Left brain reigns over sign language

Traditional notions of brain function hold that the left hemisphere controls spoken language while the right hemisphere calls the shots regarding visual and spatial skills, including those necessary for using sign language. But converging lines of evidence, underscored by a report in the July 24 NATURE, indicate that this assumption is wrongheaded.

Rather, the ability to use and understand sign language, in which hand movements and their manipulation in space are critical to meaning, appears to be rooted in the left brain hemisphere, according to the new study, headed by Antonio Damasio of the University of lowa College of Medicine.

"The left hemisphere's specialization for language appears to rest more on its ability to transform labels into meanings regardless of the modality of a language [spoken or signed]," says Damasio.

His interpretation is based on the rare opportunity to study a hearing individual who is also proficient in sign language, before and after damage to a critical portion of her right hemisphere. The 27-year-old woman, who works as an interpreter and counselor at a community agency for deaf people, sought surgical treatment after drug therapy failed to quell her persistent seizures. Images of the right-handed subject's brain, taken while she listened and responded to a spoken language task, revealed increased activity in left hemisphere language centers.

The researchers then injected a barbiturate drug, sodium amobarbital, into an artery leading into her left hemisphere in order to shut down temporarily these language areas. The subject was initially unable to identify a series of objects in either English or sign language. Correct English responses appeared about $2\frac{1}{2}$ minutes before the first correct sign language responses occurred. During that short period, the woman was able to identify many objects verbally while simultaneously using incorrect hand shapes and movements for the same objects.

Surgical removal of parts of the right hemisphere, including the hippocampus and amygdala, was conducted and the women's seizures abated. Tests of language, memory, perception and other skills, administered 3 and 12 months after surgery, showed no change from before surgery, say the researchers. Videotaped signing interviews confirmed the subject's impression that her ability to sign and understand signing had not been dimmed.

This result is consistent with several recent studies – some directed by Ursula

Bellugi of the Salk Institute for Biological Studies in La Jolla, Calif., who participated in the present experiment—of deaf individuals fluent in sign language who suffered brain damage due to strokes. Those with left hemisphere lesions had marked problems signing; lesions to the right hemisphere created difficulty with several spatial skills, but sign language was unaffected.

In the latest case, say the researchers, recovery from barbiturate action may have taken place gradually in the left hemisphere. "Classic" speech areas probably regained functioning first, they suggest, followed by visual and sensory regions that are crucial for sign language processing.

It is possible, note the scientists, that parts of the right hemisphere could regulate the learning of sign language. Once learned, however, the use and comprehension of signs are predominantly controlled by the left side.

"Both hemispheres are capable of motor and perceptual functions," says Damasio, "but one may be more important in the early stages of these functions while the other takes over later on. The lingering idea that the right hemisphere is involved in all visual and spatial tasks is changing."

— B. Bower

Challenger's last words

"Roger. 'Go' at 'throttle up.' " Those words from the space shuttle Challenger's commander, Francis R. Scobee, had been described by NASA as the last before the craft exploded during its Jan. 28 launching. But careful analysis of an on-board tape that had been recording the crew's voices has revealed a single additional comment. Said Challenger's pilot, Michael J. Smith: "Uh-oh."

That last remark, says NASA, has provided "the first potential indication of [the crew's] awareness" that something was wrong. Less than half a second earlier, the craft had made a sudden movement to the right, possibly the result of the first stages of the explosion. And almost coincident with Smith's comment, a brilliant flash was seen between the shuttle and its external fuel tank.

The actual cause of the seven crew members' deaths, however, "cannot be positively determined," says former astronaut Joseph P. Kerwin, now director of life sciences for NASA's Johnson Space Center in Houston. The forces of the orbiter's breaking up "were probably not sufficient to cause death or serious injury," says Kerwin, though the astronauts "possibly, but not certainly" lost consciousness due to loss of the cabin's atmospheric pressure. Alive or dead, however, they then hit the ocean at about 207 miles per hour, "far in excess" of "crew survivability levels."

Possible marker for dementia disease

Researchers have found abnormal proteins in the cerebrospinal fluid of people with Creutzfeldt-Jakob disease (CJD), a rare, fatal brain disorder. The proteins, they say, could prove useful as a marker, as well as a handle on what causes the mysterious disease.

Whatever causes CJD is known to be transmissible, and can take three years or more to cause problems. The disease can sometimes look like other progressive dementias such as Alzheimer's disease, but its course is quite different: Once symptoms appear, death usually occurs within a year. At the moment, definitive diagnosis can be made only by studying brain tissue.

Michael G. Harrington, D. Carleton Gajdusek and their colleagues at the National Institutes of Health in Bethesda, Md., cast a wide net in their search, studying proteins in the cerebrospinal fluid of 100 healthy people, 21 CJD patients and more than 400 patients with other neurological diseases. They used a procedure called two-dimensional electrophoresis, separating cerebrospinal fluid proteins based on relative acidity and on size.

In the July 31 New England Journal of Medicine, they report finding two proteins present in all 21 CJD patients and in 5 of 10 patients with a herpes brain infection, but not in fluid from the other people. The herpes infection is easily distinguishable from CJD.

"It suggests very strongly there will be a diagnostic potential," Harrington told Science News. The finding will first have to be replicated by other laboratories and the test will have to be made simpler, he says.

The proteins may also provide a clue about how the disease originates. Determining the makeup of the two proteins will allow the researchers to search for the genes that code for the proteins. If the genes aren't in normal cells, it could mean they're in a virus or some other infectious agent. "We don't know [the proteins' role] in the disease," says Harrington. "But something is producing them fairly selectively."

Stanley B. Prusiner of the University of California at San Francisco, who has identified a unique self-replicating protein, called a prion, that he believes causes CJD, says that while the research is interesting, it contains two troublesome points. The researchers did not find the proteins in the spinal fluid of three patients with kuru, a disease Prusiner's lab has found to be identical to CJD. And finding the proteins in people with the herpes brain infection makes it unlikely they are specific to CJD, Prusiner says.

— J. Silberner

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