Researchers eye retinal remapping

Technology being developed by NASA for spacecraft orientation and docking operations may one day help patients suffering from vision defects. Richard D. Juday of the NASA Johnson Space Center in Houston and David S. Loshin of the University of Houston College of Optometry are experimenting with a prototype "programmable remapper" that shows promise for patients with a variety of vision-reducing conditions known as field defects.

The defects — caused by syndromes such as age-related maculopathy and retinitis pigmentosa — affect the eye's retina and leave patients with part of their visual field missing or distorted. Patients with a central field defect, for example, have a relatively large "blind spot" in the middle of the visual field, making reading and facial recognition difficult. For others, the periphery of the image may be missing, or the image may be fragmented.

The programmable remapper, says Loshin, is a "field compensation" device that can take information from a given coordinate system and remap it onto another coordinate system. In the case of a retinal defect, it remaps or relocates information that otherwise would be missed. "It's taking information falling within a scatoma, or blind area, and taking it out and putting it onto viable [parts of the] retina."

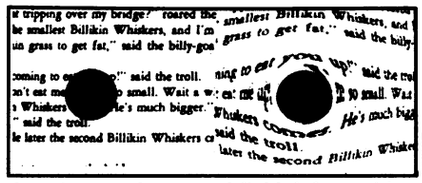
It does so essentially by "stretching" hidden information — such as typewritten words in the middle of the visual field — *around* the defective area. Although the redistribution of this information results in a distorted image, preliminary results show the increase in information provides a net benefit to at least some patients.

The prototype model is too large and expensive for everyday use; the computerized components alone fill a unit the size of a small refrigerator. However, Loshin says, chip designers have assured NASA that the complex algorithms and "look-up" boards the system uses to remap defective coordinate systems all could fit onto a single silicon chip. In that case, the entire unit — which would include a tiny, closed-circuit TV camera, a mechanism for tracking eye movements (to see where the eyes are looking) and a tiny projector that would display the remapped images for the eyes — could be mounted on a pair of glasses. NASA will conduct clinical trials of the prototype system this fall.

"We've shown static, remapped pictures to patients . . . and they claim they can read more easily," Loshin says, "although whether it's going to increase their reading rate we don't know yet." As demonstrated in a sample videotape, shown last week at the World Congress on Medical Physics and Biomedical Engineering in San Antonio, Tex., the technique's benefits are more obvious when seen in moving-picture format. As the camera scans a page, it remaps instantly — with the warped but mostly readable remapped information flowing smoothly around the defect.

Remapping faces is more difficult than remapping pictures or words, Loshin says. In preliminary work, "we were taking the [blocked-out] nose and remapping the nose all the way around the central field defect, and that doesn't really help to recognize the face." Although subsequent programs are proving more useful, he says, "I think there's going to have to be another kind of algorithm for faces, to try to get some more information from the eyes and maybe the mouth."

For patients with peripheral defects, mobility is a problem because it's difficult to perceive approaching objects or close-at-hand landscapes. By taking peripheral imagery that otherwise would be lost and remapping it toward the center of the retina, Loshin predicts, the system should enable patients to avoid approaching objects — although distortion may preclude them from knowing precisely what is coming. "I may not know what it is [that's coming]," Loshin says, "but I don't care if it's a Buick or a Volkswagen. I don't want it to hit me."



Simulation of a central field defect, with remapping (right) and without.

The technology was originally developed by the military for missile guidance and adapted by NASA to create pattern-recognition systems that would help astronauts orient their crafts for docking operations and to aid in retrieving satellites. — *R. Weiss*

Some neurons predisposed to Huntington's

Researchers long have suspected that a buildup of certain natural chemicals or an oversensitivity to them in the brain is at least partly responsible for Huntington's disease, an inherited neurological disorder afflicting about 25,000 in the United States. According to theory, these compounds flood the bundles of neurons deep within the forebrain and kill some nerve cells, causing the progressive memory loss, angry rages and muscle spasms that mark the disease.

Now, researchers from the University of Michigan in Ann Arbor report new data concerning the exact target sites on the cells where these chemicals act. In the Aug. 19 SCIENCE they describe experiments comparing the number of receptors for six chemicals in brain tissue taken from patients who died of Huntington's and from normal brains.

The two main chemicals believed responsible for the nerve cell degeneration — quinolinic acid and glutamate — occur naturally and play essential roles at their normal concentrations in the body. Quinolinic acid is a breakdown product of the amino acid tryptophan; glutamate has a metabolic function as well as acting as a neurotransmitter in the brain.

But too much of either chemical kills certain cells, and past studies have shown quinolinic acid and glutamate bind to the receptor site for a chemical abbreviated NMDA. In support of this, the Michigan scientists traced glutamate binding and found that brains with Huntington's had 93 percent fewer NMDA receptors.

This indicates that cells with those receptors had died, explains principal investigator Anne Young. "Glutamate has been hypothesized to be responsible for cell death in Huntington's," she says. "But nobody had actually measured the [NMDA] receptors in post-Huntington's brains."

Not all neurons inside the basal ganglia, a knot of several kinds of nerve cells in the forebrain, degenerate in Huntington's victims. Some cells almost completely die off, while others are unscathed. Several years ago, scientists from the University of Maryland in Baltimore injected quinolinic acid into normal rat basal ganglia and found the same patterns of cell death and survival as in Huntington's patients (SN: 1/29/83, p.70). Quinolinic acid works at the same receptor site as glutamate, Young says.

In two studies comparing a total of 13 Huntington's brains to 12 controls, Young and her colleagues found decreased numbers of receptors for five other chemicals, although none as pronounced as the decrease in NMDA receptors. Receptors for two other compounds were diminished by 67 percent and those for three others by 55 percent or less. Young plans to do the same type of comparison on patients who died before the disease progressed as far as in the Huntington's brains in the current study.

In the end, the Michigan researchers suggest that some cells may be more vulnerable to damage from glutamate and quinolinic acid because they contain more NMDA receptors where the compounds can bind to the cell. Finding a way to block those receptors, they say, might slow the disease, for which there is still no cure. — *L. Beil*