Dyslexia: A flaw in brain's blueprints

A rare opportunity to examine the intact brain of a 20-year-old man with dyslexia has yielded evidence that the condition in some cases may be related to an architectural flaw in the brain's cortex. Dyslexia — a reading impairment that frequently involves confusion of letter shapes and positions — has puzzled researchers for nearly a century. Its causes have been attributed to psychological factors, but in recent years studies have suggested possible problems in the brain, primarily involving abnormal functioning of the hemispheres. However, physical evidence to support this view has been rather skimpy.

Recently, a man with a well-documented case of developmental dyslexia since early childhood died of internal injuries from an accidental fall. Researchers at Beth Israel Hospital in Boston seized the chance to fully analyze the fiber and cell structure of the brain, which they determined was unaffected by the man's fatal accident. The brain was removed at autopsy, sectioned, and studied in its entirety; of particular interest was the "Tpt" area of the cortex, which is believed to play a critical role in speech and language functioning.

Using an observation technique called cytoarchitectonics, researchers Albert M. Galaburda and Thomas L. Kemper examined the "architecture of the cortex" in "excruciating detail," according to Galaburda, who specializes in the study of brain asymmetry.

What they found, he told SCIENCE NEWS, was a "disorganized cortex ... the layers were sort of scrambled and whirled [with] primitive, larger cells in this [left Tpt] part of the brain." The misshapen clumps of convoluted tissue, or polymicrogyria, were found exclusively in the left cerebral hemisphere, where the Tpt area is usually larger than that in the right half of a "normal" brain. But in this brain of a dyslexic, both sides were of equal size, the researchers report in the August ANNALS OF NEUROLOGY. "These findings ... lend support to the notion that language-relevant areas in the brains of patients with developmental dyslexia may be small in the two cerebral hemispheres," they say.

The researchers also discovered several lesser abnormalities, all on the left side, including the outgrowth of an unusually high number of fiber connections from the left Tpt. Previous studies indicate that the brain may attempt to compensate for injury in a certain region by growing more fibers. In addition, the abnormal tissue may also have contributed to the patient's seizures, which began when he turned 16.

The results appear to specifically relate the brain structure problems to dyslexia, the researchers suggest, because the right hemisphere and the rest of the brain appeared normal; indeed, no abnormalities at all could be detected without extremely detailed study, they report. And apart from his dyslexia, the man was of average intelligence.

Beth Israel Hospital

Abnormal "islands" of large cells, which contrast sharply with smooth area of right hemisphere at top of photo, were discovered by neurologist Galaburda.

Previous research with CT (computed tomography) scans and other tests of hemispheric specialization point to brain asymmetry problems in dyslexics, including the possibility that an "overloaded" left hemisphere could interfere with its normal functioning in the sequential and linguistic process (SN: 1/22/77, p. 55). But the Beth Israel team may be the first to pinpoint specific tissue structure abnormalities.

"This is not an explanation for all dyslexia, but it probably represents a type," says Galaburda, a neurology instructor at Harvard Medical School. He compared dyslexia to cancer in the respect that "there are several types"; symptoms may differ somewhat when comparing men to women and right-handers to left-handers (the victim in this study was left-handed), he says. "There is no *one* thing called dyslexia," Galaburda says. "And since there is no uniform behavior [associated with the disorder], there is probably no uniform structural abnormality responsible for it." Still, the tissue abnormality discovered in this study adds fuel to the argument that dyslexia is not solely — or perhaps even primarily — a psychological problem, he says. □

Amantadine: Adjunct to flu vaccination

An outbreak of influenza every winter is almost as much of a certainty as death and taxes. Most often, some variety of the type A influenza virus is responsible. If researchers know which variety to expect, they can prepare vaccines. But unfortunately, the type A influenza virus is noted for its tendency to undergo antigenic variation, and vaccines prepared against one strain of A virus are ineffective if a major antigenic change takes place.

If this happens — as it did in January 1978 when physicians had A/USSR (H3N2) vaccine ready, only to have an outbreak of A/USSR (H1N1) — we need not resign ourselves to letting the flu take its course. Amantadine hydrochloride, one of the few anti-viral drugs on the market, has been proved "very effective" in preventing type A influenza; is less effective but still useful in treating it; and should be used if there is epidemiologic and virologic evidence of influenza A in an area.

These recommendations come from the National Institutes of Health consensus development meeting on amantadine, held recently in Bethesda, Md. Vaccination remains the preferred method of flu prevention, the panel said, but amantadine has a great deal of value. The few reported side effects are transient and minor.

Amantadine is not a new drug — it was first licensed in 1966 — but it has not been widely recognized as a way of preventing type A influenza in the United States, although it is used in Britain and the USSR. This is partly because there have been no major outbreaks of type A flu since 1976, when the drug was approved for use with all strains of the virus. But amantadine is also used to treat parkinsonism (a chronic nervous system disorder), and the panel noted that physicians seem reluctant to use the drug for both purposes.

Amantadine can fill the immunologic gap between the time a viral strain is identified and the time a vaccine is ready. It can also ward off infection during the ten-day to six-week period before a vaccine "takes." The drug is best used both for prevention and in treatment of persons at high risk of complications because of underlying illness, "essential" persons like firefighters and those in closed environments like nursing homes. Amantadine is not as useful for treatment as for prevention, but it does seem to reduce fever and shorten the course of the illness slightly.

Amantadine should be used selectively, since overuse could lead to the development of amantadine-resistant variants of the type A virus. This, like the development of antibiotic-resistant bacteria, could destroy its usefulness. □