There is much clinical evidence demonstrating the preventive effects of statins against the future onset of cardiovascular diseases in various conditions (1–4). The reduction of the risk of cardiovascular diseases is substantial in coronary artery disease (CAD) but also occurs in cerebrovascular diseases, including strokes (5–7), although the strokes are heterogeneous in their pathophysiology. Indeed, symptomatic stroke can be classified into ischaemic and/or haemorrhagic stroke. Also, ischaemic stroke may be classified as lacunar, atherothrombotic, cardioembolic or of unknown cause.

The prevalence of cardioembolic stroke has increased in many countries because the main contributor to cardioembolic stroke, namely atrial fibrillation (AF), occurs more frequently in an elderly patient population (8).

It is reasonable to suppose that the risk of atherothrombotic stroke can be reduced by the use of statins because there are many overlaps in pathophysiology (9–11) and risk factors for atherothrombotic stroke and acute coronary arterial diseases (12–14); indeed, they can be considered as a local symptomatic presentation of the same generalised disorder of “atherothrombosis” (15, 16). Although a few reports have suggested an overlap between atherothrombosis and AF (14, 17), there still is no specific answer to the recurrent question of whether statins can reduce cardioembolic stroke or the onset of AF.

In this issue of Thrombosis and Haemostasis, the paper by Dentali et al. entitled ‘Use of statins and recurrence of atrial fibrillation after catheter ablation or electrical cardioversion: A systematic review and meta-analysis’ provides interesting and important information for many clinicians (18). Although there are a few randomised studies (with small sample sizes, some in specific clinical conditions) suggesting the preventive effects of statins in the future onset of AF (19, 20), a sub-study of a large-scale contemporary, global registry of patients with or at high risk of atherothrombosis suggested a negative relationship between hypercholesterolaemia and AF. This sub-study demonstrated a lower serum cholesterol concentration and a lower prevalence of hypercholesterolaemia in patients with AF than in those without (14). Another previous paper, demonstrating the electrical instability of the atrial myocardium in patients with lower serum cholesterol (21), supports the negative impact of hypercholesterolaemia in the onset of AF.

However, as statins have pleiotropic effects, they may reduce the risk of future onset of AF by effects other than lipid lowering. Thus, the precise relationship between the use of statins and the risk of AF has still to be elucidated.

Also, there remains the question of whether recurrence of AF after catheter ablation and/or electrical cardioversion is representative of the AF that occurs in the community. Indeed, a systematic review and meta-analysis of these results may provide the best available information for the physician to use to consider the impact of statin use on the onset of AF.

As the loss of quality of life after an initial cardioembolic stroke in patients with AF is often huge (8), the potential impact of the prevention of AF might be more important than the prevention of CAD in the future. Most of the risk factors for CAD—hypertension (22), diabetes (23), obesity (24), smoking (25), etc.—are also known to be risk factors for AF. These risk factors add cumulatively to increase the risk of stroke and of thromboembolism in AF (26). Hypercholesterolaemia and statin use are only a few factors not directly related to the onset of AF per se. Thus, one might argue that the routine use of statins should not be recommended for the primary prevention of cardiovascular diseases in regions where the incidence of stroke, especially AF-related stroke, is high, if statins increase the risk of future occurrence of AF. This systematic review and meta-analysis at least suggests that no such concerns are necessary so far.

Conflict of interest
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