Making the Most of Secondary Prevention

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See related article, pages 1881–1885.

Recurrent strokes are more likely to be fatal than first strokes, and survivors are more likely to be left with major disability. In this issue of Stroke, Hackman and Spence’s report of their interesting modeling study of the potential effectiveness of combining multiple medications/interventions in the secondary prevention of stroke is therefore to be welcomed. They suggest that at least 80% of recurrent cerebrovascular events might be prevented by a comprehensive, multifactorial approach. Indeed, relative reductions in risk of 90% are estimated for more intensive treatment and nearly 95% if carotid endarterectomy is included in the package. The philosophy is similar to that of the “polypill” in primary prevention although a more individualized approach is, of course, required in secondary prevention.

There are, however, a number of uncertainties. Firstly, there is, of course, the assumption that all of the interventions will have independent effects. The authors refer to some trials of 2 interventions in which the effects did appear to be independent, but it is a “leap of faith” to assume that the same will apply to 5 or 6 treatments given in combination. It is certainly possible, but no amount of modeling can give us a definite answer. Ischemic stroke has numerous etiologies, many of which are probably as yet unknown and might not be easily preventable. To what extent there will be a hard core of etiologies that will not respond to current preventive treatments is unknown.

Secondly, there is the issue of prevention versus delay. Most randomized trials of interventions have follow-up of only a few years. Strictly speaking, all that can be concluded, therefore, is that certain treatments reduce the risk of stroke over that time-scale. To what extent strokes are simply delayed rather than prevented is unknown. This is partly just semantics (delay is obviously good), but it is important that we don’t assume that prevention will necessarily be permanent. This fallacy is most relevant, of course, for treatments that “reduce mortality”—death can certainly be delayed but is unlikely ever to be “reduced”, let alone prevented.

Thirdly, there is the issue of compliance. Hackman and Spence quite rightly use estimates of treatment effects from randomized trials, but it should be emphasized that compliance with treatment is usually substantially lower in routine clinical practice than in closely monitored and motivated trial populations.

These limitations do not, however, detract from the important core message of Hackman and Spence: that a comprehensive and systematic approach to risk factor modification, including both lifestyle modification and medication, will undoubtedly be highly effective in the secondary prevention setting. We cannot perhaps predict and prevent all first strokes, but we should do our utmost to prevent all recurrent events. Indeed, we should regard recurrent strokes and other vascular events in the same way that our colleagues in obstetrics regard maternal and perinatal mortality: every single adverse outcome justifies detailed review and more than a little soul searching about whether we could have done better.

Disclosures

None.

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