iv. Use of alternating pressure mattresses and high-tech pressure-relieving systems for individuals with an elevated risk of developing pressure ulcers.

v. Minimum provisions for individuals undergoing surgery.

vi. The 24-hour approach to provision of care.

vii. Assessment of support surfaces.

viii. Management of patients in sitting positions.

ix. The pressure ulcer reduction strategy—which should incorporate a coordinated approach to the acquisition, allocation and management of pressure relieving equipment.

x. Education of healthcare professionals, carers and individuals vulnerable to or at elevated risk of developing pressure ulcers.

A systematic review by Cullum et al. [6] concluded that (i) foam alternatives to the standard hospital foam mattress can reduce the incidence of pressure sores in people at risk, as can pressure-relieving overlays on the operating table. One study suggests that air-fluidised therapy may increase pressure sore healing rates. (ii) Compression is more effective in healing venous leg ulcers than is no compression, and multi-layered high compression is more effective than single-layer compression. High-compression hosiery was more effective than moderate compression in preventing ulcer recurrence.

Given all the evidence presented here it would seem that prevention of pressure ulcers is economically attractive.

References


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The last few years have seen a quiet revolution in our appreciation of the potential for preventing recurrent events in stroke survivors, which if put into practice could have a huge impact on the overall burden of stroke disease. In 1990 MacMahon and colleagues analysed pooled data from epidemiological studies involving over 300,000 subjects and demonstrated a log-linear relationship between blood pressure levels, averaged over time, and the risk of stroke [7]. They found the same relationship in intervention trials [8]: blood pressure lowering achieved nearly all the expected reduction in stroke risk, and the benefits seemed to persist well into the ‘normotensive’ range.

Six years later, analysis of blood pressure data from the UKTIA trial showed a very similar relationship with the risk of recurrent events in people with symptomatic cerebrovascular disease [9]. Thus, in both primary and secondary prevention settings, a prolonged 5 mmHg reduction in diastolic blood pressure is associated with about a 1/3 reduction in stroke risk. In secondary prevention, baseline levels of risk are much higher, however, and the ‘numbers needed to treat’ to prevent one stroke correspondingly smaller. Thus treating 1,000 stroke or TIA survivors for a year with low-dose thiazides, indapamide or chlorthalidone should not only prevent about 20 strokes, but also save some £100,000 for the NHS.

The world-wide PROGRESS trial [10] was designed to test whether these benefits could be realised in stroke survivors, and whether there is a level of blood pressure below which treatment will reduce risk no further. The key finding was that no such threshold could be identified–trial treatment reduced the risk of recurrent events by over 25%, whatever the starting blood pressure [10]. Arguments about the relative effectiveness of perindopril and indapamide are trivial in comparison with this unequivocal result. All stroke survivors should have their blood pressure strictly controlled, at least down to conventional target levels, without using angiotensin converting enzyme (ACE)-inhibitors if possible. This will then leave room for ‘perindopril-based treatment’ to be safely added, with the expectation of a further 25–30% reduction in stroke risk.

More recently, the Heart Protection Study has produced similar findings in respect of cholesterol lowering [11]. Simvastatin treatment of patients with coronary or stroke disease, with any initial serum cholesterol level above 3.5 mmol/l, reduced major coronary and cerebrovascular events by nearly 25%. One event was prevented for about 100 patient-years of treatment, absolute benefits depending more on overall risk profile than on baseline lipid levels alone.

Indirect evidence suggests that the benefits of blood pressure lowering, lipid lowering, smoking cessation and antithrombotic treatment are additive. There is reason to hope that measures such as fish-oil supplementation, folate and other vitamins to lower homocysteine levels, may also have additive benefits (though antioxidant vitamins have proved disappointing).

Wald and Law argue that combining aspirin, a thiazide, beta-blocker, ACE-inhibitor, and folic acid into a single ‘polypill’, might reduce stroke incidence by as much as 80% [12], and propose this as a population-based primary prevention strategy for people aged over 55. If an 80% risk reduction–possibly more if new antithrombotic regimes prove successful–could be achieved in secondary prevention, the benefits would be unequivocal, so how are we rising to this challenge?

To many of us the prospect of having even one stroke is so appalling that, at first sight, efforts to prevent another one may seem unimportant. Nevertheless, ‘will it happen again?’ is a fear frequently expressed even by those whom we might expect to be overwhelmingly preoccupied with the consequences of the stroke that has already happened. This fear is not without foundation. Over a quarter of strokes are recurrent events and cross-sectional surveys indicate that some 1/3 of the burden of severe long-term disability due to cerebrovascular disease (especially of that due to mental impairment) can be attributed to recurrent stroke [13]. Much of this might be preventable. In the PROGRESS trial, one case of long-term disability was prevented for every 120 patient-years of treatment (at a cost of around £15,000) [14].

In this issue, Rudd et al. present results from the 2001 Sentinel Audit, including data on secondary prevention from 95% of trusts providing acute stroke care in England, Wales and Northern Ireland, as well as information from community follow-up [6]. Whereas 91% of eligible patients received antithrombotic medication, only 64% of those with raised cholesterol were offered lipid-lowering treatment. Follow-up information was limited, but indicated that blood pressure control was inadequate in well over half the cases where it was measured and many patients were not treated at all. Older and more disabled patients were less likely to receive appropriate preventative treatment, as were the 58% of patients who had not been in a stroke unit during their hospital stay. We clearly have some way to go before the NSF targets of eliminating age discrimination and making stroke unit care available to all are achieved.

Qualitative studies suggest several reasons why preventative medication may not be prescribed or taken [15]. We undoubtedly need to invest in information systems to improve co-ordination between hospital and primary care, and to work much harder (probably through specialist nurses) to educate patients about the importance of medication and lifestyle changes. But the greatest need is to educate physicians that secondary stroke prevention involves more than just giving aspirin, and ensure that the enormous health gains that research has shown are possible are actually achieved.

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