Physical activity for preventing strokes

Better designed studies suggest that it is effective

Stroke remains the most common life threatening neurological disorder, accounting for about 10% of all deaths worldwide. This is despite a decline in mortality rates due to stroke in most industrialised countries since the early 1900s owing to a decrease in case fatality or incidence, or both. Stroke is a leading cause of disability, and its treatment entails prolonged hospitalisation, with a commensurate financial toll. Preventing strokes is therefore of public health and economic importance.

1 Carvel J. Milburn hails more private links for NHS. Guardian 2002: p 7, 10 January.
Although genes may play a part in predicting risk of stroke, the observation that individuals who migrate have the same rates of stroke as the people in their host country implicates environmental risk factors. Epidemiological studies have identified modifiable risk factors for stroke such as raised blood pressure, obesity, glucose intolerance, smoking, and alcohol abuse. Ischaemic stroke (the commonest type) and ischaemic heart disease share similar pathophysiological traits. Clear evidence links physical activity to ischaemic heart disease. A sedentary lifestyle is therefore a possible risk factor for stroke.

The association between physical activity and stroke was first described 35 years ago in a report from the Harvard alumni study, a longitudinal study of male former college students. Alumni who had been athletes in college experienced less than half the risk of fatal stroke compared with the non-athletes. An alternative explanation—that sedentary men are more likely to smoke and drink alcohol to excess than active men, placing them at higher risk of stroke—was addressed in a recent follow up of the same cohort in which their physical activity was assessed in more detail. After statistical adjustment for these differences, physical activity continued to be inversely related to the incidence of stroke. Similar observations have been made in other populations including British men and US women.

U-shaped and null associations between physical activity and stroke have also been reported. These inconsistent findings may have several explanations. Firstly, most investigators have adjusted for characteristics that may mediate some of the beneficial effects of physical activity on stroke, such as blood pressure. This overadjustment could result in the lack of difference seen in stroke rates across groups with varying levels of activity. Secondly, some studies are hampered by a rudimentary assessment of physical activity and small numbers of strokes.

Thirdly, with one exception, studies have not accounted for changes in physical activity over time and could result in an underestimation of the association between activity and stroke. Fourthly, the discrepant pathophysiological characteristics of the major subtypes of stroke—ischaemic and haemorrhagic—make a differential relation with physical activity plausible. Studies conducted among populations varying in age, sex, ethnicity, and socioeconomic position are likely to encounter different distributions of the subtypes of stroke. This may result in inconsistent associations when all strokes are grouped into a single category and related to activity. With regard to the U-shaped relation seen in some studies, the upturn in risk seen at higher levels of activity may be partly because vigorous activity seems to be less effective than activity of low and moderate intensity in reducing blood pressure, particularly diastolic.

Further research is needed on women, ethnic groups, the subtypes of stroke, and the optimal amount of physical activity—its volume, intensity, duration, and frequency—needed to prevent stroke. Although a meta-analysis of the predictive capacity of physical activity for risk of stroke would also be of value—for the relation between blood pressure and stroke this has been done by pooling individual level data—the dimensions of physical activity and units of assessment used across studies are too disparate to do this easily.

In the absence of such studies, systematic narrative reviews do exist. The better designed studies—those with more cases and more detailed assessment of physical activity—have found an inverse relationship between activity and stroke. This has led to speculation that activity may also be effective in the secondary prevention of stroke. Clinical studies of patients with strokes suggest that formal exercise programmes have favourable effects on physical fitness, function of the lower extremities, and neurological performance. To examine fully the potential for exercise to reduce recurrence of strokes, however, extended follow up of randomised controlled trials with arms in which exercise is the only treatment used—as have been conducted sparingly in the field of cardiac rehabilitation—would be costly but informative.

The prevalence of physical activity is low in industrialised societies: in England only about one quarter of men and women currently meet guidelines for health of 30 minutes or more of aerobic activity of moderate intensity (for example, brisk walking) on five or more occasions per week. This behaviour could be modified in several places, including schools (walking to school initiatives, improved provision for physical education), recreational areas (more parks and other exercise facilities with improved maintenance), transportation infrastructure (more cycle lanes), the workplace, and general practices.

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We thank Jerry Morris and Gerry Shaper for their comments on an earlier draft.