EDITORIAL COMMENTARY

Prevention of strokes and recurrent strokes

Prevention is always preferred to treatment after the fact. Louis Pasteur commented that "when meditating over a disease, I never think of finding a remedy for it, but instead, a means of preventing it."[1] During the past half century, doctors have pursued several familiar strategies to prevent ischaemic strokes: (1) control medical disorders such as hypertension, diabetes, obesity, hyperlipidaemia, and behaviours such as smoking, excess alcohol intake, lack of exercise—known risk factors for ischaemic stroke; (2) Prescribe one treatment, a panacea. for all patients with symptomatic brain ischaemia depending on the then fashionable treatment. Vasodilators, warfarin, heparin, carotid surgery, surgical bypass, and aspirin all have had periods of favour; and (3) choose treatment according to time oriented categorisation of neurological symptoms and signs such as transient ischaemic attacks, progressing stroke, reversible ischaemic neurological deficits, and "completed" strokes.

Among these approaches only risk factor control is a viable strategy. Brain ischaemia is caused by a wide variety of stroke mechanisms and vascular occlusive lesions. The idea that one treatment would prove effective for all types of vascular lesions that cause stroke is illogical. During the past 50 years of trials, no single treatment has ever shown more than a 20%-25% effectiveness in unselected stroke patients. When will we learn that there will be no panacea, and so stop designing large expensive trials of single therapies for nondescript lumped series of brain patients with ischaemia? Treatment of brain ischaemia characterised by temporal descriptors alone is even more foolish. Time courses such as transient ischaemic attack and "completed" stroke do not predict whether brain infarction is present, and do not distinguish between various stroke aetiologies. Cardiogenic embolism, severe stenosis, or occlusion of large extracranial and intracranial arteries, intra-arterial embolism, penetrating artery disease can all cause any of the time course patterns. Temporal pattern, do not predict prognosis. The time course of today's symptoms depends on when the patient is seen. Doctors must direct treatment over time. There is no valid argument today for continuing to choose treatment in relation only to the time course of symptoms.[2 3]

During the past two decades, the advent of modern technology able to image the brain, heart, and arteries that supply the brain has made possible a different strategy for prevention of further brain ischaemia in patients with transient ischaemic attacks and strokes. Because stroke is a vascular disease, why not aim treatment at the vascular process causing brain ischaemia? The long term prognosis of patients with coronary artery disease does not depend on whether a patient has exertion induced angina, at rest angina, or a myocardial infarct today; prognosis depends on the severity of coronary artery disease. Similarly, identification of the cause of index attack(s) of brain ischaemia such as carotid artery stenosis, atrial fibrillation, protruding aortic atheroma, penetrating artery disease, etc dictate treatment aimed at controlling that specific problem. Moreover, therapeutic trials could be directed at these specific stroke aetiologies. Trials of surgery for carotid artery stenosis, angioplasty for stenotic extra and intracranial artery stenosis, and warfarin versus aspirin for patients with atrial fibrillation are examples of this logical and practical strategy.

The paper by Yamamoto and Bogousslavsky (pp 771–6 of this issue) raises a red flag about this strategy and suggests another approach to ischaemic stroke prevention. These workers showed that recurrent strokes were most often caused by the same mechanism as the index strokes. But in many patients, the second and third strokes had different aetiologies than the first stroke. After all, atherosclerosis is a systemic disease with many manifestations. Hypertensive patients with atherosclerosis often have coexistent large artery occlusive disease, coronary artery and myocardial disease, and penetrating artery disease. Atrial fibrillation and aortic atheromas are also often present. Recurrent strokes are often caused by coexistent pathology, present at the time of the index event but not aetiologically related to that event. Preventive treatment should logically be directed at all potential causes of future strokes as well as all remediable stroke risk factors.

Adopting this strategy means that all patients with atherosclerosis and brain ischaemia should have at least full non-invasive evaluation of the heart, aorta, craniocerebral arteries, and blood. The commonest cause of death in patients with brain ischaemia is atherosclerotic coronary artery disease.[4] Treatment of the systemic disease atherosclerosis in all of its manifestations is the most logical prevention strategy. Although the evaluation cost would be high, the life and brain and heart tissue saved may well be worth the economic cost and may save money in the long run.

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1 Pasteur L. *Address to the Fraternal Association of former students of the Ecole Centrale des Arts et Manufactures, Paris, May 15, 1884.*
2 Caplan LR. TIAs: we need to return to the question, "What is wrong with Mr Jones?" [editorial]. *Neurology* 1988;**38**:791–3.
3 Caplan LR. Terms describing brain ischemia by tempo are no longer useful. A polemic (with apologies to Shakespeare). *Surg Neurol* 1993;**40**:91–5.
4 Adams HP, Kassell N, Mazuz H. The patient with transient ischemic attacks: "Is this the time for a new therapeutic approach?" *Stroke* 1984;**15**: 371–5.