Psychochemical Treatment Counteracts Senility

An 81-year-old Ohio woman, diagnosed as senile, lay in her hospital bed staring blankly at the wall and unable to sign her name. She fervently clutched a stuffed dog. Several weeks later, she neatly wrote a note of gratitude to her doctors, discussed her past illness and put away the dog forever. What "cured" the woman, according to a Pittsburgh psychiatrist, is a new form of treatment for senile and pre-senile dementia that holds the promise of similar reversals—or at least substantial slowing of the senility process—in many people.

The treatment—a combination of psychotherapy and administered doses of anticoagulant—represents a break with past therapies and philosophies, says Arthur C. Walsh, a private practitioner who is also a clinical assistant professor at the University of Pittsburgh and a psychiatric consultant at the Veterans Administration Hospital in Pittsburgh.

Walsh is so convinced of the new method's effectiveness that he predicts senility and pre-senile dementia should become a preventable disease, "and the treatment should certainly extend a person's useable life."

At the American Psychiatric Association's annual meeting in Toronto this week, Walsh presented the findings of a two-year study of 49 dementia victims, many of whom were extremely deteriorated and nearly all of whom had been treated elsewhere unsuccessfully. Some "were unable to carry on even a semblance of a normal conversation."

Placed on individual, daily doses of anticoagulants, 70 percent of these otherwise hopeless patients improved, 15 percent dramatically so, report Walsh and colleagues Catherine Melaney and Bernice Walsh. Two of the patients underwent no change in condition, nine (18 percent) got worse and four persons died of various causes. The patients were treated with the anticoagulant warfarin sodium (Coumadin) in combination with individual and family psychotherapy.

The key to Walsh's approach rests in what he believes is the major cause of senility: Blood sludging or red-cell aggregation. Widely accepted theories dealing with arteriosclerosis generally trace the problem to degeneration of the cells themselves or the onset of some type of virus that slows the flow of the blood to the brain. However, the standard vasodilator and tranquilizer therapies have yielded little long-term successes. Walsh was intrigued with the work of M. H. Knisely of the Medical University of South Carolina who noted that restriction of vessels and arteries in aging can cause red cells to adhere or aggregate and impair blood flow. Knisely also pointed out that diabetes and alcoholism, among other conditions, can cause blood sludging, which Walsh says explains why brain damage is more common among persons afflicted with those two conditions. But small, previous studies also indicated that brain-damaged alcoholic and diabetes patients responded well to treatment with anticoagulants.

In preparing to further test such results, Walsh also hypothesized that since severe emotional stress can cause stroke and heart attack, it is a contributor to vessel constriction and, therefore, blood sludging. He first tested anticoagulant therapy by treating 24 patients in 1968 with the anticoagulant bishydroxycoumarin (Dicumarol). Twenty-two of the subjects showed improvement, he found. In his latest tests, the anticoagulant was changed because Coumadin "is easier to control and most doctors are familiar with its use," Walsh says. However, the somewhat less dramatic results of the later studies suggest that "perhaps Dicumarol is more effective than Coumadin for some patients," he says.

Walsh acknowledges that "there is no guaranty of a good result and there is some risk of seious complications and even death," but he emphasizes that "the majority do improve significantly." It would seem logical that a patient should have an opportunity for a trial of therapy. The psychiatrist notes that in prolonged senility a certain number of cells do die, but that others remain alive, rendered nonfunctional by sludging. Anticoagulant therapy, by breaking up the sludging, apparently rejuvenates such cells and, in effect, reverses or halts dementia in successful cases, Walsh says.

"As in other diseases, the earlier the treatment, the better the result," he says. But he and his colleagues have obtained reversals in "hopeless" older patients. "Surprisingly some of the very bad patients—several so deteriorated that we hesitated treating them—did better than others who seemed to have a better prognosis," Walsh says. Improvement usually begins from four or five weeks to four months after treatment. If improvement occurs, Walsh recommends continued anticoagulant use indefinitely, to be interrupted only by bleeding problems or prospective surgery where the ability to clot is essential. In the studies, 50 percent of the patients who had improved regressed when taken off anticoagulants.□

Lead-sabotaged vision: Low-level link

Depending on how much gets into the human body, lead can cause anemia, kidney disease, liver disease, muscle paralysis, brain damage, convulsions or death. In acute amounts, lead can also cause blindness. Now, for the first time, visual impairment has been linked to chronic low-level blood poisoning in primates.

The findings were reported last week at the spring meeting of the Association for Research in Vision and Opthalmology in Sarasota, Fla., by Joel E. Pounds of the University of Wisconsin. Pounds conducted the research with Robert Michael Jones, Philip J. Bushnell, Robert E. Bowman and James K. Allen, also of the University of Wisconsin. Pounds says their results have implications for humans, especially for children who are exposed to chronic low levels of lead.

Rhesus monkeys were reared on diets designed to produce lead concentrations in the blood stream of 14, 55 or 85 micrograms per 100 milliliters of blood for the first year of their lives. Eighteen months later, the levels of lead in their blood were presumed safe (14, 20 or 23 micrograms of lead per 100 milliliters of blood, respectively). The researchers then conducted vision experiments on both these monkeys and on controls.

The monkeys were exposed to visual stimuli at various levels of light, and their visual discrimination was compared to both their own performance under bright light and to the performance of the other monkeys under all of the light levels used. Both control monkeys and monkeys that had been reared to the 14 and 55 micrograms levels of lead showed no change in discrimination accuracy as light intensities were reduced. In contrast, the discrimination accuracy of the monkeys that had been reared to the 85 microgram level was severely impaired. This interaction of the effects of light intensity with lead treatment was statistically significant.

Because the discrimination deficit occurs in the higher lead group only at light levels below that of daylight, it reflected a loss of nighttime vision—vision provided by light-sensitive cells called rods in the retina of the eye. The researchers then examined the monkeys to determine exactly how the lead had led to decreased night vision. Since lead usually damages the central nervous system rather than the retina, they expected brain damage. Indeed, they found a decrease in the number of nerve-cell synapses in the area of the cerebral cortex known as the calcarine cortex and also damage to the blood brain barrier in the calcarine cortex. The blood brain barrier consists of tiny blood vessels that try to keep chemicals out of the brain.

Because the eyes of rhesus monkeys are strikingly similar to those of humans, Pounds proposes that chronic exposure to 85 micrograms per 100 milliliters of blood early in life in a child will probably impair night vision even if such lead levels eventually return to normal. A recent U.S. Public Health Service survey found that quite a few American children in high-risk lead areas have lead in their blood in excess of 85 micrograms per 100 milliliters. Their vision might well be impaired without them showing open symptoms of lead poisoning. □

Gene legislation: NAS urges caution

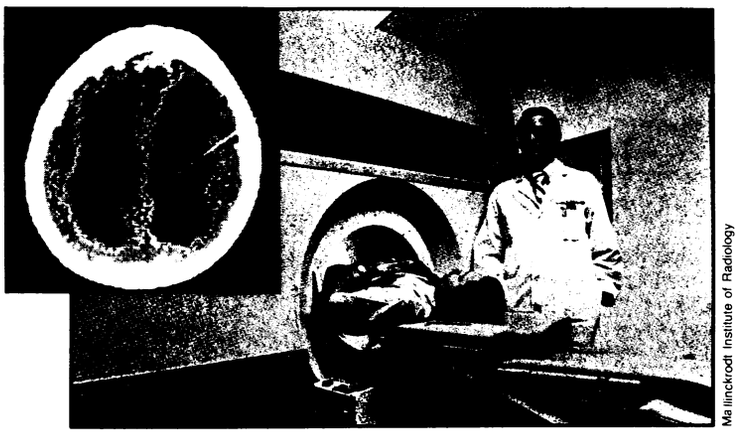
The National Academy of Sciences added its weighty opinion last week to the mountain of recommendations on regulation of recombinant DNA research. At its annual meeting, Academy members passed a resolution expressing concern about proposed federal legislation. Among the authors of the statement were molecular biologists who have argued opposing positions in recent debates on the potential hazards of the research.

The Academy resolution, although favoring extension of the NIH guidelines into law, challenges provisions that allow individual communities to impose regulations stricter than the federal law. "Above all, local option would set a dangerous pattern for the regulation of basic research in a manner that might deprive society of substantial future benefits," the statement says. The Academy also opposes the precedent of a national regulatory commission to govern an area of scientific research, calling the proposal "a wholly new and unfortunate departure."

Philip Handler, president of the Academy, made an even stronger statement in his annual report to members. "I view with great alarm the prospect of any law that would authorize government officials to determine what subject matter it is permissible to investigate as well as the manner in which such research is to be conducted," he said. "As a minimum, one can foresee constraints that will swathe research with bureaucratic complexities. . . . If pursued yet farther, science will be shattered."

Handler said most of the scientists who attended the Academy's recent forum on recombinant DNA research (SN: 3/12/77, p. 165; 3/19/77, p. 181) grudgingly concluded that federal legislation is inevitable and perhaps even desirable, partly to "terminate the feckless debate which has offered outlets for antiintellectualism and opportunity for political misbehavior while making dreadful inroads on the energies of the most productive scientists." But Handler concluded whatever the specifics of the law, "our successors will rue the day this legislation was passed." □

CT scans: Profiteers and gadgets fads



Cross-sectional X-rays of the body (inset) are produced by CT scanners. Despite their diagnostic value, are the costly scanners being over-proliferated and overused?

Mallinckrodt Institute of Radiology

The new generation of computerized tomographic (CT) scanners, which create cross-sectional X-ray photographs of the body, have brought about a diagnostic revolution (SN: 3/13/76, p. 170). They are particularly effective in detecting tumors in the head, chest and abdomen. But a new study by the Institute of Medicine warns that new standards may be required to limit where new CT scanners should be placed and when they should be used.

Since the devices were first introduced just four years ago, some 350 have been put into operation, about 20 are now being installed and 400 to 500 are on order. At a cost of up to $700,000 each and annual operating expenses of some $300,000, the devices have begun to absorb a significant share of the American health-care dollar. The Blue Cross Association thus asked the Institute of Medicine to offer some guidelines on installation and use of the new technology—guidelines almost certain to wind up as insurance company standards for reimbursing examination expenses.

The cost of an examination with a CT scan now averages $200 to $225, including a doctor's fee of $55 to $60. But the cost can run as high as $500 in some areas, allowing hospitals and some private doctors to recoup their investment in a matter of months. The chairman of the study committee, Charles A. Sanders, general director of Massachusetts General Hospital, said to "avoid profiteering," the committee was recommending a standard physician's fee of $35 and an investment amortization period of five years. Standards for use were also suggested.

The committee also recommended that CT scanners be installed primarily in large hospitals that could put them to full use—about 2,500 examinations per year. If the new fee system is implemented, says Blue Cross President Walter J. McNerney, machines now operating at low volume and high price will become unprofitable and "some of the damage can be undone."

Behind this study and its recommendations for controlling the spread and abuse of a particular technology is the larger question about competition among hospitals and doctors that frequently results in overinvestment in new gadgets. Some control is exercised over purchase of major equipment by laws requiring a hospital to obtain a state Certificate of Need (CON) before investing. But only 29 states and the District of Columbia have CON requirements and private physicians are presently exempted. (Some 15 percent of CT scanners are now installed in private offices or clinics.)

By 1980, federal law will require all states to have CON legislation, and the Institute of Medicine report specifically recommends that such laws be expanded to cover private physicians. Thus the report may well live up to its billing as "a watershed for policy decisions about appropriate distribution and use of costly medical technologies." □

Academy steps up human rights drive

In 1975, Jose Luis Massera, a prominent mathematician in Uruguay, was detained by police and held in prison for one year. After severe torture which left Massera, 62 years old, with a broken hip, he was finally charged with "subversive association" and brought to a closed trial. He is still being held incommunicado and has not been able to answer any of the scientific correspondence sent to him.

In various countries around the world, scientists are undergoing harrassment, repression and torture for outspoken political views. From outstanding scholars like Massera—known to the international community—to obscure researchers, scientists have been among those singled out for dissident views. Cut off from friends and colleagues, the scientists seldom have their story heard by the rest of the scientific community.

Last week, the Human Rights Committee of the National Academy of Sciences