## Treatment of Senile Dementia of Alzheimer Type by a Psychiatric-Anticoagulant Regimen

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## Abstract

Many patients diagnosed as senile dementia of the Alzheimer type also have a circulatory deficiency of the brain. In many such cases the author has shown that significant improvement can be obtained by adding anticoagulant therapy to the standard psychiatric treatment. A detailed case history is given to demonstrate the principles involved and a plea is made for more trials of this therapeutic regimen for the benefit of the patients and to elucidate further the role of the brain circulation in Alzheimer's disease.

There has in recent years been a swing away from considering the role of the brain circulation in Alzheimer's disease despite the fact that Alzheimer's original report indicated that this 51 year old patient had atherosclerosis of the larger cerebral arteries (1). Recent studies have shown that 40% of AD patients have, at autopsy, shown gross vascular abnormalities and in other studies capillary thickening indicates a microcirculation problem so that impaired circulation may indeed be an important factor in many AD patients. Thus we should always keep in mind the possibility of ischemia in these patients even though it may not be the primary problem.

With the lessening emphasis on the importance of having an optimum circulation to the brain recently there has been a great deal of publicity to the lay public that reflects the entirely negative attitude of physicians about the prognosis in AD that nothing can influence the progressive deterioration to death.

My experience over the last 21 years in treating over 300 dementia patients makes me

believe this attitude to be unjustifiably pessimistic. Some of these patients had been diagnosed by other physicians as having AD and yet they improved significantly under the psychiatric-anticoagulant regimen used for senile patients in general and described elsewhere (2, 3). These patients had not responded to standard forms of therapy and had been given up by their previous physicians. When they did improve on the above mentioned treatment their doctors would then say it must not have been AD in the first place and refused to consider the possibility that the treatment had indeed reversed these patients' symptoms.

This was a difficult argument to contest since most of us agree that AD cannot really be diagnosed positively except at autopsy. For this reason the patient described below is most interesting since he responded on three occasions to the addition of the anticoagulant treatment and regressed each time it was stopped. As he was too far advanced to be cared for at home he had to be transferred to a long term care hospital where he soon died. This allowed an autopsy examination which demonstrated the presence of AD. His case history, which has been reported elsewhere in greater detail (4), is as follows:

A 56 year old lawyer-engineer-executive was first seen in the psychiatric hospital where he had been admitted for final evaluation to be sure there was nothing more could be done before he would have to be confined for life to a State Hospital as his wife could no longer care for him at home. He was extremely confused but also had physical symptoms such as irregular breathing and falling spells so he was unable to go to the cafeteria with the rest of the patients. He was started on the anticoagulant and in a week was physically much better, no longer falling or having breathing problems and was

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cooperative enough to go to the cafeteria. He did not, however, improve enough to be cared for at home so the anticoagulant was stopped preparatory to his discharge to the state hospital as it was felt they would not continue it there due to the difficulty of controlling the dosage. Within a few days he deteriorated, was falling and had irregular breathing again so severe that medical consultation was obtained and the diagnosis of "ataxic breathing" was made. Because he had done so well on the anticoagulant it was decided to try this again and once more he improved and was going to the cafeteria. Once more it was stopped and again he regressed, became uncooperative and even threw his liquid medicine all over the student nurse's white apron! The nurses pleaded with me to resume the anticoagulant as he was so much easier to care for while on it. This was done and again he improved but not enough to go home so he was transferred to the State Hospital with the anticoagulant be recommendation that the continued so he could be more comfortable and easier to care for. This they did but he had some bleeding and the anticoagulant was stopped. He soon became bedridden and died within 3 months of transfer. With great effort we were able to have an autopsy at the University and this revealed that he did indeed have Alzheimer's disease so this case demonstrates quite clearly that an Alzheimer disease patient can benefit considerably from anticoagulant therapy. Actually, judging from experience in the succeeding years, it is quite likely that had this man been treated earlier he would never have had to be hospitalized — in fact he might even have been able to continue working.

This patient demonstrates several important points:

- a) As he improved each time the an ticoagulant was given and deteriorated each time it was stopped this represents scientific proof of the effectiveness of anticoagulant therapy as the patient acted as his own control.
- b) Contrary to the usual opinion that people who fall should not have anticoagulant therapy this patient was actually safer on therapy as he had better balance and no longer fell as long as he was on the anticoagulant.
- c) Other physical symptoms under control of

the nervous system, in this case his breathing rhythm, may respond well to anticoagulant therapy as it improves the function of the nervous system, d) While the risk of bleeding is always present with anticoagulant therapy there is usually much greater risk without it — this man quickly deteriorated and died when the anticoagulant was stopped. The situation is somewhat similar to the withholding of insulin from a patient with severe diabetes — the decline may be very fast or more gradual.

The conclusion one must draw is that if dementia can be arrested, or even significantly reversed, four years after the disease had begun this should also be possible at three, two or even one year after onset. And if the treatment were to begin at the appearance of the first symptom the disease process could quite likely be prevented entirely.

Because of the horrible fate awaiting people with AD and the increasing number of our population who are going to succumb to it the time has come for those physicians having research grants to do some therapeutic trials with these patients to confirm or deny the role of anticoagulant therapy in dementia, including patients with AD, who have not responded to the standard forms of therapy. To begin with one could use only a small number, say ten patients, in various stages of the disease (though not too far advanced) and determine the response to the treatment program, being sure the prothrombin time is 2 to 2 1/2 times the control time indicating a therapeutic dose of the anticoagulant is being given. In my experience there ought to be a good response in 65% of the patients. A large number of patients may not be required as you need only reverse two or three AD patients to a significant degree to prove that something positive is going on, because such improvement is unheard of. If the changes are not convincing enough one need only stop the anticoagulant and observe for any regression, as in the patient described above. I feel certain that the value of this regimen would then be apparent and then further studies could proceed to determine the optimum level of anticoagulant therapy and the duration of treatment needed. There is too much at stake for these patients to neglect investigating this approach to treatment any longer.

The risks of anticoagulant therapy are far less than the risks of senility itself as I have documented elsewhere (5) so this should not be used as an excuse for not doing further studies. The above case history is only one of sixteen histories cited in the book "Conquering Senility" (6) and those patients are only the most dramatic results; many more patients were only arrested or just slightly improved but even this was enough to keep them out of a nursing home with all the expense and degradation that entails for the patients and their families.

## References

- 1. Wilkins RH, Brody IA. Alzheimer's disease, Arch Neurol 21:109-110, 1969.
- 2. Walsh AC, Walsh BH, Melaney C. Senile-presenile dementia: follow-up data on an effective psychotherapy-anticoagulant regimen, J Am Ger Soc, 26:467-470, 1978.
- 3. Walsh AC: Prevention of senile and presenile dementia by bishydroxycoumarin (Dicumarol) therapy. J Am Ger Soc 17:477-487, 1969.
- 4. Walsh AC: Anticoagulant therapy as a potent ally effective method for the prevention of presenile dementia: two case reports. J Am Ger Soc 16:472-481, 1968.
- 5. Aker JB, Walsh AC, Beam JB. Mental Capacity: Medical and Legal Aspects of Aging, Shepard's/McGraw-Hill, Colorado Springs, 1977 (in annual supplements of 1984 and after).
- 6. Walsh AC: Conquering Senility, J. Pohl Associates, Coraopolis, PA 15108, 1985.