The death rate due to stroke in the United States and other countries has declined substantially in the past forty years. This decline has been accentuated since the introduction of specific antihypertensive therapy. The decrease has been noted in many countries, and apparently is a combination of not completely understood lifestyle factors, which have impinged on the prevalence and type of hypertensive disease and risk of stroke and the efficacy of the treatment of hypertensive disease.

Longitudinal studies in Olmstead County, Minnesota, by the Mayo Clinic Group, has documented a decline in the incidence of stroke. There is, however, relatively little evidence that either the acute case-fatality or the long-term survival after stroke has changed substantially in the past years.

The role of anticoagulation therapy in the treatment and prevention of stroke has been controversial. There appears to be a consensus that anticoagulation therapy in patients who have had a completed stroke is unlikely to be beneficial and may be potentially hazardous. The primary role of anticoagulation therapy appears to be in the prevention of stroke among patients with transient cerebral ischemia. There are only four randomized trials of anticoagulation therapy among patients with transient cerebral ischemia. These studies were primarily done in the early to middle 1960s, involved relatively few patients, were usually not blinded, and generally suggested that anticoagulation therapy was effective in decreasing the frequency of transient cerebral ischemia and possibly in the prevention of cerebral infarction but not mortality.

In 1977 the first randomized controlled trial of aspirin in cerebral ischemia were reported. Five major trials have now been published relating the use of aspirin to the prevention of stroke or recurrent transient ischemic attack (TIA) among patients with either stroke or transient cerebral ischemia on admission to the study. In general, these studies have suggested that aspirin therapy reduces the frequency of recurrent transient cerebral ischemia and may have a beneficial effect on stroke, but not on total mortality.

One trial has attempted to evaluate the efficacy of anticoagulation therapy versus antiplatelet therapy in individuals who were initially on anticoagulation therapy following transient cerebral ischemia. In this
study in Sweden, 135 patients who had been treated for two months with anti-coagulation therapy following their transient cerebral ischemic attack were randomized into a continuing anticoagulation treatment and to antiplatelet therapy. They were followed for twelve months. There were no significant differences between the two groups, three strokes occurring among the aspirin group and one in the anticoagulation group. The number of recurrent TIAs were somewhat higher in the aspirin group, while myocardial infarctions occurred more often in the anticoagulation group. A further two-year followup noted that TIA or cerebral infarctions occurred in eight patients during anticoagulation treatment, as opposed to 22 patients treated with aspirin, while there were two lethal hemorrhages on the anticoagulation therapy. The authors concluded that short-term anticoagulation treatment during the first critical period, perhaps 3-12 months after the initial ischemic symptoms is preferable followed by safer but less effective anti-platelet drugs as a long-term prophylaxis in patients with transient cerebral ischemia.

The Mayo Clinic Group, after reviewing the data on the treatment of transient cerebral ischemia proposed: 1) "that the majority of patients with vertebral-basilar TIA should be treated medically; 2) if a skilled surgeon and an experienced angiographer are available, patients with typical carotid TIA who are suitable medical risks should have angiography followed by carotid endarterectomy if an appropriate lesion is found; and, 3) non-operated patients with TIA, of less than two months duration, are treated with three months of Warfarin therapy, unless contra-indicated before aspirin is begun. Non-operated patients with continuing TIAs of two or more months duration are treated with aspirin, unless there has been a recent increase in the frequency, duration, or severity of TIA. Under these circumstances Warfarin therapy is advised for three months before aspirin is started. Aspirin therapy should then be continued until the patient has been free of TIA for at least one year. No treatment is advised for non-operated patients whose last episode of TIA was longer than 12 months ago".

Several recent papers have questioned the value of extracranial carotid endarterectomy. Specifically, there has been concern about the risk benefits of the surgical procedure. The potential high morbidity and mortality of the surgical procedure has raised serious questions about the benefits of the surgical procedure. In spite of the lack of clinical trials to demonstrate clear efficacy of this procedure, the number of carotid endarterectomies has continued to increase substantially in the United States. Furthermore, there are very wide variations in the reported morbidity and mortality following the procedure. Several investigators have suggested that the procedure is unlikely to be beneficial unless the operative and post-operative morbidity/mortality from the surgical treatment is less than three percent.

A study in Rochester, Minnesota reported that among 130 patients with transient cerebral ischemia that were not treated, 13% had a stroke within one month, 15% in three months, 23% at one year, and 43% by five years of followup. Among those treated with anticoagulants, only 4% had had a stroke within the first month, 9% by one year, and 21% by five years. The risk of stroke was much greater among the patients with TIA than would be expected in the general population. Among the 122 patients with carotid TIA there were 48 strokes and among the 64 vertebral-basilar TIAs there were 31 strokes. The net probability of stroke occurrence was somewhat higher in those with vertebral-basilar as opposed to carotid artery TIA.

The high frequency of stroke noted in the first few months after the initial TIA may be due to an ascertainment bias. The TIA patients were identified both from clinical records of patients reporting only TIA, and from stroke patients who reported prior TIA. Thus, the probability of