

Schizophrenia and brain imbalance

The possibility that biochemical problems, perhaps including a brain abnormality, are related to schizophrenia has been generally accepted for years. And for about the last decade, scattered bits of evidence have hinted that the left brain hemisphere might be a major site of any such dysfunction in schizophrenics. For example, a large body of clinical and experimental evidence indicates that schizophrenia is connected with a left brain-associated thought disorder characterized by illogical and irrational patterns, along with poor conceptualization and abstraction abilities. Other studies have uncovered apparent disturbances in lateral balance, primarily a higher incidence of left-sidedness among schizophrenics.

Now, in two separate studies of 72 schizophrenics and 48 control group members, University of Pennsylvania psychologist Raquel E. Gur reports that "schizophrenia is associated with ... both left hemisphere dysfunction and overactivation." The findings could have implications for medication, as well as psychotherapy, she says.

The first experiment involved the performance of two tasks designed to measure verbal and spatial information processing in the two hemispheres. Twenty-four schizophrenics were matched against 24 controls (12 males and 12 females in each group). In contrast to the controls, the schizophrenics showed a right hemisphere superiority on both verbal and spatial tasks, "indicating a left hemisphere dysfunction in the initial processing of verbal information," says Gur.

In the second test, lateral eye movements were recorded as measures of hemisphere activation following the presentation of various types of information and were measured in 48 schizophrenics and 24 controls. "The schizophrenics had significantly more rightward eye movements," Gur says, "indicating left hemisphere overactivation."

The two results combined, she suggests, provide a "new framework" for examining the cognitive aspects of schizophrenia. It appears that "schizophrenics overactivate the very hemisphere that is dysfunctional," Gur reports in the April *JOURNAL OF ABNORMAL PSYCHOLOGY*. "This combination of events may explain why schizophrenics are so often described as using faulty logic." Conversely, she says it may also be possible that it is the left brain overactivation that triggers dysfunction, rather than vice versa.

The role of drug treatment in left hemisphere dysfunction remains unclear, says the psychologist. Two unpublished reports on the phenothiazines, a major medication for schizophrenia, do not implicate the drugs as a potential cause of

the hemisphere abnormality. In fact, one of the reports indicates that "if anything, phenothiazines tend to attenuate laterality effects in schizophrenia." Still, Gur cautions that no research has been published in this area and that it is conceivable that "dysfunction [could] result from medication." All the schizophrenic patients in her study were on phenothiazines, she says. Her "next step" is to compare schizophrenic groups on medication with those off drugs.

Regarding psychotherapy, Gur says her results dictate a "more reasonable approach" than some therapists may have attempted in the past. "Therapy should try to correct the illogical thinking—since the left hemisphere is responsible for logic," she said in an interview. Rather than "focusing on the internal approach" that tries to "uncover" the causes of the patient's conflict, Gur says therapy should try to make sure the patient stands on his own two feet. The therapist-patient interaction should deal with things like "spending money, dressing, eating—things most of us take for granted," she says. "Then, once the patient gets out of the withdrawal stage, you might be able to get at some insight."

Gur cautions that it is too early to say whether left brain dysfunction and overactivation are a cause or effect of schizophrenia. "We can't make any claims like that," she says. "We need longitudinal, predictive studies. All we can say now is that schizophrenia is associated with left brain dysfunction." □

Ocean eddies from space



Huge ocean eddies, important in studies ranging from phytoplankton productivity to pollution-tracking to meteorology, are numerous in this remarkable photo taken by Landsat from 915 kilometers up. At least eight individual eddies can be seen, some up to 30 km across, says Richard S. Williams Jr. of the U.S. Geological Survey in Reston, Va. Three well-developed double eddies show distinct stream currents as much as 70 km long.

The case of the missing memorandum

The facts are in on the allegation that Charles A. Thomas Jr. did recombinant DNA experiments at Harvard Medical School without appropriately notifying the National Institutes of Health. Last December for the first time, the institute directed an investigator to stop recombinant DNA research (SN: 12/31/77, p. 420). While responding to an Environmental Defense Fund request for information, administrators discovered Thomas had not filed the required Memorandum of Understanding and Agreement (MUA), which outlines how proposed research meets the safety guidelines.

Last week the investigation culminated in two reports, each detailing letters, meetings and conversations among Thomas, the Harvard Medical School Committee on Recombinant DNA Activities and the NIH. An NIH report, submitted by James W. Schriver of the Division of Management Survey and Review found: "Dr. Thomas was in technical violation of the HMS and NIH guidelines dealing with recombinant DNA research."

A second report, by an *ad hoc* Harvard Medical School faculty committee, also records that the required memorandum was not submitted to NIH at the appropri-

ate time. The Harvard report says the failure was the "unintended result of many factors" and ascribes the "web of misunderstanding" to the "unprecedented nature of an untried and complicated supervisory process."

Among the tangles of the web is the fact that Thomas did not specifically state in his grant application or its 1976 renewal request that he planned to do experiments using recombinant DNA (although he did include papers describing his recombinant DNA work and he reported it to another section of NIH in response to an inquiry). When in 1977 the Harvard recombinant DNA committee approved a memorandum for low risk (P2) recombinant DNA research in Thomas's laboratory, the MUA was sent not to NIH, but only to a private foundation funding Thomas's postdoctoral fellow. Members of the Harvard recombinant DNA committee report they were unaware that low risk recombinant DNA work continued in Thomas's laboratory while they considered the submitted MUA.

The NIH Executive Recombinant DNA Committee will now analyze the two reports and make recommendations to the director of NIH. Both reports say that