presented the class with a printed alphabet, in which the vowels were red and the consonants black.

> I could not see any difference between them and could not understand what the teacher meant, until early one morning late in the autumn when the room-lights had been turned on, and, unexpectedly, I saw that some of the letters, i.e. the AEIOUY ÅÄÖ, were now suddenly a darkish grey, while the others were still solid black. This experience taught me that colours may look different under different light-sources, and that the same colour can be matched to different grey-tones in different kinds of illumination.

[15] Maxwell's demonstration of the "decomposition" and "reconstitution" of color in this way made color photography possible. Huge "color cameras" were used at first, which split the incident light into three beams and passed these through filters of the three primary colors (such a camera, reversed, served as a chromoscope, or Maxwellian projector). Though an integral color process was envisaged by Ducos du Hauron in the 1860s, it was not until 1907 that such a process (Autochrome) was actually developed, by the Lumière brothers. They used tiny starch grains dyed red, green, and violet, in contact with the photographic emulsion—these acted as a sort of Maxwellian grid through which the three color-

separation images, mosaicked together, could both be taken and viewed. (Color cameras, Lumièrecolor, Dufaycolor, Finlaycolor, and many other additive color processes were still being used in the 1940s, when I was a boy, and stimulated my own first interest in the nature of color.)

16 He was also able to find cells, in an adjacent area, that seemed to respond solely to movement. A remarkable account and analysis of a patient with a pure "motion blindness" was given by Zihl, Von Cramon, and Mai in 1983. The patient's problems are described as follows:

> The visual disorder complained of by the patient was a loss of movement vision in all three dimensions. She had difficulty, for example, in pouring tea or coffee into a cup because the fluid appeared to be frozen, like a glacier. In addition, she could not stop pouring at the right time since she was unable to perceive the movement in the cup (or a pot) when the fluid rose. Furthermore the patient complained of difficulties in following a dialogue because she could not see the movement of the face and, especially, the mouth of the speaker. In a room where more than two other people were walking, she felt very insecure and unwell, and usually left the room immediately, because "people were suddenly here or there but I have not seen them moving." The patient experienced the same problem but to an even more marked extent in crowded streets or places, which she therefore avoided as much as possible. She could not cross the street because of her inability to judge the speed of a car, but she could identify the car itself without difficulty. "When I'm looking at the car first, it seems far away. But then, when I want to cross the road, suddenly the car is very near." She gradually learned to "estimate" the distance of moving vehicles by means of the sound becoming louder.

17 A vivid account of Holmes's negative influence has been provided by Damasio, who also points out that all of Holmes's cases involved lesions in the dorsal aspect of the occipital lobe, whereas the center for achromatopsia lies on the ventral aspect.

18 The work of Antonio and Hanna Damasio and their colleagues at the University of Iowa was particularly important here, both by virtue of the

minuteness of the perceptual testing, and the refinement of the neuroimaging they used.

[19] Such chromatophenes may occur spontaneously in visual migraines, and Mr. I. himself had experienced these, on occasion, in migraines occurring before his accident. One wonders what would have been experienced if Mr. I.'s V4 areas had been stimulated—but magnetic stimulation of circumscribed brain areas was not technically possible at the time. One wonders, too, now that such stimulation is possible, whether it might be tried in individuals with congenital (retinal) achromatopsia (several such achromatopes have expressed their curiosity about such an experiment). It is possible—I am not aware of any studies on this—that V4 fails to develop in such people, with the absence of any cone input. But if V4 is present as a functional (though never functioning) unit despite the absence of cones, its stimulation might produce an astounding phenomenon—a burst of unprecedented, totally novel sensation, in a brain/mind that had never had a chance to experience or categorize such sensation. Hume wonders if a man could imagine, could even perceive, a color he had never seen before—perhaps this Humean question (propounded in 1738) could find an answer now.

[20] The power of expectation and mental set in the perception of color is clearly shown in those with partial red-green colorblindness. Such people may not, for example, be able to spot scarlet holly berries against the dark green foliage, or the delicate salmon-pink of dawn—until these are pointed out to them. "Our poor impoverished cone cells," says a dyschromatope of my acquaintance, "need the amplification of intellect, knowledge, expectation, and attention in order to 'see' the colors that we are normally 'blind' to."

[21] Malfunction in V4 can be shown by a newer technique, PET scanning (which pictures the metabolic activity of different brain areas), even if no anatomical lesion is visible on CAT or MRI scans. Unfortunately, this was not available to us at the time.

[22] Mr. I., fond of spending time in sports clubs and bars, did some research here himself and told us that he had spoken to a number of boxers who had had transient, and sometimes persistent, losses of color vision following blows to the head. Partial or total achromatopsia ("greying-out"), also temporary, is

characteristic of fainting or shock, in which there is a reduction of blood supply to the posterior, and especially the visual parts, of the brain. Greying-out also occurs in transient ischemic attacks, due to arterial insufficiency—Zeki speculates that this affects the wavelength-selective cells in the blobs of V1 and the thin stripes of V2. Transient alterations of color vision—including bizarre instabilities or transformations of color (dyschromatopsia)—may also occur in visual migraines and epilepsies and are well known to users of mescaline and other drugs. They can be a disquieting side effect of ibuprofen.

[23] It was never quite clear from Mr. I.'s descriptions of daily life whether or not he had some slight impairment of form vision. But, interestingly, when he was being tested on the Mondrians, boundaries between rectangles tended to disappear with prolonged fixation, though they would be rapidly restored if the stimulus was moved. There are two other systems besides the blob system in early visual processing: the M system, which deals with movement and depth perception particularly, but not color; and a P-interblob system, which probably deals with high-resolution form perception. Zeki thought that the dissolution of boundaries with prolonged fixation suggested a defect in the P system, and their rapid restoration with movement "a healthy and active M system."

[24] This sense of loss is not, of course, experienced by those born totally colorblind. This is brought out in another letter I received recently from a charming and intelligent woman, Frances Futterman, born totally colorblind. She contrasted her own situation with that of Jonathan I.:

> I was struck by how different that kind of experience must be, compared to my own experience of never having seen color before, thus never having lost it—and also never having been depressed about my colorless world.… The way I see in and of itself is not depressing. In fact, I am frequently overwhelmed by the beauty of the natural world.… People say I must see in shades of gray or in "black and white," but I don't think so. The word gray has no more meaning for me than the word pink or blue—in fact, even less meaning, because I have developed inner concepts of color words like pink and blue; but, for the life of me, I can't conceive of gray.

Though Mrs. Futterman's experience is certainly different from Mr. I.'s, both remark on the meaninglessness of the word "grey," a word that can no more convey anything to the achromatopic than can "darkness" to the blind, or "silence" to the deaf. Mrs. Futterman remarks, as Mr. I. came to, on the beauty of her world. "I would also be willing to bet," she says, "that if we were tested along with normals in low lighting levels, we would be able to detect far more shades of gray. Black and white photos look far too stark to me. The world I see has so much more richness and variety than black-and-white photos or TV shows.... My vision is a lot richer than normals can imagine."

[25] We may experience something like this, Zeki has recently shown, by using an inhibitory magnetic stimulation to V4, which produces a temporary achromatopsia.

[26] We also know too little about the interactions of the three major systems in early vision—the M, interblob, and blob systems. But Crick wonders whether some of the unpleasantness and abnormality, at least—the "leaden" vision of which Mr. I. complained—might not in part be due to the unmoderated action of the preserved M system, which, he emphasizes, "sees few shades of grey, [so that] its white would correspond to what was (in normal people) a dirty white." This notion gains support from the fact that people with congenital achromatopsia, who have not sustained any damage to their higher visual systems, do not have any such perceptual abnormalities. Thus Knut Nordby writes: "I have never experienced 'dirty,' 'impure,' 'stained,' or 'washed-out' colors, as reported by the artist Jonathan I."

[27] J. D. Mollon et al. describe the case of a young police cadet who, following a severe febrile illness (probably cerebral herpes) was left with achromatopsia, hemianopia, and some agnosia and amnesia. Testing him five years after the illness, Mollon reports that "he was able to name (presumably by means of verbal memory) the colours of e.g., grass, traffic lights, and the union jack, but made errors on other common objects (e.g., banana, pillar-box)." Thus here, after five years of total colorblindness, the colors of even the most familiar objects were often forgotten. Such effects have been recorded in ordinary retinal blindness, too, where after some years there may be a widespread loss of all visual memories,

including those of color.

[28] "A very intelligent blind person," Schopenhauer writes, "could almost [construct] a theory of colours from accurate statements that he heard about them." Diderot, similarly, speaking of Nicholas Saunderson, a famous blind lecturer on optics at Oxford in the early eighteenth century, feels that he had a profound theoretical knowledge and concept of space, although he never had any direct visual percept of it. (See footnote 13.)

[29] With his revulsion from color and brightness, his fondness of dusk and night, his apparently enhanced vision at dusk and night, Mr. I. sounds like Kaspar Hauser, the boy who was confined in a dimly lit cellar for fifteen years, as Anselm von Feuerbach described him in 1832:

> As to his sight, there existed, in respect to him, no twilight, no night, no darkness.... At night he stepped everywhere with the greatest confidence; and in dark places, he always refused a light when it was offered to him. He often looked with astonishment, or laughed, at persons who, in dark places, for instance, when entering a house, or walking on a staircase by night, sought safety in groping their way, or in laying hold on adjacent objects. In twilight, he even saw much better than in broad daylight. Thus, after sunset, he once read the number of a house at a distance of one hundred and eighty paces, which, in daylight, he would not have been able to distinguish so far off. Towards the close of twilight, he once pointed out to his instructor a gnat that was hanging in a very distant spider's web. (pp. 83–4)

[30] It may be that individuals with congenital achromatopsia develop heightened function of the M system, and may be extraordinarily adept at spotting movement. This is currently being investigated by Ralph Siegel and Martin Gizzi.

[31] I recently heard of an achromatopic botanist in England said to be even better than color normals at swiftly identifying ferns and other plants in woods, hedgerows, and other almost monochromatic environments. Similarly, in World War II, people with severe red-green colorblindness were pressed into service as bombardiers, because of their ability to "see through" colored camouflage and not be distracted by what would be, to the normally sighted, a confusing and

deceiving configuration of colors. One veteran of the Pacific theater reports that colorblind soldiers were indispensable in spotting the movement of camouflaged troops in the jungle. (All of these things may also be clearer to color normals at twilight.)

[32] A similar emergence of new sensibilities and imagination is described in H. G. Wells's great short story "The Country of the Blind": "For fourteen generations these people have been blind and cut off from all the seeing world; the names for all the things of sight had faded and changed.... Much of their imagination had shrivelled with their eyes, and they had made for themselves new imaginations with their ever more sensitive ears and fingertips."