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.