

CEREBROVASCULAR DISEASE - RESULTS WITH ANTITHROMBOTIC THERAPY: SOME OBSERVATIONS. B.A. Sandok. Department of Neurology, Mayo Medical School, Rochester, Minnesota.

Antithrombotic therapy is now widely used in the treatment of patients with ischemic cerebrovascular disease. Governmental approval will increase use and further convince many that the issues of therapy are now resolved. They are not. The following approved professional labelling guidelines serve to highlight the many clinical and investigational problems that remain:

(1) There is evidence that aspirin is safe and effective for reducing the risk of stroke in men who have symptoms due to fibrin-platelet emboli.

(2) Patients should have a complete medical and neurologic evaluation.

(3) The recommended dosage is 1300 mgm/day.

The evidence for effectiveness, while of statistical significance, becomes clinically less important when one notes that in the Canadian study, a disturbing 12.4% of patients under treatment with aspirin, developed subsequent stroke. This should not be unexpected since similar cerebral ischemic symptoms may be produced by differing pathophysiologic mechanisms (not all related to fibrin-platelet embolization); and treatment for one may be illogical and ineffective for another. Accurate diagnosis is imperative for selecting appropriate therapy. In the clinical situation, in spite of "complete" evaluation, uncertainty of the underlying mechanism often remains. Questions also remain about the dosage recommendations, and other treatment alternatives for both women and men.

Subsequent clinical trials will need to consider each of the above. Until then, patients with cerebral ischemic symptoms must be evaluated individually. Some may benefit from antithrombotic therapy--many will not.

DOES THE TREATMENT OF STROKE RISK FACTORS REDUCE THE INCIDENCE OF STROKE? John P. Conomy, M.D., F.A.C.P. Department of Neurology, Cleveland Clinic Foundation, Cleveland, Ohio USA

The answer to the question posed in this title is "yes", but affirmation is highly qualified. (1) Stroke is not a homogeneous clinical disease state but a dynamic process whose etiologies are manifold. Its causes vary from the commonplace atherothrombotic occlusion of brain arteries and emboli of cardiac origin to rare and complex precipitants. (2) Stroke is not a homogenous pathologic state. Parenchymal hemorrhage, hemorrhagic infarctions and bland infarctions are very different forms of brain destruction with differing outcomes. The great triumvirate of risk factors in stroke are advanced age, arterial hypertension and heart disease. All of these conditions tend to coexist with the process of atherosclerosis. Hypertension and cardiac disease are potentially amenable to early diagnosis and treatment. In clinical studies, the effective treatment of arterial hypertension is emerging as the clearest evidence that risk factor modification is correlated with a decline in stroke. Therapy for some cardiac diseases constitutes effective stroke prevention as well. Other less direct risk factors for stroke tend to parallel the severity and extent of the atherosclerotic process. These include diabetes mellitus, hyperlipidemias and obesity. Therapeutic modification of these factors has debatable influence on the incidence of stroke and may reflect the atherosclerotic process to medical intervention. Some clinical studies have suggested that certain antiplatelet agents postpone stroke and limit TIAs, at least in males. Anticoagulants may prevent TIAs but do not appear to prevent stroke. Surgery has a definable range of risks and clearly demonstrated benefits in properly selected patients in whom it may relieve TIAs and postpone a given stroke. Surgery does not alleviate an individual patient's overall stroke risk. The process of stroke prevention will remain imperfect until therapies for hypertension and heart disease are accompanied by more effective treatment of atherosclerosis.