Cardiorespiratory Fitness: A Simple, Cheap and Nonpharmacological Means to Prevent Stroke?

To the Editor:

I would like to congratulate Hooker and colleagues for their recent retrospective analysis on 46,405 males and 15,282 females who underwent maximal exercise testing and follow-up estimates of metabolic equivalents and related mortality. The authors concluded that cardiorespiratory fitness is an independent determinant of stroke incidence in initially asymptomatic and cardiovascular disease-free adults. This impressive investigation underscores the importance of cardiorespiratory training as a simple, cheap and nonpharmacological means to prevent stroke during natural aging.

Because stroke is often caused by cerebral ischemia, a brief update on the mechanisms with which physical activity might offset such ischemia is warranted. Physical training has beneficial effects on multiple cardiovascular risk factors such as dyslipidemia, hypertension, diabetes, and cardiovascular events. The effect of such exercise on clinical outcome could also be partially related to a direct and independent positive effect on endothelial dysfunction in the conduit arteries or in the peripheral microcirculation. Thus, regular physical activity is associated with increased endothelium-dependent vasodilation and nitric oxide availability. Interestingly, a link between systemic endothelial function and cerebrovascular function has been reported, indicating a common pathway between these responses. Moreover, longitudinal studies indicate that improvements in cardiovascular fitness can provide a positive effect on human cognitive abilities, potentially offsetting declines in cerebral tissue density and increasing brain volume and cerebral blood flow (CBF).

Our recent experiments are the first to confirm findings of highly controlled animal studies which have reported that habitual physical exercise can elevate CBF. These animal studies also provide evidence that such voluntary physical exercise not only improves long-term stroke outcome but also provides a prophylactic treatment strategy for increasing angiogenesis, CBF and reducing brain injury during cerebral ischemia. Although an upregulation of endothelial nitric oxide synthase activity has been implicated as the key mechanism to increase CBF and reduce brain injury during cerebral ischemia, other factors such as insulin-like growth factor-I and brain-derived neurotrophic factor have also been implicated as downstream mediators of the neuroprotective actions of exercise. Thus, the findings of Hooker and colleagues, in combination with recent experimental studies in both humans and animals, indicate that physical exercise can be used as a treatment strategy to improve CBF, potentially providing a means to reduce cerebral ischemia and improve stroke outcome. The use and prescription of physical activity as a cheap and nonpharmacological means to prevent stroke is supported.

Disclosures

None.

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