Decreased Incidence and Mortality of Stroke

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SUMMARY Dr. Whisnant argues persuasively that control of hypertension has played a major role in the decline of stroke. He offers a cohesive and logical overview on this decline and does so with data from his own vast experience and with the advantages of the Mayo Clinic system which includes a centralized population-based diagnostic index, neurological expertise and a high autopsy rate. However, neither Dr. Whisnant’s arguments nor the Mayo Clinic data justify the conclusion that “treatment of hypertension is the only significant contributor to the decline of stroke.”

BETTER DIAGNOSIS AND THERAPY may also play a role. Increased diagnostic sophistication has gradually identified more cases of senility as being due to Alzheimer’s disease and fewer cases of coma as being cerebrovascular in nature. The idea that arteriosclerosis leads to gradual narrowing of cerebral arteries, and hence senility, no doubt found refuge in previous classifications of stroke and still can harbour under rubric 437 of the International Classification of Disease (“other and ill-defined cerebrovascular disease”). As physicians learn that most cases of senility are due to Alzheimer’s disease and that when arteriosclerosis causes dementia it does so through cerebral infarcts, deaths due to Alzheimer’s disease are less likely to be attributed to stroke. Thus an apparent decrease in mortality results. Even now vascular dementia continues to be overdiagnosed.

Coma, as Dr. Whisnant points out, is also overdiagnosed as being due to stroke. As physicians learn to recognize coma as a syndrome, fewer fatal cases will be attributed to cerebrovascular disease. This, too, will contribute to a fictitious decline in stroke mortality.

A more general point about improved diagnosis and medical sophistication is that the more entities are recognized and diagnosed, the fewer will remain in any given category, which again contributes to an apparent decline.

Dr. Whisnant adds convincing evidence that the decline in stroke cannot be attributed to a classification artefact alone but he is not always willing to go as far as his own evidence could take him. For example, he quotes findings from an unpublished Mayo Clinic study (in the Rochester population since 1970) showing a sharp decline in mortality from coronary heart disease associated with only minor changes in the incidence of myocardial infarction. Given that heart disease causes almost twice as many deaths as subsequent strokes in survivors from an initial stroke would not the greater survival among these patients contribute to the decreased mortality of stroke patients? In fact, in a previous publication coauthored by Dr. Whisnant improved cardiac care, better treatment of complications and enhanced functional independence by rehabilitation therapy were offered as possible explanations for the reported improved survivorship after cerebral infarction.

Dr. Whisnant allows for the possibility that prevention and treatment of cardiac valvular disease may contribute to the decline of stroke but finds no evidence for it. The mortality from rheumatic heart disease in Canada has dropped by an impressive 75% in a 25 year period (fig. 1). Since rheumatic heart disease may lead to mitral stenosis, atrial fibrillation and other conditions generating emboli, it is not reasonable to conclude that prevention and control of rheumatic heart disease have contributed to the decline in the incidence and mortality of stroke? Figure 1 also illustrates the 51% decline in stroke mortality in parallel with an 88% drop in deaths attributable to hypertension, which emphasizes its importance, but puts it in context with other changes that occurred over the same period.

Changes in the indications for anticoagulants may also have had an impact on stroke decline. In the 1960’s the use of anticoagulants for completed strokes was common and only after it was shown that anticoagulated stroke patients had more complications and a higher mortality than control patients has the practice waned and been largely limited to the anticoagulation of patients with cardiac emboli. The treatment of cardiac emboli may in itself influence favourably the mortality and morbidity of stroke.

The findings by Drs. Matsumoto, Whisnant and colleagues...
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VASCULAR DEATH RATES, UNDER 65  
1953-1978  
(25 years)

Hypertension  
Rheumatic Heart Disease  
Stroke  
Heart Disease  
All Vascular Diseases

% Decrease  
0  
10  
20  
30  
40  
50  
60  
70  
80  
90  
100

* Width of bars indicates relative importance to cause of death

FIGURE 1. Stroke decline has been paralleled by even more dramatic drops in deaths due to hypertension and rheumatic heart disease, suggesting that the treatment of hypertension and the prevention of rheumatic fever may have contributed to the decrease in stroke mortality. (Illustration courtesy of Dr. Barnet and Raven Press.)

leagues that only 9% of patients with stroke had a preceding history of TIA's probably also represents an underestimate, since in addition to the problems discussed above, a number of stroke patients will be too ill to give a history of past health and may die before being able to do so. Among the survivors there will be the forgetful, the senile and the aphasic from whom no reliable history can be obtained.

The surgical and medical management of TIA's rests on the assumption that the natural history can be modified favourably. Although the impact of carotid endarterectomy cannot be assessed reliably, an effect would be expected since it is the most commonly performed vascular operation in the United States. Given that the risk for stroke and/or death for patients undergoing carotid surgery ranges from 2.5% to 24.4%, an unfavourable influence on stroke morbidity and mortality might be expected. Whether these hazards are counterbalanced by reducing the risk of stroke in the territory of the operated artery to 2% per year, as Dr. Whisnant states, remains a subject worthy of urgent study. The medical management of TIA also relies more on opinion than facts, but the evidence is now compelling that aspirin in doses of 1 to 1.3 G per day decreases the incidence of stroke and death by at least a third in patients with threatened stroke. Given the widespread use of aspirin in the last few years, a favourable effect on stroke decline is likely.

While contributions of treatment of TIA's to the decline of stroke may be recent and small compared to that of the treatment of hypertension, TIA's are more important for the neurologist and neurovascular surgeon because they are asked to deal with them. Most physicians are educated regarding the importance of hypertension but many doctors and the public have not yet grasped the therapeutic potential of treating TIA's. It is precisely in the area of better diagnosis and evaluating new treatments that physicians interested in cerebrovascular disease can make the greatest contribution to the continuing decline of stroke.

References
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