Vision system puts eyesight in blind spots

The brain abhors a vision vacuum. Consider a phenomenon long noted by scientists: Each eye possesses a blind spot in the retina corresponding to the head of the optic nerve, yet no black spots mar our view of the world.

The way in which the brain compensates for these natural holes in our field of vision remains largely unexplored. But a new study suggests the brain's visual system may create a physiological representation of visual information surrounding blind spots – one that automatically paints a coherent scene by filling in the optical void.

A theory that the brain simply ignores visual blind spots – and operates as if they do not exist – cannot explain the responses of healthy volunteers to artificially induced blind spots, assert the psychologists who conducted the study. Vilayanur S. Ramachandran of the University of California, San Diego, and Richard L. Gregory of the University of Bristol in England describe their investigation in the April 25 NATURE.

The pair designed a series of experiments employing computer-generated designs with deleted sections serving as artificial blind spots. Their tests indicate that visual "filling in" of a missing patch occurs separately for color and texture; that after it disappears, the blind spot continues to influence motion perception unconsciously; and that patterns beyond the immediate boundary of blind spots participate in visual makeovers.

In one test, the researchers directed four volunteers to watch a computer screen with a background of twinkling dots in different shades of gray. Each viewer focused just to the right or left of a small, blank gray square near a corner of the screen. On 10 consecutive trials, all reported that within an average of 5 seconds the square vanished and the twinkling dots took its place.

In another experiment, the computer displayed a pink background with twinkling black dots, and a small gray square with black dots moving horizontally across it as if on a conveyor belt. When the same volunteers focused just to the right or left of the square, they reported this gray patch also faded in a matter of seconds, but in two stages. First, the gray region in the square vanished and the surrounding pink seeped in; then the moving dots disappeared, and a few seconds later the twinkling dots took their place. Thus, separate "fill mechanisms" for color and texture may exist, perhaps in different parts of the visual cortex, the researchers propose.

Brain cells that extract information on the borders of the square appear critical to the fading and filling-in process, they add. In some of the prior trials, once filling in occurred the gray square with black dots was wiped off the screen and replaced by a smaller replica within its former, now invisible borders. Participants saw the new square pop into view, fade and fill in within a few seconds.

However, information from a paved-over blind spot still exerts an effect on perception, the scientists note. Using the same computer-generated arrangement, they waited until volunteers reported that the gray square had faded completely, then switched off the square and replaced it with an identical square shifted slightly to the left or right. Participants reported that the second square seemed to move or arrive at its location, rather than just appearing out of nowhere. The first square apparently stimulated an unconscious illusion of motion, Ramachandran and Gregory maintain.

Blind spots can reach beyond their borders for visual filler, the researchers report. Another computer pattern showed a circular gray disk straddling the vertical border between a pink and a yellow region. Volunteers looked just to the right or left of the disk. When a thick red ring surrounded the disk, the color red gradually filled in the entire blind spot; when concentric thin red rings surrounded the thick red ring, the blind spot appeared filled with red rings.

Neural pathways feeding into and out of the visual cortex may create the representation of visual information that fills in the blind spot, the scientists suggest.

– B. Bower

Steroid slackens pace of muscular dystrophy

A powerful steroid has proved its mettle as the first drug to slow progressive muscle weakening in youngsters with Duchenne's muscular dystrophy. Although the drug, called prednisone, carries serious side effects, researchers believe it may ultimately point the way to an equally effective but safer treatment.

"For the first time, we have a long-term study that shows [prednisone's] efficacy," says Gerald M. Fenichel of Vanderbilt University in Nashville, who presented the results this week at the American Academy of Neurology meeting in Boston. He cautions, however, that the drug cannot rid patients of the disease. "Prednisone is not a cure," he says.

One out of every 3,500 male infants in the United States has inherited a defective gene that causes Duchenne's muscular dystrophy. At about age 3, afflicted children develop a swaying or waddling motion when they walk. From that seemingly innocent early stage, the disease advances until weakened muscles can no longer hold the body upright. Even with physical therapy, most victims need a wheelchair by age 12, and most die of heart or lung complications by age 20.

Several years ago, a study directed by Daniel B. Drachman of Johns Hopkins University in Baltimore hinted at prednisone's potential in boys with Duchenne's – the most serious form of muscular dystrophy (SN: 8/22/87, p.120). Fenichel and his colleagues have now confirmed that early promise in a multicenter trial of 89 Duchenne's patients ranging from 7 to 14 years of age.

In a year-long experiment, Fenichel's team found that a daily dose of 0.75 milligrams of prednisone per kilogram of body weight seemed to offer the best shot at fighting the muscle-sapping disorder. After that initial phase, they continued to treat the boys but had to lower the maximum dose for some patients because of harsh side effects.

Three years later, the 49 boys taking the highest daily doses (at least 0.65 mg/kg) showed the most favorable response, Fenichel reports. Their muscle strength (measured by weight-lifting tests) declined by an average of 0.017 unit per year, whereas the boys on lower doses lost about 0.164 unit per year. Although the study did not include a control group, Fenichel says participants showed a clear benefit compared with Duchenne's patients in general, who typically lose about 0.4 unit per year. Some volunteers actually gained muscle strength, he adds – an improvement readily noted by their parents.

"This study reports an apparent slowing of the progression of the disease over a period of time," comments Lawrence Z. Stern of the Muscular Dystrophy Association in Tucson, Ariz. Nonetheless, he calls the results "modest." Even for children who improve, he says, the drug may delay the need for a wheelchair by only weeks or months. Drachman, on the other hand, asserts that careful prednisone treatment can postpone wheelchair confinement by two years or more.

The Muscular Dystrophy Association, citing safety concerns, does not recommend prednisone treatment. However, says Stern, if researchers could unlock the secret of the steroid's dystrophy-fighting effect, they might someday design less toxic drug treatments.

To test a hypothesis that prednisone works by suppressing a muscle-ravaging immune response, the same researchers conducted a separate study of 99 Duchenne's patients. An immunosuppressant drug called azathioprine failed to slow muscle wasting, but prednisone again showed its protective effect. Robert C. Griggs of the University of Rochester in New York, who reported the results at the Boston meeting, says this means the researchers must look beyond the immunity hypothesis.

– K.A. Fackelmann