

receive symptomatic treatment [for depression] or further subsidiary diagnoses. The problem is identifying the concurrent presence of both depression and dementia in a single person." Such a dual affliction may occur "with far greater frequency than do 'clinically pure dementing states,'" Miller adds, and is often overlooked because of the similarity of symptoms of dementia and depression.

Overall, however, depression does not appear to strike the elderly any more often than it does younger persons, reports George Winoker of the University of Iowa. It is family history of depression, rather than age of the individual, that may be critical in the occurrence of depression, he says. In a study of 90 elderly female patients diagnosed as having various types of depression, Winoker found some striking differences between persons with different family histories.

Previous studies have shown that when the drug dexamethasone is given to a non-depressed person, the body's glandular reaction is to lower the blood level of cortisol. In depressed persons, however, little or no dexamethasone-induced suppression of cortisol occurs. In his study Winoker found such abnormal suppression in 85 percent of the elderly who had a history of depression but no alcoholism in their immediate family. In contrast, abnormal levels of cortisol were induced in only 4 percent of depressed persons who had an alcoholic in their immediate family.

"Clinically, these [patients] look similar, but what we may be seeing are separate illnesses," depending upon whether the family has a history of alcoholism or pure depression, Winoker told SCIENCE NEWS. "This could have enormous meaning in treatment of such persons."

The possibility that two distinct diseases are involved in early-onset Alzheimer's and senile dementia in the elderly is also suggested in the work of the University of Minnesota's Leonard Heston. Neurologically, presenile Alzheimer's and the most common forms of senile dementia appear to be identical: Brain cells become knotted and tangled and dotted with plaques — patches of abnormal protein.

But Heston's findings indicate the genetics of the two ailments may be different. In his study, Heston examined the autopsy reports and family histories of 47 Alzheimer's victims (onset before age 65) and 68 senile dementia victims (onset after 65). He found that those who had been stricken earliest (ages 40 to 54), had the highest rate of either senile or presenile dementia disease in their immediate family — 18 percent. Victims with an onset between ages 55 and 70 carried an 11 percent familial risk, and the over-70 onset group had only a 1.5 percent risk among parents and siblings.

Moreover, the early-onset group members were found to have an incidence rate of Down's syndrome among relatives that is more than 10 times the risk rate of the

general population. In contrast, the later-onset senile dementia group had a Down's risk rate fairly close to the norm. In addition, the family cancer risks were also significantly higher among the early-onset patients.

"The [family] risk is so concentrated among the early-onset [group]," Heston says. "On genetic background, you can't exclude the possibility of two different diseases." He also suggests that some kind of "virus" may be involved. But, Heston stresses, even in families of early-onset Alzheimer's victims "very few people [family members] are affected" out of the entire population of such patients.

Whatever the primary cause of dementia, it seems apparent that victims suffer from restricted blood flow in the brain. David Ingvar of the University Hospital in Lund, Sweden, has shown that in an "at rest" condition, the brains of dementia victims show "significantly reduced" blood flow in the frontal regions (SN: 10/1/77, p. 219). The measurements are made by injecting an isotope and using a computer to analyze the flow pattern in the subject's brain.

Now, Ingvar reports similar results with brains in "activation." When presented with various stimuli — pictures, reading material and conversation — the brains of normal subjects "light up" in certain areas and display increased blood flow. Corresponding brain areas of senile dementia victims, however, show almost no reaction to such stimuli. In a somewhat surprising offshoot of this work, Ingvar has also "confirmed that the overall blood flow of chronic schizophrenics is normal." However, it appears that the more withdrawn, hallucinatory and severely debilitated the schizophrenic, the more restricted the blood flow in the frontal areas.

What causes such blood flow abnormalities is neuron degeneration that triggers a decline in brain metabolism, according to Ingvar. He suggests that corresponding abnormalities in electrical activity must also exist in senile persons. With computerized scanning and other techniques, Ingvar says that "in the future it will be possible to measure and localize the circulatory and physical events that underlie normal and abnormal functioning."

Another factor in Alzheimer's disease may be aluminum. Studies by Ohio State University's Leopold Liss and others indicate that dementia patients may be susceptible to the buildup of aluminum in their brain cells. Such results are still preliminary, however, and those attending the symposium agreed that further study of the apparent aluminum link is needed before any conclusions are drawn or preventive measures proposed.

As of now — although much progress is being made — the exact causes of dementia remain elusive. Says Ingvar: "Why suddenly some families are afflicted and others are not... we do not know at all." □

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